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Saccharide

► Carbohydrates

Saliva

► Salivary Biomarkers

Salivary Biomarkers

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Synonyms

Analytes; Biomarkers; Saliva

Definition

Behavioral medicine research is increasingly being influenced by theoretical models that explain individual differences in behavior and disease risk as a function of interrelated biological, behavioral, social, and contextual forces. This multi-level theoretical approach follows technical innovations that have made measuring the activity of many biological systems straightforward, portable, and cost efficient. Saliva, in particular, has received attention as a biospecimen; sample collection is perceived as feasible, cost-efficient, and safe, and salivary assays as reliable and accurate (see Table 1). A single oral fluid specimen can provide information about a range of physiologic systems, chemical exposures, and genetic variability relevant to basic biological function, health, and disease. The purpose of this review is to provide a road map for investigators interested in integrating this unique biospecimen into the next generation of studies in behavioral medicine.

Description

Oral fluid as a biospecimen: "Saliva" is a composite of oral fluids secreted from many different glands. The source glands are located in the upper posterior area of the oral cavity (*parotid gland* area), lower area of the mouth between the cheek and jaw (*submandibular gland* area), and under the tongue (*sublingual gland* area). There are also

In the interest of full disclosure, DAG is founder and Chief Strategy and Scientific Advisor at Salimetrics LLC (State College, PA). DAG's relationship with Salimetrics LLC is managed by the policies of the Conflict of Interest Committee at the Johns Hopkins University School of Medicine.

M. D. Gellman (ed.), *Encyclopedia of Behavioral Medicine*,
https://doi.org/10.1007/978-3-030-39903-0

Salivary Biomarkers, Table 1 Perceived advantages of oral fluids as a research specimen compared to serum

"Minimally invasive"	Considered "acceptable and noninvasive" by research participants and patients
	Collection is quick, non-painful, uncomplicated
"Safety"	Reduces transmission of infectious disease by eliminating the potential for accidental needle sticks
	CDC does not consider saliva a class II biohazard unless visibly contaminated with blood
"Self-collection"	Allows for community- and home-based collection
	Enables specimen collection in special populations
"Economics"	Eliminates the need for a health care intermediary (e.g., phlebotomist, nurse)
	Resources for collection and processing samples are of low cost and available
"Accuracy"	Salivary levels of many analytes represent the "free unbound fraction" or biological active fraction in the general circulation

Source: US Department of Health and Human Services (2000)

many minor secretory glands in the lip, cheek, tongue, and palate. A small fraction of oral fluid (i.e., crevicular fluid) comes from serum leakage, either from the cleft area between teeth and gums, or from mucosal injury or inflammation. In the presence of significant mucosal or epithelial inflammation, however, serum constituents may contribute substantially to oral fluids. Each secretory gland produces a fluid that differs in volume, composition, and constituents. Oral fluid is water-like in composition and has a pH (acidity) between 6 and 9; it has minimal buffering capacity, so substances placed in the mouth can change salivary acidity very quickly.

An understanding of how a given analyte makes its way into oral fluid is key to interpreting individual differences in that analyte, as well as its association with outcomes of interest. Many of the salivary analytes of interest in biobehavioral research are serum constituents (e.g., steroid hormones). Serum constituent analytes are transported into saliva either by *filtration* between the tight spaces between acinus or duct cells in the salivary glands, or by *diffusion* through acinus or duct cell membranes. In contrast, some analytes found in oral fluids are synthesized, stored, and released from the granules within the secretory cells of the salivary glands (i.e., enzymes, mucins, cystatins, histatins). Still others are components of humoral immunity (antibodies, complement) or compounds (cytokines) secreted by immune cells (neutrophils, macrophages, lymphocytes). In addition to these analytes, saliva contains sufficient cellular material to obtain high quantity and quality DNA (Zimmerman et al. 2007).

The *rate* of saliva secretion can significantly influence levels of salivary analytes produced locally in the mouth (e.g., alpha-amylase (sAA), secretory IgA) as well as those that migrate into saliva from blood by filtration (e.g., dehydroepiandrosterone-sulfate and other conjugated steroids) (Malamud and Tabak 1993). Oral fluid secretion is influenced by many factors, including the day-night cycle, chewing movement of the mandibles, taste and smell, medications that cause dry mouth, as well as medical conditions and treatments that affect salivary gland function (e.g., radiation therapy, Sjögren's syndrome). It is important to note, therefore, that for analytes influenced by flow rate, the measured concentration or activity of the analyte (e.g., U/mL, pg/mL) must be multiplied by the flow rate (mL/min). The resulting measure is expressed as *output as a function of time* (e.g., U/min, pg/min).

Sample Collection: Even under normative-healthy conditions, more than 250 species of bacteria are present in oral fluids (Paster et al. 2001). During upper respiratory infections, oral fluids are highly likely to contain agents of disease. Oral fluid specimens should, therefore, be handled with *universal precautions* when used in research and diagnostic applications.

Saliva collection devices have historically involved cotton-based absorbent materials. Placed in the mouth for 2–3 min, oral fluids

rapidly saturate the cotton; fluids are subsequently recovered by centrifugation or compression. Most of the time, this approach is convenient, simple, and time-efficient. However, when the sample volume is small, the specimen can be diffusely distributed in the cotton fibers, making sample recovery problematic (Harmon et al. 2007). The process of absorbing oral fluid with cotton and other materials also interferes with several salivary immunoassays (Groschl and Rauh 2006). Further, where in the mouth oral swabs are placed may affect the measured levels or activity of some salivary analytes (e.g., Beltzer et al. 2010). Standardizing swab placement instructions and monitoring compliance can minimize this threat to measurement validity.

In early studies, saliva flow was often stimulated using techniques that involved chewing or tasting various substances (e.g., gums, waxes, sugar crystals, powdered drink mixes). When not used minimally and/or consistently, some of these methods may change immunoassay performance (Granger et al. 2007). Indirectly, stimulants also influence levels of salivary analytes that depend on saliva flow rate (SIgA; dehydroepiandrosterone-sulfate (DHEA-S); Neuropeptide Y (NPY); Vasoactive Intestinal Peptide, (VIP)). Researchers are advised to avoid these techniques.

Collecting *whole saliva* by "passive drool" (Granger et al. 2007) is an alternative collection approach that minimizes many of the threats to validity described above. Briefly, participants imagine they are chewing their favorite food, slowly moving their jaws in a chewing motion, and allowing oral fluid to pool in their mouth. Next, they gently force the specimen through a short plastic drinking straw into a vial. The advantages of this procedure include the following: (1) A large sample volume may be collected relatively quickly (3–5 min). (2) Target collection volume may be confirmed by visual inspection in the field. (3) The fluid collected is a pooled specimen mixture of the output from all salivary glands. (4) The procedure does not introduce interference related to stimulating or absorbing saliva. (5) Collection materials are of very low cost. (6) Samples can be aliquoted and archived for future assays.

Blood Leakage into Oral Fluid: Blood poses a threat to the validity of salivary analyte measurements because most analytes are present in serum in much higher levels (10–100-fold) than in saliva. Specifically, to meaningfully index *systemic* (vs oral) biological activity, analyte levels in saliva must be highly correlated with levels measured in serum. This serum-saliva association depends, in part, on circulating molecules being appropriately and consistently transported into oral fluids (Malamud and Tabak 1993). When the integrity of diffusion or filtration is compromised (e.g., through blood leakage directly into salivary fluid), the level of the serological marker in saliva will be affected. Blood leakage into oral fluid is more common among individuals with poor oral health (i.e., open sores, periodontal disease, gingivitis), certain infectious diseases (e.g., HIV), and tobacco users. Samples visibly contaminated with blood present varying degrees of yellow-brownish hue. Kivlighan et al. (2004) have proposed a five-point Blood Contamination in Saliva Scale (BCSS) that rates contamination from one (no visible color) to five (deep, rich, dark yellow or brown). Under healthy conditions, BCSS ratings ($N = 42$) averaged 1.33 ($SE = .08$); after microinjury caused by vigorous tooth brushing, ratings averaged 2.42 ($SE = .19$).

Particulate Matter and Interfering Substances: As noted above, items placed in the mouth can influence the integrity of oral fluid samples. Food residue in the oral cavity may introduce particulate matter in samples, change salivary pH or composition, and/or contain substances (e.g., bovine hormones, active ingredients in medications, enzymes) that cross-react with assays. Accordingly, research participants should not eat or drink for 20 min prior to sample donation. In the event that they do eat in this time window, participants should rinse their mouths with water. Importantly, however, they must wait at least 10 min after rinsing before a specimen is collected to avoid artificially lowering estimates of salivary

analytes. Access to food and drink should be carefully planned and scheduled when study designs involve repeated sample collections over long time periods.

Sample Handling, Transport, and Storage: Typically, once specimens are collected, they should be kept cold or frozen. Refrigeration prevents degradation of some salivary analytes and restricts the activity of protolytic enzymes and growth of bacteria. Conservatively, it is recommended that samples be kept frozen. At minimum, samples should be kept cold (on ice or refrigerated) and frozen later on the day of collection. Repeated freeze-thaw cycles should be avoided. DHEA, estradiol, and progesterone are very sensitive to freeze-thaw, whereas DNA, cortisol, testosterone, and sAA are robust (up to at least three cycles). Freeze-thaw cycles should be considered in the context of plans to aliquot and archive frozen samples for future assays. It should also be noted that some salivary analytes (e.g., neuropeptides) may require specimens be collected into pre-chilled storage vials (Carter et al. 2007) or treated with neuropeptidase inhibitors (e.g., EDTA, aprotinin) to minimize degradation (Dawidson et al. 1997). For large-scale national surveys, investigators working in remote areas, or patients collecting samples at home, freezing and shipping these frozen samples can be logistically complex and cost-prohibitive. In such circumstances, the impact of the handling and storage conditions should be documented by pilot work, or alternative biospecimens should be considered.

Medications: As noted above, many medications can indirectly affect some analytes by reducing salivary flow (e.g., diuretics, hypotensives, antipsychotics, antihistamines, barbiturates, hallucinogens, cannabis, and alcohol). Further, the condition for which the medication is prescribed or taken may itself directly influence analyte levels or activity (Granger et al. 2009). Few behaviorally oriented studies involving salivary analytes comprehensively document medication usage. Further, a lack of normative data coupled with wide individual variation in salivary analyte levels makes it impractical to identify improbable values due to medication use (unless the value is not physiologically plausible).

Medications that are applied intranasally, inhaled, or applied as oral topicals (e.g., teething gels) are of particular concern. Residue in the oral cavity left by these substances may change saliva composition and/or interfere with antibody-antigen binding in immunoassays. The name, dosage, and schedule of all medications taken (prescription and nonprescription) within 48 h should be recorded and used to statistically evaluate the possibility that medication use is driving analyte-outcome relationships.

Assays for Salivary Analytes: Immunoassays are the main laboratory techniques employed to assess levels and activity of salivary analytes. Most immunoassays share two basic steps. Antibodies prepared against a specific salivary analyte are coated to the bottom of a microtiter plate well; these antibodies are used to capture the target molecules. Conversely, antigens may be coated to the wells to capture antibodies present in the sample. Most modern assays employ a labeling design known as enzyme immunoassay (EIA), which uses enzymes coupled to antigens or antibodies (i.e., the enzyme conjugate). To measure salivary cortisol, for instance, antibodies to cortisol are fixed to the plastic surface of a microtiter well. The specimen and a cortisol-enzyme conjugate are added into the well and incubated. During the incubation, cortisol from the sample and the cortisol-enzyme conjugate compete for available antibody-binding sites. The well is then rinsed to remove unbound materials. Next, a substrate is added that reacts with the enzyme conjugate to produce a color. The degree of color in each reaction well is measured in units of optical density (OD). The more cortisol in the sample, the lower the amount of cortisol-conjugate that is bound to the plate, and the lower the OD in that reaction well. To determine concentrations of cortisol in the unknown samples, samples with known concentrations of cortisol (standards) are analyzed as part of each assay. Results from the standards are used to establish a calibration curve from which concentration/volume units can be interpolated from OD.

Operationalizing Individual Differences: A "basal level" is the level or activity of an analyte that represents the "stable state" of the host during

a resting period. One approach to assessing "basal" levels is to sample early in the morning before the events of the day are able to contribute variation. However, day-to-day variability differs across salivary analytes depending on a number of factors including inherent variation in the production/release of the analytes, rate of their metabolism/degradation, and their sensitivity to environmental influences. Therefore, a single time-point measure of salivary analytes (other than invariant genetic polymorphisms) is unlikely to yield meaningful insight into an individual's true "basal level." The reliability of "basal" estimates of salivary analytes can be enhanced by sampling at the same time of day across a number of days, then aggregating (by averaging assay results or physically pooling specimens) across days.

Most salivary biomarker/analyte studies have involved a reactivity/regulation paradigm; this approach uses repeated samples to evaluate time-dependent changes in analytes (i.e., cortisol, sAA) in response to (or in anticipation of) a discrete event. The number of samples collected depends on the research question and logistical and practical issues (e.g., participant's tolerance for sampling burden). The optimal design for the measurement of salivary cortisol and sAA reactivity and regulation involves a pre-pre-[task]-post-post-post sampling scheme with samples collected on arrival to the lab (after consent) immediately before the task, then again immediately, 10-, 20- and 40-min post-challenge. Although some studies have yielded consistent mean-level differences in the patterns of cortisol response following a stressful or novel event, there are more often significant individual differences in stress-reactivity. Some individuals exhibit unexpected patterns of change (or no change), as well as continuously increasing or decreasing analyte levels over time.

An important component of variability in salivary analyte levels, both within and between individuals, is the diurnal rhythm. Most salivary hormone levels (e.g., cortisol) are high in the morning, decline before noon, and then decline more slowly in the afternoon and evening hours (Nelson 2005). By contrast, levels of sAA show the opposite pattern with low levels in the morning and higher levels in the afternoon (Nater et al. 2007). The nonlinear nature of these patterns requires multiple sampling time points to create adequate statistical models. A typical sampling design for salivary cortisol would involve sampling immediately upon waking, 30-min post-waking, midday (around noon), in the late afternoon, and immediately prior to bed.

Many analytical techniques have been used to model individual differences in diurnal rhythm including mean levels, evaluating the awakening response, and calculating summary measures of analytes over time (e.g., area under the curve, Pruessner et al. 2003). Another approach, growth curve modeling (McArdle and Bell 2000), has recently gained popularity for a number of reasons. Briefly, growth models allow the level and slope of the diurnal rhythm to be examined in the same model and their distinct effects on predictors can be evaluated; they minimize the impact of error or noise in measured values; and the presence of individual differences in the diurnal rhythm can be statistically tested (e.g., McArdle and Bell 2000).

Another analytical approach, hierarchical linear modeling (HLM) allows investigators to estimate values across the day for an individual, based on several samples (Bryk and Raudenbush 1992). Then, deviations from these expected values can be predicted from momentary states and feelings (e.g., mood states) about activities reported at that time of day. Documenting everyday events and emotions that help explain changes in analyte levels or activity across a time period of interest may strengthen causal inference when paired with samples across multiple days. For example, in studies focusing on cortisol, samples are collected approximately 20 min after a diary entry. Computerized handheld devices have made self-assessments quite feasible.

Analytes in Saliva of Interest to Behavioral Medicine: To date, most biobehavioral research has focused on a small number of salivary analytes, that is, cortisol, testosterone, DHEA, and sAA. In fact, however, the salivary proteome has recently been characterized, and includes

more than 1,000 analytes (Hu et al. 2007). These analytes provide information about the following: (1) systemic body processes, (2) local oral biology, (3) surrogate markers of physiological activity, (4) antibodies, (4) medications and environmental exposures, and (5) genetic factors. Each category of analyte is briefly discussed below and summarized in Table 2.

The first group of analytes is present in saliva because oral fluid represents an ultra-filtrate of analytes found in the bloodstream (i.e., serum constituents). Because of high serum-saliva correlations, measuring these analytes in saliva enables investigators to make interferences about systemic physiological states. Adrenal and gonadal hormones are exemplars of this category of salivary markers (e.g., see Table 2).

Most analytes in oral fluid are produced locally in the oral cavity and are secreted from salivary glands. While individual differences in these salivary analytes may reflect systemic processes, a major contributor is local oral biology (e.g., local inflammatory processes, oral health and disease). Many salivary immune and inflammatory markers such as neopterin, beta-2-microglobulin, cytokines, and C-reactive protein (see Table 2) fall into this category. Markers in this group may be less interesting to investigators outside the fields of oral biology and oral health.

A third group of salivary analytes is produced locally by salivary glands, but the levels vary predictably with systemic physiological activation. For example, sympathetic nervous system activation affects the release of catecholamines from nerve

Salivary Biomarkers, Table 2 Salivary analytes of potential interest to biobehavioral research

Endocrine		
Aldosterone	Estradiol, Estriol, Esterone	
Androstenedione	Progesterone; 17-OH Progesterone	
Cortisol	Testosterone	
Dehydroepiandrosterone, and –sulfate	Melatonin	
Adiponectin, leptin, ghrelin	Oxytocin, Vassopressin	
Immune/inflammation		
Secretory immunoglobulin A (SIgA)	Beta-2-microglobulin (B_2M)	
Neopterin	Cytokines	
Soluble tumor necrosis factor receptors	C-reactive protein (CRP)	
Autonomic nervous system		
Alpha-amylase (sAA)	Neuropeptide Y (NPY)	
Vasoactive intestinal peptide (VIP)	Chromogranin A	
Nucleic acids		
Human genomic	mRNA	
Mitochondrial	Microbial	
Bacterial	Viral	
Antibodies specific for antigens		
Measles	Hepatitis A	Herpes simplex
Mumps	Hepatitis B	Epstein-Barr
Rubella	Hepatitis C	HIV
Pharmaceuticals/environmental chemicals		
Cotinine	Alcohol	Pesticides
Meth-, amphetamine	Lithium	Metals
Methadone	Cocaine	Opiods
Marijuana (THC)	Caffeine	Phenytoin
Bisphenol-A (BPA)	Barbituates	

Sources: Cone and Huestis (2007), Malamud and Tabak (1993), Tabak (2007), and US Department of Health and Human Services (2000)

endings, and these compounds' action on adrenergic receptors influences the activity of the salivary glands. For instance, salivary alpha-amylase is considered a *surrogate marker* of ANS activation, as are salivary measures of neuropeptide Y and vasoactive intestinal peptide.

Antibodies to specific antigens (e.g., HIV antibodies) comprise another group of salivary analytes. Table 2 offers several additional examples. Antibodies in oral fluids reflect an individual's immunological history and pathogen/microbe exposure. Further, depending on the specific antibody measured, they may reflect local and/or systemic immune activity. To date, relatively few biobehavioral studies have taken advantage of the information provided by salivary antibodies.

A variety of pharmaceuticals, abused substances, and environmental contaminants can be quantitatively monitored in oral fluids (see Table 2). One example is bisphenol-A (BPA) – a constituent of polycarbonate plastic and epoxy resins used in water bottles, baby bottles, and food containers that may leach into food and drink. Daily BPA exposures below the US Human Exposure limit (50 ug/kg/day) have been linked to permanent changes in genitalia, early puberty, and reversal of sex differences in brain structure (Maffini et al. 2006).

A final group of analytes has been made possible by recent technical advances allowing high quantity and quality DNA to be extracted from whole saliva (Zimmerman et al. 2007). Saliva samples collected to assess individual differences in salivary analytes, and biomarkers can yield reliable and valid information about genetic polymorphism.

Conclusion and Future Directions: As the gateway to the body, the mouth senses and responds to the external world, and reflects what is happening inside the body. Oral fluids provide insight into environmental exposures and contaminants, and serve as an early warning system for disease and infection. Genetic analyses using oral fluids can help explain individual differences, predict outcomes of medical treatments, and identify polymorphisms that affect disease risk and resilience. As the number of substances that can be reliably measured in saliva increases, oral fluid may become an increasingly attractive alternative to collecting blood. The wealth of information provided by salivary analytes has the potential to greatly enrich behavioral medicine research.

Cross-References

- ▶ Adrenal Glands
- ▶ Adrenergic Activation
- ▶ Antibodies
- ▶ Autonomic Nervous System (ANS)
- ▶ Behavioral Immunology
- ▶ Biobehavioral Mechanisms
- ▶ Central Nervous System
- ▶ Chronobiology
- ▶ Circadian Rhythm
- ▶ Cortisol
- ▶ Genetics
- ▶ Public Health
- ▶ Stress

References and Readings

Adam, E. (2006). Transactions among adolescent trait and state emotion and diurnal and momentary cortisol activity in naturalistic settings. *Psychoneuroendocrinology, 31*, 664–679.

Arendorf, T. M., Bredekamp, B., Cloete, C. A., & Sauer, G. (1998). Oral manifestations of HIV infection in 600 South African patients. *Journal of Oral Pathology & Medicine, 27*, 176–179.

Beall, C. M., Worthman, C. M., Stallings, J., Strohl, K. P., Brittenham, G. M., & Barragan, M. (1992). Salivary testosterone concentration of Aymara men native to 3,600 m. *Annals of Human Biology, 19*, 67–78.

Beltzer, E. K., Fortunato, C. K., Guaderrama, M. M., Peckins, M. K., Garramone, B. M., & Granger, D. A. (2010). Salivary flow on alpha-amylase: Collection technique, duration, and oral fluid type. *Physiology and Behavior, 101*, 289–296.

Booth, A., Johnson, D. R., Granger, D. A., Crouter, A. C., & McHale, S. (2003). Testosterone and child and adolescent adjustment: The moderating role of parent-child relationships. *Developmental Psychology, 39*, 85–98.

Brandtzaeg, P. (2007). Do salivary antibodies reliably reflect both mucosal and systemic immunity? *Annals of the New York Academy of Science, 1098*, 288–311.

Bryk, A. S., & Raudenbush, S. W. (1992). *Hierarchical linear models*. Newbury Park: Sage.

Carter, C. S., Pournajafi-Nazarloo, H., Kramer, K. M., Ziegler, T. E., White-Traut, R., Bello, D., & Schwertz, D. (2007). Oxytocin: Behavioral associations and potential as a salivary biomarker. *Annals of the New York Academy of Science, 1098*, 312–322.

Chard, T. (1990). *An introduction to radioimmunoassay and related techniques* (4th ed.). Amsterdam, NY: Elsevier.

Cone, E. J., & Huestis, M. A. (2007). Interpretation of oral fluid tests for drugs of abuse. *Annals of the New York Academy of Science, 1098*, 51–103.

Dabbs, J. M., Jr. (1991). Salivary testosterone measurements: Collecting, storing, and mailing saliva samples. *Physiology and Behavior, 49*, 815–817.

Dawidson, I., Blom, M., Lundeberg, T., Theodorsson, E., & Angmar-Mansson, B. (1997). Neuropeptides in the saliva of healthy subjects. *Life Sciences, 60*, 269–278.

Flinn, M. V., & England, B. G. (1995). Childhood stress and family environment. *Current Anthropology, 36*, 854–866.

Granger, D. A., Kivlighan, K. T., Fortunato, C., Harmon, A. G., Hibel, L. C., Schwartz, E. B., & Whembolua, G.-L. (2007). Integration of salivary biomarkers into developmental and behaviorally-oriented research: Problems and solutions for collecting specimens. *Physiology and Behavior, 92*, 583–590.

Granger, D. A., Hibel, L. C., Fortunato, C. K., & Kapelewski, C. H. (2009). Medication effects on salivary cortisol: Tactics and strategy to minimize impact in behavioral and developmental science. *Psychoneuroendocrinology, 34*, 1437–1448.

Groschl, M., & Rauh, M. (2006). Influence of commercial collection devices for saliva on the reliability of salivary steroids analysis. *Steroids, 71*, 1097–1100.

Gunnar, M. R., & Vasquez, D. (2001). Low cortisol and a flattening of the expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology, 13*, 515–538.

Gunnar, M., Mangelsdorf, S., Larson, M., & Hertsgaard, L. (1989). Attachment, temperament, and adrenocortical activity in infancy: A study of psychoendocrine regulation. *Developmental Psychology, 3*, 355–363.

Haeckel, R., & Bucklitsch, I. (1987). Procedures for saliva sampling. *Journal of Clinical Chemistry and Biochemistry, 25*, 199–204.

Harmon, A. G., Hibel, L. C., Rumyansteva, O., & Granger, D. A. (2007). Measuring salivary cortisol in studies of child development: Watch out–what goes in may not come out of saliva collection devices. *Developmental Psychobiology, 49*, 495–500.

Harmon, A. G., Towe, N. R., Fortunato, C. K., & Granger, D. A. (2008). Differences in saliva collection location and disparities in baseline and diurnal rhythms of alpha-amylase: A preliminary note of caution. *Hormones and Behavior, 54*, 592–596.

Hellhammer, J., Fries, E., Schweusthal, O. W., Schlotz, W., Stone, A. A., & Hagemann, D. (2007). Several daily measurements are necessary to reliably assess the cortisol rise after awakening: State- and trait components. *Psychoneuroendocrinology, 32*, 80–86.

Horvat-Gordon, M., Granger, D. A., Schwartz, E. B., Nelson, V., & Kivlighan, K. T. (2005). Oxytocin is not a valid biomarker when measured in saliva by immunoassay. *Physiology and Behavior, 16*, 445–448.

Hu, S., Loo, J. A., & Wong, D. T. (2007). Human saliva proteome analysis. *Annals of the New York Academy of Science, 1098*, 323–329.

Kivlighan, K. T., Granger, D. A., Schwartz, E. B., Nelson, V., Curran, M., & Shirtcliff, E. A. (2004). Quantifying blood leakage into the oral mucosa and its effects on the measurement of cortisol, dehydroepiandrosterone, and testosterone in saliva. *Hormones and Behavior, 46*, 39–46.

Kugler, J., Hess, M., & Haake, D. (1992). Secretion of salivary immunoglobulin A in relation to age, saliva flow, mood states, secretion of albumin, cortisol, and catecholamines in saliva. *Journal of Clinical Immunology, 12*, 45–49.

Maffini, M. V., Rubin, B. S., Sonnenschein, C., & Soto, A. M. (2006). Endocrine disruptors and reproductive health: The case of bisphenol-A. *Molecular and Cellular Endocrinology, 254*, 179–186.

Malamud, D., & Tabak, L. (1993). Saliva as a diagnostic fluid. *Annals of the New York Academy of Sciences, 694*, 216–233.

McArdle, J. J., & Bell, R. Q. (2000). An introduction to latent growth models for developmental data analysis. In T. D. Little, K. U. Schnabel, & J. Baumert (Eds.), *Modeling longitudinal and multiple-group data: Practical issues, applied approaches, and specific examples* (pp. 69–107). Hillsdale: Lawrence Erlbaum Associates.

McArdle, J. J., & Nesselroade, J. (1994). Using multivariate data to structure developmental change. In S. H. Cohen & H. W. Reese (Eds.), *Life-span developmental psychology* (pp. 223–267). Hillsdale: Lawrence Erlbaum Associates.

Melnick, R., Lucier, G., Wolfe, M., Hall, R., Stancel, G., Prins, G., Gallo, M., Reuhl, K., Ho, S. M., Brown, T., Moore, J., Leakey, J., Haseman, J., & Kohn, M. (2002). Summary of the national toxicology program's report of the endocrine disruptors low-dose peer review. *Environmental Health Perspectives, 110*, 427–431.

Nater, U. M., Rohleder, N., Schlotz, W., Ehlert, U., & Kirschbaum, C. (2007). Determinants of the diurnal course of salivary alpha-amylase. *Psychoneuroendocrinology, 32*, 392–401.

Nelson, R. J. (2005). *An introduction to behavioral endocrinology*. Sunderland: Sinauer Associates.

Nemoda, Z., Horvat-Gordon, M., Fortunato, C. K., Beltzer, E. K., Scholl, J. L., & Granger, D. A. (2011). Assessing genetic polymorphisms using DNA extracted from cells present in saliva samples. *BMC Medical Research Methodology, 11*, 170.

Nieuw Amerongen, A. V., Ligtenberg, A. J. M., & Veerman, E. C. I. (2007). Implications for diagnostics in the biochemistry and physiology of saliva. *Annals of the New York Academy of Science, 1098*, 1–6.

Paster, B. J., Boches, S. K., Galvin, J. L., Ericson, R. E., Lau, C. N., Levanos, V. A., Sahasrabudhe, A., & Dewhirst, F. E. (2001). Bacterial diversity in human

subgingival plaque. *Journal of Bacteriology, 183*, 3770–3783.
Pruessner, J., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology, 28*, 916–931.
Raff, H., Homar, P. J., & Skoner, D. P. (2003). New enzyme immunoassay for salivary cortisol. *Clinical Chemistry, 49*, 203–204.
Rees, T. D. (1992). Oral effects of drug abuse. *Critical Reviews in Oral Biology and Medicine, 3*, 163–184.
Reibel, J. (2003). Tobacco and oral diseases. Update on the evidence, with recommendations. *Medical Principles and Practice, 12*(Suppl. 1), 22–32.
Santavirta, N., Konttinen, Y. T., Tornwall, J., Segerberg, M., Santavirta, S., Matucci-Cerinic, M., & Bjorvell, H. (1997). Neuropeptides of the autonomic nervous system in Sjogren's syndrome. *Annals of Rheumatoid Disease, 56*, 737–740.
Scannapieco, F. A., Papandonatos, G. D., & Dunford, R. G. (1998). Associations between oral conditions and respiratory disease in a national sample survey population. *Annals of Periodontology, 3*, 251–256.
Schwartz, E. B., Granger, D. A., Susman, E. J., Gunnar, M. R., & Laird, B. (1998). Assessing salivary cortisol in studies of child development. *Child Development, 69*, 1503–1513.
Shirtcliff, E. A., Granger, D. A., Schwartz, E. B., & Curran, M. J. (2001). Use of salivary biomarkers in biobehavioral research: Cotton based sample collection methods can interfere with salivary immunoassay results. *Psychoneuroendocrinology, 26*, 165–173.
Smyth, J. M., Ockenfels, M. C., Gorin, A. A., Cately, D., Porter, L. S., Kirschbaum, C., Hellhammer, D. H., & Stone, A. A. (1997). Individual differences in the diurnal cycle of cortisol. *Psychoneuroendocrinology, 22*, 89–105.
Sreebny, L. M., & Schwartz, S. S. (1997). A reference guide to drugs and dry mouth – 2nd edition. *Gerodontology, 14*, 33–47.
Stone, A. A., Broderick, J. E., Schwartz, J. E., Shiffman, S., Litcher-Kelly, L., & Calvanese, P. (2003). Intensive momentary reporting of pain with an electronic diary: Reactivity, compliance, and patient satisfaction. *Pain, 104*, 343–351.
Tabak, L. A. (2007). Point-of-care diagnostics enter the mouth. *Annals of the New York Academy of Science, 1098*, 7–14.
U.S. Department of Health and Human Services. (2000). *Oral health in America: A report of the surgeon general*. Rockville: U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health.
Veerman, E. C. I., Van Den Keijbus, P. A. M., Vissink, A., & Nieuw Amerongen, A. V. (1996). Human glandular saliva: Their separate collection and analysis. *European Journal of Oral Science, 104*, 346–352.
Zimmerman, B. G., Park, N. J., & Wong, D. T. (2007). Genomic targets in saliva. *Annals of the New York Academy of Science, 1098*, 184–191.

Salt, Intake

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Synonyms

Sodium; Sodium chloride

Definition

Salt is a dietary element made up of sodium and chlorine (U.S. National Library of Medicine and National Institutes of Health 2011a).

Description

A majority (90%) of sodium consumed comes from salt (Centers for Disease Control & Prevention 2011). The body needs a small amount of sodium for fluid regulation, nerve impulse transmission, and muscle function. The kidneys are responsible for retaining sodium (if body stores are low) or excreting sodium through urine (if body stores are too high). However, if the kidneys do not excrete enough sodium, the excess sodium will accumulate in the blood. This can lead to high blood pressure, from an increase in fluid volume in the arteries, ultimately putting additional stress on the heart (Mayo Clinic 2011a; U.S. National Library of Medicine and National Institutes of Health 2011a).

Recommendations

For children ages 1–3, 4–8, and 9–13, the recommended daily sodium intake is 1,500 mg, $\leq$1,900 mg, and $\leq$2,200 mg, respectively. Those ages 14 and older are recommended to consume $\leq$2,300 mg a day (U.S. Department of Agriculture and U.S. Department of Health and Human

Services 2015). However, the American Heart Association recommends the general public to reduce their sodium intake to no more than 1,500 mg per day (American Heart Association Presidential Advisory 2011). In addition, those with certain diseases (e.g., cirrhosis and congestive heart failure) may be recommended lower sodium intake levels by their primary care providers (U.S. National Library of Medicine and National Institutes of Health 2011b).

A half of a teaspoon of salt is approximately 1,200 mg of sodium, and one teaspoon of salt is approximately 2,300 mg of sodium (American Heart Association 2011). More than 85% of Americans consume 2,300 mg of sodium or more a day; the average intake of sodium for Americans over 2 years of age is 3,400 mg per day (Centers for Disease Control & Prevention 2011; U.S. Department of Agriculture and U.S. Department of Health and Human Services 2010). Diets high in sodium have been associated with an increased risk for high blood pressure, heart disease, and stroke (American Heart Association 2011). Generally, when salt intake is reduced, it only takes a few weeks for blood pressure to decrease (Centers for Disease Control & Prevention 2011).

Identifying Sources of Sodium

Most foods naturally contain sodium (U.S. National Library of Medicine and National Institutes of Health 2011b); however, this form of sodium only accounts for about 12% of daily sodium intake. An additional 11% of sodium intake comes from cooking at home and adding salt while eating. A majority (77%) of the sodium Americans consume comes from processed foods, foods bought at stores, packaged foods, and foods cooked at restaurants (Centers for Disease Control & Prevention 2010). Sodium is added to foods to act as a preservative, cure meat, retain moisture, and enhance color and flavor (American Heart Association 2011; U.S. Department of Agriculture and U.S. Department of Health and Human Services 2010). When food and beverages were grouped in 96 categories, the top six categories that contributed the most sodium to Americans' diets included yeast breads, chicken and chicken mixed dishes, pizza, pasta and pasta dishes, cold cuts, and condiments (National Cancer Institute 2010).

Reading food labels is important for determining sodium intake because milligrams of sodium in food can vary even for the same type of food. For instance, a slice of frozen pizza can range from 450 to 1,200 mg of sodium (Centers for Disease Control & Prevention 2011). However, caution should be used reading the %DV (daily value) on the food label because the percentage is based on 2,400 mg, which is 100 or 900 mg higher than the recommended daily sodium intake depending on recommended group (U.S. Department of Agriculture and U.S. Department of Health and Human Services 2015; U.S. Food and Drug Administration 2011; American Heart Association Presidential Advisory 2011). Food packaging messages can be confusing (Centers for Disease Control & Prevention 2011). For example, a package message titled unsalted or no salt added simply means no salt was added while processing the food; yet, reading the label is important because some of the ingredients may contain sodium (Mayo Clinic 2011b). Additionally, looking at the ingredients list can help determine if sodium was added. Sodium is sometimes called different names; some examples include baking soda, monosodium glutamate, and sodium nitrite (U.S. National Library of Medicine and National Institutes of Health 2011b; Mayo Clinic 2011b).

Methods for Reducing Sodium

Some methods for reducing the amount of sodium consumed can include (Mayo Clinic 2011b; National Heart, Lung and Blood Institute n.d.; National Library of Medicine and National Institutes of Health 2010; U.S. Department of Agriculture and U.S. Department of Health and Human Services 2010; American Heart Association 2011):

- Following specific heart-healthy diets (e.g., dietary approaches to stop hypertension, which is also called the DASH diet)
- Eating fresh foods
- Using food labels to purchase items low in sodium
- Ordering lower sodium items when eating out
- Using healthy salt substitutes to replace salt

Cross-References

► Hypertension

References and Further Readings

American Heart Association. (2011). Sodium (salt or sodium chloride). Retrieved 15 Apr 2011, from http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/HealthyDietGoals/Sodium-Salt-or-Sodium-Chloride_UCM_303290_Article.jsp

American Heart Association Presidential Advisory. (2011). Population-wide reduction in salt consumption recommended. Retrieved 15 Apr 2011, from http://www.newsroom.heart.org/index.php?s=43%26item=1237

Centers for Disease Control & Prevention. (2010). Sodium and food sources. Retrieved 15 Apr 2011, from http://www.cdc.gov/salt/food.htm

Centers for Disease Control & Prevention. (2011). Sodium fact sheet. Retrieved 15 Apr 2011, from http://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_sodium.htm

Mayo Clinic. (2011a). Sodium: How to tame your salt habit now. Retrieved 15 Apr 2011, from http://www.mayoclinic.com/health/sodium/NU00284

Mayo Clinic. (2011b). Sodium: How to tame your salt habit now (continued). Retrieved 15 Apr 2011, from http://www.mayoclinic.com/health/sodium/NU00284/NSECTIONGROUP=2

National Cancer Institute. (2010). Sources of sodium among the US population, 2005–06. Risk factor monitoring and methods branch website. *Applied Research Program*. Retrieved 22 Mar 2012, from http://riskfactor.cancer.gov/diet/foodsources/sodium/

National Library of Medicine, & National Institutes of Health. (2010). Tasty stand-ins for salt. *NIH Medline Plus, 5*, 15.

National Heart, Lung and Blood Institute. (2003). Your guide to lowering high blood pressure: Healthy eating. Retrieved 15 Apr 2011, from http://www.nhlbi.nih.gov/hbp/prevent/h_eating/h_eating.htm

U.S. Department of Health and Human Services, & U.S. Department of Agriculture. (2010). Available at http://health.gov/dietaryguidelines/dga2010/dietaryguidelines2010.pdf

U.S. Department of Health and Human Services, & U.S. Department of Agriculture. (2015–2020). *Dietary guidelines for Americans* (8th ed.). Dec 2015. Available at http://health.gov/dietaryguidelines/2015/guidelines/

U.S. Food and Drug Administration (2011). How to understand and use the nutrition facts label. Retrieved 15 Apr 2011, from http://www.fda.gov/food/labelingnutrition/consumerinformation/ucm078889.htm

U.S. National Library of Medicine, & National Institutes of Health (2011a). Dietary sodium. Retrieved 15 Apr 2011, from http://www.nlm.nih.gov/medlineplus/dietarysodium.html

U.S. National Library of Medicine, & National Institutes of Health (2011b). Sodium in diet. Retrieved 15 Apr 2011, from http://www.nlm.nih.gov/medlineplus/ency/article/002415.htm

Salutogenesis

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Synonyms

Hardiness; Resilience; Self-efficacy; Sense of coherence

Definition

The medical sociologist Aaron Antonovsky (1923–1994) introduced the term "salutogenesis" which derives from the Latin "salus = health" and the Greek "genesis = origin." Antonovsky was mainly interested in the question of what creates and what sustains health rather than explaining the causes of disease in the pathogenic direction (Antonovsky 1979, 1987, 1993). In his salutogenetic model, he described health as a continuum between total ease (health) and total disease rather than a health-disease dichotomy. Therefore, his most important research question was: What causes health (salutogenesis)? (rather than what are

the reasons for disease (pathogenesis)). The core concepts of salutogenesis show great conceptual overlap with the theory of "hardy personality" (Kobasa 1979, 1982), the theory of "self-efficacy" (Bandura 1977), and with the theory of "resilience" (Werner and Smith 1982).

The central terms of the salutogenetic theory are sense of coherence (SOC) and general resistance resources (GRRs). Antonovsky postulates that the status of health and well-being depends on these personal and environmental resources (Antonovsky 1979, 1987).

Description

Sense of Coherence (SOC)

Sense of coherence explains why humans in stressful situations stay well and are even able to improve their physical, mental, and social well-being. Antonovsky (1993) suggested that SOC depicts a stable and long-lasting way of looking at the world. He postulated that SOC is mainly formed in the first three decades of life and then becomes relatively stable.

Antonovsky defined SOC as a:

> Global orientation that expresses the extent to which one has a pervasive, enduring though dynamic feeling of confidence that (1) the stimuli, deriving from one's internal and external environments in the course of living are structured, predictable, and explicable; (2) the resources are available to one to meet the demands posed by these stimuli; and (3) these demands are challenges, worthy of investment and engagement. (Antonovsky 1987, p. 19)

The SOC consists of three dimensions (Antonovsky 1987) as follows:

1. *Comprehensibility (cognitive component)*: The internal and external environments are interpreted as understandable, consistent, structured, and predictable.
2. *Manageability (behavioral component)*: Individuals consider resources to be personally available to help them cope adequately with demands or problems.
3. *Meaningfulness (motivational component)*: This dimension refers to the extent to which a person feels that life makes sense, and that problems and demands are worth investing energy in. Additionally, it determines whether a situation is appraised as challenging, and whether it is worth making commitments and investments in order to cope with it.

According to Antonovsky (1987) the third component is the most important aspect of SOC.

General Resistance Resources (GRRs)

The general resistance resources (GRRs) are biological and psychosocial factors that make it easier for people to perceive their lives as predictable, controllable, and understandable. Typical GRRs are money, intelligence, social support, self-esteem, ego-strength, healthy behavior, traditions, and culture. These types of resources can help the person to deal in a better way with the challenges of life. In general, the GRRs lead to life experiences that promote a better SOC (Antonovsky 1987).

Measuring Sense of Coherence

With the Sense of Coherence (SOC) Scale, there is only one instrument that measures sense of coherence worldwide. Antonovsky (1987) developed the SOC as a self-report questionnaire with Likert-type items; higher scores indicate a better SOC. This instrument exists in a long form (SOC-29) and in a short form (SOC-13). In the long form, 11 items refer to "comprehensibility," 8 items refer to "meaningfulness," and 10 items refer to "manageability." The SOC scale is a reliable, valid, and cross-culturally applicable instrument. SOC seems to be a multidimensional concept rather than a unidimensional one (Eriksson and Lindström 2005). By 2007, the SOC questionnaire has been used in at least 44 languages all over the world (Singer and Brähler 2007).

Sense of Coherence and Health (Empirical Evidence)

Empirical evidence shows a strong association between SOC and mental health. A large number

of studies consistently reveal a negative relationship of SOC with depression, anxiety, and posttraumatic symptoms (Antonovsky 1993; Eriksson and Lindström 2007; Tagay et al. 2006, 2009). In a recent review, Eriksson and Lindström (2007) synthesized empirical findings on SOC and examined its capacity to explain health and its dimensions. SOC was strongly related to perceived health. The stronger the SOC, the better the perceived health in general. This relation was manifested in study populations regardless of age, sex, ethnicity, nationality, and study design. Therefore, numerous authors assert that there is substantial empirical support for the idea that SOC promotes health. A strong SOC is associated with successful coping with the inevitable stressors that individuals encounter in the course of their daily lives, and therefore, with better outcomes (Antonovsky 1993; Eriksson and Lindström 2007). All in all, SOC seems to have a main, moderating, or mediating role in the explanation of health, and it seems to be able to predict health (Schnyder et al. 1999; Tagay et al. 2011).

Cross-References

- ▶ Coping
- ▶ Health
- ▶ Optimism
- ▶ Self-Esteem
- ▶ Stress
- ▶ Well-Being

References and Readings

Antonovsky, A. (1979). *Health, stress, and coping*. San Francisco/Washington/London: Jossey-Bass.

Antonovsky, A. (1987). *Unraveling the mystery of health*. San Francisco/London: Jossey-Bass.

Antonovsky, A. (1993). The structure and properties of the sense of coherence scale. *Social Science & Medicine, 36*, 725–733.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*, 191–215.

Eriksson, M., & Lindström, B. (2005). Validity of Antonovsky's sense of coherence scale. *Journal of Epidemiology and Community Health, 59*, 460–466.

Eriksson, M., & Lindström, B. (2007). Antonovsky's sense of coherence scale and it's relation with quality of life – a systematic review. *Journal of Epidemiology and Community Health, 61*, 938–944.

Kobasa, S. C. (1979). Stressful life events, personality, and health. *Journal of Personality and Social Psychology, 37*, 1–11.

Kobasa, S. C. (1982). The hardy personality: Toward a social psychology of stress and health. In G. S. Sanders & J. Suls (Eds.), *Social psychology of health and illness* (pp. 3–32). Hillsdale: Erlbaum.

Schnyder, U., Büchi, S., Mörgeli, H., Senky, T., & Klaghofer, R. (1999). Sense of coherence-a mediator between disability and handicap? *Psychotherapy and Psychosomatics, 68*, 102–110.

Singer, S., & Brähler, E. (2007). *Die "Sense of coherence scale". Testhandbuch zur deutschen Version*. Göttingen: Vandenhoeck & Ruprecht.

Tagay, S., Erim, Y., Brähler, E., & Senf, W. (2006). Religiosity and sense of coherence – protective factors of mental health and well-being? *Zeitschrift für Medizinische Psychologie, 4*, 165–171.

Tagay, S., Mewes, R., Brähler, E., & Senf, W. (2009). Sense of coherence in female patients with bulimia nervosa: A protective factor of mental health? *Psychiatrische Praxis, 36*, 30–34.

Tagay, S., Düllmann, S., Schlegl, S., Nater-Mewes, R., Repic, N., Hampke, C., Brähler, E., Gerlach, G., & Senf, W. (2011). Effects of inpatient treatment on eating disorder symptoms, health-related quality of life and personal resources in anorexia and bulimia nervosa. *Psychotherapie, Psychosomatik, Medizinische Psychologie, 61*(7), 319–327.

Werner, E., & Smith, R. (1982). *Vulnerable but invincible. A longitudinal study of resilient children and youth*. New York: McGraw Hill.

Sample Size Estimation

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S

Synonyms

Sample-size calculation; Sample-size determination; Study size

Definition

Sample-size estimation is the process by which a researcher decides how many subjects to include

in a given clinical trial. Sample-size estimation is a critical part of the design of clinical trials, and, like all design issues, this must be addressed in the study protocol before the trial commences.

Description

Many sources use the terms sample-size determination or sample-size calculation when discussing this issue. The term sample-size estimation emphasizes that deciding on the sample size that will be employed in a clinical trial is a process of estimation that involves both statistical and clinical informed judgment and not a process of simply calculating the "right" answer. It is true that mathematical calculations are made in this process, and, for a given set of values that are placed into the appropriate formula in any given circumstance, a precise answer will be given. However, the values that are placed into the formula are chosen by the researcher.

Some of the values that need to be entered into the formula are typically chosen from a standard set of possibilities, with the researcher deciding which of several generally acceptable values is best suited for the intentions of a given trial. Other values are estimates based on data that may be available in existing literature or may have been collected in an earlier trial. These include the estimated treatment effect and the variability associated with the estimated treatment effect.

The likelihood of a successful outcome (at least from the point of view that "success" means obtaining a statistically significant result) can be increased by increasing the sample size. When designing a study, the researcher wants to ensure that a large enough sample size is chosen to be able to detect an important difference that does in fact exist. It is certainly possible that a trial can fail to demonstrate such a difference simply because the sample size chosen was too small. Therefore, it might appear reasonable to think that a very big sample size is a good idea. However, increasing the sample size increases the expenses, difficulties and overall length of a trial. Somewhere, for each researcher and each study, an acceptable sample size needs to be chosen that balances the likelihood of a statistically significant result with the cost and time involved in conducting the clinical trial.

Several variables need to be considered in the process of sample-size estimation. The values of these variables in any given case can be chosen by the researcher based on several considerations. Relevant terms include:

- Type I errors and Type II errors. A Type I error occurs when a significant result is "found" when it does not really exist, and a Type II error occurs when one fails to find a significant difference that actually exists.
- The probability of making a Type I error, α. This is also the level of statistical significance chosen, typically 0.05, but it is possible to choose 0.01 or even more conservative values.
- The probability of making a Type II error, β. A probability value must be between 0 and 1: therefore, β will be between 0 and 1.
- Power, calculated as $1 - \beta$. Since the probability represented by β will be between 0 and 1, power will also be between 0 and 1 since it is defined as $1 - \beta$. The power of a statistical test is the probability that the null hypothesis is rejected when it is indeed false. Since rejecting the null hypothesis when it is false is extremely desirable, it is generally regarded that the power of a study should be as great as practically feasible.

Sample-size estimation can be performed for any study design. In each case, the respective formula will be used to estimate the sample size required. For the formula used in the type of study design discussed in some entries in the Methodology section (i.e., a comparison of a new behavioral medicine treatment or intervention with an existing one), each of the variables we have discussed will have certain influences on the sample size, N, that will be given by the formula. These influences, i.e., their relationships with N given that all of the others remain the same, can be summarized as follows:

- The smaller the chosen value of α, the larger the value of N that will be given.

- The smaller the chosen value of β, the larger the value of N that will be given. This is because power is defined as $1 - \beta$. As β decreases, power increases; as power increases, the larger the value of N that will be given.
- The larger the standardized effect size, the smaller the value of N that will be given. The standardized treatment effect is the estimated treatment effect divided by the variability associated with it.

Cross-References

- ▶ False-Negative Error
- ▶ False-Positive Error
- ▶ Probability
- ▶ Study Protocol

Sample-Size Calculation

- ▶ Sample Size Estimation

Sample-Size Determination

- ▶ Sample Size Estimation

Sarcopenia

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Synonyms

Anabolic resistance; Disuse atrophy; Skeletal muscle atrophy

Definition

Sarcopenia is a syndrome characterized by a progressive generalized loss of skeletal muscle mass and strength with a risk of adverse outcomes such as physical disability, poor quality of life, and death. Sarcopenia is derived from Greek "sarx" for flesh and "penia" for loss.

Description

The global population aged 60 years or older is expected to more than double from 901 million in the year 2015 to 2.1 billion by 2050 (www.un.org). A common result of the aging process is the loss of skeletal muscle mass. This is associated with metabolic disease such as type 2 diabetes (Park et al. 2009), impaired physical performance (e.g., walking and rising from a chair), self-reported disability (Janssen et al. 2002), and the increased risk of accidental falls (Szulc et al. 2005) and fractures (Fiatarone Singh et al. 2009). Hip fractures are among the most common site of fracture in elderly individuals (Johnell and Kanis 2006), and elderly hip fracture patients have a threefold higher all-cause age- and sex-standardized risk of mortality than the general population (Panula et al. 2011). Consequently, maintaining muscle mass will be very important for lowering the socioeconomic burden of an aging population.

The age-related loss of skeletal muscle mass and physical function was termed sarcopenia by Rosenberg (1989). The European Working Group on Sarcopenia in Older People (EWGSOP) now defines sarcopenia as "a syndrome characterized by progressive and generalized loss of skeletal muscle mass and strength with a risk of adverse outcomes such as physical disability, poor quality of life and death" (Cruz-Jentoff et al. 2010). The EWGSOP suggests three stages of severity for sarcopenia that depends on the cutoff points appropriate to the measurement technique used to measure muscle mass, strength, and physical performance. The "presarcopenia" stage is present in individuals who combine a low muscle mass

without impact on skeletal muscle strength or physical performance. "Sarcopenia" is the stage in which individuals combine a low muscle mass with either a low muscle strength or low physical performance. Finally, "severe sarcopenia" is present in individuals who combine a low muscle mass with a low muscle strength and low physical performance. In a systematic review, the estimated prevalence of sarcopenia using EWGSOP criteria was 1–29% in community-dwelling individuals and 14–33% of those receiving long-term care (Cruz-Jentoft et al. 2014). Using other diagnostic criteria, Baumgartner et al. (1998) observed the prevalence of sarcopenia in >50% of males and females aged >80 years.

Sarcopenia can be masked by weight stability in the presence of a concomitant increase in fat mass. However, the prolonged accumulation of fat mass can lead to obesity, resulting in "sarcopenic obesity." This presents further complications as excess fat mass which, in association with chronic low-level pro-inflammatory cytokines, can result in the development of insulin resistance in a variety of tissues such as the endothelium of the microvasculature and within skeletal muscle fibers. This leads to periods of hyperglycemia, hyperinsulinemia, and hyperlipidemia and the development of type 2 diabetes and cardiovascular disease (Wagenmakers et al. 2006, 2016).

The genesis of sarcopenia can be attributable to physical inactivity and inadequate nutrition among other factors (Table 1).

Sarcopenia, Table 1 Summary of the potential mechanisms underpinning sarcopenia

Whole body
Reduced physical activity and muscle disuse
Loss of motor neurons
Reduced growth hormone and insulin-like growth factor-I production
Chronic pro-inflammatory state
Increased glucocorticoid production and receptor activity
Malnutrition
Muscular
Reduced number and proliferative capacity of skeletal muscle satellite cells
Mitochondrial DNA mutations and apoptosis
Increased intracellular production of glucocorticoids
Impaired insulin-mediated increase in microvascular perfusion
Blunting of the effects of amino acids on muscle protein synthesis
Blunting of the effects of insulin on muscle protein breakdown
Impaired transcriptional responses of muscle to exercise and nutrition

Effect of Age on Skeletal Muscle

Skeletal muscle mass peaks between 20 and 30 years of age and the loss of muscle mass begins in the fourth decade (Janssen et al. 2000). The loss of muscle mass is more notable in lower than upper limbs (Janssen et al. 2000) with leg skeletal muscle mass lost at an average rate of approximately 1% per year for males and females during their eighth decade (Goodpaster et al. 2006). Most individuals aged over 70 years will possess about 80% of the muscle mass of those aged 20–30 years.

Leg strength can be lost at an average rate of approximately 2.8% and 3.8% per year for females and males aged >70 years over a 3-year period (Goodpaster et al. 2006). This exceeds the annual loss of muscle mass and suggests a reduction in muscle quality (force per unit area of tissue) with age. Muscle power also declines with age, and low muscle power is associated with a two- to threefold greater risk of impaired physical performance than low muscle strength (Bean et al. 2003). The effect of age on muscle strength and power has been termed dynapenia (Manini and Clark 2012). The loss of skeletal muscle mass and strength is evident even in those who engage in regular aerobic or resistance exercise.

Sarcopenia and dynapenia have been linked to the age-related loss of spinal motor neurons (Aagaard et al. 2010). This leads to a reduction in the number and size of muscle fibers and results in impaired muscle mechanical performance (Aagaard et al. 2010). Some of the denervated muscle fibers are reinnervated by surviving motor units, leading to the formation of very

large motor units and the appearance of fiber-type grouping (Andersen 2003). Of the remaining muscle fibers, atrophy is more notable in type II fibers. In people aged 88 years, type II fibers had atrophied in size to about 43% of a matched group of 25-year-olds, but type I fibers had atrophied in size to about 75% of their younger counterparts (Andersen 2003). These effects, in combination with an age-related decline in neuromuscular function, lead to impaired muscle mechanical performance and a reduced ability to complete normal daily living tasks such as walking and rising from a chair (Aagaard et al. 2010). Further age-related changes include the infiltration of fat and connective tissue within the muscle and a reduction in type II fiber satellite cell content (Koopman and van Loon 2009). A reduction in the oxidative capacity of skeletal muscle is also observed with age and is likely attributable to a reduction in mitochondrial content and/or function. This results in poor endurance and increased fatigability, compromising the ability to live an independent lifestyle. Poor oxidative capacity is also mechanistically linked to the development of insulin resistance and type 2 diabetes (Wagenmakers et al. 2006, 2016).

Protein Metabolism in the Elderly

Contemporary evidence suggests basal rates of muscle protein synthesis (MPS) and muscle protein breakdown (MPB) in the elderly equal that of younger individuals and does not explain the age-related loss of muscle mass (Wall et al. 2014). Instead, sarcopenia might be attributable to anabolic resistance, the reduced ability to mount a youthful response of MPS to protein ingestion, and/or muscle loading (Witard et al. 2016). The mechanisms underlying anabolic resistance remain to be elucidated, but impairments may exist at the cellular level (e.g., protein/amino acid digestion, absorption, and transport) and molecular level (e.g., Akt-mTOR-p70S6K signaling pathway) (Wall et al. 2014; Witard et al. 2016). These may impede the availability of amino acids for MPS and blunt the sensing and transduction of amino acid- and/or insulin-dependent signaling, thereby limiting the stimulation of MPS and the inhibitory effects of insulin on MPB. As exercise increases the sensitivity of the muscle to the anabolic effects of protein intake, increasing the physical activity rates of the elderly may help counteract anabolic resistance (Wall et al. 2014).

Muscle loss and anabolic resistance may also develop through periods of reduced physical activity and muscle disuse. An accelerated loss of muscle mass and blunting of postprandial MPS in elderly individuals after 2 weeks of reduced daily physical activity have been reported (Breen et al. 2013). Suggestions have also been made that an accelerated loss of muscle mass during periods of illness leading to bed rest ($<$10 days) is irreversible and will lead to stepwise reductions in muscle mass (Wall et al. 2013).

The age-associated decline in daily physical activity and total energy expenditure also leads to a gradual reduction of both the calorie and protein intake, if the dietary protein content is kept constant. Consequently, sedentary and frail elders may fail to consume the current recommended daily allowance (RDA) for protein intake (0.8 g/kg body mass). An increase of the protein intake of the diet from ±15 to ≥ 20 En% is enough to prevent this deficit (Houston et al. 2008). In addition, such an increase will be more effective in maximally stimulating (post-exercise) MPS and, therefore, attenuating the loss of muscle mass (Houston et al. 2008; Wall et al. 2014; Witard et al. 2016).

Resistance Training in the Elderly

Resistance exercise training is a potent stimulator of MPS, leading to increased muscle mass (mostly attributable to type II fiber hypertrophy) and strength in the young, elderly, frail elderly, and older individuals presenting with comorbidities. Resistance training therefore offers an effective strategy to counteract sarcopenia and improve functional muscle capacity. The American College of Sports Medicine (Chodzko-Zajko

et al. 2009) recommends progressive resistance training at least 2 days per week using 8–10 exercises involving the major muscle groups over 8–12 repetitions. Gains in muscular endurance of up to 200% have also been reported after completing moderate- to high-intensity resistance training (Chodzko-Zajko et al. 2009). An increase in mitochondrial protein content and oral glucose tolerance has also been reported in the elderly after resistance training (Frank et al. 2016).

Conclusion

Sarcopenia contributes to the increased risk of falls, fractures, and functional impairment and increased dependency. The loss of muscle mass is also associated with type 2 diabetes. Anabolic resistance to protein and exercise may contribute to the development of sarcopenia, and this may be further exacerbated through periods of reduced physical activity or bed rest. The accelerated loss of muscle mass during these periods may be irreversible. However, resistance exercise is a potent stimulator of MPS, and progressive resistance training results in improved muscle mass and strength and will give older individuals the confidence to continue to engage in aerobic exercise such as walking. The combination of resistance and aerobic exercise is optimal to maintain metabolic health until a very high age.

Cross-References

- ▶ Atrophy
- ▶ Cytokines
- ▶ Glucocorticoids
- ▶ Insulin
- ▶ Physical Inactivity

References and Further Readings

Aagaard, P., Suetta, C., Caserotti, P., Magnusson, S. P., & Kjaer, M. (2010). Role of the nervous system in sarcopenia and muscle atrophy with aging: Strength training as a countermeasure. *Scandinavian Journal of Medicine & Science in Sports, 20*, 49–64.

Andersen, J. L. (2003). Muscle fibre type adaptation in the elderly human muscle. *Scandinavian Journal of Medicine & Science in Sports, 13*, 40–47.

Baumgartner, R. N., Koehler, K. M., Gallagher, D., Romero, L., Heymstleld, S. B., Ross, R. R., Garry, P. G., & Lindeman, R. D. (1998). Epidemiology of sarcopenia among the elderly in New Mexico. *American Journal of Epidemiology, 147*(8), 755–763.

Bean, J. F., Leveille, S. G., Kiely, D. K., Bandinelli, S., Guralnik, J. M., & Ferrucci, L. (2003). A comparison of leg power and leg strength within the InCHIANTI study: Which influences mobility more? *Journals of Gerontology. Series A, Biological Sciences and Medical Sciences, 58*(8), 728–733.

Breen, L., Stokes, K. A., Churchward-Venne, T. A., Moore, D. R., Baker, S. K., Smith, K., Atherton, P. J., & Phillips, S. M. (2013). Two weeks of reduced activity decreases leg lean mass and induces "anabolic resistance" of myofibrillar protein synthesis in healthy elderly. *Journal of Clinical Endocrinology and Metabolism, 98*(6), 2604–2612.

Chodzko-Zajko, W. J., Proctor, D. N., Fiatarone Singh, M. A., Minson, C. T., Nigg, C. R., & Salem, G. J. (2009). Exercise and physical activity for older adults. *Medicine & Science in Sports & Exercise, 41*, 1510–1530.

Cruz-Jentoff, A. J., Baeyens, J. P., Bauer, J. M., Boirie, J. M., Cederholm, T., Landi, F., Martin, F. C., Michel, J. P., Rolland, Y., Schneider, S. M., Topinkova, E., Vandewoude, M., & Zamboni, M. (2010). Sarcopenia: European consensus on definition and diagnosis: Report of the European working group on sarcopenia in older people. *Age and Ageing, 39*(4), 412–423.

Cruz-Jentoft, A. J., Landi, F., Schneider, S. M., Zúñiga, C., Arai, H., Boirie, Y., Chen, L. K., Fielding, R. A., Martin, F. C., Michel, J. P., Sieber, C., Stout, J. R., Studenski, S. A., Vellas, B., Woo, J., Zamboni, M., & Cederholm, T. (2014). Prevalence of and interventions for sarcopenia in ageing adults: A systematic review. Report of the International Sarcopenia Initiative (EWGSOP and IWGS). *Age and Ageing, 43*(6), 748–759.

Fiatarone Singh, M. A., Singh, N. A., Hansen, R. D., Finnegan, T. P., Allen, B. J., Diamond, T. H., Diwan, A. D., Lloyd, B. D., Williamson, D. A., Smith, E. U., Grady, J. N., Stavrinos, T. M., & Thompson, M. W. (2009). Methodology and baseline characteristics for the sarcopenia and hip fracture study: A 5-year prospective study. *Journal of Gerontolgical Advances in Biological Sciences and Medical Sciences, 64A*(5), 568–574.

Frank, P., Andersson, E., Pontén, M., Ekblom, B., Ekblom, M., & Sahlin, K. (2016). Strength training improves muscle aerobic capacity and glucose tolerance in elderly. *Scandinavian Journal of Medicine & Science in Sports, 26*(7), 764–773.

Goodpaster, B. H., Park, S. W., Harris, T. B., Kritchevsky, S. B., Nevitt, M., Schwartz, A. V., Simonsick, E. M., Tylavsky, F. A., Visser, M., & Newman, A. B. (2006).

The loss of skeletal muscle strength, mass, and quality in older adults: The health, aging and body composition study. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences, 61*(10), 1059–1064.
Houston, D. K., Nicklas, B. J., Ding, J., Harris, T. B., Tylavsky, F. A., Newman, A. B., Lee, J. S., Sahyoun, N. R., Visser, M., Kritchevsky, S. B., & Health ABC Study. (2008). Dietary protein intake is associated with lean mass change in older, community-dwelling adults: The Health, Aging, and Body Composition (Health ABC) Study. *American Journal of Clinical Nutrition, 87*(1), 150–155.
Janssen, I., Heymsfield, S. B., Wang, Z., & Ross, R. (2000). Skeletal muscle mass and distribution in 468 men and women aged 18–88 yr. *Journal of Applied Physiology, 89*(1), 81–88.
Janssen, I., Heymsfield, S. B., & Ross, R. (2002). Low relative skeletal muscle mass (sarcopenia) in older persons is associated with functional impairment and physical disability. *Journal of the American Geriatrics Society, 50*(5), 889–896.
Johnell, O., & Kanis. (2006). An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. *Osteoporosis International, 17*, 1726–1733.
Koopman, R., & van Loon, L. J. (2009). Aging, exercise, and muscle protein metabolism. *Journal of Applied Physiology, 106*, 2040–2048.
Manini, T. M., & Clark, B. C. (2012). Dynapenia and aging: An update. *Journals of Gerontology. Series A, Biological Sciences and Medical Sciences, 67*(1), 28–40.
Panula, J., Pihlajamäki, H., Mattila, V. M., Jaatinen, P., Vahlberg, T., Aarnio, P., & Kivela, S. L. (2011). Mortality and cause of death in hip fracture patients aged 65 or older – A population-based study. *BMC Musculoskeletal Disorders, 12*(105), 1–6.
Park, S. W., Goodpaster, B. H., Lee, J. S., Kuller, L. H., Boudreau, R., de Rekeneire, N., Harris, T. B., Kritchevsky, S., Tylavsky, F. A., Nevitt, M., Cho, Y. W., & Newman, A. B. (2009). Excessive loss of skeletal muscle mass in older people with type 2 diabetes. *Diabetes Care, 32*, 1993–1997.
Rosenberg, I. (1989). Summary comments: Epidemiological and methodological problems in determining nutritional status of older persons. *American Journal of Clinical Nutrition, 50*, 1231–1233.
Szulc, P., Beck, T. J., Marchand, F., & Delmas, P. D. (2005). Low skeletal muscle mass is associated with poor structural parameters of bone and impaired balance in elderly men – The MINOS study. *Journal of Bone and Mineral Research, 20*(5), 721–729.
Wagenmakers, A. J., vanRiel, N. A., Frenneaux, M. P., & Stewart, P. M. (2006). Integration of the metabolic and cardiovascular effects of exercise. *Essays in Biochemistry, 42*, 193–210.
Wagenmakers, A. J., Strauss, J. A., Shepherd, S. O., Keske, M. A., & Cocks, M. (2016). Increased muscle blood supply and transendothelial nutrient and insulin transport induced by food intake and exercise: Effect of obesity and ageing. *Journal of Physiology, 594*, 2207–2222.
Wall, B. T., Dirks, M. L., & van Loon, L. J. C. (2013). Skeletal muscle atrophy during short-term disuse: Implications for age-related sarcopenia. *Ageing Research Reviews, 12*, 898–906.
Wall, B. T., Cermak, N. M., & van Loon, L. J. C. (2014). Dietary protein considerations to support active ageing. *Sports Medicine, 44*(2), S185–S194.
Witard, O. C., McGlory, C., Hamilton, D. L., & Phillips, S. M. (2016). Growing older with health and vitality: A nexus of physical activity, exercise and nutrition. *Biogerontology, 17*, 529–546.
www.un.org United Nations, Department of Economic and Social Affairs: Population Division. (2015). *World population prospects: The 2015 revision, key findings and advance tables.* Accessed 25 Aug 2016.

Saturated Fats

▶ Fat, Dietary Intake

Saturated Fatty Acids

▶ Fat: Saturated, Unsaturated

SBM

▶ Society of Behavioral Medicine

S

Scale Development

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Synonyms

Questionnaire development

Definition

Scale development is an essential stage in the assessment of constructs and variables in behavior medicine and in any social and health science. Scales are used for assessment of self-reported variables including mood, daily disability, various types of symptoms, adherence to recommended diet, etc. Though there is no explicit "rule" for the stages of scale development, certain steps need to be included for claiming that a scale is reliable and valid. The reliability of a scale is very important and refers to its repeatability and lack of measurement error. This is tested by internal reliability tests (Cronbach's α) and by a test-retest reliability of scores over time. Validity is an essential aspect of a scale and refers to the extent to which it measures what it claims to measure. This is tested by several manners including "face validity," concurrent validity, construct validity, and criterion validity. When developing a scale, it is essential to have a clear definition of the concept it refers to. Thus, for example, an anxiety scale should not have items assessing depression since these are not the same construct. After choosing an acceptable definition for the construct, a group of "experts" on the construct meets to provide items or even topics related to the construct, from which the researcher creates items. The chosen items will reflect the most common topics or items suggested by the expert panel. The panel can be experts from the field (psychologists, physicians, etc.) and patients who experienced the issue under investigation, thus reflecting experienced "experts." Then, the investigator can ask another group of experts or patients to rate the relevance of each item to the construct, reflecting face validity. The items with a mean relevance above a chosen criterion will be selected for the preliminary scale. Next, the researcher administers the scale to a larger sample, with theoretically relevant additional tests. This will enable to test the internal reliability, concurrent validity against another scale assessing the same construct, and the construct validity against scales assessing theoretically related constructs. Finally, an acceptable criterion (e.g., ill vs. healthy sample) will enable to test the scale's criterion validity. Predictive validity can also be tested by examining whether the scale's scores predict a certain event or outcome in the future, beyond the effects of known confounders. For example, Barefoot et al. (1989) tested the predictive validity of a shorter hostility scale derived from the original Ho scale in the Minnesota Multiphasic Personality Inventory. They found that the brief scale which included cynicism, hostile affect, and aggressive responding predicted death better than the original full Ho scale, supporting the brief scale's predictive validity. These steps are needed for scale development, to verify a scale's reliability and validity, for use in research and clinical evaluations.

Cross-References

▶ Reliability and Validity

References and Further Readings

Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, W. G., & Williams, R. B., Jr. (1989). The Cook-Medley hostility scale: Item content and ability to predict survival. *Psychosomatic Medicine, 51*, 46–57.

Clark, L. A., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. *Psychological Assessment, 3*, 309–319.

Scatter

▶ Dispersion

Scenario Design

▶ Scenario-Based Design

Scenario-Based Design

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Synonyms

Scenario design; Scenario-based design; User-centered design approaches

Definition

Scenario-based design is a validated and user-centered design approach, used early in the design process of a technology or product, which relies upon "scenarios" or stories to capture the elements of interaction between a person and a future product.

Description

Scenario-based design (SBD) refers to a family of validated user-centered design approaches that use "scenarios," or stories, to inform the design of a technology or product. These scenarios are narratives, which may be in the form of text, storyboards, videos, etc. The scenarios are created and utilized to concretely define a user's experience of an interaction with a technology, what happens during that interaction, and how it happens. Indeed, given that scenarios are like stories, they have characters, character goals and actions, and a plot within a larger setting. Through the use of scenarios, the use of the product becomes contextualized and more explicit, thereby informing ultimate design decisions (Carroll 1985; Rosson and Carroll 2002).

Use in the Design of Behavioral Intervention Technologies

SBD is increasingly being utilized in the field of behavioral medicine, primarily in designing behavioral intervention technologies (BITs). As BITs tend to be designed by interdisciplinary teams, ranging from engineers to psychologists (Schueller et al. 2014), SBD lends well to the design process across disciplines. Indeed, SBD's use of scenarios to contextualize a BIT interaction is similar to the use of case vignettes to establish clinical practice techniques (a training practice common in medical and clinical fields), while also being a design approach familiar to technologists, usability experts, and engineers. Further, the use of SBD provides a common framework and language for the interdisciplinary team to utilize throughout the design process (Carroll 2000). Recent examples of the use of SBD include BITs targeting well-being and mental health in youth (Blythe and Wright 2006; Bødker 2000; Orlowski et al. 2015) and apps targeting symptoms of depression and anxiety in adults (Mohr et al. 2017; Stiles-Shields et al. 2016). Additionally, in examining the impact of the use of SBD in the design of medication alerts for prescribers, system usability improved, errors were reduced, and the prescribers reported a decrease in perceived work load (Russ et al. 2014). For the purposes of direct application of SBD to the design process of BITs, the remainder of the description of SBD will utilize the design of a mobile treatment app for adults with depression as an example.

Framework

The framework of SBD involves three primary phases: Analysis, Design, and Prototype and Evaluation (Rosson and Carroll 2002). The first phase of the framework is *Analysis*, in which claims about current practices and possible stakeholders are identified through the use of *Problem Scenarios*. Problem Scenarios depict the story of a user in a current practice. To represent the user, personas are often created to elucidate the benefits and challenges that may be incorporated and/or addressed through the design process. For the

example of the design of an app for depression, the persona demographics could be informed by samples of adults interested in treatments delivered through technology, treatment-seeking patients in traditional community or clinic settings, and/or national prevalence rates of adults with depression. These personas would be individualized (e.g., "Sara," a 21-year-old Latina with moderate depression who moved to a new city within the last year), and then used in Problem Scenarios designed to highlight benefits and challenges associated with seeking depression treatment through current practices (e.g., face-to-face therapy, medication, and/or self-help resources). A *Claims Analysis* would follow, with the intention to reveal positive and negative features about current practice. For example, a design team might consider varying positive (e.g., a therapist can directly address "Sara's" concerns as they are brought up in the moment) and negative (e.g., "Sara" might need to wait several weeks for an initial therapy session, possibly impacting her motivation and symptomology) features of a face-to-face therapy session (e.g., a "current practice" for depression treatment). A stakeholder analysis would also follow. *Stakeholders* include anyone who might be impacted by the product. Stakeholders might have conflicting interests from other stakeholders, and may be Primary (use the product), Secondary (provide input or receive output from the product), or Tertiary (no direct involvement but effected by successes or failures). For example, in the design of an app for adults with depression, stakeholders might include: patients with depression (Primary); researchers and clinical providers (Secondary); and users of future iterations of the app, the immediate social network of the patient, referral sources, developers and technologists, and other mHealth researchers (Tertiary). Through the Analysis process of SBD, designers elucidate the positive and negative elements of current practices for stakeholders, while identifying how a new product will maintain, enhance, or alter current practice (Carroll 1985; Rosson and Carroll 2002).

The second phase of the SBD framework is *Design*, in which multiple iterations of designs are completed through analyses of Design Claims informed by *Activity Scenarios, Information Design Scenarios, and Interaction Design Scenarios*. Activity Scenarios are persona interactions with the new technology, and therefore serve as initial depictions of the transformation from the current practice to the new design features. The intended output of Activity Scenarios is *Design Claims*, which detail new features and their purposes. For example, an Activity Scenario for the design of an app for depression may depict "Sara" locating the app on an app store and learning about the functionality through pop up notifications at app launch; a Design Claim from this scenario would include brief, informative notifications at initial launch. Information Design Scenarios add nuance to the Activity Scenarios, such that they make perceptual details more explicit. The ultimate aim of this process is to identify how a user might perceive or interpret the product and/or information provided. For example, an Information Design Scenario would describe specifically what the launch notification reads to "Sara" and how she might interpret that information. Finally, Interaction Design Scenarios extend the design process initiated by the previous scenarios by explicitly detailing the actions a user might take, and how the system would respond. For example, these scenarios would provide step-by-step information about "Sara's" launch of the app, what is presented, how she proceeds, etc. Throughout the Design phase of the SBD framework, multiple iterations are completed across personas to inform and enrich the design (Carroll 1985; Rosson and Carroll 2002).

The final phase of the SBD framework is *Prototype and Evaluation*. Resulting from the multiple iterations of scenarios created during the Analysis and Design phases, *Prototypes* may be created as preliminary models of aspects of the designed product. Prototypes may range from simple (e.g., paper drawings, tending to be less costly and time intensive) to complex/realistic (e.g., an initial version of a mobile app, tending to have greater validity). The purpose of creating prototypes in the SBD process is to engage in usability evaluations of the designs. These evaluations may be formative (i.e., occurring during the design and development) or summative (i.e., occurring at the end of the development stage; Tullis and Albert 2008; Please see "Usability Testing" entry for more information). This evaluative process enables teams to finalize

their designs and prepare for the ultimate creation of a new product.

Benefits

Multiple benefits of SBD have been highlighted in the literature. First, scenarios evoke careful attention and reflection for design teams through their use of depictions of persona experiences with the product. This process often highlights how well design ideas align with ultimate user and designer goals. Second, scenarios allow for flexibility, in that they are easily revised, but can also promote concrete solutions to identified problems. Third, scenarios can be created from multiple perspectives and levels, and for varying purposes. They can anchor design discussions in specific work products, across multiple disciplines, and design team members. Finally, scenarios are easily abstracted and categorized, promoting the recognition, use, and reuse of generalizations or patterns that emerge in interactions with the designed product (Carroll 1985, 2000; Rosson and Carroll 2002). SBD therefore stands as a promising and accessible means for interdisciplinary teams to design BITs, or other products which may promote behavioral health.

Cross-References

► Usability Testing

References and Further Reading

Blythe, M. A., & Wright, P. C. (2006). Pastiche scenarios: Fiction as a resource for user centred design. *Interacting with Computers, 18*(5), 1139–1164. https://doi.org/10.1016/j.intcom.2006.02.001.

Bødker, S. (2000). Scenarios in user-centred design—Setting the stage for reflection and action. *Interacting with Computers, 13*(1), 61–75. https://doi.org/10.1016/S0953-5438(00)00024-2.

Carroll, J. M. (1985). *Scenario-based design: Envisioning work and technology in system development*. New York: Wiley.

Carroll, J. M. (2000). Five reasons for scenario-based design. *Interacting with Computers, 13*(1), 43–60. https://doi.org/10.1016/S0953-5438(00)00023-0.

Mohr, D. C., Tomasino, K. N., Lattie, E. G., Palac, H. L., Kwasny, M. J., Weingardt, K., et al. (2017). IntelliCare: An eclectic, skills-based app suite for the treatment of depression and anxiety. *Journal of Medical Internet Research, 19*(1). https://doi.org/10.2196/jmir.6645.

Orlowski, S. K., Lawn, S., Venning, A., Winsall, M., Jones, G. M., Wyld, K., et al. (2015). Participatory research as one piece of the puzzle: A systematic review of consumer involvement in design of technology-based youth mental health and well-being interventions. *JMIR Human Factors, 2*(2). https://doi.org/10.2196/humanfactors.4361.

Rosson, M. B., & Carroll, J. M. (2002). *Usability engineering: Scenario-based development of human-computer interaction*. San Francisco: Morgan Kaufmann.

Russ, A. L., Zillich, A. J., Melton, B. L., Russell, S. A., Chen, S., Spina, J. R., et al. (2014). Applying human factors principles to alert design increases efficiency and reduces prescribing errors in a scenario-based simulation. *Journal of the American Medical Informatics Association, 21*(e2), e287–e296. https://doi.org/10.1136/amiajnl-2013-002045.

Schueller, S. M., Begale, M., Penedo, F. J., & Mohr, D. C. (2014). Purple: A modular system for developing and deploying behavioral intervention technologies. *Journal of Medical Internet Research, 16*(7). https://doi.org/10.2196/jmir.3376.

Stiles-Shields, C., Montague, E., & Mohr, D. C. (2016). *The use of scenario-based design for the development of behavioral intervention technologies*. International Society for Research on Internet Interventions (ISRII) 8th scientific meeting, Washington, DC.

Tullis, T., & Albert, B. (2008). *Measuring the user experience: Collecting, analyzing, and presenting usability metrics*. Burlington: Morgan Kaufmann Publishers.

Schneiderman, Neil

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Neil Schneiderman was born in Brooklyn, New York, on February 24, 1937. He has been

married to his wife Eleanor since 1960 and is the father of three children and grandfather of five. Schneiderman received his A.B.. degree from Brooklyn College, spent 2 years in the US Army, earned his Ph.D. degree in Psychology from Indiana University, and received postdoctoral training in Neurophysiology and Neuropharmacology in the Physiological Institute of the University of Basel, Switzerland. Schneiderman was appointed as assistant professor at the University of Miami, Coral Gables, Florida, in 1965, rising through the ranks to become professor in 1974. He subsequently received secondary appointments as professor of Medicine, Public Health Sciences, Psychiatry and Behavioral Sciences, and Biomedical Engineering. In 1989, he was awarded an endowed chair as the James L. Knight Professor of Health Psychology. Since 1986, he has served as the director of the Division of Health Psychology in the Department of Psychology and as director of the University of Miami Behavioral Medicine Research Center. He also served extensively as chair of the NIH-funded University of Miami General Clinical Research Center Advisory Committee. Schneiderman has directed pre- and postdoctoral NIH training grants involving cardiovascular disease from the National Heart, Lung, and Blood Institute (NHLBI) since 1979 and HIV/AIDS from the National Institute of Mental Health (NIMH) from 1993 to 2017.

Schneiderman was the second editor in chief of the journal *Health Psychology* before becoming founding editor in chief of the *International Journal of Behavioral Medicine*. Within the NIH, he served as a member of the Biopsychology Study Section, NHLBI Research Training Review Committee, and NIMH Health Behavior and Prevention Review Committee. In the American Psychological Association (APA), he was chair of the Board of Scientific Affairs and is a fellow in the Divisions of Experimental Psychology (3), Behavioral Neuroscience and Comparative Psychology (6), and Health Psychology (38) as well as a former president of Division 38. A founding fellow of the Academy of Behavioral Medicine Research, Schneiderman later served as president of that organization. Schneiderman also served as president of the International Society of Behavioral Medicine (ISBM). He is a fellow of the Society of Behavioral Medicine and of the American College of Clinical Pharmacology. He is also the recipient of the APA Distinguished Scientific Contribution Award (1994), Society of Behavioral Medicine Distinguished Scientist Award (1997), ISBM Outstanding Scientific Achievement Award (2004), and American Psychosomatic Society Distinguished Scientist Award (2014).

Major Accomplishments

Schneiderman's first two empirical research articles were published in *Science* in 1962. Written with his academic mentor, Isidore Gormezano, the papers described animal models of eyelid and nictitating membrane Pavlovian conditioning in rabbits. These preparations were suitable for concomitantly studying behavioral and neurophysiological processes in conscious, minimally restrained animals. Subsequently, Schneiderman added heart rate conditioning to the repertoire of animal models, and for the next several decades, he and his colleagues traced neuronal pathways involved in Pavlovian conditioning of cardiovascular responses in rabbits. This began with identifying the cells of origin of vagal cardioinhibitory motoneurons in the rabbit medulla, using histochemistry, microstimulation, and single neuron extracellular electrophysiological recordings, and continued with mapping the central nervous system pathways that mediated conditioned and unconditioned cardiovascular adjustments. Key collaborators included James Schwaber, Marc Kaufman, Howard Ellenberger, and Michael Spyer. The study of central nervous system pathways mediating differentiated patterns of cardiovascular adjustments also led Schneiderman and his colleagues including Marc Gellman, Barry Hurwitz, Maria Llabre, and Pat Saab to conduct an important series of psychophysiological studies in humans. They described differentiated patterns of neurohormonal and cardiovascular responses to separate behavioral stressors as a

function of race, gender, and hypertensive status. These responses were also shown to be influenced by such psychosocial factors as harassment and hostility. Subsequently, Schneiderman collaborated with Philip McCabe, Armando Mendez, and other Miami scientists in documenting the psychosocial prevention of atherosclerosis progression in a rabbit model of coronary artery disease.

Because of Schneiderman's interest in relationships among biological regulation, psychosocial factors, and disease processes, it was not surprising that he also joined with colleagues including MaryAnn Fletcher, Gail Ironson, and Nancy Klimas relatively early in the HIV/AIDS epidemic to study relationships between psychosocial variables and endocrine-immune regulation in HIV-infected patients, when AIDS was beginning to ravage the Miami community. This, in turn, led Schneiderman, Michael Antoni, and their collaborators to begin to use group-based cognitive behavior therapy and relaxation training in randomized controlled trials to influence psychosocial, endocrine, and immune factors and even to reduce HIV viral load to undetectable levels in patients who were failing their regimen of highly active antiretroviral drugs.

Schneiderman's broad research experience, including intervention studies with clinical patients, prepared him to serve as principal investigator of the Miami Field Center for the NIH/NHLBI "Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD)" randomized trial. The trial compared cognitive behavior therapy and usual care in post-myocardial infarction patients. Although that trial produced null results in terms of morbidity and mortality, Schneiderman and colleagues conducted a secondary analysis that suggested that the trial appeared to decrease morbidity and mortality in White men, but not in women or minority patients. Based on the supposition that the null result in the ENRICHD trial was due to the protocol not being sufficiently tailored to women, Schneiderman joined with Kristina Orth-Gomér and other Swedish colleagues to conduct the "Stockholm Women's Intervention Trial for Coronary Heart Disease (SWITCHD)." This trial, which used group-based cognitive behavior therapy and relaxation training in women previously hospitalized for myocardial infarction or coronary revascularization, showed a significant decrease in mortality rate for the intervention compared with a usual care group. Similar results have now also been reported by others.

In addition to Schneiderman's contributions in basic science and in clinical trials research, he has been actively involved in population-based observational studies such as those with Ronald Goldberg and Jay Skyler. As the principal investigator of the Miami Field Center of the NIH/NHLBI multicenter Hispanic Community Health Study/Study of Latinos, Schneiderman and his colleagues in Miami, including Frank Penedo, David Lee, and Marc Gellman, as well as investigators in the Bronx, Chicago, and San Diego are characterizing the health status and disease burden of Hispanic adults living in the United States, describing the positive and negative consequences of immigration and acculturation in relation to lifestyle and access to health care and assessing likely causal factors of disease in this diverse population. This study, sponsored by the NIH, was initiated in 2006 and is funded until 2024. Between 2008 and 2011, 16,415 women and men 18–74 years of age, who self-identified as being Hispanic or Latino, completed a 6.5 h baseline clinical exam. Participants were recruited from a stratified random sample of households in defined committees in the Bronx, Chicago, Miami, and San Diego, thus including a wide range of ancestral backgrounds. The study assessed risk factors and prevalence of heart, lung, blood, and sleep disorders, liver and kidney dysfunction, diabetes, cognitive impairment, dental problems, and hearing disorder. A second clinic exam was conducted on 80% of the original cohort between 2014 and 2017, and a third exam is being conducted between 2019 and 2023. The emphasis of clinic exams 2 and 3 is on long-term follow-up and disease incidence. In summary, Schneiderman's contributions have been in terms of basic science, population-based observational studies, and randomized clinical trials.

Cross-References

▶ International Society of Behavioral Medicine

References and Further Readings

Ironson, G. H., Gellman, M. D., Spitzer, S. B., Llabre, M. M., Pasin, R. D., Weidler, D. J., et al. (1989). Predicting home and work blood pressure measurements from resting baselines and laboratory reactivity in black and white Americans. *Psychophysiology, 26*, 174–184.

Orth-Gomér, K., Schneiderman, N., Wang, H., Walldin, C., Blom, M., & Jernberg, T. (2009). Stress reduction prolongs life in women with coronary disease: The Stockholm women's intervention trial for coronary heart disease (SWITCHD). *Circulation. Cardiovascular Quality and Outcomes, 2*, 25–32. https://doi.org/10.1161/CIRCOUTCOMES.108.812859.

Schneiderman, N., Fuentes, I., & Gormezano, I. (1962). Acquisition and extinction of the classically conditioned eyelid responses in the albino rabbit. *Science, 136*, 650–652.

Schneiderman, N., Saab, P. G., Catellier, D. J., Powell, L. H., DeBusk, R. F., Williams, R. B., et al. (2004). Psychosocial treatment within sex by ethnicity subgroups in the enhancing recovery in coronary heart disease (ENRICHD) clinical trial. *Psychosomatic Medicine, 66*, 475–483.

Schwaber, J., & Schneiderman, N. (1975). Aortic nerve activated cardioinhibitory neurons and interneurons. *American Journal of Physiology, 229*, 783–789.

Scientific Psychology

▶ Psychological Science

Screen Time

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Synonyms

Computer use; Screen-based behaviors; Sedentary behavior; TV viewing

Definition

The time spent engaged in screen-based behaviors. ∗*These behaviors can be performed while being sedentary or physically active.*

Screen time refers to the time spent engaged in "screen-based" behaviors, such as watching television (TV) and using a smartphone, tablet, or computer (Tremblay et al. 2017). Screen time is the most studied component sedentary behavior (i.e., waking behavior $\leq$1.5 metabolic equivalents, while in a sitting, reclining, or lying posture), with researchers typically examining the link between screen-based sedentary behaviors and several health indicators – such as adiposity and cardio-metabolic (including diabetes and metabolic syndrome) and a cardiovascular outcome (van Ekris et al. 2017).

Much of the existing research in this domain includes observational studies investigating the adverse health consequences of TV viewing time among children and adults. Overall, results of both cross-sectional and prospective studies are suggestive of a positive (adverse) relationship between TV viewing and adiposity indicators among youth, such as overweight/obesity, body-mass-index, and body fat (%) (Biddle et al. 2017, 2018). In addition, prospective studies in adults suggest higher levels of TV viewing are linked to increased risk of cardiovascular, cancer, and all-cause mortality (Dunstan et al. 2010). However, authors consistently emphasize that the associations observed between TV viewing with studied health outcomes may be bi-directional in nature (i.e., we cannot rule out reverse causality) and might be mediated or moderated by coexisting behaviors, such as physical activity (Biddle et al. 2018). These considerations are not exclusive to studies examining the health implications of TV viewing, but extend to other screen-based behavior, such as using a computer, smartphones, tablet, or screen-based entertainment systems.

With this in mind, future research that considers the mediating roles of concomitant health behaviors in the associations between screen time and various health outcomes is critical, particularly when we consider how advances in modern technology have changed the manner in which people access and engage in screen-based

behaviors. For example, increased use of smartphones and tablets – not of which will be engaged in through sitting – have contributed to overall declines in TV viewing (Ofcom 2018). Consequently, screen-based behaviors can be engaged when being sedentary (e.g., at home, watching TV) or while being active (e.g., using a phone while walking, exergames), across many contexts (e.g., home, school, work, travel) and for many different purposes (e.g., work/study, recreation). The changing technological landscape that is facilitating access to screen-based behaviors is therefore essential to consider when studying the role of "screen time" – and the diverse behaviors this may encompass – for health indicators in different populations.

In their recent "Terminology Consensus Project," the Sedentary Behaviour Research Network highlight caveats to the definition of screen time, emphasizing the importance of describing whether screen-based behaviors are undertaken while sitting, stationary, or being physically active, as well the context (and type) of screen time. Specific definitions include:

- ***Sedentary screen time:*** Time spent using a screen-based device (e.g., smartphone, tablet, computer, television) while being sedentary in any context (e.g., school, work, recreational). **∗∗Currently, the most commonly researched definition of screen time.**
- ***Active screen time:*** Time spent using a screen-based device (e.g., smartphone, tablet, computer, television) while not being stationary in any context (e.g., school, work, recreational). Examples of active screen time include playing active video games and running on a treadmill while watching television.
- ***Stationary screen time:*** Time spent using a screen-based device (e.g., smartphone, tablet, computer, television) while being stationary in any context (e.g., school, work, recreational).
- ***Recreational screen time:*** Time spent in screen-based behaviors that are not related to school or work.

In conducting future research centered on the health consequences of screen time, the development of new measures aligned with these nuanced definitions will be essential. Such measures should serve to assess the specific type of screen-based behavior, as well as the behavioral and environmental context in which it occurs.

Cross-References

- ▶ Lifestyle, Sedentary
- ▶ Sedentary Behaviors
- ▶ Sedentary Time

References and Further Readings

Biddle, S. J. H., Garcia Bengoechea, E., Pedisic, Z., Bennie, J., Vergeer, I., & Wiesner, G. (2017). Screen time, other sedentary behaviours, and obesity risk in adults: A review of reviews. *Current Obesity Reports, 6*(2), 134–147. https://doi.org/10.1007/s13679-017-0256-9.

Biddle, S. J. H., Pearson, N., & Salmon, J. (2018). Sedentary behaviors and adiposity in young people: Causality and conceptual model. *Exercise and Sport Sciences Reviews, 46*(1), 18–25. https://doi.org/10.1249/JES.0000000000000135.

Dunstan, D. W., Barr, E. L., Healy, G. N., Salmon, J., Shaw, J. E., Balkau, B., et al. (2010). Television viewing time and mortality: The Australian diabetes, obesity and lifestyle study (AusDiab). *Circulation, 121*(3), 384–391. https://doi.org/10.1161/CIRCULATIONAHA.109.894824.

Ofcom. (2018). *Media nations: UK 2018*. Retrieved from https://www.ofcom.org.uk/__data/assets/pdf_file/0014/116006/media-nations-2018-uk.pdf.

Tremblay, M. S., Aubert, S., Barnes, J. D., Saunders, T. J., Carson, V., Latimer-Cheung, A. E., et al. (2017). Sedentary Behavior Research Network (SBRN) – Terminology Consensus Project process and outcome. *The International Journal of Behavioral Nutrition and Physical Activity, 14*(1), 75. https://doi.org/10.1186/s12966-017-0525-8.

van Ekris, E., Altenburg, T. M., Singh, A. S., Proper, K. I., Heymans, M. W., & Chinapaw, M. J. M. (2017). An evidence-update on the prospective relationship between childhood sedentary behaviour and biomedical health indicators: A systematic review and meta-analysis. *Obesity Reviews, 18*(6), 712–714. https://doi.org/10.1111/obr.12526.

Screen-Based Behaviors

▶ Screen Time

Screening

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Synonyms

Early detection

Definition

Screening refers to the process of surveying a population or sample of a population, in the attempt to identify people at risk for or with a given health condition. Screening is a crucial part of epidemiology, as it informs about the prevalence and risk factors of various health conditions in a population. Furthermore, screening is crucial for preventive medicine, since it enables to identify people who may benefit from primary, secondary, or tertiary interventions. Screening for primary prevention reflects identifying people without a risk factor (e.g., hypertension, depression), to prevent the risk factor and subsequent illnesses. Screening for secondary prevention could be among people with a risk factor, to prevent an illness. And screening for tertiary prevention would be done to prevent relapse or mortality in people already ill (e.g., after a first myocardial infarction). Screening could be in relation to psychosocial factors such as hostility or anxiety, to behavioral risk factors of disease such as smoking or excessive alcohol drinking, and to genetic profiles. For implementing screening tests in clinical use to reliably predict disease risk or prognosis, it is crucial to know the relative risk for a disease in people high and low on a screening risk factor as well as the correct value of "false positives" (Wald and Morris 2011). It is of utmost importance to identify the criteria or cutoffs for screening in clear, precise, and operational manners (e.g., smoking more than ten cigarettes/day, depression score above 10 on the Center for Epidemiological Studies Depression Scale). Screening then enables either to study specific subpopulations at risk for health conditions or for treating them. One important criterion for screening tests is their accuracy. A test is thought to be 95% accurate if in 95% of the times it predicts correctly who has a disease (sensitivity) and if 95% of the time it predicts correctly who does not have a disease (specificity). Screening also enables to increase one's therapeutic and statistical effects, since by excluding people below a certain cutoff, researchers can prevent a "floor effect" of therapeutic effectiveness. Regretfully, such exclusion is often not practiced in psychological intervention trials. In clinical practice, the cutoffs used to screen are a function of previously defined cutoffs from research or clinical studies, a function of how severe a risk the researchers aim to identify, and the available therapeutic resources that can be allocated for treating the "screened in" subpopulation later. Furthermore, in randomized controlled trials (RCT), the more strict screening criteria are, the longer could be the trial's duration as the sought patient profile becomes more scarce. Thus, the screening criteria are a function of the research question and available resources for such screening and subsequent treatment, and this is a vital part of clinical epidemiology and research and of therapeutic preventative interventions.

Cross-References

- Cancer Prevention
- Epidemiology
- Population-Based Study
- Population Health

References and Further Readings

Wald, N. J., & Morris, J. K. (2011). Assessing risk factors as potential screening tests. A simple assessment tool. *Archives of Internal Medicine, 171*, 286–291.

Screening, Cognitive

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Synonyms

Cognitive impairment tests; Cognitive status tests; Dementia screening tests; Mental status examination

Definition

Cognitive screening is a brief, performance-based assessment of one or more domains of neurobehavioral or cognitive functioning. These assessments typically are completed using standardized cognitive screening tests that can be completed at bedside or in the clinic in 20–30 min or less, often accompanied by interview information elicited from family members or other informants who know the examinee well and can comment on their observations about the examinee's behaviors or changes in their behaviors.

Description

Cognitive screening tests are very commonly used in behavioral medicine, neuropsychology, neuropsychiatry, and primary care medicine. Surveys indicate that cognitive screening instruments are used by over 50% of practitioners in neuropsychiatry and such tests have become a mainstay in the practice of medicine over the course of the last 35 years. Because cognitive screening tests are brief and require a minimum of specialized testing equipment, they can in most cases be administered at bedside, in a busy clinic, or in the emergency department and serve to identify those patients who might benefit from more extensive workups, including neuroimaging, metabolic assays and blood work, or more extensive neuropsychological testing. Cognitive screening tests are used as one central component in the initial differential diagnosis of delirium versus dementia and are perhaps most frequently used in the initial screening for dementias and mild cognitive impairment (MCI), both of which are underdiagnosed in their earliest stages in primary care practice due to the subtlety of their initial presenting symptoms.

Changes in cognitive functioning are frequently seen as a consequence of a number of neurological and general medical diseases, including dementias and degenerative diseases of the cerebral cortex and subcortical regions of the brain. In addition to central nervous system diseases, cognition may also be affected by other systemic diseases, including respiratory, cardiovascular, and renal diseases as well as some infectious diseases, diseases of the liver and pancreas, nutritional deficiencies, metabolic diseases and diabetes, adverse effects of medications, and exposure to toxic substances. Judicious use of cognitive screening instruments can provide evidence to suggest an underlying medical disorder heretofore undiagnosed and may help guide the use of medications and medication dosages, as well as provide information that may prompt the treatment of reversible conditions, such as reversible dementias and pseudodementias.

Cognitive screening tests can help detect deficits associated with disorders that are commonly missed in a standard psychiatric intake interview, especially in emergency room settings, including patients who present with mild disorientation or evidence of possible substance abuse. In addition, many primary psychiatric disorders have significant effects on cognition, such as affective disorders and schizophrenia, and some focal neurological disorders such as focal strokes, neoplasias, and seizure disorders may have combined cognitive and affective sequelae.

Among the most commonly used and well-researched brief cognitive screening tests are the

Mini-Mental State Examination (MMSE), the Cognitive Capacities Screening Examination (CCSE), and the Short Portable Mental Status Questionnaire (SPMSQ), but there are numerous cognitive screening tests available to practitioners at the present time, and these are listed in Table 1. Although there is considerable variability in the component sections of the cognitive screening tests listed in Table 1, in general each contains some assessment of orientation (does the patient know who they are, where they are, and know the day and date), attention and concentration, language skills, memory and immediate recall of verbal information, and visuospatial or drawing/copying skills. Most cognitive screening tests are designed to be completed within 10 min or less. The BIMC, the ACE-R, the CASI, the Cognistat, the RBANS, the HSCS, and the CAMCOG-R contain more extensive subtests and may require up to 30 min to complete. The Mattis Dementia Rating Scale requires 20–45 min to complete and provides assessment of attention, initiation perseveration, visuospatial construction, reasoning, and memory.

Since 1988, there have been several cognitive screening tests designed to be administered by phone or telehealth link, often used in epidemiological studies as more extensive follow-up instruments after an initial administration face-to-face of a brief screening instrument such as the MMSE or the SASSI. Six such instruments are listed in Table 1.

Also listed in Table 1 are five-guided interview or informant-based cognitive screening instruments which are designed to document information from family members or caregivers of patients regarding observed cognitive decline, changes in behavior, or – in the case of the Deterioration Cognitive Observee (DECO) instrument – changes in activity level, long-term memory, short-term memory, visuospatial processing, and new skill learning. Although these can be used as stand-alone measures, they are perhaps best used to complement the findings from cognitive screening tests directly administered to the patient in question.

There is now considerable interest in the development of cognitive screening tests for specific at-risk populations, and recent examples include a

Screening, Cognitive, Table 1 Cognitive screening tests

Brief cognitive screening tests
AB Cognitive Screen (ABCS)
Abbreviated Mental Test Score (AMTS)
Addenbrooke's Cognitive Examination III (ACE-III)
Animal Fluency Test
Blessed Information-Memory-Concentration Test (BIMC)
Blessed Orientation-Memory-Concentration Test (OMC)
Brief Alzheimer Screen (BAS)
Brief Cognitive Assessment Tool (BCAT)
Brief Cognitive Rating Scale (BCRS)
Brief Interview for Mental Status (BIMS)
Brief Memory and Executive Test (BMET)
Bowles-Langley Technology/Ashford Memory Test
Cambridge Cognitive Examination-Revised (CAMCOG-R)
Clock Drawing Test (CDT)
Cognitive Abilities Screening Instrument (CASI)
Cognitive Assessment Screening Test (CAST)
Cognistat (also known as the Neurobehavioral Cognitive Status Examination or NCSE)
Cognitive Capacity Screening Examination (CCSE)
Cognitive Disorders Examination (Codex)
Cognitive Failures Questionnaire (CFQ)
Cognitive Performance Scale (CPS)
Cognitive Screening Battery for Dementia in the Elderly
Community Screening Interview for Dementia (CSI'D')
Computer-Administered Neuropsychological Screen for Mild Cognitive Impairment (CANS-MCI)
Continuous Recognition Test
Dementia Questionnaire (DQ)
DemTect
Double Memory Test
Eurotest
Fototest
Free and Cued Selective Reminding Test/Five Words Test
Fuld Object Memory Evaluation
Galveston Orientation and Amnesia Test (GOAT)
General Practitioner Assessment of Cognition (GPCOG)
Geriatric Evaluation of Mental Status (GEMS)
Hasegawa Dementia Scale-Revised (HDS-R)
High Sensitivity Cognitive Screen (HSCS)
Hopkins Verbal Learning Test (HVLT)
Imon Cognitive Impairment Screening Test (ICIS)
Isaacs' Set Test of Verbal Fluency
Kingston Standardized Cognitive Assessment
Kokmen Short Test of Mental Status (STMS)
Mattis Dementia Rating Scale (DRS)

(continued)

Screening, Cognitive, Table 1 (continued)

Memory and Executive Screening (MES)
Memory Impairment Screen (MIS)
Memory Orientation Screening Test (MOST)
Mental Alteration Test (MAT)
Mental Status Questionnaire (MSQ)
Middlesex Elderly Assessment of Mental State (MEAMS)
Mini-Addenbrooke's Cognitive Examination (M-ACE)
Mini-Cog
Mini-Mental Status Examination (MMSE)
Mini-Severe Impairment Battery (Mini-SIB)
Modified Mini-Mental Status Examination (3MS)
Modified WORLD Test (WORLD)
Montpellier Screen (Mont)
Montreal Cognitive Assessment (MoCA)
Neurobehavioral Cognitive Status Examination (NCSE)
Philadelphia Brief Assessment of Cognition
Poppelreuter Overlapping Figure
Queen Square Screening Test for Cognitive Deficits
Quick Mild Cognitive Impairment Screen (Qmci)
Quick Test for Cognitive Speed (AQT)
Rapid Dementia Screening Test (RDST)
Repeatable Battery for the Assessment of Neuropsychological Status (RBANS)
Revised Mattis Dementia Rating Scale (DRS-2)
Rowland Universal Dementia Assessment Scale (RUDAS)
Saint Louis University Mental Status Examination (SLUMS)
Severe Impairment Battery (SIB)
Seven-Minute Screen (7MS)
Severe MMSE
Short and Sweet Screening Instrument (SASSI)
Short Blessed Test (SBT)
Short Cognitive Battery (B2C)
Short Cognitive Evaluation Battery (SCEB)
Short Memory Questionnaire (SMQ)
Short Portable Mental Status Questionnaire (SPMSQ)
Short Test of Mental Status (STMS)
Six-item Cognitive Impairment Test (6CIT)
Six-Item Screener (SIS)
Sweet 16
Takeda Three Colors Combination Test
TE4D-Cog
Test for the Early Detection of Dementia from Depression (TE4D-Cog)
Test Your Memory Test (TYM)
Three Word Recall (3WR)
Time and Change Test (T&C)
Trail Making Test (TMT)

(continued)

Screening, Cognitive, Table 1 (continued)

Tree Drawing Test (TDT; Koch's Baum Test)
Verbal Fluency Categories (VFC)
Verbal Fluency Animals (VFA)
Visual Association Test
Cognitive screening tests for specialized patient populations
High Sensitivity Cognitive Screen
HIV Dementia Scale
Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT)
Mini-Mental Parkinson (MMP)
Informant- or proxy-rated screening instruments
The Alzheimer Disease 8 (AD8)
Blessed Dementia Rating Scale (BDRS)
Deterioration Cognitive Observee (DECO)
Informant Questionnaire for Cognitive Decline in the Elderly (IQCODE)
Quick Dementia Rating Scale (QDRS)
Telephone and mail screening instruments
Dementia Questionnaire
Five-Minute Telephone Version of the Short Blessed Test (SBT)
Minnesota Cognitive Acuity Screen
Structured Telephone Interview for Dementia Assessment (STIDA)
Telephone Interview for Cognitive Status (TICS)
Telephone MMSE (TMMSE)

cognitive screening test designed to assess changes in cognition in Parkinson patients (MMP), two cognitive screening tests designed to detect the early signs of AIDS-related dementia in AIDS patients (the High Sensitivity Cognitive Screen test and the HIV Dementia Scale), and a recently developed test to screen for post-concussion cognitive changes, the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT).

With the aging of the population have come an increased interest in cognitive screening in geriatric populations and the increased need to identify early signs of dementia and early signs of mild cognitive impairment, especially as new treatments are developed that are capable of modifying the progression of dementias. In primary care medicine, the standard of practice in the very near future may well include cognitive screening of all patients over the age of 75 in addition to screening of all younger patients when there is a reason to suspect cognitive impairment.

Cross-References

▶ Neuropsychology

References and Further Reading

Cullen, B., O'Neill, B., Evans, J. J., Coen, R. F., & Lawlor, B. A. (2007). A review of screening tests for cognitive impairment. *Journal of Neurology, Neurosurgery, and Psychiatry, 78*, 790–799.

Demakis, G. J., Mercury, M. G., & Sweet, J. J. (2000). Screening for cognitive impairments in primary care settings. In M. E. Maruish (Ed.), *Handbook of psychological assessment in primary care settings* (pp. 555–582). London: Lawrence Erlbaum.

Larner, A. (Ed.). (2017). *Cognitive screening instruments: A practical approach*. New York: Springer.

Lonie, J. A., Tierney, K. M., & Ebmeier, K. P. (2009). Screening for mild cognitive impairment: A systematic review. *International Journal of Geriatric Psychiatry, 24*, 902–915.

Malloy, P. F., Cummings, J. L., Coffey, C. E., Duffy, J., Fink, M., Lauterbach, E. C., et al. (1997). Cognitive screening instruments in neuropsychiatry: A report of the Committee on Research of the American Neuropsychiatric Association. *Journal of Neuropsychiatry and Clinical Neurosciences, 9*, 189–197.

Mitchell, A. J., & Malladi, S. (2010). Screening and case finding tools for the detection of dementia. Part I: Evidence-based meta-analysis of multidomain tests. *American Journal of Geriatric Psychiatry, 18*, 759–782.

Mitrushina, M. (2009). Cognitive screening methods. In I. Grant & K. M. Adams (Eds.), *Neuropsychological assessment of neuropsychiatric and neuromedical disorders* (pp. 101–126). New York: Oxford University Press.

Tombaugh, T. N., & McIntyre, N. J. (1992). The mini-mental state examination: A comprehensive review. *Journal of the American Geriatrics Society, 40*, 922–935.

Seasonal Affective Disorder

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Synonyms

Bipolar disorder, with seasonal pattern; Major depressive disorder, with seasonal pattern

Definition

The most common presentation of seasonal affective disorder (SAD) is recurrent depressive episodes in winter followed by spring remission (Rosenthal et al. 1984). SAD is diagnosed according to the American Psychiatric Association not as a separate disorder, but rather as a course specifier to describe the pattern of depressive episodes in patients meeting criteria for major depressive disorder, or bipolar I or II disorder (American Psychiatric Association [DSM-IV-TR] 2000). Criteria for the seasonal specifier include (1) recurrence of major depressive episodes at a specific time of year; (2) full remission (or change to mania/hypomania) from depression also recurring at a specific time of year; (3) at least two major depressive episodes meeting criteria 1 and 2 within the last 2 years, with no occurrence of nonseasonal depression within the same period; and (4) experiencing a greater number of major depressive episodes meeting SAD criteria than that of nonseasonal depression throughout the individual's lifetime (American Psychiatric Association [DSM-IV-TR] 2000).

Description

Epidemiology. SAD is characterized by depressed mood, anhedonia, and fatigue, as well as higher rates of appetite increase, weight gain, and hypersomnia compared to nonseasonal major depression (Magnusson and Partonen 2005). Ten to twenty percent of patients with depression seeking outpatient treatment have a seasonal pattern of recurrences, and SAD accounts for 10–22% of all unipolar and bipolar mood disorders (Roecklein et al. 2010). A notable characteristic of sleep in SAD is that, in contrast with the predominance of insomnia in nonseasonal depression, a majority of individuals with SAD experience hypersomnia (68–80%; e.g., Rosenthal et al. 1985). Given that seasonality, the tendency to vary in mood and behavior across the seasons (Kasper et al. 1989; Rosen et al. 1990), is normally distributed, a range of mild to severe seasonal changes are likely to occur in behavioral medicine research and practice.

Etiology. Etiological models propose that seasonal changes in the environment, being light

levels or other conditioned cues, trigger onset in fall or winter (Rohan et al. 2009; Sohn and Lam 2005). Lewy et al. (1987) proposed that winter changes in day length lead to a delay in internal circadian rhythms relative to clock time or other rhythms like sleep and wake. Wehr et al. (2001) proposed that winter changes in day length are encoded by nocturnal melatonin release duration as a "circadian signal of change of season," leading to behavioral and physiological changes in humans similar to those of seasonally breeding mammals. Transforming environmental light levels to neural signals is mediated by a retinal pathway to the central clock, and this pathway could be differentially sensitive across individuals, leading some to be vulnerable to insufficient input in winter (Hebert et al. 2002). These circadian and retinal hypotheses may interact with one another, as well as with the monoamine hypothesis (i.e., serotonin and dopamine) and cognitive behavioral mechanisms (Rohan et al. 2009).

Treatment. The recommended first-line treatment for SAD is light therapy, while antidepressants are also commonly used (Lam and Levitt 1999). Light therapy typically requires daily exposure to 10,000-lux of white or full-spectrum fluorescent light for at least 30 min, although efforts to refine the wavelength and reduce duration are being tested clinically. Among antidepressants, Bupropion XL is the first FDA-approved drug for the treatment of winter depression. A double-blind, placebo-controlled, multisite trial testing Bupropion XL on adults with a history of SAD demonstrated that the overall proportion of depression recurrences following treatment was lower for those taking Bupropion (16%) than for those using a placebo (28%; Modell et al. 2005), although the low rate of recurrence indicates a significant placebo response. In addition, cognitive behavioral therapy is as effective as light therapy for acute treatment during a depressive episode, and has prophylactic effects 1 year later in reducing the risk of a subsequent episode (Rohan et al. 2004). Given that multiple empirically validated treatments are available, detecting seasonal patterns in clinical settings can improve patient outcomes.

Implications for behavioral medicine. Seasonal variations in mood and behavior are relevant to Behavioral Medicine research and clinical practice. Such implications can be divided into specific biopsychosocial components including biological characteristics (e.g., genetic risk for seasonality, neurotransmitter and neurohormonal seasonal fluctuations), behaviors (e.g., seasonal changes in physical activity, sleep, substance use, and eating behavior), and social factors (e.g., seasonal changes in social activity rhythms). Candidate behavioral mechanisms in SAD include behavioral disengagement (i.e., lack of response-contingent positive reinforcement) as well as emotional and psychophysiological reactivity to light and seasonal visual stimuli. Several biological mechanisms in SAD have also been proposed (Rohan et al. 2009). The circadian phase shift hypothesis suggests that in the fall and winter months, the timing of different circadian rhythms (e.g., melatonin release, sleep-wake cycle) is out of phase, or desynchronized from other rhythms and/or environmental factors (e.g., dusk/dawn cycle). Another hypothesis is that individuals with SAD have retinas that are less sensitive to light; low environmental light levels in the winter then lead to subthreshold levels of light information transmitted to the brain. The photoperiodic hypothesis proposes that some individuals with SAD maintain biological mechanisms to track changes in photoperiod, a circadian signal of change between seasons, evidenced by individuals with SAD who demonstrate a longer duration of nocturnal melatonin release in the winter months. Rohan et al. (2009) proposed that behavioral and cognitive mechanisms contribute to a psychological vulnerability that, when integrated with biological vulnerabilities, may explain the onset, maintenance, or remission of SAD. Although these separate mechanisms have been shown to play a role in SAD, it is not yet clear if these factors are mechanistic in the cause or maintenance of the disease.

Cross-References

- ▶ Circadian Rhythm
- ▶ Depression: Symptoms
- ▶ Psychiatric Diagnosis
- ▶ Unipolar Depression

References and Further Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th). Washington, DC: Author.

Hebert, M., Dumont, M., & Lachapelle, P. (2002). Electrophysiological evidence suggesting a seasonal modulation of retinal sensitivity in subsyndromal winter depression. *Journal of Affective Disorders, 68*(2–3), 191–202.

Kasper, S., Wehr, T. A., Bartko, J. J., Gaist, P. A., & Rosenthal, N. E. (1989). Epidemiological findings of seasonal changes in mood and behavior. A telephone survey of Montgomery County, Maryland. *Archives of General Psychiatry, 46*(9), 823–833.

Lam, R. W., & Levitt, A. J. (Eds.). (1999). *Clinical guidelines for the treatment of seasonal affective disorder*. Vancouver: Clinical & Academic.

Lewy, A. J., Sack, R. L., Miller, L. S., & Hoban, T. M. (1987). Antidepressant and circadian phase-shifting effects of light. *Science, 235*(4786), 352–354.

Magnusson, A., & Partonen, T. (2005). The diagnosis, symptomatology, and epidemiology of seasonal affective disorder. *CNS Spectrums, 10*(8), 625–634.

Modell, J. G., Rosenthal, N. E., Harriett, A. E., Krishen, A., Asgharian, A., Foster, V. J., et al. (2005). Seasonal affective disorder and its prevention by anticipatory treatment with bupropion XL. *Biological Psychiatry, 58*(8), 658–667.

Roecklein, K. A., Rohan, K. J., & Postolache, T. T. (2010). SAD: Is seasonal affective disorder a bipolar variant? *Current Psychiatry, 9*(2), 42–54.

Rohan, K. J., Roecklein, K. A., & Haaga, D. A. F. (2009). Biological and psychological mechanisms of seasonal affective disorder: A review and integration. *Current Psychiatry Reviews, 5*(1), 37–47.

Rohan, K. J., Tierney Lindsey, K., Roecklein, K. A., & Lacy, T. J. (2004). Cognitive-behavioral therapy, light therapy, and their combination in treating seasonal affective disorder. *Journal of Affective Disorders, 80*, 273–283.

Rosen, L. N., Targum, S., Terman, M., Bryant, M., Hoffman, H., Kasper, S., et al. (1990). Prevalence of seasonal affective disorder at four latitudes. *Psychiatry Research, 31*(2), 131–144.

Rosenthal, N. E., Sack, D. A., Gillin, J. C., Lewy, A. J., Goodwin, F. K., & Davenport, Y. (1984). Seasonal affective disorder. A description of the syndrome and preliminary findings with light therapy. *Archives of General Psychiatry, 41*(1), 72–80.

Rosenthal, N. E., Sack, D., James, S., Parry, B., Mendelson, W., Tamarkin, L., et al. (1985). Seasonal affective disorder and phototherapy. *Annals of the New York Academy of Sciences, 453*, 260–269.

Sohn, C. H., & Lam, R. W. (2005). Update on the biology of seasonal affective disorder. *CNS Spectrums, 10*(8), 635–646.

Wehr, T. A., Duncan, W. C., Jr., Sher, L., Aeschbach, D., Schwartz, P. J., Turner, E. H., et al. (2001). A circadian signal of change of season in patients with seasonal affective disorder. *Archives of General Psychiatry, 58*(12), 1108–1114.

Secondary Care

▶ Clinical Settings

Secondary Gain

▶ Symptom Magnification Syndrome

Secondary Parkinsonism

▶ Parkinson's Disease: Psychosocial Aspects

Secondary Prevention Programs

▶ Cardiac Rehabilitation

Secondhand Smoke

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Synonyms

Environmental tobacco smoke; Involuntary exposure to tobacco smoke; Passive smoking

Definition

The exposure to, and effects of, inhalation of cigarette smoke by an individual other than the active smoker. The term is also applied, more specifically, to smoke exhaled by an active smoker that remains in the environment.

Description

Secondhand smoke includes sidestream smoke from the end of a lit cigarette and exhaled smoke (United States Department of Health and Human Services 2006, 2010; World Health Organization International Agency for Research on Cancer 2004). Harmful components identified specifically in cigarette smoke measured in the air include gases (e.g., carbon monoxide), droplets, and respirable particles which result from the release, combustion, and partial combustion of the tobacco leaves and cigarette paper, as well as flavorants, additives, and other chemicals introduced at agricultural, manufacturing, or packaging stages (California Environmental Protection Agency 2005a). Secondhand smoke, has been shown to contain elevated levels of a large number of known and probable human carcinogens as well as many other toxins with proven causal links to human health conditions (California Environmental Protection Agency 2005b; Institute of Medicine 2010; United States Department of Health and Human Services 2010; United States Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment & U.S. EPA 1992; World Health Organization International Agency for Research on Cancer 2004). It has been estimated that, in 2004, one third of all children and nonsmoking adults worldwide were exposed to secondhand smoke, and that this avoidable exposure was responsible for over 600,000 premature deaths or 1% of all deaths (Oberg et al. 2010). As with active smoking, there is no confirmed risk-free level of exposure to secondhand cigarette smoke (United States Department of Health and Human Services 2006, 2010).

Concentrations of secondhand smoke (and resulting levels of toxic exposure) vary widely and are influenced by: the number of cigarettes and rate of active smoking; the time elapsed since cigarettes were lit and extinguished; the volume of the affected air space; ventilation, air exchange rates, and direction of air flow; and the duration of exposure (California Environmental Protection Agency 2005a). Air concentrations in occupational and private settings, where smoking is permitted, often exceed occupational safety standards for specific agents, and biomarker levels in heavily exposed nonsmokers can overlap levels observed in active smokers (California Environmental Protection Agency 2005b; United States Department of Health and Human Services 2006). Exposure levels in outdoor settings can vary from negligible to concentrations similar to indoor levels if the passive smoker is exposed to the stream of smoke (Institute of Medicine 2010).

Measuring exposure for research, surveillance, and program evaluation may be achieved through air sampling or use of markers of human exposure in biological samples. Air monitoring assesses for concentrations for one or more specific component of tobacco smoke or respirable particulates of specific sizes (Institute of Medicine 2010; United States Department of Health and Human Services 2006). The most widely used biomarkers include exhaled carbon monoxide, and cotinine (a metabolite of nicotine) in saliva, urine, or blood samples. Other biomarkers used in research include, metabolites other than cotinine and concentrations of known carcinogens, as well as through use of other biological media (e.g., testing for accumulated cotinine and nicotine in hair samples from exposed individuals, even newborns as an indication of late prenatal exposure).

Over 40 years of evidence exists on the adverse health effects caused by passive smoke exposure. This evidence has been summarized in prominent reports by international health agencies including International Agency for Research on Cancer (IARC) (International Agency for Research on Cancer 2004, 2009), the Office of the United States Surgeon General (United States

Department of Health and Human Services 2006, 2010), and others (California Environmental Protection Agency 2005b; Institute of Medicine 2010). The major classes of health effects linked to passive smoking include cancers, respiratory disorders, cardiovascular diseases, reproductive effects, and adverse effects on pre-and postnatal growth and development.

Carcinogenic effects of passive smoking can be expected to be consistent with those for active smoking (International Agency for Research on Cancer 2004), and for both, the increased risk is dose-dependent. Secondhand smoke has a confirmed causal role in the development of lung cancer in exposed nonsmokers (California Environmental Protection Agency 2005b; International Agency for Research on Cancer 2004; United States Department of Health and Human Services 2006) and is suggested to increase the risks of nasal and nasopharyngeal cancers in adults (California Environmental Protection Agency 2005b; United States Department of Health and Human Services 2006). There is also evidence to suggest secondhand smoke increases the risk of all childhood cancers, studied collectively, as well as specific childhood cancers (California Environmental Protection Agency 2005b; United States Department of Health and Human Services 2006). For several cancers with definitive evidence linking them to active smoking (including various digestive, kidney, and bladder cancers), there is not sufficient quantitative data to show a causal association with secondhand smoke exposure in humans (International Agency for Research on Cancer 2009). The role of secondhand smoke in breast cancer remains more controversial with The California EPA (California Environmental Protection Agency 2005b) being the first health agency to draw the conclusion of a causal association, while other agencies have not concluded that there is a strong link between active or passive cigarette smoke exposure and breast cancer (International Agency for Research on Cancer 2009; United States Department of Health and Human Services 2010).

Conclusive evidence of noncancer respiratory effects of passive smoking include lower respiratory tract illness in children and adults, prevalence of asthma in children, and severity of asthma in children and adults (United States Department of Health and Human Services 2006). Secondhand smoke also causes recurrent otitis media and middle ear effusion in children (United States Department of Health and Human Services). Passive smoke exposure has also been found to cause adverse and lasting effects on lung function and lung development in children associated with prenatal passive smoking and secondhand exposure in childhood (United States Department of Health and Human Services). Secondhand smoke has been identified as a risk factor for sudden infant death syndrome, with sufficient evidence to suggest a causal association, and associated with a small reduction in birth weight when nonsmoking mothers are exposed while pregnant (United States Department of Health and Human Services).

In terms of cardiovascular diseases, secondhand smoke exposure is accepted as a cause of coronary heart disease morbidity and mortality in adult women and men as well as acute cardiac events (Institute of Medicine 2010; United States Department of Health and Human Services 2006). Even brief exposure to environmental cigarette smoke can lead to vascular function changes and arrhythmic effects associated with acute cardiovascular events in susceptible individuals (Institute of Medicine 2010; United States Department of Health and Human Services 2010).

The World Health Organization Framework Convention on Tobacco Control (FCTC) requires that all signatory nations adopt and enforce measures to protect their populations from exposure to tobacco smoke (World Health Organization 2003, 2011). Protecting the population from secondhand smoke is identified as a key, evidence-based, measure to reduce death, disease, and disability caused by tobacco (World Health Organization 2003). FCTC guidelines (and others International Agency for Research on Cancer 2009; United States Department of Health and Human Services 2006, 2010) recommend that this be achieved through making all environments 100% smoke free as opposed to reliance on ventilation or creation of designated smoking spaces, which have

proven ineffective. Legislation and other measures should apply equally to outdoor spaces wherever there is evidence of exposure (United States Department of Health and Human Services 2006, 2010; World Health Organization 2011).

A number of educational, legislative, occupational, and clinical interventions to eliminate exposure to tobacco smoke have been implemented and evaluated. Evidence from several countries has shown that legislated bans in a variety of settings including workplaces, bars, and restaurants have been effective in terms of: achievement of compliance, improved air quality, reduced human biomarker levels of exposure, and a corollary effect of reducing smoking prevalence among individuals exposed to the restrictions (Institute of Medicine 2010; International Agency for Research on Cancer 2009; United States Department of Health and Human Services 2006). There is also growing evidence that event rates for acute cardiac events have been reduced successfully by legislative and other interventions to eliminate smoking in workplaces and other settings and reduce secondhand smoke exposure (Institute of Medicine 2010; United States Department of Health and Human Services 2010).

Cross-References

- ▶ Cancer and Smoking
- ▶ Heart Disease and Smoking
- ▶ Institute of Medicine
- ▶ Smoking and Health
- ▶ Smoking Behavior
- ▶ Smoking Cessation
- ▶ Tobacco Control
- ▶ Tobacco Use
- ▶ World Health Organization (WHO)

References and Readings

California Environmental Protection Agency. (2005a). *Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part A: Exposure assessment*. Sacramento: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment.

California Environmental Protection Agency. (2005b). *Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part B: Health effects*. Sacramento: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment.

Institute of Medicine. (2010). *Secondhand smoke exposure and cardiovascular effects: Making sense of the evidence*. Washington, DC: Committee on Secondhand Smoke Exposure and Acute Coronary Events, Board on Population Health and Public Health Practice, Institute of Medicine of the National Academies.

International Agency for Research on Cancer. (2004). *IARC monographs on the evaluation of carcinogenic risks to humans* (volume 83: Tobacco smoke and involuntary smoking). Lyon: International Agency for Research on Cancer.

International Agency for Research on Cancer. (2009). *IARC handbook of cancer prevention. volume 13: Evaluating the effectiveness of smoke-free policies*. Lyon: International Agency for Research on Cancer, World Health Organization.

Oberg, M., Jaakkola, M. S., Woodward, A., Peruga, A., & Pruss-Ustun, A. (2010). Worldwide burden of disease from exposure to second-hand smoke: A retrospective analysis of data from 192 countries. *Lancet, 377*(9760), 139–146.

United States Department of Health and Human Services. (2006). *The health consequences of involuntary exposure to tobacco smoke: A report of the Surgeon General*. Rockville: United States Department of Health and Human Services, Public Health Service, Office of the Surgeon General.

United States Department of Health and Human Services. (2010). *How tobacco smoke causes disease: The biology and behavioral basis for smoking-attributable diseases. A report of the Surgeon General*. Rockville: United States Department of Health and Human Services, Public Health Service, Office of the Surgeon General.

United States Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment & U.S. EPA. (1992). *Respiratory health effects of passive smoking (also known as exposure to secondhand smoke or environmental tobacco smoke ETS). EPA/600/6-90/006F*. Washington, DC: United States Environmental Protection Agency (US EPA).

World Health Organization. (2003). *WHO framework convention on tobacco control*. Geneva: Author.

World Health Organization. (2011). *WHO framework convention on tobacco control: Guidelines for implementation Article 5.3; Article 8; Articles 9 and 10; Article 11; Article 12; Article 13; Article 14–2011 edition*. Geneva: Author.

World Health Organization International Agency for Research on Cancer. (2004). *IARC monographs on the evaluation of carcinogenic risks to humans. volume 83: Tobacco smoke and involuntary smoking*. Lyon: International Agency for Research on Cancer.

Sedentary Activity

▶ Lifestyle, Sedentary

Sedentary Behavior

▶ Screen Time

Sedentary Behaviors

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Synonyms

Physical inactivity

Definition

Sedentary behaviors are an increasingly common problem worldwide, with important health consequences. These behaviors include long durations of sitting in front of the TV or the computer, playing computer or TV games, and a general lack of peripheral limb movements. These behaviors have risen due to a multitude of reasons including technological advancements, greater dependence on transportation, urbanization and hence smaller distances to work or schools spent walking, the omnipresence of TV and computers, and our dependence on such means for information, work, leisure, and communication. Various measures and scales exist to assess sedentary behaviors, and these depend on the type of behaviors assessed, the time frame the questions refer to (days, weeks, etc.), and the response format (e.g., a Likert scale or hours). This variability in assessment and use of different cutoffs could of course impact on the prevalence of sedentary behaviors identified in various samples. The prevalence of sedentary behaviors was found to be 58% in a nationally representative sample of Americans aged between 20 and 59 years. When looking just at sitting, one in four Americans spends 70% of their waking time sitting. Furthermore, people in developed countries may spend 4 h a day watching TV and 1 h a day in their vehicle. Importantly, the metabolic and health consequences of sedentary behaviors are distinct from the effects of lack of physical exercise (Owen et al. 2010). In a 21-year follow-up study, the number of hours riding in a car, alone or in combination with hours in front of a TV, significantly predicted cardiovascular disease mortality, independent of confounders (Warren et al. 2010). In contrast, taking daily breaks from sedentary behaviors is related to reduced waist circumference and to improved metabolic outcomes, independent of total amount of sedentary behaviors and of physical exercise (Healy et al. 2008). Mental health problems such as anxiety and depression are also associated with more sedentary behaviors, independent of general physical activity level (de Wit et al. 2011). Finally, an important study examined prospectively over 25 years the relationship between sedentary behavior (TV viewing >3 h/day) and long-term cognitive functions using various measures of processing speed and executive functioning. Indeed, sedentary behavior significantly predicted longer processing speed and poorer executing functioning (inhibition), independent of multiple confounders such as education, age, sex, and hypertension (Hoang et al. 2016). Thus, sedentary behaviors are an important topic for research and intervention in behavior medicine.

Cross-References

▶ Cardiovascular Risk Factors
▶ Lifestyle, Sedentary
▶ Obesity: Causes and Consequences
▶ Physical Activity and Health

References and Further Readings

de Wit, L., van Straten, A., Lamers, F., Cuijpers, P., & Penninx, B. (2011). Are sedentary television watching and computer use behaviors associated with anxiety and depressive disorders? *Psychiatry Research, 186*, 239–243.

Healy, G. N., Dunstan, D. W., Salmon, J., Cerin, E., Shaw, J. E., Zimmet, P. Z., et al. (2008). Breaks in sedentary time: Beneficial associations with metabolic risk. *Diabetes Care, 31*, 661–666.

Hoang, T. D., Reis, J., Zhu, N., Jacobs, D. R., Jr., Launer, L. J., Whitmer, R. A., Sidney, S., & Yaffe, K. (2016). Effect of early adult patterns of physical activity and television viewing on midlife cognitive function. *Journal of the American Medical Association – Psychiatry, 73*, 73–79.

Owen, N., Healy, G. N., Matthews, C. E., & Dunstan, D. W. (2010). Too much sitting: The population health science of sedentary behavior. *Exercise and Sport Sciences Reviews, 38*, 105–113.

Warren, T. Y., Barry, V., Hooker, S. P., Sui, X., Church, T. S., & Blair, S. N. (2010). Sedentary behaviors increase risk of cardiovascular disease mortality in men. *Medicine and Science in Sports and Exercise, 42*, 879–885.

Sedentary Time

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Definition

Sedentary Time (vs. Sedentary Behavior)

Sedentary behavior has been defined as "any waking behavior characterized by an energy expenditure $\leq$1.5 metabolic equivalents (METs), while in a sitting, reclining or lying posture" (Tremblay et al. 2017). Common sedentary behaviors include watching television, reading a book, working at a computer, and driving motorized transport.

Sedentary time refers to the sum of all sedentary behaviors that are undertaken throughout the course of a day. For example, time spent traveling to work by car, sitting working at an office desk, and watching television during leisure time all represent different sedentary behaviors but accumulate to contribute toward "total sedentary time." However, within the field of sedentary behavior research, the terms sedentary time and sedentary behavior are often used interchangeably, and incorrectly.

Description

Measurement of Sedentary Time

Methods employed to assess sedentary time include both self-report measures and objective devices. Until recently, self-report measures were more frequently used in large-scale studies or epidemiological research due to their ease of application and relatively low-cost. Examples include diaries and questionnaires, such as the Bouchard Physical Activity Record and the International Physical Activity Questionnaire, respectively. However, the validity of the data collected using these measures is somewhat limited, due to social desirability bias and errors in participant recall. Moreover, most self-report measures largely focus on assessing specific sedentary behaviors (e.g., screen-based behaviors such as television viewing) rather than overall time spent sedentary.

Objective devices, such as accelerometers and posture sensors, are being more readily employed for the measurement of sedentary time due to their superior validity and reliability relative to self-report measures. Accelerometers (e.g., the ActiGraph) assess sedentary time on the basis of accelerations recorded over prespecified time periods (epochs). These accelerations are subsequently converted to accelerometer "counts," which are interpreted against validated "count thresholds" or "cut points," to determine the frequency and duration of sedentary time. Numerous cut points have been developed for this purpose, for example, Troiano et al. (2008) defines adults' sedentary time as $\leq$99 counts per minute (cpm), and Freedson et al. (2005) have proposed cut points of $\leq$149 cpm to classify sedentary time in

children. Such cut points have been derived by establishing the upper limit of accelerometer counts recorded while undertaking activities requiring $\leq$1.5 METs (measured with indirect calorimetry). Consequently, accelerometers enable measurement of sedentary time on the basis of energy expenditure, but they typically do not afford the ability to determine the posture at which the behavior was undertaken. This may lead to misclassification of low energy, non-sitting behaviors (e.g., standing) as contributors toward sedentary time. Similarly, posture sensors (e.g., the activPAL) are not able to quantify the energy cost of sitting/lying behaviors they measure and therefore may misclassify seated active behaviors (e.g., cycling) as sedentary time. Bearing these limitations in mind, devices that have the ability to combine both accelerometry and posture classification should provide the most comprehensive and accurate assessment of sedentary time.

Sedentary Time and Health

The problem of sedentariness is receiving increased attention due to the high prevalence of this behavior among youth, adults, and older adults, coupled with growing evidence for the role of sedentary time in the development of poor health (Biswas et al. 2015; Hoare et al. 2016). For example, epidemiological research indicates that adolescents accumulate 6 h/day of accelerometer-assessed sedentary time, and sedentary time estimates increase with age (Collings et al. 2014). For adults, the National Health and Nutrition Examination Survey (Healy et al. 2011) indicated that sedentary time represents around 50–60% of waking hours when measured with accelerometry. Still, older adults represent the most sedentary age group, with sedentary time estimates of almost 10 h per day (Harvey et al. 2015).

The adverse health consequences of high sedentary time include increased risk of developing cardiovascular disease, type 2 diabetes, metabolic syndrome, and compromised mental health (Biswas et al. 2015; Hoare et al. 2016). Furthermore, a review of recent epidemiological studies indicated a probable causal positive association between sedentary time and all-cause mortality (Biddle et al. 2016). Importantly, the deleterious health consequences of sedentary time are observed to be independent of participation in moderate-vigorous physical activity (i.e., activity $\geq$3 METs).

Still, despite growing evidence for negative health consequences of sedentary time, studies are yet to determine exactly "how much" sedentary time is bad for us. As a result, current guidelines can only recommend reducing overall sedentary time. Research examining the dose-response association between sedentary time and poor health is therefore necessary to refine sedentary time guidelines.

Cross-References

- ▶ Cardiovascular Risk Factors
- ▶ Lifestyle, Sedentary
- ▶ Sedentary Behaviors
- ▶ Type 2 Diabetes

References and Further Reading

Biddle, S. J., Bennie, J. A., Bauman, A. E., Chau, J. Y., Dunstan, D., Owen, N., et al. (2016). Too much sitting and all-cause mortality: Is there a causal link? *BMC Public Health, 16*, 635.

Biswas, A., Oh, P. I., Faulkner, G. E., Bajaj, R. R., Silver, M. A., Mitchell, M. S., et al. (2015). Sedentary time and its association with risk for disease incidence, mortality, and hospitalization in adults: A systematic review and meta-analysis. *Annals of Internal Medicine, 162*(2), 123–132.

Collings, P. J., Wijndaele, K., Corder, K., Westgate, K., Ridgway, C. L., Dunn, V., et al. (2014). Levels and patterns of objectively-measured physical activity volume and intensity distribution in UK adolescents: The ROOTS study. *International Journal of Behavioral Nutrition and Physical Activity, 11*(23), 1–12.

Freedson, P. S., Pober, D., & Janz, K. F. (2005). Calibration of accelerometer output for children. *Medicine and Science in Sports and Exercise, 37*(11 Suppl), S523–S530.

Harvey, J. A., Chastin, S. F., & Skelton, D. A. (2015). How sedentary are older people? A systematic review of the amount of sedentary behaviour. *Journal of Aging and Physical Activity, 23*(3), 471–487.

Healy, G. N., Matthews, C. E., Dunstan, D. W., Winkler, E. A., & Owen, N. (2011). Sedentary time and cardio-metabolic biomarkers in US adults: NHANES 2003-06. *European Heart Journal, 32*(5), 590–597.

Hoare, E., Milton, K., Foster, C., & Allender, S. (2016). The associations between sedentary behaviour and mental health among adolescents: A systematic review. *International Journal of Behavioral Nutrition and Physical Activity, 13*(1), 108.

Tremblay, M. S., Aubert, S., Barnes, J. D., Saunders, T. J., Carson, V., Latimer-Cheung, A. E., et al. (2017). Sedentary behaviour research network (SBRN) – Terminology consensus project process and outcome. *International Journal of Behavioral Nutrition and Physical Activity, 14*(75), 1–17.

Troiano, R. P., Berrigan, D., Dodd, K. W., Mâsse, L. C., Tilert, T., & McDowell, M. (2008). Physical activity in the United States measured by accelerometer. *Medicine and Science in Sports and Exercise, 40*(1), 181–188.

Seek Feedback

► Self-Monitoring

Selection Bias

► Bias

Selective Serotonin Reuptake Inhibitors (SSRIs)

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Synonyms

Celexa®; Citalopram; Escitalopram; Fluoxetine; Fluvoxamine; Lexapro®; Luvox®; Paroxetine; Paxil®; Prozac®; Sertraline; Zoloft®

Definition

The SSRIs are a family of medications that act primarily (but not exclusively) by inhibiting serotonin reuptake pumps (Finley 2009; Sussman 2009). These medications are used to treat a variety of psychiatric disorders including depression, generalized anxiety disorder, obsessive-compulsive disorder (OCD), panic disorder, premenstrual dysphoric disorder, bulimia nervosa, social phobia, and posttraumatic stress disorder (Sussman). Clinicians should consult the product labeling for each individual medication to determine the indications that each drug is currently approved to treat, since not all of the SSRIs are indicated in the treatment of all of these disorders (although in some cases there is data in the literature suggesting efficacy for a given agent for the treatment of conditions for which it does not have an indication). In the treatment of most types of depression, the SSRIs have similar efficacy to older classes of medications such as the tricyclic antidepressants. However better tolerability at therapeutic doses and lower toxicity in overdose has led to an increased use of SSRIs and decreased use of these older agents over the past several decades (American Psychiatric Association [APA] 2010; Baldessarini 2006; Finley 2009). As with all antidepressants, full therapeutic effects are not observed for as long as 8 weeks in the treatment of depression or longer in the treatment of other disorders (such as OCD), however some symptoms may start to improve sooner (APA 2010; Finley 2009; Kirkwood et al. 2008). In the treatment of depression, all of the SSRIs are thought to be approximately equally effective and therefore initial choice of therapy is often based on factors such as patient preference, prior response to the medication, cost, side effect profile of the individual agents, and the probability that a drug interaction will occur between the antidepressant being chosen and the other medications that the patient is on (APA 2010; Finley 2009). Although the mechanism of action of the SSRIs is similar, lack of efficacy following treatment with one of the drugs in this class does not necessarily predict lack of efficacy by another medication in this class (APA 2010).

There are currently six medications classified as SSRIs that are approved for marketing in the United States (i.e., fluoxetine, paroxetine, sertraline, citalopram, escitalopram, and fluvoxamine) (APA 2010). The SSRIs are similar in many respects; however, important differences between agents are present. Some of these differences are in the side effect profiles of the various drugs and in the likelihood of each drug contributing to a drug-drug interaction with other medications that a patient is taking.

All of the SSRIs have been commonly associated with side effects such as gastrointestinal complaints (e.g., nausea and diarrhea), disturbances in sleep and headache (APA 2010; Finley 2009). Although an individual patient could have any of the sides effects listed, fluoxetine is generally considered the most likely to cause insomnia while paroxetine is often considered to be the most sedating. In many patients, these side effects decrease after the first week of therapy. Additionally, all of the SSRIs have been associated with sexual dysfunction (in both men and women) with the most common symptom reported being delayed orgasm, although decreased interest in sex or erectile dysfunction can also occur (APA 2010; Finley 2009). Since many patients may not spontaneously report sexual side effects, clinicians should inform patients that these may occur and determine if these have been problematic. Serotonin syndrome which includes neurobehavioral (e.g., lethargy and mental status changes), autonomic (e.g., sweating, blood pressure, and heart rate changes), and neuromuscular (e.g., rigidity and tremor) signs and symptoms has been rarely reported with the use of an SSRI as mono-therapy (Chyka 2008). However, the risk of serotonin syndrome increases when SSRIs are used in combination with other serotonergic agents, particularly with monoamine oxidase inhibitors (MAOIs) (APA 2010; Chyka 2008). SSRIs have also been associated with increased bleeding risk, likely due to the presence of serotonin transporters on blood platelets (Sussman 2009). Other side effects such as increased sweating, osteoporosis, bruxism, akathisia, and hyponatremia have been reported occasionally as have a wide range of other side effects (APA 2010; Finley 2009). As with all antidepressants, the SSRIs carry a warning regarding increased suicidality, particularly in children, adolescents, and young adults (under age 24) during the initial stages of therapy (Sussman 2009).

Substantial differences between the SSRIs are present in the pharmacokinetic properties of the agents and in the likelihood that the drug can contribute to drug-drug interactions. For example, fluoxetine is notable for its long half-life (i.e., 4–6 days) and that of its active metabolite norfluoxetine (4–16 days) (Teter et al. 2008). A longer half-life means that any side effects will persist for a longer period of time after discontinuation of the medication. Discontinuation of SSRIs has been associated with withdrawal symptoms characterized by headache, anxiety, flu-like symptoms, and paresthesias (APA 2010). Therefore, it is advisable to taper the medication when possible. The likelihood of a withdrawal syndrome is less likely in SSRIs with longer half-lives (such as fluoxetine) since the decline in the concentration of medications occurs more gradually.

Many of the SSRIs have been found to interact with other medications, some of which may be commonly co-administered with the SSRIs. The cytochrome P450 (CYP450) superfamily of enzymes is responsible for the metabolism of a large number of medications. There are numerous specific isoenzymes within the CYP450 superfamily of enzymes and the degree to which each is affected by an individual SSRI varies considerably. For example, paroxetine and fluoxetine are both strong inhibitors of CYP2D6 (with fluoxetine being a moderate inhibitor of several other CYP450 isoenzymes) (APA 2010; Finley 2009). Since antidepressants are frequently co-administered with other medications, care should be taken to identify and manage any potential drug-drug interactions. It is important to consider the impact of drug interactions both when initiating an enzyme inhibitor (since concentrations of affected medications can increase) and when discontinuing an enzyme inhibitor (since concentrations of affected medication can decrease).

Cross-References

- ▶ Anxiety Disorder
- ▶ Depression: Symptoms

References and Readings

American Psychiatric Association. (2010). *Practice guideline for the treatment of patients with major depressive disorder* (3rd ed.). Washington, DC: Author. *The American Journal of Psychiatry, 167*(Suppl. 10), 1–124.

Baldessarini, R. J. (2006). Drug therapy of depression and anxiety disorders. In L. S. Goodman, A. Gilman, L. L. Brunton, J. S. Lazo, & K. L. Parker (Eds.), *Goodman & Gilman's the pharmacological basis of therapeutics* (11th ed.). New York: McGraw-Hill, Medical.

Chyka, P. A. (2008). Clinical toxicology. In J. T. DiPiro, R. L. Talbert, G. C. Yee, G. R. Matzke, B. G. Wells, & L. M. Posey (Eds.), *Pharmacotherapy: A pathophysiologic approach* (7th ed.). New York: McGraw-Hill Medical.

Finley, P. R. (2009). Mood disorders: Major depressive disorders. In M. A. Koda-Kimble, L. Y. Young, B. K. Alldredge, R. L. Corelli, B. J. Gugielmo, W. A. Kradjan, & B. R. Williams (Eds.), *Applied therapeutics: The clinical use of drugs* (9th ed.). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Kirkwood, C. K., Makela, E. H., & Wells, B. G. (2008). Anxiety disorders: Posttraumatic stress disorder and obsessive-compulsive disorder. In J. T. DiPiro, R. L. Talbert, G. C. Yee, G. R. Matzke, B. G. Wells, & L. M. Posey (Eds.), *Pharmacotherapy: A pathophysiologic approach* (7th ed.). New York: McGraw-Hill Medical.

Sussman, N. (2009). Selective serotonin reuptake inhibitors. In B. J. Sadock, V. A. Sadock, P. Ruiz, & H. I. Kaplan (Eds.), *Kaplan & Sadock's comprehensive textbook of psychiatry* (9th ed.). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Teter, C. J., Kando, J. C., Wells, B. G., & Hayes, P. E. (2008). Depressive disorders. In J. T. DiPiro, R. L. Talbert, G. C. Yee, G. R. Matzke, B. G. Wells, & L. M. Posey (Eds.), *Pharmacotherapy: A pathophysiologic approach* (7th ed.). New York: McGraw-Hill Medical.

Self, The

- ▶ Self-Identity

Self-Assessment

- ▶ Self-examination

Self-Attitude

- ▶ Self-Concept
- ▶ Self-image

Self-Blame

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Synonyms

Responsibility

Definition

Self-blame is the attribution that the consequences one experiences are a direct result of one's actions or character. In the context of behavioral medicine, this may be either beneficial or harmful depending on if it leads to positive behavior change or increased negative affectivity and lack of behavior change.

Description

Self-blame is indirectly related to perceived control, where individuals who self-blame more often also are more likely to believe they have greater control over their lives. Because enhancements in perceived self-control are adaptive to psychological well-being, one may assume that self-blame may also be adaptive. However, this is not always the case.

Janoff-Bulman (1979) proposed two types of self-blame: (1) an adaptive, control-oriented response where the focus is on the individual's behavior and (2) a maladaptive, esteem-oriented response where the focus is on the individual's character. Self-blame is adaptive when individuals recognize that they had some control over

the situation but failed to act appropriately. Thus, these individuals can modify their behavior for future events. On the other hand, self-blame is maladaptive when individuals blame their character flaws for the outcome; this is referred to as characterological self-blame. These flaws are generally seen as stable, so these individuals make no efforts to change. This can lead to recurrence of the same problems and feelings of helplessness and depression.

Although Janoff-Bulman's (1979) theory of self-blame seems plausible, there is little support for these notions in the literature. Two studies did not support the theory that behavioral self-blame is adaptive in breast cancer patients; rather, behavioral self-blame was positively associated with symptoms of anxiety and depression (Bennett et al. 2005; Glinder and Compas 1999). Moreover, Bennett et al. found that both forms of self-blame were unrelated to perceptions of control, which directly contradicts the theory of self-blame.

However, one study of head and neck cancer patients supported Janoff-Bulman's (1979) theory. Low behavioral self-blame (smoking specific) was related to greater likelihood of continued smoking after the cancer diagnosis (Christensen et al. 1999). The relationship held for those patients with high and low perceived control over cancer recurrence, although those with low perceived control had almost a three times greater probability of continued smoking after a cancer diagnosis than those with higher perceived control over cancer recurrence. Furthermore, in this study, it appeared that specific behavior self-blame was more predictive of behavior than a general behavioral self-blame. The authors suggest this may be because of the patients' knowledge of how specific behaviors related to the likelihood of having cancer and that patients do not attribute their behavior in general as the cause of cancer. This study illustrates the importance of understanding how patients' attributions of self-blame, perceived control, and knowledge of their condition may interact in predicting behavior change following a cancer diagnosis.

In the literature self-blame may be used interchangeably with responsibility. However, there are important differences between self-blame and responsibility that should be recognized. Self-blame differs from responsibility in that self-blame suggests that one intentionally brings about negative consequences, whereas responsibility is related more to perceived control over the event (Voth and Sirois 2009). Indeed, Voth and Sirois demonstrated in their study of patients with inflammatory bowel disease that self-blame is related to poor psychological adjustment, whereas responsibility is related to better psychological adjustment. Self-blame was associated with increased use of avoidant coping strategies, whereas responsibility was associated with decreased use of avoidant coping strategies. Avoidant coping was related to poorer psychological adjustment.

Theories of self-blame in behavioral medicine suggest that self-blame has maladaptive and adaptive qualities. Self-blame can be adaptive when individuals recognize their past actions caused their negative consequences, and they also recognize that their behavior is modifiable. Thus, individuals can make positive behavior changes in these cases, which can improve health. However, self-blame is maladaptive when it is primarily characterological in nature. This may lead individuals to feel helpless and to have poorer psychological adjustment to disease. Thus, it is imperative for researchers to properly define the self-blame construct for their research in order to further understand self-blame's role in behavioral medicine.

References and Further Reading

Bennett, K. K., Compas, B. E., Beckjord, E., & Glinder, J. G. (2005). Self-blame and distress among women with newly diagnosed breast cancer. *Journal of Behavioral Medicine, 28*, 313–323.

Christensen, A. J., Moran, P. J., Ehlers, S. L., Raichle, K., Karnell, L., & Funk, G. (1999). Smoking and drinking behavior in patients with head and neck cancer: Effects of behavioral self-blame and perceived control. *Journal of Behavioral Medicine, 22*, 407–418.

Glinder, J. G., & Compas, B. E. (1999). Self-blame attributions in women with newly diagnosed breast cancer: A prospective study of psychological adjustment. *Health Psychology, 18*, 475–481.
Janoff-Bulman, R. (1979). Characterological versus behavioral self-blame: Inquiries into depression and rape. *Journal of Personality and Social Psychology, 37*, 1798–1809.
Voth, J., & Sirois, F. M. (2009). The role of self-blame and responsibility in adjustment to inflammatory bowel disease. *Rehabilitation Psychology, 54*, 99–108.

Self-Care

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Synonyms

Self-management

Definition

Self-care as described by Orem (1995) is "action of persons who have developed or developing capabilities to use appropriate, reliable and valid measures to regulate their own functioning and development in stable or changing environments" (p. 43). Self-care is both caring "for" oneself and "by" oneself. Self-care promotes well-being and is a perceived condition of personal existence characterized by experiences of contentment, pleasure, and happiness. It is associated with health and with sufficiency of resources. This definition is consistent with Diener's (2009) concept of subjective well-being as an individual's global judgment of values and standards that are significant to life satisfaction.

A paradigm that is emerging in health-care delivery for people with chronic conditions is that they are their own principal caregivers; health-care professionals act as consultants and advisors in supporting them in self-care and self-management of their condition. This paradigm of collaborative care and self-management education involves shared decision making between providers and patients. Self-management education includes providing patients with information, problem-solving skills, and behavioral strategies to enhance their lives.

Diabetes is an excellent example of a health condition that requires self-management skills to maintain optimal control through healthy eating, being active, taking medications, monitoring, problem solving, reducing risks, and healthy coping. http://www.diabeteseducator.org/DiabetesEducation/Patient_Resources/AADE7_PatientHandouts.html Self-management education can occur in group settings where peers can provide emotional support and practical information for problem solving. In addition to knowledge and skills, self-care behaviors are determined by attitudes and beliefs, social and environmental influences, and self-efficacy expectations.

Description

Self-care is the thoughts and actions a person takes to achieve or maintain health and well-being.

Cross-References

▶ Self-Management

References and Further Readings

Bodenheimer, T., Lorig, K., Holman, H., & Grumbach, K. (2002). Patient self-management of chronic disease in primary care. *Journal of the American Medical Association, 288*, 2469–2475.
Diener, E. (2009). *The science of well-being*, (Social indicators book series, Vol. 37). New York: Springer.
Orem, D. E. (1995). *Nursing: Concepts and practice* (6th ed.). St. Louis: Mosby.

Self-Concept

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Synonyms

Self-attitude; Self-identity; Self-image

Definition

Self-concept can be defined as one's beliefs about oneself.

Description

Self-concept is a difficult yet important terms to define as self-concept attempts to explain human behavior (micro). Defining self-concept is difficult as a large number of terms use the term 'self' to define some sort of individualistic behavior (Burns 1980). However, in its simplest form, self-concept can be defined as one's beliefs about oneself. Carl Rogers (1951) suggested that oneself, or the "self," plays a role in the development of personality and behavior.

Self-concept can be seen as what an individual understands him or herself to be, cultivated by the appraisal of oneself (Epstein 1973). Self-concept is an organized system of learned beliefs, perceptions, and feelings that aid in the understanding of oneself. Simply, self-concept is the perception an individual has of one's personal characteristics, formed and shaped by society and attitudes. The understanding of oneself is established by one's character, personality, traits, and appearance. Self-concept is developed from an individual's "I," "me," and/or "mine" experience (Burns 1980).

As individuals age, their self-concept is organized and reorganized by their social and nonsocial experiences (Burns 1980). Characteristics in an individual's social environment structure understanding of who the individual is (Epstein 1973). The self-concept or the "who am I" assessed by the individual can be defined and then redefined as the individual encounters many life experiences. In this sense, self-concept can be designated as a multifaceted phenomenon that is dynamic and can change due to experiences, environments, and social affiliations (Markus and Wurf 1987). Thus, self-concept is not fixed and is based individual on the context or situation. Moreover, Burns (1980) suggests that individuals may have many overlapping self-concepts that have been shaped and developed by various beliefs, experiences, and events. Therefore, self-concept can be seen as a multifaceted and individualized process.

An individual can develop a positive or negative self-concept based entirely on the evaluations of oneself. Bailey (2003) states that self-concept is associated with individualistic qualities that can be assessed rather than measured. "Non-measurable" aspects to one's self can be seen as physical attributes, religious preferences, and/or personality traits (Bailey 2003). Thus, self-concept is a learned trait. Rogers (1951) suggests that the self, through self-concept, must be maintained in order to avoid anxiety and stress. This ability to maintain one's self-concept can be achieved through the maintenance of self-esteem (Rosenberg 1979). It has been suggested that self-concept consists of one's self-image and one's self-esteem (Burns 1980). Self-image can be defined as the perception individuals have of themselves physically, psychologically, philosophically, and politically, developed through the agency of their societal experiences and development (Fisher 1986; Statt 1990). Self-esteem is operationally defined as an individual's orientation toward oneself (Rosenberg 1965); self-worth, motivations, and perceptions encompass the conceptualization of an individual's understanding of who they are (Rosenberg 1965). Thus, the process

in which individuals view themselves constructed by the perceptions they have of their self-worth aids in the development of self-concept.

Self-concept can be measured in children and adults by means of various psychometric scales such as the Tennessee Self Concept Scale (Fitts 1991) and the Piers-Harris Children's Self-Concept Scale (Piers 1984).

Cross-References

- ▶ Self-Blame
- ▶ Self-esteem
- ▶ Self-examination
- ▶ Self-Identity
- ▶ Self-image

References and Readings

Bailey, J. A. (2003). Self-image, self-concept, and self-identity revisited. *Journal of the National Medical Association, 95*, 383–386.

Burns, R. B. (1980). *Psychology for the health professions*. Lancaster: MTP Press.

Epstein, S. (1973). The self-concept revisited – Or a theory of a theory. *The American Psychologist, 28*, 404–416.

Fisher, S. (1986). *Development and structure of the body image* (Vol. 1). Hillsdale: Erlbaum.

Fitts, W. H. (1991). *Tennessee self-concept scale manual*. Los Angeles: Western Psychological Services.

Harriman, P. L. (1947). *The new dictionary of psychology*. New York: The Philosophical Library.

Markus, H., & Nurius, P. (1986). Possible selves. *The American Psychologist, 41*, 954–969.

Markus, H., & Wurf, E. (1987). The dynamic self-concept: A social psychological perspective. *Annual Review of Psychology, 38*, 299–337.

Piers, E. V. (1984). *Revised manual for the Piers-Harris children's self-concept scale*. Los Angeles: Western Psychological Services.

Piers, E. V. (1986). *The Piers-Harris children's self-concept scale, revised manual*. Los Angeles: Western Psychological Services.

Rogers, C. R. (1951). *Client-centered counselling*. Boston: Houghton-Mifflin.

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton: Princeton University Press.

Rosenberg, M. (1979). *Conceiving the self*. New York: Basic Books.

Rosenberg, M. (1986). *Conceiving the self*. Malabar: Krieger.

Rosenberg, M. (1989). *Society and the adolescent self-image*. Middeltown: Wesleyan University Press.

Statt, D. (1990). *The concise dictionary of psychology*. New York: Routledge.

Self-Conception

▶ Self-image

Self-Consciousness

▶ Self-image

Self-Construal

▶ Self-Identity

Self-Control

▶ Behavioral Inhibition
▶ Self-Regulation Model

Self-Control Capacity

▶ Self-Regulatory Capacity

Self-Control Failure

▶ Self-Regulatory Fatigue

Self-Determination Theory

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Synonyms

Cognitive evaluation theory

Definition

Self-determination theory is a theory of human motivation that describes two distinct types of motivation: autonomous (regulated through natural and internal processes such as inherent enjoyment or satisfaction) and controlled (regulated through externally held demands and social expectations). Autonomous motivation can be elicited and sustained through social–environmental factors including high autonomy, competence, and relatedness and may contribute to long-term maintenance of a behavior change.

Description

Self-determination theory (SDT) is a theory of human motivation that describes motivation in two distinct types: autonomous and controlled (Deci and Ryan 2008). Autonomous motivation, which includes intrinsic and well-internalized extrinsic motivation, is regulated through natural and internal processes such as inherent satisfaction and can be thought of as an individual's innate desire to engage in healthy behaviors independent of external influences. Autonomous motivation can be elicited and sustained through social contextual conditions including social support from significant others that facilitates intrinsic motivation (e.g., autonomy, competence, and relatedness) in contrast to conditions that undermine one's innate propensity for it such as authoritarian or highly controlled interpersonal interactions (Ryan and Deci 2000). An intrinsically motivated individual may engage in healthy eating because it aligns with his/her self-concept and is enjoyable. Controlled (extrinsic) motivation is regulated through externally held demands and expectations that are contingent on rewards or punishments. An extrinsically motivated individual may engage in healthy eating for approval from peers or to avoid health consequences rather than inherent enjoyment or self-satisfaction. While both types of motivation represent an individual's intention to act, health behavior outcomes resulting from autonomous versus controlled motivation may be qualitatively different. A growing evidence base shows that autonomous motivation may more likely contribute to the maintenance of health behaviors compared to controlled motivation (Ng et al. 2012).

SDT provides a framework for understanding motivational influences on health behaviors, such as healthy diet, physical activity, safe sex practices, and substance use. The conceptualization of motivation on a continuum allows for distinctions to be made in the type and quality of motivation that may contribute to different outcomes. SDT makes distinctions between autonomous and controlled motivation with autonomous motivation being more inherently enjoyable, long-lasting, and internally regulated, while controlled motivation consists of motivation that is primarily driven by externally held demands, social pressures, and reinforcers (Deci and Ryan 2008). For example, controlled motivation based on social pressures has been shown to be negatively related to fruit and vegetable intake compared to autonomous motivation which positively predicted this health behavior (McSpadden et al. 2016).

SDT originally conceptualized the motivation continuum as ranging from amotivation to extrinsic to intrinsic motivation. Amotivation is conceptualized as having no intention to act and has been associated with a lack of outcome value, as well as a lack of beliefs about the link between behavior and desired outcome, or competence in performing the behavior. On the one end of the continuum, motivation is hypothesized to be

regulated extrinsically and controlled by rewards and punishments or other externally regulated processes. Extrinsic motivation may be broken down into subcategories based on increasing levels of intrinsic regulation: extrinsic, introjected (i.e., somewhat external regulation or internal rewards and/or punishments), and identified (somewhat internal regulation and holds personal importance; Deci and Ryan 1985; Ryan and Deci 2000). At the other end of the continuum, intrinsic motivation is regulated or controlled by an individual's inherent satisfaction, novelty, and drive. Previous research has supported beneficial effects of intrinsic motivation compared to extrinsic motivation in substance abuse treatment outcomes (Zeldman et al. 2004) and in predicting mental health and job outcomes in the workplace (Vansteenkiste et al. 2007). However, some researchers have combined extrinsic and introjected into controlled-type motivation and combined identified and intrinsic into autonomous-type motivation (Deci and Ryan 2008). This has resulted in a shift of the primary motivation differentiation moving toward autonomous and controlled in conceptualizing intrinsic and extrinsic in a more dynamic fashion.

SDT emphasizes the role of social context in understanding health behavior motivation and suggests its influence on behavior is through affecting social contextual conditions that may help to elicit and sustain intrinsic motivation. These conditions are described as psychological needs that are inherent to being human and consist of the needs for competence, autonomy, and relatedness. Social relationships (e.g., social support), environmental characteristics (e.g., built environment, resources), and cultural practices and norms (e.g., gender roles) can influence these psychological needs and in turn facilitate or undermine one's sense of intrinsic motivation for engaging in healthy behaviors (Deci and Ryan 1985, 2008; Ryan and Deci 2000). An intervention to increase physical activity, the Active by Choice Today trial, focused on increasing social support (relatedness), teaching behavioral skills (competence), and encouraging choice (autonomy) to provide conditions that facilitate the development of autonomous motivation to be physically active for a lifetime (Wilson et al. 2008). This trial showed a significant intervention effect on increasing accelerometry estimates of physical activity in youth during the afterschool program as compared to a general health education program (Wilson et al. 2011). Similarly, researchers and clinicians interested in reducing substance use have utilized autonomy-supportive strategies for resisting peer pressure (competence) for substance use while preserving positive peer relationships (relatedness) and increasing their choices for alternative activities (autonomy; Williams et al. 2000).

Application of SDT to interventions with other behavioral change theories, such as Social Cognitive Theory (SCT) or Family Systems Theory (FST), has also been implemented in recent trials. For example, the Families Improving Together for Weight Loss trial combined essential elements from SCT including self-monitoring and goal setting and FST including family communication skills and social support with SDT strategies that promoted parental autonomy-supportive communication to enhance motivation for health behavior change in overweight African American adolescents (Wilson et al. 2015). Integrating evidenced-based theoretical frameworks will allow researchers to design comprehensive interventions that are relevant and effective in improving long-term health behaviors.

There is growing evidence that interventions that address cognitive and social factors related to health behavior change may also have ripple effects, such as impacting additional physical, mental, and social health outcomes that were not the intended targeted outcomes (Wilson 2015). This suggests that these health behavior interventions may have far-reaching benefits and impact population health more broadly. Mata et al. (2009) found that increased motivation for exercise behavior also had positive effects on eating self-regulation in a weight control intervention, suggesting that utilizing SDT in health behavior interventions may support multiple health behavior change. Further, a recent meta-analysis of studies utilizing SDT in health interventions showed positive relationships between supportive health

climates that emphasized autonomous motivation and mental and physical health, suggesting that positive health climate-based interventions may be a cost-effective strategy to improve a variety of health outcomes (Ng et al. 2012). In addition, a recent study found that applying SDT to financial incentives was a cost-effective strategy for increasing adherence to existing treatments (Kullgren et al. 2016). However, it is important to utilize theories of motivation, such as SDT, in behavioral economics studies to better understand which types of incentive structures may yield consistent positive changes across different settings (Haff et al. 2015).

In conclusion, SDT has been shown to be a promising theory for individual health behavior change that emphasizes the importance of a positive social context in fostering motivation over time and across health behaviors. Applications of SDT, especially when coupled with other evidenced-based theoretical frameworks of behavior change, show strong promise for future interventions that could be important for improving population level health outcomes.

Cross-References

- ▶ Health Behaviors
- ▶ Motivational Interviewing

References and Further Reading

Deci, E. L., & Ryan, R. M. (1985). *Intrinsic motivation and self-determination in human behavior*. New York: Plenum Press.

Deci, E. L., & Ryan, R. M. (2008). Facilitating optimal motivation and psychological well-being across life's domains. *Canadian Psychology, 49*(1), 14–23.

Haff, N., Patel, M. S., Lim, R., Zhu, J., Troxel, A. B., Asch, D. A., & Volpp, K. G. (2015). The role of behavioral economic incentive design and demographic characteristics in financial incentive-based approaches to changing health behaviors: A meta-analysis. *American Journal of Health Promotion, 29*(5), 314–323.

Kullgren, J. T., Williams, G. C., Resnicow, K., An, L. C., Rothberg, A., Volpp, K. G., & Heisler, M. (2016). The promise of tailoring incentives for healthy behaviors. *International Journal of Workplace Health Management, 9*(1), 2–16.

Mata, J., Silva, M. N., Vieira, P. N., Carraça, E. V., Andrade, A. M., Coutinho, S. R., . . ., & Teixeira, P. J. (2009). Motivational "spill-over" during weight control: Increased self-determination and exercise intrinsic motivation predict eating self-regulation. *Health Psychology*, *28*(6), 709.

McSpadden, K. E., Patrick, H., Oh, A. Y., Yaroch, A. L., Dwyer, L. A., & Nebeling, L. C. (2016). The association between motivation and fruit and vegetable intake: The moderating role of social support. *Appetite, 96*, 87–94.

Ng, J. Y., Ntoumanis, N., Thøgersen-Ntoumani, C., Deci, E. L., Ryan, R. M., Duda, J. L., & Williams, G. C. (2012). Self-determination theory applied to health contexts a meta-analysis. *Perspectives on Psychological Science, 7*(4), 325–340.

Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist, 55*(1), 68–78.

Vansteenkiste, M., Neyrinck, B., Niemiec, C. P., Soenens, B., Witte, H., & Broeck, A. (2007). On the relations among work value orientations, psychological need satisfaction and job outcomes: A self-determination theory approach. *Journal of Occupational and Organizational Psychology, 80*(2), 251–277.

Wilson, D. K. (2015). Behavior matters: The relevance, impact, and reach of behavioral medicine. *Annals of Behavioral Medicine, 49*(1), 40–48.

Williams, G. C., Cox, E. M., Hedberg, V. A., & Deci, E. L. (2000). Extrinsic life goals and health-risk behaviors in adolescents. *Journal of Applied Social Psychology, 30* (8), 1756-1771.

Wilson, D. K., Kitzman-Ulrich, H., Williams, J. E., Saunders, R., Griffin, S., Pate, R., et al. (2008). An overview of "The Active by Choice Today" (ACT) trial for increasing physical activity. *Contemporary Clinical Trials, 29*(1), 21–31.

Wilson, D. K., Van Horn, M. L., Kitzman-Ulrich, H., Saunders, R., Pate, R., Lawman, H. G. . . ., & Mansard, L. (2011). Results of the "Active by Choice Today" (ACT) randomized trial for increasing physical activity in low-income and minority adolescents. *Health Psychology*, *30*(4), 463–471.

Wilson, D. K., Kitzman-Ulrich, H., Resnicow, K., Van Horn, M. L., George, S. M. S., Siceloff, E. R., . . ., & Coulon, S. (2015). An overview of the Families Improving Together (FIT) for weight loss randomized controlled trial in African American families. *Contemporary Clinical Trials*, *42*, 145–157.

Zeldman, A., Ryan, R. M., & Fiscella, K. (2004). Motivation, autonomy support, and entity beliefs: Their role in methadone maintenance treatment. *Journal of Social and Clinical Psychology, 23*(5), 675–696.

Self-Directed Violence

- ▶ Suicide

Self-Efficacy

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Definition

Self-efficacy: Self-efficacy is the belief in personal ability to successfully perform challenging life tasks. Self-efficacy plays an important role in a person's emotions, cognitions, motivational activities, and behaviors across a variety of activities.

Description

Self-efficacy is rooted within Social Cognitive Theory (Bandura 1986) in which people are characterized as active agents of control within their own lives – dynamically influencing their personal environments by organizing responses to opportunities, reflecting on past performances, and self-regulating behavior. Self-efficacy influences response organization, develops in part from reflection, and contributes to self-regulation, particularly, by fostering approach-oriented behavior and persistence in the face of obstacles. Attribution Theory (Weiner 1992), which includes the properties of locus, stability, controllability, also provides a framework for understanding self-efficacy. Locus reflects the cause of a situation as internal or external to a person. Stability reflects how changeable the situation is perceived to be. Controllability is an indicator of whether the person can willfully change a situation. A negative situation such as reaching an unhealthy weight could be interpreted as internal (*Being this heavy is my fault*), stable (*I've always been too heavy*), and uncontrollable (*It doesn't matter what I eat, I just get heavier*), resulting in poor self-efficacy for weight control in the future. In contrast, reaching an unhealthy weight could be interpreted as external (*Weight gain is common with age*), unstable (*This weight came on, it can come off!*), and controllable (*Now that I realize I'm eating too much and moving too little, I can change that behavior*), producing opportunities for improvements in future self-efficacy.

Social Learning Theory (Bandura 1977b) explains development of self-efficacy via four principle pathways: mastery experiences, modeling, social persuasion, and physiology. Past experiences are most predictive of future experiences. People engage in tasks and actions, interpret the outcome, and develop perceptions of their competence within the task domain. Mastery experiences result from multiple successes and promote self-efficacy. Failures are detrimental to developing self-efficacy, although the negative impact of failures is diminished when failures occur attempting a task that has been successfully completed on multiple occasions. The primacy of mastery experiences in self-efficacy development speaks to the titration between efficacy development and actual performance. Self-efficacy develops in domains in which skills are acquired often through the combination of effortful practice and natural talent. Self-efficacy development can be fostered by modeling, particularly when successful completion of activities is modeled by someone viewed as admirable and possessing desired capabilities of the observer. Conversely, a model who fails to perform a desired activity can weaken an observer's sense of competence for performing the activity. Social persuasions involve effective encouragement (not empty praise) that can be instrumental in fostering self-efficacy when the tasks are achievable or nearly achievable. Negative social persuasion deflates self-efficacy. Social persuasion is an important pathway by which parents, teachers, coaches, employment supervisors, and others can facilitate or damage developing self-efficacy. Physiological responses during task attempts influence self-efficacy development. Emotional states indicative of negative arousal such as stress and fear are indicators that the task is difficult and may indicate anticipated failure. In contrast, excitement, anticipation of fun, happiness, or a sense of work "flow" indicate positively developing efficacy. Individuals experiencing negative adjustment periods – such as depressive states or grief – will have difficulty developing self-efficacy. Self-efficacy

can develop more freely when negative emotional states are resolved.

Self-efficacy influences behavior across many domains, including the choices of activities to become involved in effortful work to complete activities, persistence in the face of setbacks or failures, and resilience following adversity (Schunk and Pajares 2005). Individuals tend to avoid activities for which they anticipate poor performance and approach tasks for which they anticipate success. Thus, individuals with low self-efficacy in a given domain may avoid it all together, thus contributing to narrowing of life skills. High self-efficacy contributes to task engagement. High self-efficacy is also associated with effortful engagement in tasks – particularly when intrinsic motivation to engage in the task is also present. Persistence in the face of setbacks is more likely when individuals have high self-efficacy for the task. With the expectation for eventual failure (a component of low self-efficacy), persistence is unlikely. In addition, when failures or enduring obstacles are encountered, low self-efficacy may contribute to a sense of failure and withdrawal from the situation whereas high self-efficacy may contribute to resilience.

Self-efficacy is an integral component of a number of models designed to explain health behavior – primarily because self-efficacy contributes to the willingness to try to change undesirable health behavior, successful implementation of health behavior change, and persistently maintaining health behavior change over time. The Theory of Reasoned Action was extended to form the Theory of Planned Behavior. The extension incorporated self-efficacy as a key factor in changing health behavior along with personal attitudes toward change and the attitudes of significant others toward the change (Ajzen 1991). Self-efficacy was also incorporated into the Health Belief Model during the 1980s – reflecting understanding of the importance of the construct in promoting health (Rosenstock et al. 1988). The Health Behavior Change Model (Prochaska and Velicer 1997) incorporates self-efficacy as a contributor to stages of change. Self-efficacy can influence a person's thoughts about needing change (in the contemplation stage). A person high in self-efficacy will anticipate success and may progress more quickly to the active preparation stage – involving active planning for change behaviors. In addition, self-efficacy will influence effective active change (action stage) as persons high in self-efficacy may more effectively respond to setbacks with persistence and resilience. This quality of those high in self-efficacy also contributes to effective sustainment of the changed behavior (maintenance stage).

Self-efficacy is generally best understood in specific domains and the concept is well supported across multiple domains including school performance, athletic achievement, occupational arenas, and in health behaviors including health maintenance (such as diet and exercise), recovery from acute events such as surgery, and coping with chronic illness or dangerous diagnoses. There is some evidence for a generalized self-efficacy as individuals expect better competence for activities when they have demonstrated aptitude in other activities in the past (Smith 1989). Self-efficacy contributes a theoretically grounded explanation for the myriad ways in which people shape their own environments by seeking out opportunities for success, persisting in the face of hard work and adversity, learning from past experiences, and responding with resilience to failure.

Cross-References

- ▶ Efficacy
- ▶ Efficacy Cognitions
- ▶ Hopelessness
- ▶ Locus of Control
- ▶ Salutogenesis
- ▶ Self-Concept
- ▶ Self-Image
- ▶ Theory of Reasoned Action

References and Further Readings

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes, 50*, 179–211.

Ajzen, I., & Fishbein, M. (1980). *Understanding attitudes and predicting social behavior*. Englewood Cliffs: Prentice-Hall.

Bandura, A. (1977a). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*, 191–215.
Bandura, A. (1977b). *Social learning theory*. Englewood Cliffs: Prentice-Hall.
Bandura, A. (1986). *Social foundations of thought and actions: A social cognitive theory*. Englewood Cliffs: Prenctice-Hall.
Fishbein, M., & Ajzen, I. (1975). *Belief, attitude, intention, and behavior: An introduction to theory and research*. Reading: Addison-Wesley.
Prochaska, J. O., & Velicer, W. F. (1997). The transtheoretical model of health behavior change. *American Journal of Health Promotion, 12*, 38–48.
Rosenstock, I. M., Strecher, V. J., & Becker, M. H. (1988). Social learning theory and the health belief model. *Health Education Quarterly, 15*, 175–183.
Schunk, D. H., & Pajares, F. (2005). Competence perceptions and academic functioning. In A. J. Elliot & C. S. Dweck (Eds.), *Handbook of competence and motivation* (pp. 84–104). New York: Guilford Press.
Smith, R. E. (1989). Effects of coping skills training on generalized self-efficacy and locus of control. *Journal of Personality and Social Psychology, 56*, 228–233.
Walker, J. (2001). *Control and the psychology of health: Theory, measurement and applications*. Buckingham: Open University Press.
Weiner, B. (1986). *An attributional theory of motivation and emotion*. New York: Springer.
Weiner, B. (1992). *Human motivation: Metaphors, theories, and research*. Newbury Park: Sage.

Self-esteem

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Synonyms

Pride; Self-respect

Definition

Self-esteem can be defined as a positive self-evaluation or a concept broader than confidence. It refers to an individual's cognitive appraisal that is constant over time. A positive self-appraisal indicates higher self-esteem, and a negative self-appraisal indicates lower self-esteem. Self-esteem is not perceived anytime, but it essentially influences one's actions, consciousness, or attitude. One who is perceived to have high self-esteem pursues goals aggressively and actively. Further, they are perceived to be amiable by themselves or by others. In this sense, self-esteem becomes indispensable to mental health or social adaptation.

In the previous study concerning self-esteem, an individual's self-esteem was considered in terms of not only his or her tendency and degrees of appraisal, which could be positive or negative, but also its relationship with the individual's cognitive faculty. James (1890) propounded that "self-esteem is successes divided by desire." This formula suggests that just thinking that one could succeed in the desired field increases self-satisfaction. This formula is similar to the theory on the gap between ideal self and real self (Rogers et al. 1951).

An individual's self-esteem strongly correlates with the affection, unconditional acceptance, and nurturing attitude that the parents display. Self-esteem can be measured in various ways, the most representative being Rosenberg's (1965) questionnaire and Coopersmith's (1967) scales. Because each scale may be different in terms of its dimensionalities or factors, it is necessary to consider the characteristics of the scales used or interpreted on a case-by-case basis.

Cross-References

- ▶ Attitudes
- ▶ Cognitive Appraisal
- ▶ Self-Concept

References and Further Reading

Coopersmith, S. (1967). *The antecedents of self-esteem*. San Francisco: WH Freeman.
James, W. (1890). *The principles of psychology*. New York: Holt.

Rogers, C. R., Dorfman, E., Gordon, T., & Hobbs, N. (1951). *Client-centered therapy: Its current practice, implications, and theory.* Boston: Houghton Mifflin.
Rosenberg, M. (1965). *Society and adolescent self-image*. Princeton: Princeton University Press.

Self-Evaluate

► Self-Monitoring

Self-Evaluation

► Self-examination

Self-examination

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Synonyms

Self-assessment; Self-evaluation; Self-monitoring; Self-rating

Definition

Self-examination can be seen as a process of evaluation or appraisal of one's qualities, traits, and characteristics.

Description

Self-examination can be seen as a process of evaluation or appraisal of one's qualities, traits, and characteristics. The evaluation of one's qualities, traits, and characteristics helps develop one's self-image and aids in the development of one's self-awareness or self-concept. Self-examination may result in a positive or negative self-feeling, which may enhance or decrease individuals' ideas about themselves. The evaluation of oneself can be seen as the attempt to understand one's motivations and behaviors. In addition, the examination of oneself is likely to affect self-esteem, which may affect self-image. Individuals who deem themselves as worthy may have a greater self-esteem, which may maintain one's "self." Rogers (1951) suggests that the self must be maintained in order to avoid anxiety and stress. Further, theorists often define self-examination as the evaluation or rating of oneself. This evaluation or rating is often defined as the degree to which an individual is self-aware and may result in a negative or positive self-actualization. This formed self-awareness helps develop the basis of individual self-regulation or the ability of one to control one's behaviors and actions (Hull 2002). Therefore, an individual defines one's self-concept from the degree to which the individual self-examines their personal traits, motivations, behaviors.

The act of self-examination may result in individuals self-monitoring themselves. Self-monitoring can be seen as the degree to which an individual manages or controls the image presented to others in social circumstances (Rawn et al. 2007). An individual who is a "high self-monitor" may be adept at self-monitoring and motivated to alter individual behavior in order to impact the responses of peers in social situations (Rawn et al. 2007). An individual who is a "low self-monitor" remains constant in individual behavior and will not alter individual behavior in social situations (Rawn et al. 2007). Further, the act of self-examination may result in self-criticism, or the awareness that individual traits and/or characteristics do not compare to the ideal self-image one may have for oneself. This self-criticism may be a result of the self-examination of one's strengths and weaknesses and may increase the evaluation of one's image. One may examine and reexamine individual strengths and

weaknesses in order to measure self-actualization, or the fulfillment of one's potential (Maslow 1943, 1976). Therefore, the greater the self-actualization, the more defined one's self-concept may be, and possibly the greater the self-acceptance an individual may have for oneself. Self-acceptance is defined as the attitude toward one's self and ones individual qualities (English und English 1958).

Research that explores how self-evaluation impacts everyday situations has become common. For example, Judge, Locke, and Durham (1997), in a study exploring workplace job satisfaction, found that individuals with greater self-evaluations were more likely to have a higher self-esteem, self-efficacy, personal control, and emotional stability and were motivated to perform well in the workplace. Judge et al. (1997) conceptually define self-esteem, self-efficacy, locus of control, and "neuroticism-stability" as core self-evaluations. Judge et al. suggest that core self-evaluations are the "fundamental, subconscious conclusions individuals reach about themselves, other people, and the world" (Judge et al. 1998).

Cross-References

- ▶ Self-blame
- ▶ Self-concept
- ▶ Self-esteem
- ▶ Self-identity
- ▶ Self-image

References and Readings

English, H. B., & English, A. C. (1958). *A comprehensive dictionary of psychology and psychoanalytic terms*. New York: Longmans, Green.

Hull, J. G. (2002). Modeling the structure of self-knowledge and the dynamics of self-regulation. In A. Tesser, D. A. Stapel, & J. V. Wood (Eds.), *Self and Motivation: Emerging Psychological Perspectives* (pp. 173–206). Washington, DC: American Psychological Association.

Judge, T. A., Locke, E. A., & Durham, C. C. (1997). The dispositional causes of job satisfaction: A core evaluations approach. *Research in Organizational Behavior, 19*, 151–188.

Judge, T. A., Locke, E. A., Durham, C. C., & Kluger, A. N. (1998). Dispositional effects on job and life satisfaction: The role of core evaluations. *Journal of Applied Psychology, 83*, 17–34.

Maslow, A. H. (1943). A theory of human motivation. *Psychological Review, 50*, 37.

Maslow, A. H. (1976). Self-actualization psychology. In J. Fadiman & R. Frager (Eds.), *Personality and personal growth*. New York: Harper Collins.

Rawn, C. D., Mead, N., Kerkhof, P., & Vohs, K. D. (2007). The influence of self-esteem and ego threat on decision making. In K. D. Vohs, R. F. Baumeister, & G. Loewenstein (Eds.), *Do Emotions Help or Hurt Decision Making? A Hedgefoxian Perspective* (pp. 157–182). New York: Russell Sage Foundation Press.

Rogers, C. (1951a). Perceptual reorganization in client-centered therapy. In R. R. Blake & G. V. Ramsey (Eds.), *Perception: An approach to personality*. New York: Ronald Press.

Rogers, C. (1951b). *Client-centered therapy (pp. 13–71)*. Boston: Houghton Mifflin Company.

Self-Experimentation

▶ Quantified Self

Self-Identity

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Synonyms

Self-concept; Self-construal; Self-perspective; Self-schema; Self-system; Self, The; Sense of self

Definition

Self-identity can be conceptualized as a dynamic, contextually based system (Baumeister 1998). It is a complex structure centered in memory and cognition that helps define who we are, how we relate to others, and our place in the world (Swann and Bosson 2008). It is also considered a key motivating force that influences personality and behavior. Self-identity is thought to drive our interactions with others (Andersen and Chen 2002), goals and future roles (Markus and Wurf 1987), and experience of emotions (Higgins 1989). Self-identity is also believed to regulate and motivate behavior by providing key self-regulation through a feedback system (Carver and Scheier 2002).

Description

A person's self-views are considered fundamental to how he or she interprets events, experiences emotion, and behaves. Individuals have distinct identities in different social roles, and differentiation into multiple role-related selves (e.g., self as a student, self as an athlete) is a process of normal development that begins in adolescence or earlier (Oosterwegel and Oppenheimer 2002). Self-identity differs in content and structure across individuals, and likely varies as a function of culture or gender (Cross and Madson 1997).

Research has examined the importance of the organization of positive and negative attributes within self-identity, namely, compartmentalization (i.e., negative attributes enclosed within a single role) and integration (i.e., negative attributes spread across multiple roles; see Showers and Zeigler-Hill 2007). The structure of self-identity is considered contextual and fluctuates between situations as different aspects of the self-structure are activated, strongly relating to mood and self-esteem. A person with a compartmentalized self-structure would experience more positive moods when a role containing predominantly positive attributes is activated frequently, but experience negative moods when a role containing negative attributes is activated. In contrast, both positive and negative attributes are frequently activated in an integrated self-structure, moderating the adverse emotional consequences of activating negative beliefs.

Self-identity is also conceptualized as dynamic. Andersen and Chen (2002) take an interpersonal developmental approach to understanding self-identity in different situations. In their theory of the relational self, they suggest that self-identity develops through interactions with significant others, who set *exemplars*, or cognitive templates stored in long-term memory. These exemplars are set in motion by environmental cues, so that behavior with different individuals varies based upon the active exemplar. Therefore, *who we are now* may differ when with different people.

In addition to describing *who we are now*, views of self-identity also describe *who we may become*. Higgins (1987) suggests that a driving force in self-identity is comparison of the *actual* self to the *ideal* self and *ought* self. A discrepancy between the selves is thought to cause negative psychological states, which initiate behavior that is designed to reduce the discrepancy. Similarly, Carver and Scheier (1998) describe that all individuals strive toward goals, which organize and motivate behavior. Comparisons between future goals and current behavior create emotions that drive future behavior. Positive emotions are experienced and behavior is reinforced when an action is judged to move us closer to a goal; negative emotions are experienced and behavior may change when an action is inconsistent with attaining a goal. Therefore, self-identity is part of a regulatory system that not only reflects, but also drives, who we are and who we will become.

Markus and Nurius (1986) suggest that current self-identity is strongly influenced by *possible selves*, or who we either want to become, or fear becoming, in the future. In this view, self-identity is fluid, continuously developing as possible selves are achieved, modified, or relinquished. Possible selves are thought to directly influence current behavior by providing movement toward or away from possible selves (i.e., approaching hoped-for selves or avoiding feared selves). Exposure to possible selves has been shown to influence exercise behavior (Ouellette et al. 2005) and school involvement in adolescents (Oyserman

et al. 2002) over time. Possible selves are thus considered to play key roles in self-regulatory processes of motivation and behavior (Hoyle and Sherrill 2006). By comparing current self-identity with future selves, possible selves provide a framework in which to interpret and contextualize *who we are now*.

Self-identity is important for Behavioral Medicine because one's self-conceptions can influence how one responds to chronic illness, and can be altered by the experience of chronic illness. As views of the future are highly impacted by circumstances, major life events are likely to lead to changes in these views and, likewise, to changes in self-identity (Tesser et al. 2002). The diagnosis of a chronic illness is an example of this type of event. Adverse outcomes are likely if an illness contains attributes that reflect negatively onto an individual's self-identity. For example, individuals with lung cancer are more likely than individuals with prostate or breast cancer to associate stigma and self-blame with their illness, leading to negative psychological outcomes (Else-Quest et al. 2009). However, a growing body of literature suggests that positive outcomes such as perceptions of personal growth, improvements in life priorities and important relationships, and positive changes in personality (i.e., increased patience, tolerance, and empathy) can also occur as a result of dealing with adverse circumstances such as a chronic illness (Pakenham 2005; Tedeschi and Calhoun 2004).

Self-identity is also relevant to health promotion and illness prevention behaviors. Self-identity plays key roles in self-regulatory processes of motivation and behavior (Hoyle and Sherrill 2006), with the potential to influence health behavior in both positive and negative ways. For example, contemplating an image of a future possible self as an exerciser or non-exerciser influenced exercise behavior at four-week follow-up (Ouellette et al. 2005). Additionally, individuals' self-identity related to both smoking and quitting smoking independently predicted future attempts to quit. In contrast, sexually active teens who viewed STD's as more stigmatized were significantly less likely to have received STD screening over the last year (Cunningham et al. 2009). Goals that individuals wish to achieve, and views of who they are as they achieve these goals, are thought to be continuously present in the self-concept, providing a feedback loop that regulates these behaviors.

Cross-References

- ▶ Benefit Finding
- ▶ Chronic Disease Management
- ▶ Posttraumatic Growth
- ▶ Self-Concept
- ▶ Self-Image
- ▶ Self-Regulation
- ▶ Stigma

References and Further Readings

Andersen, S. M., & Chen, S. (2002). The relational self: An interpersonal social-cognitive theory. *Psychological Review, 109*, 619–645.

Baumeister, R. R. (1998). The self. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (Vol. 1, 4th ed., pp. 680–740). New York: McGraw-Hill.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior*. New York: Cambridge University Press.

Carver, C. S., & Scheier, M. F. (2002). Coping processes and adjustment to chronic illness. In A. J. Christensen & M. H. Antoni (Eds.), *Chronic physical disorders: Behavioral medicine's perspective* (pp. 47–68). Malden: Blackwell.

Cross, S. E., & Madson, L. (1997). Models of the self: Self-construals and gender. *Psychological Bulletin, 122*, 5–37.

Cunningham, S. D., Kerrigan, D. L., Jennings, J. M., & Ellen, J. M. (2009). Relationships between perceived STD-related stigma, STD-related shame and STD screening among a household sample of adolescents. *Perspectives on Sexual and Reproductive Health, 41*, 225–230.

Else-Quest, N. M., LoConte, N. K., Schiller, J. H., & Hyde, J. S. (2009). Perceived stigma, self-blame, and adjustment among lung, breast, and prostate cancer patients. *Psychology and Health, 24*, 949–964.

Higgins, E. T. (1987). Self-discrepancy: A theory related self and affect. *Psychological Review, 94*, 319–340.

Higgins, E. (1989). Continuities in self-regulatory and self-evaluative processes: A developmental theory relating self and affect. *Journal of Personality, 57*, 407–444.

Hoyle, R. H., & Sherrill, M. R. (2006). Future orientation in the self-system: Possible selves, self-regulation, and behavior. *Journal of Personality, 74*(6), 1674–1696.

Markus, H., & Nurius, P. (1986). Possible selves. *American Psychologist, 41*, 954–969.
Markus, H., & Wurf, E. (1987). The dynamic self-concept: A social psychological perspective. *Annual Review of Psychology, 38*, 299–337.
Oosterwegel, A., & Oppenheimer, L. (2002). Jumping to awareness of conflict between self-representations and its relation to psychological wellbeing. *International Journal of Behavioral Development, 26*, 548–555.
Ouellette, J. A., Hessling, R., Gibbons, F. X., Reis-Bergan, M., & Gerrard, M. (2005). Using images to increase exercise behavior: Prototypes versus possible selves. *Personality and Social Psychology Bulletin, 31*, 610–620.
Oyserman, D., Terry, K., & Bybee, D. (2002). A possible selves intervention to enhance school involvement. *Journal of Adolescence, 25*, 313–326.
Pakenham, K. I. (2005). Benefit finding in multiple sclerosis and associations with positive and negative outcomes. *Health Psychology, 24*, 123–132.
Showers, C. J., & Zeigler-Hill, V. (2007). Compartmentalization and integration: The evaluative organization of contextualized selves. *Journal of Personality, 75*(6), 1181–1204.
Swann, W. B., & Bosson, J. K. (2008). Identity negotiation: A theory of self and social interaction. In R. W. Robins & L. A. Pervin (Eds.), *Handbook of personality psychology: Theory and research* (3rd ed., pp. 448–471). New York: Guillford Press.
Tedeschi, R. G., & Calhoun, L. G. (2004). Posttraumatic growth: Conceptual foundations and empirical evidence. *Psychological Inquiry, 15*, 1–18.
Tesser, A., Crepaz, N., Collins, J. C., Cornell, D., & Beach, S. R. H. (2002). Confluence of self-esteem regulation mechanisms: On integrating the self-zoo. *Personality and Social Psychology Bulletin, 26*, 1476–1489.

Self-image

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Synonyms

Self-attitude; Self-concept; Self-conception; Self-consciousness; Self-identity

Definition

Self-image is how an individual thinks they should be (English and English 1958). An individual's self-image is comprised of many attitudes, opinions, and ideals.

Description

Like self-concept, self-image is conceptually a difficult term to define due to the large number of varied terms using "self" as a phrase to define some sort of behavior (Burns 1980). However, self-image is important as it delineates how a self-aware individual views themselves or their image, which further establishes one's self-concept (Harriman 1947). Self-image is how an individual thinks they should be (English and English 1958). An individual's self-image is comprised of many attitudes, opinions, and ideals. Self-image develops at a young age and is a process which develops throughout the lifespan. Beginning at a young age, self-image can be seen as a physical process (Statt 1990). However, self-image is developed not only from body image and physical aspects but also from concepts shaped by society and attitudes.

Self-image is a measurable assessment of one's characteristics. Measurable aspects belonging to an individual build and maintain an individual's self-image. Measurable aspects can be identified as achievements and appearance (Bailey 2003). Self-image is how an individual perceives him/herself based on measurable traits developed at an early age (Bailey 2003). Like self-concept, an individual's self-image may be a learned trait structured from an individual's attitude toward a group, idea, object, and so forth (Rosenberg 1989). Thus, defined behavior may emerge from self-image; however, this behavior is not seen as fixed as it may deviate with the occurrence of different experiences and roles (Burns 1980). In addition, self-image may be affected by an individual's self-esteem. Self-esteem is defined as an individual's orientation toward oneself

(Rosenberg 1965). Rosenberg (1989) suggested that the more uncertain an individual is in regard to who they perceive he/she is, the more likely the individual will have a lowered self-esteem. A lower degree of self-esteem may render a negative self-image, diverging from the ideal self-image individuals may hold for themselves (Burns 1980). The ideal self can be defined as who an individual aspires to be. The ideal self is one part of self-concept and helps individuals better evaluate who they are (Burns 1980).

How individuals perceive who they are may depend on the individual's social environment and/or culture. Self-image can further be characterized as the perception an individual has of who he/she is physically, psychologically, philosophically, and politically, developed through the agency of individual societal experiences and development (Fisher 1986; Statt 1990). Individuals may develop a self-image that is associated with societal roles and norms (Markus and Kitayama 1991). Individuals in collectivist or individualistic cultures may establish who they are based on the culture in which they were raised. A collectivist culture accentuates individual's social roles and responsibilities within the context of social groups, while an individualistic culture accentuates individual identity and achievements (Nevid 2009). Thus, experiences and culture aid in the development of one's self-image. In defining individual roles, culture imparts a strong effect on the individual self-image.

Self-image has been measured in many populations by means of various psychometric scales such as the Rosenberg Self-Esteem Scale (Rosenberg 1989). Further, theory, such as the Social Identity Theory (Tajfel and Turner 1979) and the Objective Self-Awareness Theory (Duval and Wicklund 1972) have emerged from interpretations of self-image and self-consciousness.

Cross-References

- ▶ Body Image and Appearance-Altering Conditions
- ▶ Self-Concept

References and Readings

Bailey, J. A. (2003). Self-image, self-concept, and self-identity revisited. *Journal of the National Medical Association, 95*, 383–386.

Burns, R. B. (1980). *Psychology for the health professions*. Lancaster: MTP Press.

Duval, T. S., & Wicklund, R. A. (1972). *A theory of objective self-awareness*. New York: Academic Press.

English, H. B., & English, A. C. (1958). *A comprehensive dictionary of psychology and psychoanalytic terms*. New York: Longmans, Green.

Fisher, S. (1986). *Development and structure of the body image* (Vol. 1). Hillsdale: Erlbaum.

Fitts, W. H. (1991). *Tennessee self concept scale, manual*. Los Angeles: Western Psychological Services.

Harriman, P. L. (1947). *The new dictionary of psychology*. New York: The Philosophical Library.

Markus, H., & Kitayama, S. (1991). Culture and the self: Implication for cognition, emotion, and motivation. *Psychology Review, 98*, 224–253.

Markus, H., & Nurius, P. (1986). Possible selves. *The American Psychologist, 41*, 954–969.

Markus, H., & Wurf, E. (1987). The dynamic self-concept: A social psychological perspective. *Annual Review of Psychology, 38*, 299–337.

Nevid, J. S. (1990). *Essentials of psychology: Concepts and applications* (2nd ed.). Boston: Houghton Mifflin Company.

Nevid, J. S. (2009). *Psychology: Concepts and applications* (3rd ed.). Belmont: Cengage.

Rogers, C. (1951a). Perceptual reorganization in client-centered therapy. In R. R. Blake & G. V. Ramsey (Eds.), *Perception: An approach to personality*. New York: Ronald Press.

Rogers, C. (1951b). *Client-centered therapy* (pp. 13–71). Boston: Houghton Mifflin Company.

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton: Princeton University Press.

Rosenberg, M. (1979). *Conceiving the self*. New York: Basic Books.

Rosenberg, M. (1986). *Conceiving the self*. Malabar: Krieger.

Rosenberg, M. (1989). *Society and the adolescent self-image*. Middeltown: Wesleyan University Press.

Statt, D. (1990). *The concise dictionary of psychology*. New York: Routledge.

Tajfel, H., & Turner, J. C. (1979). An integrative theory of intergroup conflict. In W. G. Austin & S. Worchel (Eds.), *The social psychology of intergroup relations* (pp. 33–47). Monterey: Brooks/Cole.

Self-Inflicted Injurious Behavior

- ▶ Suicide

Self-Management

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Synonyms

Self-care

Definition

The process of actively engaging in activities aimed at controlling the negative effects of an illness, particularly a chronic illness, on one's health.

Description

Self-management concerns the acquisition of knowledge, as well as application of skills, necessary to engage in a complex set of health-promoting behaviors in the context of daily living. This process of integrating a number of complex behaviors in an effort to maintain wellness often involves problem-solving, decision-making, resource utilization, and communicating with multiple health-care providers. The ability to adapt health behaviors based on physiological or psychological information is a key element of self-management. The importance of self-management is undergirded by its role in health outcomes: It has been demonstrated that those who successfully engage in self-management activities experience better health-related outcomes for a number of chronic conditions.

Based on early studies of those living with chronic conditions, it has been proposed that the tasks related to self-management generally fall within three primary categories addressing: (1) medical management, which includes activities such as taking medications or adhering to a special diet; (2) role management, which includes actions allowing one to adopt roles that accommodate for ones condition; and (3) emotional management, which includes actions aimed at coping with emotions associated with an illness, such as uncertainty, depression, and fear (Corbin and Strauss 1988). It follows, then, that a wide array of psychosocial factors play a role in one's ability to successfully engage in self-management including, but not limited to, social support, motivation, confidence (self-efficacy), and depression. In addition, the ability to read and understand written information (literacy), understand and manipulate numerical information (numeracy), verbal memory, planning, and motor speed have all been associated with disease self-management behavior.

Although disease-specific self-management education is widely accepted as beneficial, its effect on health outcomes has been difficult to establish, primarily due to variability in the nature of the programs and populations tested. Because of the complexity associated with self-management, education and support aiming to train patients to manage their chronic disease attempt to address the many factors and tasks associated with self-management as well as tailor training to the individual needs of patients. However, some common elements among self-management education programs include general information about an illness (e.g., physiology), establishing and personalizing a treatment or self-care "plan" that addresses the behaviors necessary for improved disease outcomes and strategies to facilitate health-related behavior change and maintenance (Funnell et al. 2009; Lorig and Holman 2003). Many programs are guided by a specific theoretical framework and plan training to target variables believed to be important facilitators and barriers to self-management such as means of promoting patients' perceived self-efficacy (confidence) in engaging in health-promoting activities (Bandura 1997). A recent focus has been on the feasibility and broad dissemination of programs facilitating self-management and behavior change.

Driven by rising prevalence, costs, and poor outcomes associated with chronic illnesses,

health-care settings have recently begun to focus on models of service delivery that best support patients' self-management needs. Although these efforts have been given a number of names, some common elements include easy access to health-care providers, providing disease-specific education, incorporating interdisciplinary teams of health-care providers (e.g., physicians, social workers, physical therapists), proactive reminders to both clinicians and patients about health maintenance needs (e.g., routine blood tests), and strategies for supporting the adoption and maintenance of health-promoting behaviors, such as behavioral goal setting between health-care providers and patients (Bodenheimer et al. 2002a, b; Patient-Centered Primary Care Collaborative 2010; The MacColl Institute for Healthcare Innovation 2010).

Cross-References

- ▶ Behavior Change
- ▶ Chronic Disease Management
- ▶ Disease Management
- ▶ Fatigue
- ▶ Health Behaviors
- ▶ Health Promotion
- ▶ Self-Care
- ▶ Self-efficacy

References and Readings

Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.

Bodenheimer, T., Wagner, E. H., & Grumbach, K. (2002a). Improving primary care for patients with chronic illness. *Journal of the American Medical Association, 288*(14), 1775–1779.

Bodenheimer, T., Wagner, E. H., & Grumbach, K. (2002b). Improving primary care for patients with chronic illness: The chronic care model, Part 2. *Journal of the American Medical Association, 288*(15), 1909–1914.

Corbin, J., & Strauss, A. (1988). *Unending work and care: Managing chronic illness at home*. San Francisco: Jossey-Bass.

Funnell, M. M., Brown, T. L., Childs, B. P., Haas, L. B., Hosey, G. M., & Jensen, B. (2009). National standards for diabetes self-management education. *Diabetes Care, 32*(Suppl. 1), S87–S94.

Lorig, K. R., & Holman, H. (2003). Self-management education: History, definition, outcomes, and mechanisms. *Annals of Behavioral Medicine, 26*(1), 1–7.

Patient-Centered Primary Care Collaborative. (2010). *Patient-centered medical Home*. Retrieved 24 Jan-2010, from http://www.pcpcc.net/patient-centered-medical-home

The MacColl Institute for Healthcare Innovation. (2010). *The chronic care model*. Retrieved 24 Jan- 2010, from http://www.improvingchroniccare.org/index.php?p=The_Chronic_Care_Model%26s=2

Self-Management Education

- ▶ Diabetes Education

Self-medication

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Synonyms

Self-treatment

Definition

Self-medication is the use of medications, treatments, and/or substances by an individual without a medical prescription. Self-medication is the most popular form of self-care, which is defined as the personal preservation of health through prevention and self-treatment of ailments (Ryan et al. 2009). In regards to self-care, substances used to self-medicate include but are not limited to over-the-counter (OTC) medications, nutritional supplements, and other nonprescription medications. The number of OTC medications has increased significantly, allowing more individuals to practice self-medication. These nonprescription medications can be purchased at various

locations such as pharmacies, supermarkets, and retail superstores (Wazaify et al. 2005).

This increase in self-medicating practices entails both advantages and disadvantages. The benefits of self-medication include increased access to treatment, increased patient involvement in their own health care, economical choices, and evidence of cost-effectiveness compared to prescription treatments in some situations. The drawbacks of self-treatment are the threat of misuse and abuse of medications, incorrect self-diagnosis, a delay in appropriate treatment, and an increased risk of drug-drug interactions (Hughes et al. 2001). Due to the many risks listed above, safety is a major concern with self-medication. To ensure safety with self-treatment, patient education about the medication is a necessity (Bradley and Blenkinsopp 1996). Pharmacists are in a pivotal position to help ensure appropriate and safe use of various medications by patients that are obtained without a prescription.

Cross-References

- ▶ Nutritional Supplements
- ▶ Self-care
- ▶ Self-management

References and Readings

Bradley, C., & Blenkinsopp, A. (1996). Over the counter drugs: The future for self medication. *British Medical Journal, 312*, 835–837.

Hughes, C. M., McElnay, J. C., & Fleming, G. F. (2001). Benefits and risks of self medication. *Drug Safety, 24*(14), 1027–1037.

Ryan, A., Wilson, S., Taylor, A., & Greenfield, S. (2009). Factors associated with self-care activities among adults in the United Kingdom: A systematic review. *BMC Public Health, 9*, 96.

Wazaify, M., Shields, E., Hughes, C. M., & McElnay, J. C. (2005). Societal perspectives on over-the-counter (OTC) medicines. *Family Practice, 22*, 170–176.

Self-Monitor

- ▶ Self-Monitoring

Self-Monitoring

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Synonyms

Seek feedback; Self-evaluate; Self-monitor

Definition

Self-monitoring can refer to (1) self-monitoring expressive behavior and self-presentation (the extent to which people observe and control their expressive behavior and self-presentation) and (2) self-monitoring goal progress (periodically noting current state and comparing these perceptions with whichever goals are currently relevant).

Description

Self-Monitoring Expressive Behavior and Self-Presentation

People differ in the extent to which they self-monitor (observe and control) their expressive behavior and self-presentation. High self-monitors think about how they appear to others and take care to portray themselves in a socially appropriate manner. Thus, high self-monitors are likely to monitor their facial expression, content of speech, tone of voice, expressed emotionality, and so on. In contrast, low self-monitors do not monitor these things, either because they lack the ability to do so or because they are not motivated to do so.

A number of studies have examined differences between high and low self-monitors. For example, low self-monitors tend to behave in ways that are more consistent with their attitudes

and are better able to imagine how their own behavior relates to particular traits (e.g., the extent to which they are sociable). In contrast, individuals who score high on self-monitoring scales are particularly sensitive to social cues and are better able to imagine the prototypic type of person that would be described as holding a particular trait (e.g., a sociable person), perhaps because they are keen to be able to tailor their own behavior so as to demonstrate certain traits (e.g., to appear sociable). High self-monitors are even more likely to choose friends who facilitate the construction of their own situationally appropriate appearances and romantic partners with an attractive physical appearance. The idea that people self-monitor their expressive behavior has been hugely influential, and these studies just only hint at the wealth of differences.

The self-monitoring scale was developed to measure these individual differences. There are 25 items including "I guess I put on a show to impress or entertain people"; "in different situations and with different people, I often act like very different persons"; and "even if I am not enjoying myself, I often pretend to be having a good time." The scale was revised by Lennox and Wolfe who proposed a shorter 13-item scale with 6 items measuring sensitivity to the expressive behavior of others (e.g., "I am often able to read people's true emotions correctly through their eyes") and 7 items measuring the ability to modify self-presentation (e.g., "once I know what the situation calls for, it's easy for me to regulate my actions accordingly"). Lennox and Wolfe also proposed a separate 20-item "Concern for Appropriateness" scale measuring cross-situational variability (e.g., "different people tend to have different impressions about the type of person I am") and attention to social comparison information (e.g., "I usually keep up with clothing style changes by watching what others wear"). Despite this revision (and others), there remains a debate over exactly what the self-monitoring scale measures.

Self-Monitoring Goal Progress

A separate, but potentially overlapping, literature has examined whether and how people monitor their current standing in relation to their personal goals, for example, whether and how people assess if they are on track to maintain a healthy weight. While monitoring goal progress can involve feedback from external sources, people can, and do, self-monitor. Self-monitoring in this sense involves periodically noting one's current state and comparing these perceptions with whichever goals are currently relevant. For example, a person with the goal to reach a particular weight for their health might monitor caloric intake and physical activity. Monitoring goal progress, therefore, involves a series of processes from deciding to seek information (e.g., I need to monitor the suitability of the food I consume), becoming aware of and directing attention toward relevant information (e.g., looking at nutritional information), interpreting the information (e.g., this indicates a high-/low-fat/sugar content), and so on. Relevant goals provide both a comparative standard and also a schema for making sense of the information available. The person can monitor behavior (e.g., food chosen) or the outcomes of behavior (e.g., at routine health checkups). Monitoring may also vary on a temporal dimension (e.g., hourly, daily, weekly, or monthly) and can occur with respect to goals represented at different levels of specificity, for example, monitoring progress toward relatively high-level values comprising principles (or "be" goals, e.g., to be healthy), specific behavioral goals (or "do" goals, e.g., to take more exercise), or even the performance of motor programs (e.g., "one last rep").

Monitoring goal progress is central to a number of models of goal striving and self-regulation (e.g., control theory), but only a few studies have examined the effect of manipulating the likelihood that people would or could self-monitor. Polivy et al. (1986) investigated the effect of being able to monitor consumption on unhealthy eating. Participants were asked to taste some chocolates and to "eat as many as necessary to ensure accurate ratings." Unbeknown to participants, the researchers manipulated how easy it was for participants to monitor their consumption; some participants were asked to leave their chocolate wrappers on the table, others to place them in a wastebasket that was already half full of

wrappers. The main finding was that participants asked to leave their wrappers on the table (and so, presumably, found it easy to monitor how many chocolates they had eaten) ate less than those asked to put the wrappers in the wastebasket. Quinn et al. (2010) found that self-monitoring helped people to break bad habits. Across two studies, prompting participants to use vigilant monitoring (thinking "don't do it" and watching carefully for mistakes) proved more effective in helping participants to avoid habitual responses (e.g., staying up too late, eating too much) than prompting stimulus control (removing oneself from the situation or removing the opportunity to perform the behavior). Prompting or facilitating behavioral monitoring has also proven an influential technique for promoting goal attainment. In summary, people who self-monitor their current standing in relation to their goals tend to be better able to achieve their goals and make changes to their behavior than people who do not.

Cross-References

- ▶ Behavior Change
- ▶ Self-examination
- ▶ Self-Regulation

References and Further Reading

Abraham, C., & Michie, S. (2008). A taxonomy of behavior change techniques used in interventions. *Health Psychology, 27*, 379–387.

Ajzen, I., Timko, C., & White, J. B. (1982). Self-monitoring and the attitude behavior relation. *Journal of Personality and Social Psychology, 42*, 426–435.

Ashford, S. J., & Cummings, L. L. (1983). Feedback as an individual resource: Personal strategies of creating information. *Organizational Behavior and Human Performance, 32*, 370–398.

Carver, C. S., & Scheier, M. F. (1982). Control theory: A useful conceptual framework for personality, social, clinical, and health psychology. *Psychological Bulletin, 92*, 111–135.

Carver, C. S., & Scheier, M. F. (1990). Origins and functions of positive and negative affect: A control process view. *Psychological Review, 97*, 19–35.

Gangestad, S. W., & Snyder, M. (2000). Self-monitoring: Appraisal and reappraisal. *Psychological Bulletin, 126*, 530–555.

Kluger, A. N., & DeNisi, A. (1996). The effects of feedback interventions on performance: A historical review, a meta-analysis, and a preliminary feedback intervention theory. *Psychological Bulletin, 119*, 254–284.

Lennox, R. D., & Wolfe, R. N. (1984). Revision of the self-monitoring scale. *Journal of Personality and Social Psychology, 46*, 1349–1364.

Polivy, J., Herman, C. P., Hackett, R., & Kuleshnyk, I. (1986). The effects of self-attention and public attention on eating in restrained and unrestrained subjects. *Journal of Personality and Social Psychology, 50*, 1253–1260.

Quinn, J. M., Pascoe, A., Wood, W., & Neal, D. T. (2010). Can't help yourself? Monitor those bad habits. *Personality and Social Psychology Bulletin, 36*, 499–511.

Snyder, M. (1974). Self-monitoring expressive behavior. *Journal of Personality and Social Psychology, 30*, 526–537.

Snyder, M. (1979). Self-monitoring processes. *Advances in Experimental Social Psychology, 12*, 85–128.

Snyder, M., & Cantor, N. (1980). Thinking about ourselves and others: Self-monitoring and social knowledge. *Journal of Personality and Social Psychology, 39*, 222–234.

Snyder, M., Gangestad, S., & Simpson, J. A. (1983). Choosing friends as activity partners: The role of self-monitoring. *Journal of Personality and Social Psychology, 45*, 1061–1072.

Snyder, M., Berscheid, E., & Glick, P. (1985). Focusing on the exterior and the interior: Two investigations of the initiation of personal relationships. *Journal of Personality and Social Psychology, 48*, 1427–1439.

Self-Monitoring of Blood Glucose

- ▶ Glucose Meters and Strips

Self-Murder

- ▶ Suicide

Self-Perspective

- ▶ Self-Identity

Self-Rating

► Self-examination

Self-Regulation

► Self-Regulation Model

Self-Regulation Model

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Synonyms

Model of self-regulation; Self-control; Self-Regulation

Definition

Self-regulation is a dynamic and systematic process that involves efforts to modify and modulate thoughts, emotions, and actions in order to attain goals.

Description

Self-regulation refers to the dynamic cognitive, affective, and behavioral processes that underlie goal attainment. It is important to note that not all types of situations involving goal attainment or problem solving constitute self-regulation. What makes self-regulation unique is that the target of problem solving is set by or focuses on the individual (i.e., self), its "machinery" (e.g., physical problems such as symptoms), and subjective feelings or affect.

Models of self-regulation are based on the idea of cybernetic control and propose that actions are regulated by a TOTE (Test, Operate, Test, Exit) feedback control loop. Central to the idea of feedback loop is the corrective actions that result from the detection and evaluation of discrepancies between input (internal or external) and a reference value. In the context of health, "feeling good" (i.e., not having any symptoms) can be considered a reference value or goal. Thus, when a person experiences a headache (i.e., discrepancy between current state and feeling good), he or she will engage in corrective actions to rid himself or herself of the headache such as taking a pain reliever or resting. If the headache subsides (i.e., test has determined that the discrepancy was eliminated), then the loop ends (i.e., exit). If not, then a new loop will begin.

These self-regulation principles have been widely applied to the study and explanation of multiple psychological phenomena. Psychological models of self-regulation diverge in terms of their foci of interest and emphases; however, they do share core features. Common features of psychological self-regulation models are: (1) goal setting, (2) developing and enacting strategies to achieve these goals, (3) developing criteria to determine proximity to the goal, and (4) determining, based on goal proximity, whether corrective actions are needed or whether the goal needs to be revised. Self-regulation is an iterative process that may require constant evaluation to ensure that the distance between the person's status and the goal is the desired one. One additional commonality shared by behavioral models is the importance they ascribe to affective experiences as integral to self-regulation. Affect, as a core component of the motivational system, can be the reference value that triggers self-regulatory behaviors, be the product of progress toward a goal or lack thereof (i.e., negative affect resulting from not attaining a goal), and/or influence cognitions and behaviors involved in self-regulatory activities (e.g., symptom perception). Models that integrate affective experiences with self-regulation propose that problem-focused and emotion-focused goals and the behavioral processes used by individuals to attain such goals operate in a parallel yet

interrelated fashion (see commonsense model of self-regulation, stress-behavior model advanced by Lazarus and Folkman, or work on self-regulation of affect conducted by Carver and Scheier).

Hierarchical Structure of Goals

Goals are usually differentiated and hierarchically organized in terms of their level of abstraction (Carver and Scheier 1990a). At the highest level of abstraction, one can find goals related to self-concepts (e.g., ideal self and undesired self), self-assessments (e.g., self-rated health), and general affect (e.g., depressed or happy mood). Specific, concrete actions such as daily activities performed by an individual (e.g., buying low fat food) are at the lower level of the hierarchy. Attaining higher-order goals requires that lower-level goals are accomplished; that is, lower-level goals constitute routes to higher-order ones (e.g., buying and consuming low fat food in order to achieve better health). Abstract goals also provide internal consistency and coherence to lower-level goals and to the actions performed to achieve specific higher-order goals. The relationship between goals of different levels of abstraction is quite dynamic. Thus, while a single abstract goal can be attained by pursuing multiple lower-level, concrete goals, it is also possible to simultaneously attain multiple, distinct higher-order goals by setting and pursuing the same lower-level, concrete goals. For instance, a person can get closer to their ideal, healthy self and farther from their undesired, decrepit self by engaging in similar lower-level self-regulatory activities (e.g., engaging in regular exercises and eating a healthful diet).

Feedback Loops and Behavioral Strategies Involved in Goal Attainment

In self-regulation, goals provide the reference values for feedback loops and motivate and guide actions. Individuals may engage in two types of overall feedback loops depending on whether the reference value (i.e., goal) represents a desired state (i.e., approach) or whether it represents an undesired one (i.e., avoidance, Carver 2006). Behavior directed toward desired goals is regulated by a negative feedback loop. In this case, a reduction of the discrepancy between the current state (i.e., input) and the goal (i.e., reference value) dominates the individual's actions. Behaviors involved in the avoidance of reference value, on the other hand, are controlled by a positive feedback loop. Thus, efforts are deployed to maintain or enlarge a discrepancy between the input and the reference value. Many times avoiding an undesired state may require that individuals set desired goals. For example, infirmity (undesired goal) can be avoided by establishing a regime of regular exercise and a healthy diet (desired goals). One can argue that positive feedback loops are part of larger negative feedback loops that motivate individuals to reduce discrepancy between their current status and the reference value. In the case discussed above, an individual for whom avoiding infirmity is critical will need to develop achievable, concrete goals in order to appraise progress. Thus, the reference value for "avoiding infirmity" can take the form of "being symptom-free," a goal that is regulated by a negative feedback loop.

The two strategies discussed above assume that goals set by an individual are attainable; however, there are situations in which, regardless of effort, goal attainment can become difficult or impossible. In such situations, abandoning activities directed at the pursuit of the goal will result in better adjustment than maintaining goal-directed efforts. Research has shown that goal disengagement can result in improved well-being if goal-directed efforts are focused on alternative goals when available. If alternative goals are unavailable, inability to disengage from goal pursuit can result in frustration, negative affect, and increased stress (Miller and Wrosch 2007). For older adults, goal disengagement and goal reengagement may be a key strategy for adjustment to physical changes and for successful aging.

Goal attainment and affect. As indicated above, affect can be an indicator of progress toward the attainment of a goal. Carver and Scheier (1990b) have proposed a second feedback

process that monitors whether a person's efforts are being successful in attaining a goal. Success in closing the gap between one's status and a given goal results in positive affect. Slow progress or failure to attain a goal, on the other hand, results in negative affect. Individuals differ in terms of the reference values they use to determine what constitutes acceptable or unacceptable progress. Accordingly, similar rates of discrepancy reduction can result in two very different responses if the individuals use different criteria to appraise the effectiveness of their actions.

Research on self-discrepancies provides an interesting example of how proximity to a goal can elicit positive or negative moods (Mora et al. in press). These data have shown that individuals who felt they were farther from being at their worst (i.e., undesired self) reported less anxiety and depression and more happiness than their counterparts who felt closer to their feared self. These affective experiences can, in turn, influence subsequent evaluations of progress and actions. Negative mood arisen from perceptions that progress toward a goal is slow can result in maladaptive behaviors especially if new actions are focused on reducing negative feelings. Depressive feelings may alter perceptions of control over their environment and future outcomes, and lead individuals to give up on their efforts to attain a goal. Despite its critical and multifaceted role in self-regulation, there is a dearth of research examining the multiple interactive ways in which cognitive/behavioral and affective self-regulation operates.

Organization of Goals and Actions: Goal Cognitions

Germane to the relationships among goals of different levels of abstraction are goal cognitions (Karoly 1993) and action plans (Leventhal 1970). Goal cognitions are mental models or schemas that organize domain-specific goals and actions or procedures to attain those goals (in health literature, goal cognitions are referred as illness representations). For instance, a schema for an abstract, higher-order goal of "being healthy" would involve several less abstract concrete goals such as engaging in exercise, eating healthful food, and improving coping skills. These goals, in turn, would be connected in the mental schema to more concrete, lower-level goals such as setting days to go the gym, eating more complex carbohydrates, and learning how to meditate to reduce stress. Action plans translate aspects of goal pursuit (e.g., beliefs, attitudes, evaluation criteria) into actual, concrete behaviors. These behaviors help link goals at different levels of abstraction and provide continuity to the pursuit of abstract goals. In the current example, being healthy requires the person to set and attain multiple concrete goals. Goal cognitions will specify the action plans required to attain the concrete goals in route to reach the higher-order, abstract goal. Thus, to attain the various goals on route to being healthy, the person will need to engage in actions such as joining a gym, building a routine of going to the gym, and choosing the specific workout routine (e.g., cardio and/or weightlifting).

Conscious and Nonconscious Aspects of Self-Regulation

Although the terms "goal setting" and "self-regulation" suggest conscious volition, processes involved in self-regulation are the result of both automatic and intentional behaviors. Awareness of every single process involved in self-regulation would unnecessarily tax the organism and result in maladaptation. Action plans required for the attainment of a goal can become automated behaviors if goal pursuit is an ongoing activity. For instance, the management of a chronic condition such as hypertension requires that people take medications every day. To ensure that doses are not missed, a person may decide to put pills in a case and place this case on top of the counter next to the coffee maker. By doing this, taking the pill can become an automated behavior that imposes a minimal burden on the cognitive system of the individual. It is in consciousness, however, where automatic and intentional processes are integrated. Ensuring that doses have not been missed, the person needs to engage in a conscious decision-making process (e.g., pick up the pill case and count the remaining pills).

Recently, investigators interested in the volitional aspects of self-regulation have been devoting increased attention to the idea of self-control. Although sometimes self-control and self-regulation are used interchangeably, self-control refers to the capacity to consciously and effortfully regulate one's affect, cognitions, and behaviors (Hagger et al. 2010). In attempting to explain the reasons individuals engage in maladaptive behaviors (e.g., smoking), researchers have argued that self-control is a limited resource (Ego Strength model: Baumeister et al. 2007). The Ego Strength model argues that self-control works as "a muscle" that can become tired after repeated use (i.e., ego depletion). In other words, a person's capacity to engage in self-control becomes reduced following previous acts of self-control. Although the idea of ego depletion has received support from experimental studies, a recent meta-analysis suggests that the strength model does not fully explain the mechanisms underlying self-control and additional theoretical perspectives need to be integrated into the strength model (Hagger et al. 2010). The results from the meta-analysis suggest that motivation, fatigue, self-efficacy, and affect are involved in self-control and ego-depletion processes. Further research in this area is needed to better understand how self-control and ego depletion operate in real world contexts in which long-term and unplanned attempts at self-control occur frequently.

Moderators of Self-Regulation

Self-regulation occurs within particular settings and is, thus, influenced by individual, social, and cultural factors. Personality, an individual factor, has been implicated in various processes of self-regulation such as appraisal, coping, and behaviors (Cervone et al. 2006). For instance, research has shown that individuals high in neuroticism (one of the big five personality traits) consistently and reliably perceive and report more physical symptoms than their low neuroticism counterparts (Watson and Pennebaker 1989). Because physical symptoms are a departure from normal functioning, increases in symptomatology may trigger prompt self-regulatory actions (e.g., care seeking) if symptoms are perceived to be indicators of imminent threat. Research on coping has revealed that individual differences such as optimism and pessimism influence goal pursuit. Specifically, individuals who have positive views about the future tend to engage in problem-focused coping, whereas individuals who perceive the future as uncertain tend to either engage in emotion-focused coping or else disengage from goal pursuit (Rasmussen et al. 2006). Personality can also have an impact on the selection of specific behaviors used by individuals to deal with threat. There is evidence that individuals high in optimism and conscientiousness who face stressful situations select coping procedures that improve well-being and adjustment (O'Connor et al. 2009).

Social factors and culture can also influence the various stages of self-regulation from goal setting to selection and performance of corrective actions through multiple pathways. Social comparisons can help individuals to determine the origin of their physical symptoms (e.g., food poisoning if everybody at dinner has the same symptoms or stomach flu if symptoms are unique to one person) and, thereby, select the necessary corrective actions to deal with such symptoms (Leventhal et al. 1997). Research has also demonstrated that social relations (i.e., social network size) can influence long-term self-regulatory activities such as participation in cardiac rehabilitation among patients with acute coronary syndrome (Molloy et al. 2008).

Culture has been shown to influence the type of stimuli that trigger self-regulation, the content of goals, the specific corrective actions, and the appraisal criteria involved in goal attainment. Anthropological research has shown that the interpretation and attribution of bodily symptoms varies across culture. This body of evidence suggests that culturally determined illness models are powerful determinants of the ways people experience illness conditions. For example, work on depression has shown that among certain ethnic groups (e.g., Chinese), depression is usually experienced as a somatic disorder (Kleinman 1977). This difference in experience can shape what

actions are taken to remediate the problem (e.g., seeing a primary care physician instead of a mental health specialist) and the criteria to determine the success of the remedial actions (e.g., improvement in physical versus affective well-being). The impact of culture on the construction and development of the self has led some authors to question the idea that self-regulation is set by and focuses on the individual. In collectivistic cultures where the self is construed as closely interrelated to others, goal setting can be motivated by a need to please the person's social network (Trommsdorff 2009). Future research needs to determine whether the mechanisms of self-regulation guided by interrelatedness are different from those underlying self-regulation of intraindividual processes.

Self-Regulation and Health

Although most models of self-regulation have originated outside the domain of behavioral medicine (see the Commonsense Model of Self-regulation for an exception), the use of self-regulatory principles and ideas to understand health-related behaviors has been relatively widespread. Models such as the Health Beliefs Model, Theory of Reasoned Action, and Social Cognitive Theory which focus on specific aspects of self-regulation such as social (e.g., norms) and psychological (e.g., self-efficacy) determinants have been utilized to understand self-regulation of health behaviors. In general, the use of these models has focused more on the description of predictors of goals rather than on the underlying mechanisms that explain self-regulatory behaviors. In addition, goal pursuit is usually examined by these models as a single-event rather than a continuous process (Maes and Karoly 2005). Recent efforts, however, have been directed at the understanding and examination of self-regulation as a process by investigating how goal-directed behaviors in health domains are initiated and maintained. Evidence has provided convincing support that the various phases of self-regulation are controlled by different processes. Studies examining health behaviors such as smoking cessation and engagement in exercise activities have revealed that factors such as self-efficacy and attitudes toward the specific behavior are important determinants of initiation. Maintenance of health behaviors, on the other hand, is influenced by factors such as satisfaction with behavioral change. A recent study examining data from participants in a smoking cessation program provided further evidence of the complexity of self-regulation processes (Baldwin et al. 2009). In this study, the authors found that maintenance of health-related behaviors was a heterogeneous process and that the factors that influenced satisfaction with behavioral maintenance varied according to whether they had quit smoking recently or not.

Final Remarks

Literature and research on self-regulation has grown rapidly over the past 20 years. New research has expanded the understanding of self-regulation mechanisms but more research is needed. Some of the research questions that remained underexplored include changes in self-regulation over time, factors that contribute to long-term self-regulatory behaviors (e.g., adherence to medical treatment for chronic conditions), and the multiple and simultaneous pathways through which cognitive and affective self-regulation interact.

New opportunities provided by advances in technology, especially in brain imaging, are opening exciting areas of inquiry (e.g., cognitive neuroscience of self-regulation). Similarly, increased understanding of the psychophysiology of stress offers an invaluable opportunity to understand how self-regulatory processes get under the skin.

Cross-References

- ▶ Active Coping
- ▶ Behavioral Intervention
- ▶ Common-Sense Model of Self-regulation
- ▶ Optimism
- ▶ Self-Efficacy
- ▶ Self-Regulatory Capacity
- ▶ Self-Regulatory Fatigue

References and Further Readings

Baldwin, A. S., Rothman, A. J., & Jeffery, R. W. (2009). Satisfaction with weight loss: Examining the longitudinal covariation between people's weight-loss-related outcomes and experiences and their satisfaction. *Annals of Behavioral Medicine, 38*, 213–224.

Baumeister, R. F., & Vohs, K. D. (2004). *Handbook of self-regulation: research, theory, and applications*. New York: Guilford Press.

Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The strength model of self-control. *Current Directions in Psychological Science, 16*(6), 351–355. https://doi.org/10.1111/j.1467-8721.2007.00534.x.

Boekaerts, M., Zeidner, M., & Pintrich, P. R. (1999). *Handbook of self-regulation*. San Diego\London: Academic Press.

Carver, C. (2006). Approach, avoidance, and the self-regulation of affect and action. *Motivation and Emotion, 30*(2), 105–110. https://doi.org/10.1007/s11031-006-9044-7.

Carver, C. S., & Scheier, M. (1990a). Principles of self-regulation: Action and emotion. In E. T. Higgins, R. M. Sorrentino, et al. (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (Vol. 2, pp. 3–52). New York: Guilford Press.

Carver, C. S., & Scheier, M. F. (1990b). Origins and functions of positive and negative affect: A control-process view. *Psychological Review, 97*(1), 19–35.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior*. New York: Cambridge University Press.

Cervone, D., Shadel, W. G., Smith, R. E., & Fiori, M. (2006). Self-regulation: Reminders and suggestions from personality science. *Applied Psychology: An International Review, 55*(3), 333–385. https://doi.org/10.1111/j.1464-0597.2006.00261.x.

Folkman, S., & Lazarus, R. S. (1988). The relationship between coping and emotion: Implications for theory and research. *Social Science & Medicine, 26*(3), 309–317.

Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. D. (2010). Ego depletion and the strength model of self-control: A meta-analysis. *Psychological Bulletin, 136*(4), 495–525. https://doi.org/10.1037/a0019486.

Karoly, P. (1993). Mechanisms of self-regulation: A systems view. *Annual Review of Psychology, 44*, 23–52.

Kleinman, A. M. (1977). Depression, somatization and the "new cross-cultural psychiatry". [Case reports comparative study]. *Social Science and Medicine, 11*(1), 3–10.

Leventhal, H. (1970). Findings and theory in the study of fear communications. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 5, pp. 120–186). New York: Academic.

Leventhal, H. (1983). Behavioral medicine: Psychology in health care. In D. Mechanic (Ed.), *Handbook of health, health care, and the health professions* (pp. 709–743). New York: The Free Press.

Leventhal, H., Hudson, S., & Robitaille, C. (1997). Social comparison and health: A process model. In B. P. Buunk & F. X. Gibbons (Eds.), *Health, coping, and well-being: Perspectives from social comparison theory* (pp. 411–432). Mahwah: Lawrence Erlbaum Associates.

Maes, S., & Karoly, P. (2005). Self-regulation assessment and intervention in physical health and illness: A review. *Applied Psychology, 54*(2), 267–299. https://doi.org/10.1111/j.1464-0597.2005.00210.x.

Miller, G. E., & Wrosch, C. (2007). You've gotta know when to fold'em. *Psychological Science, 18*(9), 773–777. https://doi.org/10.1111/j.1467-9280.2007.01977.x.

Molloy, G. J., Perkins-Porras, L., Strike, P. C., & Steptoe, A. (2008). Social networks and partner stress as predictors of adherence to medication, rehabilitation attendance, and quality of life following acute coronary syndrome. *Health Psychology, 27*(1), 52–58. https://doi.org/10.1037/0278-6133.27.1.52.

Mora, P. A., Musumeci-Szabo, T. J., Popan, J., Beamon, T., & Leventhal, H. (in press). Me at my worst: Exploring the relationship between the undesired self, health, and mood among older adults. *Journal of Applied Social Psychology.*

O'Connor, D., Conner, M., Jones, F., McMillan, B., & Ferguson, E. (2009). Exploring the benefits of conscientiousness: An investigation of the role of daily stressors and health behaviors. *Annals of Behavioral Medicine, 37*(2), 184–196. https://doi.org/10.1007/s12160-009-9087-6.

Rasmussen, H. N., Wrosch, C., Scheier, M. F., & Carver, C. S. (2006). Self-regulation processes and health: The importance of optimism and goal adjustment. *Journal of Personality, 74*(6), 1721–1747. https://doi.org/10.1111/j.1467-6494.2006.00426.x.

Schunk, D. H., & Zimmerman, B. J. (1994). *Self-regulation of learning and performance: issues and educational applications*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Trommsdorff, G. (2009). Culture and development of self-regulation. *Social and Personality Psychology Compass, 3*(5), 687–701. https://doi.org/10.1111/j.1751-9004.2009.00209.x.

Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review, 96*(2), 234–254.

Self-Regulatory Ability

► Self-Regulatory Capacity

Self-Regulatory Capacity

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Synonyms

Self-control capacity; Self-regulatory ability; Strength model of self-control

Definition

Self-regulatory capacity refers to people's ability to exert control over their thoughts, feelings, and actions, for example, the capacity to inhibit prejudice, make oneself feel better, or select healthy food. However, self-regulatory capacity is thought to be a limited resource that (i) is temporarily depleted by exertions of self-control (an effect termed "ego-depletion") and (ii) differs in strength from person to person.

Description

Limited Self-Regulatory Capacity

Self-regulatory capacity refers to an individual's ability to exert control over their behavior, thoughts, and feelings. The capacity for self-regulation differs between individuals and can differ intraindividually dependent on situational factors, such as the experience of self-regulatory fatigue. Self-regulation is closely related to goal-driven behavior and is characterized by the process of attempting to work toward a desired held goal. For example, in terms of health behavior, an individual may regulate their diet to reach a specific weight or resist a personal temptation such as cigarettes to reach their goal of overcoming a smoking addiction. Self-regulatory capacity would influence how successfully an individual pursues such goals, alongside other factors such as the prepotency of the action that needs to be overcome. The modal model of self-regulatory capacity (strength model) poses that persistence with self-regulation can lead to a temporary state of self-regulatory fatigue and lapses of self-regulation can occur. In terms of the prior examples, this could include short-term gratifications such as deviations from a planned diet or the resumption of smoking.

Self-regulatory capacity can be measured using a wide variety of cognitive or behavioral tests. Examples include persistence at impossible (e.g., completing unsolvable anagrams) or aversive (e.g., consuming unpleasant drinks, squeezing a handgrip, cold pressor) tasks; response time or errors made in inhibition tasks (Stroop task, stop signal); resisting temptation (limiting alcohol consumption); and suppression of emotional expression. The key criterion for a task measuring self-regulatory capacity is the requirement for the person to override an otherwise dominant response (e.g., the desire to give up, to read the words in the Stroop task). Tasks that are simply difficult (e.g., complex math puzzles) or stressful (e.g., watching an emotional video) are unlikely to reflect the same ability. The strength model argues that self-regulation draws from a universal pool – that exerted effort at one self-regulation task impairs ability at performing a subsequent, seemingly distinct, task. Presenting these tasks in sequence is a commonly used method to examine self-regulatory capacity.

Self-regulatory capacity has an important relation to health behavior. Many behaviors which are considered to have long-term benefits, such as eating a healthy diet or maintaining an exercise regimen, are notably absent from people's routines, while behaviors that are demonstrably harmful in the long term such as smoking, unhealthy diets, unprotected sexual intercourse, or substance abuse are common in society. The contrast between these behaviors can be seen in the temporal distance between the costs and benefits: behaviors associated with improved health

typically only show a beneficial change over the longer term, while the costs (such as the self-regulatory effort in changing behavior) are high in the short term, whereas behaviors that show a high cost of being harmful to health in the long term offer immediate benefits (such as the satiation of an immediate desire or need). In the context of temporal self-regulation theory, self-regulatory capacity acts as a moderator of the link between an individual's intentions for health behavior, which are shaped by the temporal differences between perceived costs and benefits for an action, and the individual's actual observed behavior. A greater capacity for self-regulation is predicted to be associated with the ability to overcome prepotent responses, such as habitual smoking, and follow intentions for behavior, such as intention to quit smoking. Limited capacity for regulation is predicted to be associated with lapses in self-control and the resumption of prepotent responses; so despite the intention to quit smoking, this would not translate into actionable behavior.

Nature of Self-Regulatory Capacity

The precise nature of self-regulatory capacity remains elusive – since the development of the strength model in 1994, a multitude of studies using the two-task paradigm indicate a limited self-regulatory capacity; a meta-analysis of these suggests a reliable effect. However, more recent research challenges the size and reliability of effects previously observed.

Self-regulatory capacity has been linked to the biological substrate of blood glucose levels – Gailliot and colleagues (2007) identify that depletion of self-regulatory capacity was correlated with blood glucose levels and that performance on self-regulatory tasks could be restored with blood glucose supplements (namely, lemonade). The strength model has since been updated to situate a central monitor of available, or potentially available, glucose as a candidate for momentary self-regulatory capacity.

Self-regulatory capacity's apparent dependence on glucose availability suggests a similarity with executive functions known to be affected by available glucose levels (e.g., the attentional system). Self-regulation is considered to exist alongside this diverse array of higher cognitive processes and may work in concert with others, particularly that of planning and error detection. Self-regulation can be considered as a goal-driven process because it is used by an individual who seeks to override one state of behavior, thoughts, or feelings with another goal state. As such, an individual is required to recruit multiple executive processes to reach goal states: representations of one's current state and goal state must be held alongside a plan for how to achieve the goal state, detection of discrepancy (or error) between these two states must occur, and self-regulation to change the current experienced state toward the goal is required. Apparent failures in the wider system of self-regulation may not be restricted to a depleted self-regulatory capacity; related systems such as that of error detection may impair self-regulation if there is an incorrect perception of error between the current state and desired state.

Like other aspects of executive function, self-regulatory capacity is associated with the frontal lobes. Both neuroimaging and lesion studies indicate that self-regulatory actions such as the control of behavioral and emotional output is closely associated with the ventromedial prefrontal cortex. In addition, the ventromedial-orbitofrontal cortex has been particularly associated with the self-regulatory process of suppressing previously rewarding responses. Damage to this area often results in a diminished or absent ability to inhibit immediately rewarding actions, even though the semantic knowledge of an action's inappropriateness may still be intact. These regions associated with self-regulatory capacity show high connectivity will executive areas related to the processes involved in self-monitoring, which support the process of self-regulation. The anterior cingulate cortex is associated with the degree and nature of conflict between competing responses to situations such as the short-term desire to satiate an immediate need against the longer-term goal for a healthier lifestyle. The dorsolateral prefrontal cortex has been associated with the resolution of detected response conflict alongside attentional

and planning behavior. These regions working in cohort are considered to form the neural circuitry underpinning self-regulation.

Individual Differences in Self-Regulatory Capacity

In addition to situational demands on self-regulatory capacity, there is also a body of research suggesting that there may be stable individual differences in self-regulatory capacity. Some people simply seem better than others at, for example, selecting healthy food, cheering themselves up, and acting in an egalitarian manner. In support of this idea, Hofmann et al. (2008) found that individual differences in working memory capacity moderated participants' ability to self-regulate their sexual interest, consumption of tempting food, and anger expression. In a similar vein, a number of authors have proposed measures of self-regulatory capacity such as the Self-Control Behavior Inventory, the Self-Control Questionnaire, and the Brief Self-Control Scale (BSCS). In each case, differences have been found between people that reliably map onto various self-control outcomes such as task performance, impulse control, interpersonal relations, and so on.

Building Self-Regulatory Capacity

Much like with general executive function training, there is a significant interest in the possibility that self-regulatory capacity can be improved in individuals through practice. Meta-analysis of training studies indicates that training can have a positive effect on some elements of self-regulatory capacity, particularly when measured using the two-task paradigm.

Cross-References

- ▶ Cold Pressor Task
- ▶ Ego-Depletion
- ▶ Emotion
- ▶ Limited Resource
- ▶ Prejudice
- ▶ Self-Control
- ▶ Self-Report Inventory
- ▶ Stroop Color-Word Test
- ▶ Working Memory

References and Further Reading

Baumeister, R. F., & Vohs, K. D. (2018). Strength model of self-regulation as limited resource: Assessment, controversies, update. In *Self-regulation and self-control* (pp. 78–128). New York: Routledge.

Brandon, J. E., Oescher, J., & Loftin, J. M. (1990). The self-control questionnaire: An assessment. *Health Values, 14*, 3–9.

Carter, E. C., & McCullough, M. E. (2014). Publication bias and the limited strength model of self-control: Has the evidence for ego depletion been overestimated? *Frontiers in Psychology, 5*, 823.

Fagen, S. A., Long, N. J., & Stevens, D. J. (1975). *Teaching children self-control: Preventing emotional and learning problems in the elementary school*. Columbus: Charles E. Merrill.

Friese, M., Frankenbach, J., Job, V., & Loschelder, D. D. (2017). Does self-control training improve self-control? A meta-analysis. *Perspectives on Psychological Science, 12*(6), 1077–1099.

Gailliot, M. T., Baumeister, R. F., DeWall, C. N., Maner, J. K., Plant, E. A., Tice, D. M., Brewer, L. E., & Schmeichel, B. J. (2007). Self-control relies on glucose as a limited energy source: Willpower is more than a metaphor. *Journal of Personality and Social Psychology, 92*, 325–336.

Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. D. (2010). Ego depletion and the strength model of self-control: A meta-analysis. *Psychological Bulletin, 136*, 495–525.

Hagger, M. S., Chatzisarantis, N. L., Alberts, H., Anggono, C. O., Batailler, C., Birt, A. R., . . ., Calvillo, D. P. (2016). A multilab preregistered replication of the ego-depletion effect. *Perspectives on Psychological Science*, *11*(4), 546–573.

Hall, P. A., & Fong, G. T. (2013). Temporal self-regulation theory: Integrating biological, psychological, and ecological determinants of health behavior performance. In *Social neuroscience and public health* (pp. 35–53). New York: Springer.

Hofmann, W., Friese, M., Gschwendner, T., Wiers, R. W., & Schmit, M. (2008). Working memory capacity and self-regulatory behavior: Toward an individual differences perspective on behavior determination by automatic versus controlled processes. *Journal of Personality and Social Psychology, 95*, 962–977.

Tangney, J. P., Baumeister, R. F., & Boone, A. L. (2004). High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *Journal of Personality, 72*, 271–324.

Self-Regulatory Fatigue

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Synonyms

Ego-depletion; Limited resource; Self-control failure

Definition

Self-regulatory fatigue refers to the temporary depletion of individuals' capacity for self-control. In a state of self-regulatory fatigue, individuals find it harder to resist making impulsive purchases, inhibit prejudice, or regulate their own emotions (an effect often termed "ego-depletion"). Self-regulatory fatigue arises from the extended use of self-regulation, which is thought to be a limited resource.

Description

Limited Self-Regulatory Capacity

Self-regulatory fatigue describes the temporary impairment in self-control performance after prior exertions of self-control (an effect also referred to as "ego-depletion"). The strength model of self-regulation draws the analogy between muscular fatigue and self-regulatory fatigue. Muscular fatigue occurs after repeated or prolonged acts of physical exertion, where longer or more effortful exertions lead to a greater experience of fatigue. Similarly, persistent acts of self-regulation, such as the overriding of impulses, result in a mental fatigue, limiting the effectiveness of further regulatory efforts. Again, longer or more effortful exertions of self-regulation lead to a greater experience of self-regulatory fatigue. Like muscular fatigue, rest and recuperation are hypothesized to restore the temporary depletion in self-regulatory strength.

It is important to distinguish between subjective and actual self-regulatory fatigue, although the two can coincide. Self-regulatory persistence and ego-depletion is associated with the physiological indications and subjective feelings of fatigue such as somnolence and decreased attention. Subjective fatigue is thought to mediate between exertions of self-regulation and subsequent decrements in self-regulatory ability. However, subjective fatigue – or the belief that one is fatigued – can have effects that are independent of actual fatigue. Despite experiencing prior effortful self-regulation, individuals perceiving themselves to be low in fatigue can engage in subsequent regulation more effectively than those perceiving themselves to be high in fatigue. A second contributing factor to the state of self-regulatory fatigue is motivation. Individuals may cease persistence at self-regulation if they believe that the effort exerted during self-regulation costs more than the outcome of self-regulation is worth. Self-regulatory fatigue is, therefore, not merely a state of exhaustion in which the individual is willing to engage a task and yet unable to persist but can also lead to acquiescence – the person willfully consents to relax self-control because they are not motivated to do otherwise.

Measuring Self-Regulatory Fatigue

The strength model poses that exertions of self-regulation draw from a limited self-regulatory capacity. However, this hypothesis of capacity has proven difficult to objectively measure. A range of cognitive or behavioral tests have been implemented to measure self-regulatory fatigue. Common examples include persistence at impossible (e.g., unsolvable puzzles) or unpleasant (e.g., consuming unpleasant drinks, cold pressor) tasks; response time or errors made in inhibition tasks (modified Stroop task, go/no-go); resisting temptation (limiting alcohol consumption); and regulating facial expression. In such cases, the primary means for measuring self-regulatory fatigue is a comparison of

performance at a self-regulatory task between individuals in a depleted state (those having exerted prior self-regulation) against individuals in a non-depleted state (those having completed a difficult task such as complex math problems that does not require self-regulation). As a result, self-regulatory fatigue and the presence of a limited resource are only measured indirectly; thus the relationship between subjective fatigue, motivation, and self-regulatory fatigue remains unclear.

Attempts at more direct and biological measures of self-regulatory fatigue have been made. Blood glucose levels have been reported to show a correlation with self-regulatory fatigue, suggesting a positive link between available caloric energy to the brain and available mental resource. However, glucose supply itself is not thought to be a causal link to self-regulatory fatigue, as glucose dynamics are too small to account for differences between depleted and non-depleted states. The strength model for self-regulatory fatigue has more recently been revised to incorporate a monitor of potentially available (rather than direct levels of) glucose as a possible driver of self-regulatory fatigue. Alternative physiological measures, such as glycogen store levels and heart rate variability, have also been suggested as means of measuring resource availability and self-regulatory fatigue.

Overcoming Self-Regulatory Fatigue

While the primary means to counteract self-regulatory fatigue are rest and recuperation, the effects of self-regulatory fatigue may be moderated or counteracted by a number of other means. The induction of a positive mood by others, including humor and laughter, observation of others undergoing successful self-regulation, salient social goals, primed ideas of success, a broadened mindset through self-affirmation, external attribution of the causes of depletion, perception of a low state of fatigue, simultaneous tensing or firming of muscles, and monetary incentives all serve to encourage individuals to persist at self-regulation. However, in many cases, this increased persistence is considered to deprioritize conservation of remaining resources rather than a reduction in fatigue. Poor performance in an unannounced third self-regulatory task after the standard two-task design often indicates that depleted participants have persisted beyond their typical point of self-regulatory fatigue rather than overcoming fatigue. Further interventions, such as the prior practice of self-regulation tasks or the formulation of implementation intentions ("if. . . then. . ." plans), serve to encourage persistence at self-regulation through moderating the effort required to expend during successful self-regulation.

Wider Implications of Self-Regulatory Fatigue

Self-regulatory fatigue has been implicated in a number of problems for both the individual and society. While warnings about substance abuse, risky sexual practice, and unhealthy diets are extensively promoted, there equally exists a widespread failure of individuals to adhere to these guidelines or rules. Experimental studies such as those examining alcohol or unhealthy food consumption demonstrate that these behaviors are affected by both self-regulatory fatigue and individuals' chronic tendencies. A chronic tendency that is typically suppressed, such as a high temptation to drink alcohol, becomes more likely to shape behavior when an individual is depleted. In contrast, an individual with low trait temptation to drink alcohol might show no change in alcohol consumption when depleted because it is not necessary to engage in self-regulation. Further examples of behaviors affected by self-regulatory fatigue include impulsive or overspending, emotional regulation such as anger management, interpersonal interaction, self-presentation or impression formation, and stereotype suppression.

Cross-References

- ▶ Ego-Depletion
- ▶ Fatigue
- ▶ Limited Resource
- ▶ Self-Control
- ▶ Self-Report Inventory
- ▶ Working Memory

References and Further Reading

Baddeley, A. (2007). *Chapter 13: Working memory, thought and action*. New York: Oxford University Press.

Baumeister, R. F., & Vohs, K. D. (2018). Strength model of self-regulation as limited resource: Assessment, controversies, update. In *Self-regulation and self-control* (pp. 78–128). New York: Routledge.

Baumeister, R. F., Heatherton, T., & Tice, D. M. (1994). *Losing control: How and why people fail at self-regulation*. London: Academic.

Baumeister, R. F., Gailliot, M., DeWall, N., & Oaten, M. (2006). Self-regulation and personality: How interventions increase regulatory success, and how depletion moderates the effects of traits on behavior. *Journal of Personality, 74*, 1773–1802.

Baumeister, R. F., Tice, D. M., & Vohs, K. D. (2018). The strength model of self-regulation: Conclusions from the second decade of willpower research. *Perspectives on Psychological Science, 13*(2), 141–145.

Beedie, C. J., & Lane, A. M. (2012). The role of glucose in self-control: Another look at the evidence and an alternative conceptualization. *Personality and Social Psychology Review, 16*(2), 143–153.

Gailliot, M. (2008). Unlocking the energy dynamics of executive functioning: Linking executive functioning to brain glycogen. *Perspectives on Psychological Science, 3*, 245–263.

Self-Report

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Synonyms

Self-report inventory

Definition

Self-report includes an individual's reports about what they are feeling, what they are doing, and what they recall happening in the past (Stone et al. 2009).

These are captured by validated self-report questionnaires, of which there are many. Indeed, one of the challenges facing behavioral medicine is the bewildering variety of measurement instruments (Dekker 2009). Although validated, the limitations of self-report questionnaires are that the researcher is dependent on the research participant to be completely truthful and unbiased and to be able to accurately remember details.

References and Further Reading

Dekker, J. (2009). Measurement instruments in behavioral medicine. *International Journal of Behavioural Medicine, 16*, 89–90. https://doi.org/10.1007/s12529-009-9049-1.

Stone, A., Turkkan, J., Bachrach, C., Jobe, J., Kuftzman, H., Cain, V., et al. (2009). *The science of self-report*. Taylor & Francis e-library. Retrieved from http://books.google.co.uk/books

Self-Report Inventory

▶ Self-Report

Self-Reported Patient Outcome Measure

▶ Health Assessment Questionnaire

Self-Respect

▶ Self-esteem

Self-Schema

▶ Self-Identity

Self-System

▶ Self-Identity

Self-Tracking

▶ Quantified Self

Self-Treatment

▶ Self-medication

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Biographical Information

Dr. Martin Seligman

Martin E. P. Seligman was born August 12, 1942, in Albany, NY. He received his A.B.. from Princeton University in 1964 and his Ph.D. in Psychology from the University of Pennsylvania in 1967 (Shah n.d.). After receiving his doctorate, he began his career as an assistant professor at Cornell University in Ithaca, NY. He soon returned to the University of Pennsylvania where he was promoted to associate professor and to professor of Psychology and then to director of the Clinical Training Program. He is currently the Zellerbach Family Professor of Psychology in the Department of Psychology at the University of Pennsylvania and the director of the Positive Psychology Center (University of Pennsylvania 2007). Early in his career, Seligman studied depression and defined the theory of learned helplessness and pessimism. His worked progressed from a focus on pessimism to optimism, and hence from depression to happiness. He believed that psychology focused too much on mental illness and not enough on health and flourishing; thus, he pioneered the field of positive psychology in 2000 (Seligman and Csikzentmihalyi 2000).

Major Accomplishments

In 1996, Seligman was elected president of the American Psychological Association (APA) by the largest vote in history. As president, he chose the theme of positive psychology and called for an integration of human flourishing, strengths, and virtues into the science and practice of psychology. He noted that psychology and psychiatry had focused primarily on mental illness (e.g., depression, suffering, victimization, anger, anxiety), but had forgotten positive forms of mental health (e.g., positive emotion, engagement, positive relationships, purpose, accomplishment) (Seligman 2008). His mission in the APA paralleled his personal mission to promote positive psychology.

In 2008, he began his next mission: to promote a new movement in psychology, positive health (Seligman 2008). He argued that people desire more than just the absence of suffering and pain; they desire well-being and flourishing that in itself can be protective against mental illness and disease. Thus, he defined positive health as the subjective, biological, and functional realms that can predict positive aspects of mental health, e.g., longevity, positive emotion, prognosis, and suggested areas for which positive health could be incorporated into studies of well-being and illness.

Seligman has published 20 books and over 200 articles on motivation and personality, including best sellers such as *Learned Optimism*, *Authentic Happiness*, and *The Optimistic Child*

(Shah n.d.). He is one of the most often-cited psychologists and the thirteenth most likely name to appear in a general psychology textbook (TED Conferences LLC 2008). Seligman also created the Masters of Applied Positive Psychology program at the University of Pennsylvania. Many institutions, including the National Institute on Aging, the National Science Foundation, and the National Institute of Mental Health, have supported his research. He has received numerous awards, including two Distinguished Scientific Contribution awards from the APA, the William James Fellow Award, and the James McKeen Cattell Fellow Award from the Association for Psychological Science, the MERIT Award from the National Institute of Mental Health, the Laurel Award of the American Association for Applied Psychology and Prevention, and the Lifetime Achievement Award of the Society for Research in Psychopathology.

Cross-References

- ▶ Learned Helplessness
- ▶ Optimism
- ▶ Positive Psychology

References and Readings

Seligman, M. E. P. (1975). *Helplessness: On depression, development, and death*. San Francisco: W. H. Freeman.

Seligman, M. E. P. (1990). *Learned optimism*. New York: Knopf.

Seligman, M. E. P. (1993). *What you can change and what you can't: The complete guide to successful self-improvement*. New York: Knopf.

Seligman, M. E. P. (1996). *The optimistic child: Proven program to safeguard children from depression and build lifelong resilience*. New York: Houghton Mifflin.

Seligman, M. E. P. (2002). *Authentic happiness: Using the new positive psychology to realize your potential for lasting fulfillment*. New York: Free Press.

Seligman, M. (2008). Positive health. *Applied Psychology. An International Review, 57*, 3–18.

Seligman, M., & Csikzentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist, 55*, 5–14.

Shah, N. (n.d.). *Seligman, Martin E. P.* Retrieved 15 July 2011 from http://www.pabook.libraries.psu.edu/palitmap/bios/Seligman_Martin.html

TED Conferences, LLC. (2008). *Speakers: Martin Seligman: Psychologist*. Retrieved 15 July 2011 from http://www.ted.com/index.php/speakers/martin_seligman.html

The Trustees of the University of Pennsylvania. (2006). *Authentic happiness*. http://www.authentichappiennes.sas.upenn.edu/seligman.aspx

University of Pennsylvania. (2007). *Positive psychology center: Seligman bio*. Retrieved 15 July 2011 from http://www.ppc.sas.upenn.edu/bio.htm

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Biographical Information

Dr. Hans Selye 1973

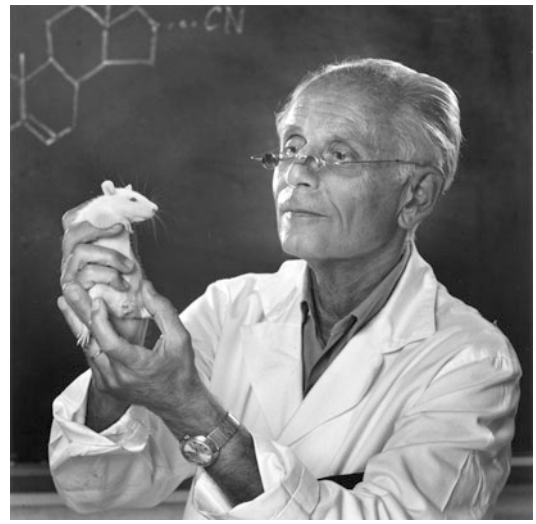

Photo by Yousuf Karsh

Hans Selye was born on January 26, 1907, in Vienna, Austria. His mother Maria Felicitas Langbank was an Austrian, his father Hugo Selye was a Hungarian military surgeon, and his grandfather and great grandfather were family doctors. Selye completed his elementary and secondary school education in Komarno (Slovakia). Since 1924 he studied medicine in Prague, Rome,

and Paris, and obtained his diploma at the German University of Prague in 1929. He started his research at the Institute of Experimental Pathology in Prague, and got his doctorate in biochemistry in 1931.

A Rockefeller Research Fellowship allowed Selye to continue his scientific career at the Department of Biochemical Hygiene of the Johns Hopkins University in Baltimore in 1931, and then from 1932 at the Department of Biochemistry of the McGill University in Montreal. He became lecturer then associate professor in biochemistry, and also in histology. He received Canadian citizenship in 1939, and became Doctor of Sciences in 1942. From 1945 to 1976 he directed the Institute of Experimental Medicine and Surgery (IMCE) at the University of Montreal, which gained international reputation under his leadership. After his retirement he remained active as founding president of the International Institute of Stress in Montreal (1976), and as co-founder of the Hans Selye Foundation (1979) until his death on October 15, 1982.

Selye held honorary doctorates from 18 universities, was a member of the Academy of Science of the Royal Society of Canada and 43 scientific societies, was an honorary citizen of many cities and countries, and received numerous high-ranking awards and distinctions. He was nominated for the Nobel Prize in physiology or medicine for 10 consecutive years (first in 1949), but he never received it. In 2006 he was inducted as a member of the Canadian Medical Hall of Fame.

Major Accomplishments

Selye is one of the most well-known, most productive scientists of the twentieth century. He wrote his name in the history of science by introducing the concept of stress. As a result of his work the word "stress" previously used in other contexts gained a physiological meaning and has been adopted in all languages. His research work had great impact in the field of endocrinology, physiology, biochemistry, epidemiology of chronic diseases, and behavioral medicine, not only on scientific thinking but also among lay people all around the world. Selye authored or coauthored 39 books and over 1,600 scientific articles, and his work has been estimated to have been cited in more than 300,000 scientific papers, although Somorjai (2007) could compile a list of only 800 of all his publications.

Selye's most important finding, the discovery of the stress syndrome, was accidental during his attempts to isolate a special female hormone from the placenta (Selye 1964, 1967b). The originality of his thinking was that he started to research the significance of the nonspecific reactions that he observed. His first short note describing "the nonspecific response to nocuous agents" was published in Nature in 1936, followed in the same year by a longer article in The British Journal of Experimental Pathology describing the General Adaptation Syndrome. In the following years, he systematically developed his comprehensive theory on stress (Selye 1950) and envisaged the existence of diseases of adaptation (Selye 1946).

His researches on steroids turned his interest to the inflammatory process (Selye 1943, 1949, 1965), then to the phenomena of calciphylaxis (Selye 1962) and of thrombohemorrhagy (Selye 1967a). Through these experiments he confirmed his hypothesis that diseases such as heart disease, rheumatoid arthritis, anaphylaxis, depression, autoimmune diseases, and Alzheimer disease are in fact all diseases of adaptation. Although his experiments were always on animals, over the years Selye became more and more interested in how his research results could be applied to medicine and to society. While his original definition of stress – "nonspecific response of the living organism to any stimuli, for example, effort, focused attention, pain, illness, failure, joy, success, that cause changes," – implied that the stressor can be either pleasant or unpleasant, since similar physiological/biochemical changes are produced, he later made the distinction between good stress (eustress) and bad stress (distress) (Selye 1974).

Selye expanded his model to include Perception, Conditioning Factors, and Coping Mechanisms. This so-called *Selye – Smith Conceptual Model of Stress Variables* served as theoretical

basis to the comprehensive course "Stress Management for Optimal Health" offered by Selye's Institute first to health professionals and then to the lay public (Smith and Selye 1979). Selye proposed that stress education and stress management should be important elements of preventive medicine.

Research was a passion for Selye. He was very much interested in great discoveries, the history and psychology of science, and in personal characteristics of scientists. These topics appeared in his lectures, books, and also in his every day discussions. He emphasized that original ideas were the most important elements in research that must be tested and proved by well-designed experiments. He also gave special attention to research methodology. On one hand, he was reluctant to adapt complicated technological methods, and he emphasized in all forums that one always should try to view the entire organism in its complexity and not to get lost with tiny little details without considering how they relate to the whole organism (Selye 1967b). On the other hand he introduced new methods in experimental surgery (Selye et al. 1960). He also carefully selected his laboratory assistants based on their skillfulness.

Effective information processing was another key element of Selye's exceptional productivity. He systematically developed his library that became world famous. He worked out his own "Symbolic Shorthand System for Medicine and Physiology" (SSS) that was subsequently published and used until the start of the computer era (Selye and Mishra 1957). He also followed a very structured daily schedule at his Institute as well as in his private life (Selye 1964). Selye was also a charismatic teacher. He shared his knowledge and his devotion to science with his fellow workers and students, exerting a deep influence on their lives and careers. He invited talented young researchers from all over the world to work in his Institute. He also invited many world renowned professors (the so-called Claude Bernard Professors). Thus, young researchers had the opportunity to meet famous personalities. They not only delivered one or two lectures, but participated in daily routine of the Institute and at informal dinners and discussions at Selye's house. Selye also traveled over the world and very often he obtained the recognition of his audience not only by his research results and presentation style, but also by giving his lecture in the language of the respective country. He not only shared his experiences with the scientific community, but could transmit his knowledge about stress, the process of scientific research, and related subjects to the general lay population by writing several popular books such as The Stress of Life (1956), From Dream to Discovery (1964), In Vivo: The Case for Supramolecular Biology (1967b), and Stress without Distress (1974) that have been translated into many languages.

Selye's scientific heritage is formally maintained by the Hans Selye Foundation, Montreal, Canada, www.stresscanada.org.

Cross-References

- ▶ Coping
- ▶ Stress
- ▶ Stress: Appraisal and Coping

References and Readings

Berczi, I. Stress and disease: The contribution of Hans Selye to: Neuroimmune Biology. A personal reminiscence. Retrieved March 30, 2012 from http://home.cc.umanitoba.ca/~berczii/page2.htm

Selye, H. (1936a). A syndrome produced by diverse nocuous agents. *Nature, 138*, 32.

Selye, H. (1936b). Thymus and adrenals in the response of the organism to injuries and intoxications. *British Journal of Experimental Pathology, 17*, 234–248.

Selye, H. (1943). Morphological changes in the fowl following chronic overdosage with various steroids. *Journal of Morphology, 73*, 401.

Selye, H. (1946). The general adaptation syndrome and the diseases of adaptation. *Journal of Clinical Endocrinology, 6*, 117–230.

Selye, H. (1949). Effect of ACTH and cortisone upon an anaphylactoid reaction. *Canadian Medical Association Journal, 61*, 553–556.

Selye, H. (1950). *Stress*. Montreal: Acta.

Selye, H. (1956). *The stress of life*. New York: McGraw Hill.

Selye, H. (1962). *Calciphylaxis*. Chicago: University of Chicago Press.

Selye, H. (1964). *From dream to discovery*. New York: McGraw Hill.

Selye, H. (1965). *The mast cells*. Washington, DC: Butterworths.
Selye, H. (1967a). Experimental thrombohemorrhagic phenomena. *The American Journal of Cardiology, 20*(2), 153–160.
Selye, H. (1967b). *In vivo: The case for supramolecular biology*. New York: Livesight.
Selye, H. (1974). *Stress without distress* (p. 364). Philadelphia: Lippincott.
Selye, H. (1979). *The stress of my life: A scientist's memory*. New York: Van Nostrand Reinhold.
Selye, H., & Mishra, R. K. (1957). Symbolic shorthand system for medicine and physiology. *Federation Proceedings, 16*(3), 704–706.
Selye, H., Bajusz, E., Grasso, S., & Mendell, P. (1960). Simple techniques for the surgical occlusion of coronary vessels in the rat. *Angiology, 11*, 398–407.
Smith, M. J. T., & Selye, H. (1979). Stress: Reducing the negative effects of stress. *The American Journal of Nursing, 11*, 1953–1955.
Somorjai, N. (2007). Bibliography Hans Selye. Retrieved March 30, 2012 from http://www.selyesociety.hu/pdf/Selye_bibliography_2011.pdf.
Stauder, A., & Kovács, P. B. (Eds.). (2007). *Stress. A memorial book on the birth centenary of Hans Selye*. Budapest: Downtown Artists' Society.
The American Institute of Stress: Hans Selye and The Birth of Stress. Retrieved March 30, 2012 from http://www.stress.org/hans.htm.
Université de Montréal: Hans Selye: Une vie en images. Retrieved March 30, 2012 from http://www.archiv.umontreal.ca/exposition/Hans_Selye/index.html

SEM

▶ Structural Equation Modeling (SEM)

Semenogelase

▶ Prostate-Specific Antigen (PSA)

Seminin

▶ Prostate-Specific Antigen (PSA)

Seminoma

▶ Cancer, Testicular

Senescence

▶ Immunosenescence

Senior

▶ Elderly

Sense of Coherence

▶ Salutogenesis

Sense of Coherence – Measurement

▶ Resilience: Measurement

Sense of Self

▶ Self-Identity

Separation

▶ Divorce and Health

SEPs

▶ Needle Exchange Programs

Sera

▶ Serum

Serious Games

▶ Health Gaming

Serostatus: Seronegative and Seropositive

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Synonyms

HIV status

Definition

Serostatus refers to the extent to which HIV antibodies can be detected in an individual's serum. This detection is an indicator of HIV infection.

Description

An individual is considered to be seronegative when the amount of HIV antibodies are not sufficient enough to be detected using an antibody test, although indication of HIV infection can be determined through the use of more sensitive culture, antigen, and viral gene detection techniques (Fultz 1989; Kaslow and Francis 1989; O'Malley 1988). Individuals who are seronegative may not produce HIV antibodies for months or year after HIV infection, or they may stop producing these antibodies after some unknown time interval. There is a long incubation period associated with being seronegative; individuals may not show physical symptoms associated with HIV/AIDS for several months or years after the initial HIV infection. As such, it is difficult to determine the ability of seronegative individuals to spread HIV (Kaslow and Francis 1989).

An individual is considered to be seropositive when HIV antibodies are detected on a HIV antibody test (Fultz 1989; O'Malley 1988). Individuals who have detectable HIV antibodies are considered to be "HIV-positive." The period of time between HIV infection and seroconversion (the presence of detectable antibodies in the serum) may range from 2 weeks to 3 months; this period of time is referred to as the "window period" (Stine 2003).

Some individuals who are seropositive, or "HIV-positive," exhibit symptoms associated with an acute mononucleosis-like syndrome immediately after being infected with HIV. This syndrome has been known as acute HIV syndrome or acute retroviral syndrome; its symptoms include sweats, lethargy, headaches, muscle aches, fever, and sore throat (Fultz 1989; Stine 2003). Not all seropositive individuals experience this syndrome. Other seropositive individuals remain asymptomatic for several weeks. All seropositive individuals, whether symptomatic or asymptomatic, are infected with HIV and can infect others through blood and genital secretions and by transmission from mother to fetus (Fultz 1989; O'Malley 1988). In 2001, the Center for Disease Control and Prevention proposed the Serostatus Approach to Fighting the HIV Epidemic (SAFE) program to encourage awareness that individuals may be HIV-positive even if they appear to be outwardly healthy. This program attempted to reduce the spread of HIV by extending prevention services and improving treatment adherence for individuals with a seropositive status and by providing training to

individuals who give these services (Normand et al. 1995; Stine 2003).

Being notified of a seropositive status is associated with behavioral and psychological changes. For instance, individuals who receive a positive test result may reduce their high-risk sexual activity. However, they also may experience increased stress and depression. Also, these individuals may feel compelled to disclose their HIV status to family, friends, and potential sexual parents. Some barriers associated with the disclosing HIV status include the disclosure of a stigmatized identity (such as being gay or an intravenous drug user), the fear of losing health insurance or employment, and the concern of being stigmatized by family and friends (Stall et al. 1989).

References and Readings

Black, P. H., & Levy, E. M. (1988). The HIV seropositive state and progression to AIDS: An overview of factors promoting progression. In P. O'Malley (Ed.), *The AIDS epidemic: Private rights and the public interest* (pp. 97–107). Boston: Beacon.

Fultz, P. (1989). The biology of human immunodeficiency viruses. In R. A. Kaslow & D. P. Francis (Eds.), *The epidemiology of AIDS: Expression, occurrence, and control of human immunodeficiency virus type 1 infection* (pp. 3–17). New York: Oxford University Press.

Kaslow, R. A., & Francis, D. P. (1989). Epidemiology: General considerations. In R. A. Kaslow & D. P. Francis (Eds.), *The epidemiology of AIDS: Expression, occurrence, and control of human immunodeficiency virus type 1 infection* (pp. 117–135). New York: Oxford University Press.

Normand, J., Vlahov, D., & Moses, L. E. (1995). *Preventing HIV transmission: The role of sterile needles and bleach*. Washington, DC: National Academy Press.

O'Malley, P. (1988). *The AIDS epidemic: Private rights and the public interest*. Boston: Beacon.

Stall, R., Coates, T. J., Mandel, J. S., Morales, E. S., & Sorensen, J. L. (1989). Behavioral factors and intervention. In R. A. Kaslow & D. P. Francis (Eds.), *The epidemiology of AIDS: Expression, occurrence, and control of human immunodeficiency virus type 1 infection* (pp. 266–281). New York: Oxford University Press.

Stine, G. J. (2003). *AIDS update2003: An annual overview of acquired immune deficiency syndrome*. Upper Saddle River: Prentice Hall.

Serotonin

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Definition

Serotonin (5-hydroxytryptamine or 5-HT) is a neurotransmitter that is particularly important in central nervous system modulation.

Description

Serotonin (5-hydroxytryptamine or 5-HT) is synthesized from the amino acid tryptophan. It is a major neurotransmitter, considered an indolamine monoamine, that is particularly important in central nervous system modulation, especially in the brain. Serotonin is also found in peripheral sites and regarded as a neuromodulator. In the periphery, serotonin is found in enteric neurons, part of the autonomic nervous system that governs the functions of the gastrointestinal tract. Serotonin is also found in blood platelets and cells of the gut.

In the brain serotonin plays an important neuromodulatory role involved in the regulation of mood, appetite, and sleep and in the cognitive functions of learning and memory.

Serotonin plays an important role in mental processes and has been implicated in many psychiatric disorders. Modulation of serotonin at synapses is thought to be a major action of several classes of pharmacological antidepressants, including selective serotonin reuptake inhibitors (SSRIs). It is uncertain whether low serotonin levels contribute to depression or if depression causes a fall in serotonin levels.

Cross-References

- ▶ Antidepressant Medications
- ▶ Central Nervous System
- ▶ Depression: Treatment
- ▶ Sleep

References and Further Reading

Pilowsky, P. (Ed.). (2019). *Serotonin: The mediator that spans evolution*. London: Academic. https://doi.org/10.1016/C2013-0-14440-6.

Serotonin Transporter Gene

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Synonyms

SERT; SLC6A4 (solute carrier family 6, member 4)

Definition

In humans, the gene that encodes for the serotonin transporter is called the serotonin transporter gene which is modulated by the functional serotonin-transporter-linked polymorphism (5-HTTLPR), a variable number of tandem repeats in the 5′ promoter region.

Description

The neurotransmitter *serotonin* (5-hydroxytryptamine, 5-HT) is probably best known for its modulation of neural activity and the modulation of various neuropsychological processes such as mood, perception, emotion, and cognition. Serotonin is also implicated in the pathogenesis of many psychiatric and neurological disorders and furthermore, it is involved in brain development and plasticity of brain areas related to cognitive and emotional processes (Berger et al. 2009; Trevor et al. 2010). Additionally, the serotonin transporter is considered to be the initial site of action of broadly used antidepressant drugs, such as selective serotonin uptake inhibitors (SSRIs), and several potentially neurotoxic compounds.

Following neuronal stimulation, serotonin is transmitted into the synaptic cleft and then binds to receptors on the membrane of the postsynaptic neuron. Serotonin is then removed from the synaptic cleft via special proteins, called *transporters*. Serotonin transporters are located in the serotonin neuron; they transport serotonin from the synaptic cleft back into the presynaptic neuron in both the brain and many peripheral tissues terminals. In humans, the gene that codes for the serotonin transporter contains a number of common variants (polymorphisms), including the serotonin-transporter-linked promoter region (*5-HTTLPR)* of the serotonin transporter gene (SLC6A4). This polymorphism is located upstream of the transcription start site on the long arm of the 17th chromosome (17q11.1-q12). The majority of alleles are composed of either fourteen ("short" or "*S*" allele) or sixteen repeated ("long" or "*L*" for allele) units, which differentially modulate on the expression and function of 5-HTT. The short form of *5-HTTLPR* has been associated with a reduced transcription of the 5-HTT gene, which leads to a decreased 5-HTT expression and availability and also a reduced 5-HT uptake (Lesch et al. 1996). In addition to the *S* and *L* alleles, there is an A > G single nucleotide polymorphism (SNP), a single nucleotide variation in a genetic sequence, upstream of the repetitive region that comprises the *5-HTTLPR*. The derived L_G allele has been associated with decreased 5-HTT transcription relative to the L_A allele. The frequency of *5-HTTLPR* alleles can vary substantially across ethnic groups, thus, showing population stratification.

The short allele variant of *5-HTTLPR* was first reported to be associated with personality traits such as neuroticism and harm avoidance (Lesch

et al.,). Subsequent work has reported *5-HTTLPR* allelic variation to a wide range of phenotypes including aggression, anxiety, and affective disorders. Most studies have implied that *5-HTTLPR* has only a moderate impact on these behavioral predispositions of 3–4% or less of the total variance. The less active *S* allele has been associated, either by itself or in interaction with adverse life events, with abnormal levels of anxiety, fear, and depression. Despite the association between presence of the *S* allele and psychopathology, studies of patient responsiveness to SSRIs suggest no strong link between *5-HTTLPR* genotype and drug effectiveness.

However, if the *S* allele produced only deleterious consequences, evolutionary pressures should have led to its removal from the gene pool. Thus, more recent studies have begun to accrue evidence for favorable phenotypes associated with the *S* allele. For example, studies revealed an improved performance in (social) cognition in individuals with the *S* allele (Homberg and Lesch 2010). On the other hand, the *L* allele, originally viewed as the protective allele, also has negative associations with at-risk phenotypes, such as cardiovascular health (e.g., increased cardiovascular reactivity and greater probability of myocardial infarction) (Fumeron et al. 2002; Williams et al. 2001) or certain psychiatric diseases (e.g., psychosis or posttraumatic stress disorder, PTSD) (Goldberg et al. 2009; Grabe et al. 2009). In addition to allelic association on observed phenotypes, there is growing evidence for gene-by-environment (G x E) interactions, suggesting that individuals possessing the *S* allele are predisposed to an increased risk for major depression or suicidal ideation as a function of early life stress. The first evidence for this G x E interaction in humans was presented by Caspi et al. (2003) who investigated more than 800 individuals over 23 years and found that life stress and depression was moderated by the *5-HTTLPR* genotype. Individuals with the *S* allele showed a higher probability of depressive symptoms, diagnosis of depression and suicidal attempts when exposed to stressful life events. However, replication studies have shown somewhat inconsistent results and thus, these findings are still a matter of debate. In addition to behavioral studies, non-invasive functional MRI (fMRI) studies have also shown that structural and functional characteristics of neural circuits involved in emotion and cognition can be moderated by the interaction of life stress and 5-HTTLPR genotype.

Most recently, investigators have come to recognize the potential gene regulatory role of epigenetic mechanisms in mediating environmental effects on brain function and on behavior (Rutter et al. 2006). For example, it has been argued that environmental influences bear the potential to persistently modify neuronal units during early development by epigenetic programming of emotionality (Weaver et al. 2004; Weaver 2007). This has first been shown with respect to the glucocorticoid receptor gene and individual differences in rodents' stress reactivity: variations in maternal care have been shown to modify the expression of genes that regulate behavioral and endocrine responses to stress and hippocampus synaptic development. Thus, alterations of particular genomic regions within the 5-HTT in response to varying environmental conditions might serve well as a major source of variation in biological and behavioral phenotypes. Indeed, there is now emerging evidence linking *5-HTTLPR* genotype to individual differences in epigenetic methylation (Philibert et al. 2007, 2008).

Studies that use biological endophenotypes, such as stress-induced HPA activation, might be more strongly associated with a specific polymorphism than a psychiatric disorder (Uher and McGuffin 2010). However, given the fact that brain serotonin and more specifically *5-HTTLPR* shows pleiotropic behavioral effects, we need to learn and understand more about the biological function and how *5-HTTLPR* becomes associated with various different phenotypes. The modulation of these multiple behavioral processes might very likely be regulated by multiple serotonin receptors that are expressed in multiple brain regions. In summary, *5-HTTLPR* seems to play an important, though not yet fully understood, role in behavioral medicine. We will likely gain new serotonergic drugs and disease treatments as well as a more thorough understanding of complexity of human biology from this research.

Cross-References

- ▶ Depression
- ▶ Gene-Environment Interaction
- ▶ Serotonin

References and Readings

Berger, M., Gray, J. A., & Roth, B. L. (2009). The expanded biology of serotonin. *Annual Review of Medicine, 60*, 355–366.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science, 301*(5631), 386–389.

Fumeron, F., Betoulle, D., Nicaud, V., Evans, A., Kee, F., Ruidavets, J. B., et al. (2002). Serotonin transporter gene polymorphism and myocardial infarction: Etude Cas-Temoins de l'Infarctus du Myocarde (ECTIM). *Circulation, 105*(25), 2943–2945.

Goldberg, T. E., Kotov, R., Lee, A. T., Gregersen, P. K., Lencz, T., Bromet, E., et al. (2009). The serotonin transporter gene and disease modification in psychosis: Evidence for systematic differences in allelic directionality at the 5-HTTLPR locus. *Schizophrenia Research, 111*(1–3), 103–108.

Grabe, H. J., Spitzer, C., Schwahn, C., Marcinek, A., Frahnow, A., Barnow, S., et al. (2009). Serotonin transporter gene (SLC6A4) promoter polymorphisms and the susceptibility to posttraumatic stress disorder in the general population. *The American Journal of Psychiatry, 166*(8), 926–933.

Homberg, J. R., & Lesch, K. P. (2010). Looking on the bright side of serotonin transporter gene variation. *Biological Psychiatry, 69*, 513–519.

Lesch, K. P., Bengel, D., Heils, A., Sabol, S. Z., Greenberg, B. D., Petri, S., et al. (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science, 274*(5292), 1527–1531.

Philibert, R., Madan, A., Andersen, A., Cadoret, R., Packer, H., & Sandhu, H. (2007). Serotonin transporter mRNA levels are associated with the methylation of an upstream CpG island. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics, 144B*(1), 101–105.

Philibert, R. A., Sandhu, H., Hollenbeck, N., Gunter, T., Adams, W., & Madan, A. (2008). The relationship of 5HTT (SLC6A4) methylation and genotype on mRNA expression and liability to major depression and alcohol dependence in subjects from the Iowa Adoption Studies. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics, 147B*(5), 543–549.

Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry, 47*(3–4), 226–261.

Trevor, A. J., Katzung, B. G., & Masters, S. B. (2010). *Katzung and Trevor's pharmacology examination and board review* (9th ed.). New York: McGraw-Hill Medical.

Uher, R., & McGuffin, P. (2010). The moderation by the serotonin transporter gene of environmental adversity in the etiology of depression: 2009 update. *Molecular Psychiatry, 15*(1), 18–22.

Weaver, I. C. (2007). Epigenetic programming by maternal behavior and pharmacological intervention. Nature versus nurture: Let's call the whole thing off. *Epigenetics, 2*(1), 22–28.

Weaver, I. C., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., et al. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience, 7*(8), 847–854.

Williams, R. B., Marchuk, D. A., Gadde, K. M., Barefoot, J. C., Grichnik, K., Helms, M. J., et al. (2001). Central nervous system serotonin function and cardiovascular responses to stress. *Psychosomatic Medicine, 63*(2), 300–305.

SERT

- ▶ Serotonin Transporter Gene

Sertraline

- ▶ Selective Serotonin Reuptake Inhibitors (SSRIs)

Serum

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Synonyms

Antiserum; Sera

Definition

Serum is blood plasma with the coagulatory proteins removed. It is a clear, pale-yellow, thin, and sticky fluid that moistens the surface of serous

membranes or that is secreted by such membranes when they become inflamed. In blood, serum is obtained after coagulation, upon separating whole blood into its solid and liquid components. This is achieved whereby blood is drawn from the subject and is allowed to naturally form a blood clot. After blood is allowed to clot and stand, a centrifuge is used to extract the red blood cells and the blood clot, which contains platelets and fibrinogens. In practice, blood serum is used in numerous diagnostic tests as well as blood typing.

Cross-References

- ▶ Antibodies
- ▶ Antigens

References and Further Reading

Abbas, A. K., & Lichtman, A. H. (2004). *Basic immunology: Functions and disorders of the immune system* (2nd ed.). Philadelphia: Saunders.

Service Attendance

- ▶ Religious Ritual

Sex

- ▶ Sexual Behavior

Sex Hormones

- ▶ Estrogen

Sexual Activity

- ▶ Sexual Behavior

Sexual Behavior

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Synonyms

Sex; Sexual activity

Definition

There is a diverse array of activities that can be classified as sexual behavior: masturbation, oral-genital stimulation (oral sex), penile-vaginal intercourse (vaginal sex), and anal stimulation or anal intercourse. Sexual behaviors may also include activities to arouse the sexual interest of others or attract partners. Individuals engage in sexual behaviors for a variety of reasons, differ in their acceptability based on societal norms, and change across the lifespan.

Description

What is Sexual Behavior: Types of Sexual Behaviors

Sexual behavior includes a wide variety of activities individuals engage in to express their sexuality (Crooks and Baur 2008). Abstinence and celibacy are terms used for individuals who do not engage in certain or any sexual behaviors. Kissing and touching are sexual behaviors that stimulate the erogenous zones of one's partner. Masturbation is a sexual behavior referring to stimulation of one's genitals to create sexual pleasure. Individuals may also engage in oral stimulation of a partner's genitals; terms used to describe oral-genital stimulation include: oral sex (referring broadly to oral-genital stimulation), cunnilingus (oral stimulation of the vulva), and fellatio (oral stimulation of the penis). Anal stimulation includes either touching around the anus or penile insertion in the anus (often referred to as anal sex). Penile-vaginal intercourse

involves insertion of the penis into a female's vagina; this behavior too has a variety of other synonymous terms (e.g., vaginal sex, coitus). The frequency that these and other sexual behaviors are engaged in has enormous individual variability and may differ based upon many factors (e.g., social acceptability, age).

Sexual Behavior: The Role of Societal Norms

Societal norms for acceptable sexual behaviors differ across cultures. Paraphilia refers to less common sexual behaviors within a given society or culture; an example of such behavior is a fetish. In some cultures, the nature of the relationship affects which behaviors are deemed acceptable. For instance, sexual behavior may be deemed appropriate only within the context of marriage. Similarly, societal perspectives on sexual orientation may influence whether sexual behaviors are viewed as socially acceptable.

Reasons for Sexual Behavior Engagement

Individuals engage in sexual behaviors for a multitude of reasons. Sexual behavior may be engaged in to experience sexual pleasure, sexual arousal, or orgasm. Procreation or a desire for children may motivate sexual behavior. Sexual behaviors may also be used to earn money or acquire other goods or services; prostitution refers to the exchange of a sexual behavior for monetary or other compensation. Additionally, pornography may motivate engagement in sexual behaviors. Unfortunately, sexual behaviors also occur in nonconsensual or coerced contexts (e.g., rape) and in the form of abuse (e.g., child sexual abuse) or sexual exploitation (e.g., pedophilia). Sexual behavior engagement may also have unintended consequences (e.g., unplanned pregnancy) or pose health risks associated with the acquisition of sexually transmitted diseases, including human immunodeficiency virus (HIV).

Developmental Perspectives on Sexual Behavior Engagement

Engagement in sexual activity changes across one's lifespan, and there is considerable variation in sexual development (Crooks and Baur 2008). During childhood, sexual behaviors may include self-stimulation of the genitals or engagement in play that may be viewed as sexual in nature (e.g., "playing doctor" with a peer). Puberty typically occurs during adolescence and results in dramatic physical changes including the development of secondary sex characteristics. Adolescence is typically linked to increases in sexual activity, both self-stimulation behaviors and sexual behavior with partners. During adulthood, and as people age, there is considerable individual variation of sexual behavior engagement.

Cross-References

- ▶ Condom Use
- ▶ HIV Infection
- ▶ HIV Prevention
- ▶ Sexual Functioning
- ▶ Sexual Hookup
- ▶ Sexual Risk Behavior

References and Readings

Crooks, R., & Baur, K. (2008). *Our sexuality* (10th ed.). Pacific Grove: Thomson.

Sexual Dysfunction

- ▶ Sexual Functioning

Sexual Functioning

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Synonyms

Sexual dysfunction

Definition

Sexual functioning is characterized by absence of difficulty moving through the stages of sexual desire, arousal, and orgasm, as well as subjective satisfaction with the frequency and outcome of individual and partnered sexual behavior.

Description

Sexual functioning is an important aspect of quality of life. Our understanding of sexual functioning is influenced by not only the current state of medical knowledge but also the social values upheld in our culture. Healthy sexual functioning is characterized by a lack of pain or discomfort during sexual activity and a lack of physiological difficulty moving through the three-phase sexual response cycle of desire, arousal, and orgasm. In addition, sexual functioning is indicated by subjective feelings of satisfaction with the frequency of sexual desire and sexual behavior, as well as subjective pleasure during individual and partnered sexual activity.

Kaplan's three-phase model is the basis for current models of healthy sexual response. The desire phase consists of sexual fantasies and desire to engage in sexual behavior. The arousal phase involves subjective feelings of pleasure along with physiological changes conducive to sexual intercourse. Males experience penile tumescence and erection, and females experience pelvic vasocongestion and vaginal lubrication. The orgasm phase consists of peak feelings of sexual pleasure and a release of sexual tension. Males ejaculate semen, whereas females experience contractions of the outer vaginal wall; additionally, in both males and females, the anal sphincter contracts. Individuals may experience physiological and/or psychological difficulties at any or all of the three phases of sexual response. A resolution period, characterized by relaxation and, for males, a refractory period, follows orgasm.

Etiology of Sexual Dysfunction

The etiology of sexual problems is often a complex combination of biological/medical, psychological, and social factors. For example, the sexual dysfunction may be secondary to a chronic health condition or psychotropic medication. In other cases, performance anxiety, low mood, or previous traumatic experiences may impair sexual functioning. Moreover, conflicts within a relationship as well as within the larger sociocultural context may affect an individual's sexual functioning. Due to the variety of potential predisposing, precipitating, and maintaining factors, clinicians are encouraged to take a biopsychosocial approach to assessment and treatment of problems in sexual functioning.

Sexual Dysfunction Disorders

Consistent with the medical model of disease, most research and scholarship focuses on sexual dysfunction rather than healthy sexual functioning. The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (2000) describes nine main disorders of sexual dysfunction, which are grouped into four categories: desire, arousal, orgasm, and pain. All nine disorders share some common diagnostic criteria: the dysfunction is persistent and recurrent; the dysfunction is not substance-induced, due to a general medical condition, or part of another Axis I mental disorder; and the dysfunction causes clinically significant distress or interpersonal difficulty. Sexual dysfunctions are also classified according to their onset (lifelong or acquired), context (generalized or situational), and etiology (due to psychological factors or due to combined psychological and medical factors). Additional diagnostic options include sexual dysfunction due to a general medical condition, substance-induced sexual dysfunction, and sexual dysfunction not otherwise specified.

Desire disorders are characterized by lack of interest in sex, absence of sexual fantasies and sexual behavior, or fear of sexual contact. In hypoactive sexual desire disorder, there is a low level of sexual fantasy and desire for sex. In

sexual aversion disorder, genital sexual contact is feared and actively avoided.

Arousal disorders are characterized by difficulty attaining or maintaining sexual arousal. Male erectile disorder is the most researched type of sexual dysfunction and receives the most media attention, particularly since the advent of Sildenafil (Viagra) in 1998. Erectile dysfunction is the inability to maintain an erection adequate for sexual penetration until completion of sexual activity. Female sexual arousal disorder is the inability to attain or maintain vaginal lubrication until completion of sexual activity.

Orgasm disorders are characterized by delay in or absence of orgasm on one extreme, or, on the other extreme, by occurrence of orgasm before the individual wants. In female orgasmic disorder and male orgasmic disorder, orgasm is delayed or absent despite normal sexual arousal and sufficient sexual stimulation. Premature ejaculation describes the occurrence of ejaculation with minimal stimulation before, upon, or soon after penetration and before the individual wants to orgasm.

Sexual pain disorders are characterized by genital pain that is not due to a general medical condition. In dyspareunia, which affects both males and females, genital pain occurs during sex. In vaginismus, involuntary muscle spasms of the vagina prevent penetration or may cause pain if it is attempted.

Prevalence of Sexual Dysfunction

Epidemiological surveys suggest problems with sexual functioning are common among the general population. For example, prevalence rates of premature ejaculation, erectile dysfunction, and female orgasmic disorder are 5%, 5%, and 10%, respectively, among community samples (Wincze and Carey 2001). Symptoms that do not meet full diagnostic criteria for a sexual dysfunction disorder are likely much more common. Patients struggling with sexual health problems may be reluctant to seek medical consultation due to embarrassment or privacy concerns. Many health care providers are also uncomfortable discussing sexuality, so patients' sex-related questions and concerns may be neglected in clinical settings.

Assessment of Sexual Functioning

Clinicians are advised to employ multimethod assessment of sexual functioning by including medical, psychosocial, and psychophysiological components. All three perspectives provide valuable information that aids in diagnosing sexual dysfunction, hypothesizing the etiology of the problem, and developing an appropriate treatment plan.

A medical evaluation is an essential piece of the sexual functioning assessment. A general physical examination allows for biological causes (e.g., general medical conditions, such as diabetes or cancer, as well as other vascular, neurologic, or hormonal conditions) to be ruled out. A gynecological or urological exam ensures no anatomical complications. Physical symptoms, such as bleeding or pain, can also be addressed. Medical providers should attend to any notable medical history (e.g., surgeries) as well as any prescription medications or substance use that may affect sexual functioning.

For the psychosocial evaluation, an interview is essential to learn about the onset, frequency, intensity, and duration of the presenting complaint(s). In addition, clinicians should assess pertinent areas including family history, adolescence, significant relationships in adulthood, sexual history, and sexual abuse or trauma. Although an individual patient may present with sexual complaints, difficulties with sexual functioning are often better understood in the context of the individual's sexual relationship. In many cases (e.g., when working with a patient who is married or in a committed relationship), involving the patient's sexual partner in the psychosocial evaluation (with the patient's permission) facilitates a better resolution. It is often advisable to interview the patient's sexual partner separately to find out more about the presenting complaint. A joint interview with the patient and his or her partner also provides additional insight into the couple's

interaction style, relationship quality, and non-sex-related problems that may cause interpersonal tension. For some patients, self-administered questionnaires may be used to supplement the psychosocial interview. Questionnaires may provide an easier method whereby to disclose sensitive information compared to a face-to-face interview.

The third potential component of the evaluation is psychophysiological assessment. Depending on the presenting complaint, psychophysiological measures can be quite helpful in differential diagnosis. For example, nocturnal penile tumescence is the gold standard for differential diagnosis for male patients with erectile dysfunction. Inability to obtain erections during sleep indicates a medical cause, whereas ability to maintain erections during sleep suggests a psychosocial cause.

Treatment of Sexual Dysfunction

Often the first therapeutic intervention occurs during the comprehensive assessment. During the psychosocial and medical evaluations, clinicians normalize the patient's problem, provide information, and correct misunderstandings. Formal treatment plans will depend on the presumed cause of the sexual dysfunction. Medical treatments include options such as pharmacotherapy (e.g., Viagra for erectile dysfunction), gels and creams (e.g., lubricating gels to compensate for problems with female arousal), hormone replacement therapy (e.g., testosterone to increase sexual desire), and surgery (e.g., penile implants to treat organic erectile dysfunction). Psychological treatments include psychoeducation about healthy sexual functioning, behaviorally focused sex therapy with the patient or the patient and his or her partner (e.g., using the stop-start technique to treat premature ejaculation), interpersonally focused couples therapy with the patient and his or her partner (e.g., working on trust or communication), or more traditional individual therapy with the patient (e.g., treating mood, anxiety, or trauma symptoms).

Cross-References

▶ Sexual Behavior

References and Readings

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Balon, R., & Segraves, R. T. (Eds.). (2005). *Handbook of sexual dysfunction* (Medical psychiatry series, Vol. 30). Boca Raton: Taylor & Francis.

Balon, R., & Segraves, R. T. (Eds.). (2009). *Clinical manual of sexual disorders*. Arlington: American Psychiatric Publishing.

Carey, M. P., & Gordon, C. M. (1995). Sexual dysfunction among heterosexual adults: Description, epidemiology, assessment, and treatment. In L. Diamant & R. McAnulty (Eds.), *The psychology of sexual orientation, behavior, and identity: A handbook* (pp. 165–196). Westport: Greenwood.

Fagan, P. J. (2004). *Sexual disorders: Perspectives on diagnosis and treatment*. Baltimore: The Johns Hopkins University Press.

IsHak, W. W. (Ed.). (2008). *The guidebook of sexual medicine*. Beverly Hills: A & W Publishing.

Maurice, W. L. (1999). *Sexual medicine in primary care*. St. Louis: Mosby.

Nusbaum, M., & Rosenfeld, J. A. (2005). *Sexual health across the lifecycle: A practical guide for clinicians*. Cambridge: Cambridge University Press.

Rowland, D. L., & Incrocci, L. (Eds.). (2008). *Handbook of sexual and gender identity disorders*. Hoboken: Wiley.

Wincze, J. P., & Carey, M. P. (2001). *Sexual dysfunction: Guide for assessment and treatment* (2nd ed.). New York: Guilford.

Sexual Hookup

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Synonyms

Casual sex; Hooking up

Definition

A sexual hookup is a sexual interaction between partners who are not dating or in a committed romantic relationship. There is no universal definition of sexual hookups, but qualitative research has begun to converge on the most common interpretation of hookup, which has three main components. First, hookups may involve a range of sexual behaviors, from kissing to sex. Kissing and sexual touching occur more frequently, but oral and vaginal sex occur during a significant minority of hookups. Anal sex during a hookup is rare. The variety of sexual behaviors that can occur during hookups causes ambiguity. From a research or public health perspective, behavioral specificity is needed to distinguish among different levels of sexual risk behavior. Condom use is rare during oral sex hookups, suggesting a potential risk for sexually transmitted diseases.

Second, hookup partners are not dating or in a committed romantic relationship. They may be friends, acquaintances, or strangers, or they may have been in a romantic relationship in the past. The most common connection between partners is friendship. Third, hookups do not signify an impending romantic commitment, so partners typically do not expect a relationship to result from the encounter. Instead, hookups are expected to serve a utilitarian function of sexual pleasure. However, individuals may desire a relationship with their hookup partner, and some engage in hookups with the hope that a relationship will eventually develop. Besides these three main criteria, hookups are also understood in terms of what they lack (emotional attachment and commitment). Emerging adults' descriptions of typical hookups are highly consistent, even between those who have and have not hooked up.

Several biomedical, sociocultural, and college environment changes occurring over the past 50 years have contributed to emergence of the hookup culture. Notably, emerging adults increasingly choose to postpone serious committed relationships to focus on self-development. Hookups offer a convenient way to obtain sexual intimacy without the commitment or time investment required by a relationship. Accordingly, hooking up has become very common among adolescents and emerging adults. A minority of middle and high school students and the majority of college students report hookup experience. Hooking up has replaced traditional dating as the main way to explore relationships and sexual behavior on college campuses. Hookup behavior among similarly aged noncollege attending youth is rarely studied.

Research has investigated several characteristics of sexual hookups. Hookups frequently co-occur with alcohol use, and alcohol use is a strong predictor of hookup behavior. Hookups are often spontaneous, but some individuals plan to hook up (either with a particular partner or with anyone). A variety of sexual, emotional, and social motives may lead individuals to hook up, such as sexual desire, intoxication, excitement, and desire to feel attractive. Some hookup partners interact only once, but some hook up multiple times, which is sometimes known as "friends with benefits." Hookups are also related to casual sex, as both lack emotional attachment. The main differences are the greater variety of sexual behaviors and partner types involved in hookups compared to casual sex and the extent to which hooking up has become a normative experience for youth.

Cross-References

- ▶ Condom Use
- ▶ Sexual Behavior
- ▶ Sexual Risk Behavior

References and Readings

Bogle, K. A. (2008). *Hooking up: Sex, dating, and relationships on campus*. New York: New York University Press.

Owen, J. J., Rhoades, G. K., Stanley, S. M., & Fincham, F. D. (2010). "Hooking up" among college students: Demographic and psychosocial correlates. *Archives of Sexual Behavior, 39*, 653–663.

Stinson, R. D. (2010). Hooking up in young adulthood: A review of factors influencing the sexual behavior of college students. *Journal of College Student Psychotherapy, 24*, 98–115.

Sexual Maturation

- ▶ Puberty

Sexual Orientation

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Definition

Sexual orientation is the sexual attraction, emotional, and/or romantic state that a person endures toward women, men, or both sexes. Sexual orientation also pertains to a person's sense of identity that is tied to these attractions, behaviors, and membership into a community with similar individuals (American Psychological Association [APA] 2011). Therefore, sexual orientation is the compilation of a person's sexual behavior and sense of sexual identity.

These two core components of sexual orientation are developed across a person's life span, yet many believe it to be an innate and fixed state (APA 2011). Nonetheless, an individual's sexual orientation is often characterized with a label, such as heterosexuality, homosexuality, or bisexuality. Sometimes asexuality is also considered as a separate entity of sexual orientation. Those labels normally, but not always, incorporate and include the individuals' sexual identity and sexual behavior of her or his sexual orientation. Because sexuality may be viewed as a fluid construct, an individual's sexual behavior may change over time while maintaining the same sexual identity. An example of this phenomenon would be when a heterosexual male experiments sexually with another male. Another exception to this generalization is when a self-identified lesbian woman has sex with a male. As such, these categories exist on a continuum of sexuality.

Scientists and psychologists have created measures of sexual orientation to better assess an individuals' sexuality. Typically, these measures will include a range of "solely heterosexual" to "solely homosexual" with "bisexuality" falling somewhere in between these two categories and "asexuality" not being included. A variety of measurements exist to assess sexual orientation. However, an individual's sexual orientation may change over time, and as such, measuring this construct at a single point in time (i.e., cross-sectional study) does have its limitations. Nonetheless, more studies are including a variety of measures that assess the sexual behavior and identity dimensions of sexual orientation.

The most well-recognized measurement of sexual orientation for males and females is the Kinsey scale (Kinsey et al. 1948, 1953). The scale ranges from 0 for "exclusively heterosexual with no homosexual" to 6 for "exclusively homosexual." The original scale does not include an "asexual" rating or category nor does it take into account any changes of sexual orientation over a period of time. Since then, other measurements of sexual orientation have been created and reviewed. For more information and references, please refer to the further readings section.

Cross-References

► Sexual Behavior

References and Readings

American Psychological Association. (2011). *Sexual orientation and homosexuality*. Retrieved 4 Feb 2011 from http://www.apa.org/topics/sexuality/orientation.aspx

Chung, Y. B., & Katayama, M. (1996). Assessment of sexual orientation in lesbian/gay/bisexual studies. *Journal of Homosexuality, 30*(4), 49–62.

Kinsey, A., Pomeroy, W., & Martin, C. (1948). *Sexual behavior in the human male*. Philadelphia: Saunders. ISBN 978-0253334121.

Kinsey, A., Pomeroy, W., Martin, C., & Gebhard, P. (1953). *Sexual behavior in the human female*. Philadelphia: Saunders. ISBN 978-0253334114.

Sell, R. L. (1996). The sell assessment of sexual orientation: Background and scoring. *Journal of Gay, Lesbian, and Bisexual Identity, 1*(4), 295–310.

Sexual Risk

► Sexual Risk Behavior

Sexual Risk Behavior

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Synonyms

Sexual risk; Unprotected sex

Definition

Sexual risk behavior is any sexual behavior (typically condom-unprotected oral, vaginal, or anal intercourse) that puts one at risk for an adverse health outcome. Adverse health outcomes may include an unwanted pregnancy or contracting a sexually transmitted disease (STD), including human immunodeficiency virus (HIV).

Vaginal intercourse is the only sexual behavior that puts an individual at risk for unwanted pregnancy. There are many methods for reducing the risk of unwanted pregnancy, including hormonal contraceptives, correct and consistent condom use, surgical methods such as a vasectomy or tubal ligation, and other methods such as an intrauterine device or a diaphragm.

Sexual behavior falls on a continuum from no risk to low risk to high risk for contracting an STD. The risk level of the sexual behavior depends on the STD under consideration. For example, with respect to HIV, masturbation incurs virtually no risk, oral sex is low risk, and vaginal and anal intercourse are high-risk behaviors. However, oral sex is a high-risk behavior for contracting some STDs such as gonorrhea.

The risk level of a particular sexual behavior also depends on with whom the behavior occurs. Any sexual behavior that occurs when an individual is alone is generally no risk because the individual is not at risk of contracting an STD from him- or herself. In addition, any sexual behavior that occurs within the context of a mutually monogamous relationship, in which both individuals are not infected with any STDs (especially when confirmed by testing), confers no risk of contracting an STD for either individual. Sexual behavior puts an individual at risk for contracting an STD only when his or her sexual partner is infected with an STD.

The risk of a particular sexual behavior also depends on the individual's sexual network. Sexual risk increases with an increasing number of sexual partners, and/or an increasing number of a sex partner's partners, because there is an increasing likelihood that one of these individuals is infected with an STD. In other words, the larger the sexual network, the greater the sexual risk.

STDs can have serious consequences, including epididymitis in men, pelvic inflammatory disease and pregnancy complications in women, infertility, and cancer (Centers for Disease Control and Prevention 2010d). In addition, the presence of another STD facilitates the transmission of HIV through sexual exposure (Centers for Disease Control and Prevention 2007). HIV weakens the immune system, ultimately leading to death when the immune system is so weakened it is unable to fight off infections or cancers (Centers for Disease Control and Prevention 2010a). STDs can also have negative relationship and social consequences due to the stigma surrounding some STDs.

The majority of STDs are either bacterial or viral (Holmes et al. 2008). In general, bacterial STDs can be cured with antibiotics, although some STDs are becoming resistant to antibiotics that had previously successfully treated the disease (Centers for Disease Control and Prevention 2010b). Viral STDs have no cure, although there are medications that can help to manage outbreaks or viral load. STDs may sometimes be cleared from the body with no treatment (Centers for Disease Control and Prevention 2010c).

Condoms are an effective way to reduce the risk of contracting an STD when they are used consistently and correctly. Although the value of condoms for the prevention of some STDs that can be transmitted through genital-to-genital

contact, such as human papillomavirus and herpes simplex virus, has been debated, recent evidence suggests that condoms reduce the risk of STD infection from these pathogens as well (Holmes et al. 2008).

Numerous factors influence sexual risk behavior. These factors can be broadly categorized into individual-level factors, partner- or relationship-level factors, and social or structural factors. Researchers have typically focused on only one level of influence at a time, although the integration of individual-level, partner or relationship-level, and social or structural factors has recently been attempted in the Network-Individual-Resource Model for HIV prevention (Johnson et al. 2010).

The relation between individual-level factors and sexual risk behavior has been extensively researched. Numerous health behavior theories, including the health belief model, social-cognitive theory, the theory of planned behavior, and the information-motivation-behavioral skills model, have been used to explain why individuals engage in sexual risk behavior (Fisher and Fisher 2000). Constructs from these models such as perceived risk, benefits of and barriers to risk reduction, self-efficacy, social norms, attitudes, intentions, and skills have been associated with sexual risk behavior (Fisher and Fisher 2000).

Because sexual risk behavior usually occurs within a dyad, researchers have begun to consider partnership-level influences on sexual behavior. At the partnership level, variables such as intimate partner violence and the balance of power in a relationship may influence sexual risk behavior. There are few existing theories that incorporate partner influences on sexual risk behavior; one exception is a framework recently proposed by Karney et al. (2010), which posits that sexual risk behavior is influenced by the ability to communicate about and coordinate sexual behavior, which, in turn, is influenced by the individual beliefs and motivations of each partner as well as by the nature of the relationship.

Although it is commonly accepted that social and structural factors influence sexual risk behavior, because of the complexity and breadth of these factors, it is difficult to develop a general model that predicts how these factors influence sexual risk behavior. Several broad frameworks have been suggested that specify different levels of structural influence (Gupta et al. 2008). Structural factors that influence sexual risk behavior vary depending on the social, cultural, and economic conditions faced by the population under study. Some structural factors associated with sexual risk behavior include gender inequality and poverty (Gupta et al. 2008). In the United States, one factor that has received considerable recent attention is the male-to-female ratio. Social and structural factors such as the high mortality rate among African American males due to disease and violence, high rate of incarceration among African American males, and high rates of poverty and unemployment among African American males (making them less desirable as potential husbands) have led to an unbalanced ratio of available African American men to women. This shortage of men relative to women may reduce women's power in relationships and their ability to insist on monogamy, ultimately leading to high rates of partner concurrency (Adimora and Schoenbach 2002).

Behavioral medicine researchers and practitioners have played an important role in the design and evaluation of interventions to reduce sexual risk behavior. Numerous interventions have been developed to target the individual-level determinants of sexual risk behavior. These interventions, particularly those that include motivational and skills elements, are effective in reducing sexual risk behavior (Crepaz et al. 2007, 2009; Johnson et al. 2006). Few interventions have been developed to target the partnership-level determinants of sexual risk behavior (Karney et al. 2010); this is an important area for future research. Although there are challenges to implementing and evaluating structural interventions, some programs, such as microcredit programs for women and policies requiring condoms be used for sex work, have successfully reduced sexual risk behavior in some settings (Gupta et al. 2008).

Additional research on structural-level sexual risk reduction interventions is needed, as is research on interventions that target multiple levels of influence.

References and Readings

Adimora, A. A., & Schoenbach, V. J. (2002). Contextual factors and the black–white disparity in heterosexual HIV transmission. *Epidemiology, 13*, 707–712.

Centers for Disease Control and Prevention. (2007). *CDC fact sheet: The role of STD prevention and treatment in HIV prevention*. Retrieved 6 Jan 2011 from http://cdc.gov/std/HIV/stds-and-hiv-fact-sheet.pdf

Centers for Disease Control and Prevention. (2010a). *Basic information about HIV and AIDS*. Retrieved 6 Jan 2011 from http://www.cdc.gov/hiv/topics/basic/index.htm

Centers for Disease Control and Prevention. (2010b). *CDC fact sheet: Basic information about antibiotic-resistant gonorrhea (ARG)*. Retrieved 6 Jan 2011 from http://cdc.gov/std/Gonorrhea/arg/basic.htm

Centers for Disease Control and Prevention. (2010c). *CDC fact sheet: Genital HPV*. Retrieved 6 Jan 2011 from http://cdc.gov/std/hpv/hpv-fact-sheet-press.pdf

Centers for Disease Control and Prevention. (2010d). *CDC fact sheets: Sexually transmitted diseases*. Retrieved 6 Jan 2011 from http://cdc.gov/std/healthcomm/fact_sheets.htm

Crepaz, N., Horn, A. K., Rama, S. M., Griffin, T., Deluca, J. B., Mullins, M. M., et al. (2007). The efficacy of behavioral interventions in reducing HIV risk sex behaviors and incident sexually transmitted disease in Black and Hispanic sexually transmitted disease clinic patients in the United States: A meta-analytic review. *Sexually Transmitted Diseases, 34*, 319–332.

Crepaz, N., Marshall, K. J., Aupont, L. W., Jacobs, E. D., Mizuno, Y., Kay, L. S., et al. (2009). The efficacy of HIV/STI behavioral interventions for African American females in the United States: A meta-analysis. *American Journal of Public Health, 99*, 2069–2078.

Fisher, J. D., & Fisher, W. A. (2000). Theoretical approaches to individual-level change in HIV risk behavior. In J. L. Peterson & R. J. DiClemente (Eds.), *Handbook of HIV prevention* (pp. 3–55). New York: Kluwer Academic/Plenum.

Gupta, G. R., Parkhurst, J. O., Ogden, J. A., Aggleton, P., & Mahal, A. (2008). Structural approaches to HIV prevention. *The Lancet, 372*, 764–775.

Holmes, K. K., Sparling, P. F., Stamm, W. E., Piot, P., Wasserheit, J. N., et al. (2008). Introduction and overview. In K. K. Holmes, P. F. Sparling, W. E. Stamm, P. Piot, J. N. Wasserheit, L. Corey, et al. (Eds.), *Sexually transmitted diseases* (4th ed., pp. xvii–xxv). New York: McGraw Hill.

Johnson, B. T., Carey, M. P., Chaudoir, S. R., & Reid, A. E. (2006). Sexual risk reduction for persons living with HIV: Research synthesis of randomized controlled trials, 1993 to 2004. *Journal of Acquired Immune Deficiency Syndromes, 41*, 642–650.

Johnson, B. T., Redding, C. A., DiClemente, R. J., Mustanski, B. S., Dodge, B., Sheeran, P., et al. (2010). A network-individual-resource model for HIV prevention. *AIDS and Behavior, 14*, S204–S221.

Karney, B. R., Hops, H., Redding, C. A., Reis, H. T., Rothman, A. J., & Simpson, J. A. (2010). A framework for incorporating dyads in models of HIV-prevention. *AIDS and Behavior, 14*, S189–S203.

Sexuality and Stress

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Introduction

According to many therapy manuals and self-help guides on sexuality, stress is the number one factor inhibiting sexual desire. However, no sufficient empirical evidence can be found to support this claim. Since both sexuality and stress are important factors of everyday life and contribute substantially to health (e.g., McEwen 1998; World Health Organization [WHO] 2006), this entry attempts to provide a summary of the current state of knowledge regarding how sexuality and stress might be intertwined.

Sexuality

Human sexuality plays a central role throughout life (WHO 2006) and "encompasses sex, gender identities and roles, sexual orientation, eroticism, pleasure, intimacy and reproduction" (WHO 2006, p. 5). There are three key components of sexual experience and behavior, based on the sexual response cycle (Masters and Johnson 1966), modified by Kaplan (1979), and repeatedly reported in the scientific literature: sexual desire, sexual arousal, and orgasm. According to Bancroft, sexual desire

and arousal are "two 'windows' into the complexity of sexual arousal [. . .], one focusing on the incentive motivation component (desire) and the other on the arousal component (excitement)" (2009, p. 65). An orgasm, often seen as the goal of sexual activity, is associated with pleasure and reduced tension (Bancroft 2009). The importance of these factors is also evident in the division of sexual dysfunctions presented in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association 2013).

Among other factors, sexuality is influenced by psychological, social, and biological factors (WHO 2006) such as reproductive hormones, which play an essential role in human sexual development, sexual function, and sexual experience and behavior (Bancroft 2009). The main reproductive hormones include steroid (e.g., testosterone, estradiol) and peptide hormones (e.g., vasopressin, oxytocin). Androgens, such as testosterone, and estrogens, such as estradiol, are produced by the testes as well as the ovaries and play an important role for sexuality in both men and women. Testosterone seems to be especially relevant for sexual desire and arousal in both men and women (Bancroft 2009).

Given that sexuality plays such an essential role throughout life, maintaining sexual health is highly important. Sexual health can be defined as "a state of physical, emotional, mental and social well-being in relation to sexuality; it is not merely the absence of disease, dysfunction or infirmity" (WHO 2006, p. 5). It "requires a positive and respectful approach to sexuality and sexual relationships, as well as the possibility of having pleasurable and safe sexual experiences, free of coercion, discrimination and violence" (WHO 2006, p. 5).

A sexual dysfunction, by contrast, is characterized as a sexual problem that occurs recurrently or in the majority of sexual contacts, leads to significant distress, lasts for at least half a year and cannot directly be explained by another mental disorder, severe relationship distress, or other significant stressors, a disease, or substance or medication intake (American Psychiatric Association 2013). The most common sexual dysfunctions have been found to involve lack of sexual desire (prevalence rates between 10% and 61.5%) and lack of sexual arousal (prevalence rates between 12% and 49%) in women, and premature ejaculation (prevalence rates between 1% and 10%) and erectile dysfunction (prevalence rates between 1% and 10% for individuals younger than 40 years old) in men (McCabe et al. 2016).

Stress

Acute stress can be caused by potential stressors such as major life events or situations necessitating a "fight or flight" response, while chronic stress can be caused by potential stressors such as daily hassles. Whether an individual perceives a potential stressor as a stressor is determined by the individual's perception of the situation as well as the individual's general health (McEwen 1998). Specifically, according to the transactional model of stress and coping, in a step called "primary appraisal", a situation is evaluated as irrelevant, positive, or, as in the case of a stressor, dangerous (Lazarus and Folkman 1984). In a concomitant step called "secondary appraisal", available resources and coping strategies are analyzed. Depending on "primary appraisal" and "secondary appraisal", a stressful situation can be categorized as harm or loss, a threat or a challenge by an individual and can thus differ between individuals (Lazarus and Folkman 1984). Acute and chronic stress can both have negative long-term consequences (McEwen 1998).

The Association between Sexuality and Stress

Sexual experience and behavior seems to be negatively associated with stress (e.g., Abedi et al. 2015), although there is some contrasting evidence regarding sexual desire, which can be seen as a component of sexual experience and behavior (Morokoff and Gillilland 1993; Raisanen et al. 2018).

Results from cross-sectional studies: Specifically, stress has been negatively associated with the frequency of sexual intercourse in women (Abedi et al. 2015). Furthermore, while one study reported that stress was negatively associated with sexual desire in women (Abedi et al. 2015), another

study found a positive association between stress and sexual desire in both women and men (Morokoff and Gillilland 1993). In women, in the study by Abedi et al. (2015), sexual arousal and lubrication were negatively associated with stress, while Hamilton and Meston (2013) also found a negative association between genital arousal and levels of stress as well as cortisol levels, but did not find a significant association between psychological arousal and stress. Moreover, the study by Abedi et al. (2015) also reported a negative association between stress and satisfaction as well as stress and orgasm, and a positive association between stress and sexual pain in women.

Results from experiments inducing stress: One of the few experimental studies to have investigated the association of sexual experience and behavior with stress found that receiving a sexual reward in the form of viewing mildly erotic images compared to viewing neutral images before participating in the Trier Social Stress Test (TSST) positively affected stress in men: Participants in the reward condition showed significantly lower cortisol reactivity during the TSST compared to participants in the neutral condition (Creswell et al. 2013).

Results from longitudinal studies: In addition, some studies have examined the link between sexual experience and behavior and stress longitudinally. Specifically, stress was negatively associated with sexual desire for partnered sexual activity in women but positively associated with sexual desire for partnered sexual activity in men, and not associated with sexual desire for non-partnered sexual activity in men or women (Raisanen et al. 2018). Furthermore, the authors found that testosterone was negatively associated with sexual desire for partnered sexual activity in women and with sexual desire for non-partnered sexual activity in women with lower levels of stress, but positively associated with sexual desire for non-partnered sexual activity in women with high levels of stress. In men, testosterone was not associated with sexual desire for partnered sexual activity, but positively associated with sexual desire for non-partnered sexual activity. Stress moderated the association between testosterone and sexual desire for non-partnered sexual activity but not between testosterone and sexual desire for partnered sexual activity (Raisanen et al. 2018). In addition, research has shown a negative association of same-day stress with occurrence of sexual intercourse, genital stimulation, and orgasm with and without the partner in women (Burleson et al. 2007). Stress was also negatively associated with frequency of sexual activity, satisfaction, and relationship satisfaction in women (Bodenmann et al. 2010). Contrasting these findings, Hall, Kusunoki, Gatny, and Barber found that women with moderate to severe stress symptoms showed a significantly higher frequency of sexual intercourse (2014). Furthermore, in line with these results, intimacy, including sexual intercourse, was significantly associated with lower levels of daily salivary cortisol (Ditzen et al. 2008). Also, sexual intercourse led to better blood pressure reactions under stress compared to engagement in other or no sexual activities (Brody 2006). In another study, experiencing a stressful day increased the probability of engaging in sexual intercourse the next day, although this probability was higher for men than for women (Ein-Dor and Hirschberger 2012). Furthermore, sexual intercourse on 1 day led to lower levels of stress the next day for both genders. While this link between stress and sexual intercourse was unaffected by relationship satisfaction for women, men with lower relationship satisfaction showed a higher stress relief following sexual intercourse (Ein-Dor and Hirschberger 2012). In addition, physical affection on 1 day predicted lower stress on the following day in women (Burleson et al. 2007). Similarly, genital stimulation and orgasm with a partner predicted lower stress the next day, but only for women with a shorter relationship duration; no such effect was found for women with longer relationship duration. Stress, however, did not significantly affect any of the variables on the following day in this study in women (Burleson et al. 2007).

Outlook

So far, study findings on the link between sexual experience and behavior and stress are not

straightforward. Few studies have investigated this link bidirectionally or longitudinally. Moreover, there is little research investigating this relationship in daily life, thus limiting the ecological validity of previous studies. Additionally, studies comparing the sex-stress link between men and women are scarce. Hence, future research will need to focus on investigating the bidirectional link between sexual experience and behavior and stress in both men and women, ideally in a setting characterized by high ecological validity. Furthermore, while the assessment of stress requires a multidimensional measurement approach (Nater 2018), previous analyses have rarely sampled and included biological stress parameters such as cortisol or alpha-amylase, even though it is widely known that these parameters not only represent the human stress reaction biologically but have also been associated with negative health consequences (e.g., Kudielka and Kirschbaum 2014). Similarly, testosterone has been linked to both sexual desire and arousal (e.g., Bancroft 2009). Thus, future studies should incorporate a comprehensive measurement of biological parameters (e.g., cortisol, alpha-amylase, and testosterone) and thus fulfill the requirements of a multidimensional measurement approach. Finally, to date, no studies have investigated the sex-stress link in the everyday life of patients diagnosed with a sexual dysfunction, even though acquiring knowledge in this area might inform further valuable treatment options. Hence, future research should investigate the association between sexual experience and behavior and stress in individuals with diagnosed sexual dysfunctions such as female sexual interest/arousal disorder, as this is one of the most frequently occurring sexual dysfunctions.

Cross-References

- ▶ Daily Stress
- ▶ Immune Responses to Stress
- ▶ Mental Stress
- ▶ Perceived Stress
- ▶ Perceptions of Stress
- ▶ Psychological Stress
- ▶ Sexual Behavior
- ▶ Sexual Functioning
- ▶ Stress
- ▶ Stress Reactivity
- ▶ Stress Responses
- ▶ Stress, Emotional
- ▶ Stress: Appraisal and Coping
- ▶ Stress-Related Disorders
- ▶ Trier Social Stress Test

References and Further Reading

Abedi, P., Afrazeh, M., Javadifar, N., & Saki, A. (2015). The relation between stress and sexual function and satisfaction in reproductive-age women in Iran: A cross-sectional study. *Journal of Sex & Marital Therapy, 41*(3), 384–390. https://doi.org/10.1080/0092623X.2014.915906.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5* (5th ed.). Washington, DC: American Psychiatric Publishing.

Bancroft, J. (2009). *Human sexuality and its problems* (3rd ed.). Edinburgh: Churchill Livingstone.

Bodenmann, G., Atkins, D. C., Schär, M., & Poffet, V. (2010). The association between daily stress and sexual activity. *Journal of Family Psychology, 24*(3), 271–279. https://doi.org/10.1037/a0019365.

Brody, S. (2006). Blood pressure reactivity to stress is better for people who recently had penile-vaginal intercourse than for people who had other or no sexual activity. *Biological Psychology, 71*, 214–222. https://doi.org/10.1016/j.biopsycho.2005.03.005.

Burleson, M. H., Trevathan, W. R., & Todd, M. (2007). In the mood for love or vice versa? Exploring the relations among sexual activity, physical affection, affect, and stress in the daily lives of mid-aged women. *Archives of Sexual Behavior, 36*(3), 357–368. https://doi.org/10.1007/s10508-006-9071-1.

Creswell, J. D., Pacilio, L. E., Denson, T. F., & Satyshur, M. (2013). The effect of a primary sexual reward manipulation of cortisol responses to psychosocial stress in men. *Psychosomatic Medicine, 75*(4), 397–403. https://doi.org/10.1097/PSY.0b013e31828c4524.

Ditzen, B., Hoppmann, C., & Klumb, P. (2008). Positive couple interactions and daily cortisol: On the stress-protecting role of intimacy. *Psychosomatic Medicine, 70*(8), 883–889. https://doi.org/10.1097/PSY.0b013e318185c4fc.

Ein-Dor, T., & Hirschberger, G. (2012). Sexual healing: Daily diary evidence that sex relieves stress for men

and women in satisfying relationships. *Journal of Social and Personal Relationships, 29*(1), 126–139. https://doi.org/10.1177/0265407511431185.
Hall, K. S., Kusunoki, Y., Gatny, H., & Barber, J. (2014). Stress symptoms and the frequency of sexual intercourse among young women. *Journal of Sexual Medicine, 11*(8), 1982–1990. https://doi.org/10.1111/jsm.12607.
Hamilton, L. D., & Meston, C. M. (2013). Chronic stress and sexual function in women. *Journal of Sexual Medicine, 10*(10), 2443–3454. https://doi.org/10.1111/jsm.12249.
Kaplan, H. S. (1979). *Disorders of sexual desire and other new concepts and techniques in sex therapy*. New York: Brunner/Mazel.
Kudielka, B. M., & Kirschbaum, C. (2014). Sex differences in HPA axis responses to stress: A review. *Biological Psychology, 69*, 113–132. https://doi.org/10.1016/j.biopsycho.2004.11.009.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. Berlin: Springer.
Masters, W. H., & Johnson, V. E. (1966). *Human sexual response*. Boston: Little, Brown.
McCabe, M. P., Sharlip, I. D., Lewis, R., Atalla, E., Balon, R., Fisher, A. D., et al. (2016). Incidence and prevalence of sexual dysfunction in women and men: A consensus statement from the fourth international consultation on sexual medicine 2015. *The Journal of Sexual Medicine, 13*(2), 144–152. https://doi.org/10.1016/j.jsxm.2015.12.034.
McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*(3), 171–179. https://doi.org/10.1056/NEJM199801153380307.
Morokoff, P. J., & Gillilland, R. (1993). Stress, sexual functioning, and marital satisfaction. *The Journal of Sex Research, 30*(1), 43–53. https://doi.org/10.1080/00224499309551677.
Nater, U. M. (2018). The multidimensionality of stress and its assessment. *Brain, Behavior, and Immunity, 73*, 159–160. https://doi.org/10.1016/j.bbi.2018.07.018.
Raisanen, J. C., Chadwick, S. B., Michalak, N., & van Anders, S. M. (2018). Average associations between sexual desire, testosterone, and stress in women and men over time. *Archives of Sexual Behavior, 47*(6), 1613–1631. https://doi.org/10.1007/s10508-018-1231-6.
Stoléru, S., Fonteille, V., Cornélis, C., Joyal, C., & Moulier, V. (2012). Functional neuroimaging studies of sexual arousal and orgasm in healthy men and women: A review and meta-analysis. *Neuroscience & Biobehavioral Reviews, 36*(6), 1481–1509. https://doi.org/10.1016/j.neubiorev.2012.03.006.
World Health Organization (WHO). (2006). *Defining sexual health: Report of a technical consultation of sexual health, 28–31 January 2002*. Sexual Health Document Series. Geneva. Retrieved from http://www.who.int/reproductivehealth/publications/sexual_health/defining_sexual_health.pdf

Sexually Transmitted Disease/Infection (STD/STI)

▶ AIDS: Acquired Immunodeficiency Syndrome
▶ HIV Infection

Sexually Transmitted Diseases (STDs)

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Synonyms

Sexually transmitted infections; Venereal diseases

Definition

A sexually transmitted disease is a disease that is transmitted through sexual contact (World Health Organization).

Our knowledge of sexually transmitted diseases (STDs) is still evolving. Currently, at least 35 pathogens that can be transmitted sexually have been identified (Holmes et al. 2008). Although some sexually transmissible pathogens can be transmitted through other routes besides sexual contact, generally a disease is classified as an STD when the primary method of transmission in a population is sexual contact (Holmes et al.).

There are five different types of pathogens that can be transmitted sexually: (a) bacteria, such as gonorrhea or chlamydia; (b) viruses, such as human immunodeficiency virus (HIV), herpes simplex virus, or human papillomavirus (HPV); (c) protozoa, such as trichomoniasis; (d) fungi, such as *Candida albicans* (although the primary mode of transmission for this pathogen is not sexual); and (e) ectoparasites, such as pubic lice (Holmes et al. 2008). Depending on the pathogen, STDs can be transmitted through bodily fluids

(e.g., blood, semen, cervicovaginal fluid), feces, or skin-to-skin contact. Thus, the transmission of an STD usually involves vaginal or anal intercourse, oral sex (including oral-genital contact and oral-anal contact), or genital-to-genital contact.

STD symptoms vary depending on the pathogen involved, as well as the sex of the infected person and the site of infection. Symptoms associated with some of the more common STDs include blisters or ulcers, pain or burning during urination, discharge, abdominal pain, and pain during intercourse. However, many individuals who are infected with an STD do not have any symptoms (Centers for Disease Control and Prevention 2010d). Such "asymptomatic" individuals may unknowingly infect a sexual partner with the STD.

Testing for STDs is generally conducted in medical facilities (although community- and home-based testing protocols are now available). Depending on the STD, testing may involve urethral or cervical swabs, swabs taken from the site of an ulcer, urine testing, blood testing, and clinical examination (Centers for Disease Control and Prevention 2010d). In the United States, some STDs must be reported (by health-care providers) to county, state, or federal health authorities; for example, positive test results for chlamydia, gonorrhea, and syphilis must be reported to the Centers for Disease Control and Prevention for disease surveillance and monitoring (Centers for Disease Control and Prevention 2009).

STDs can have serious consequences, including epididymitis in men, pelvic inflammatory disease and pregnancy complications in women, infertility, and cancer (Centers for Disease Control and Prevention 2010d). In addition, individuals who are co-infected with HIV and another STD are more likely to transmit HIV through sexual exposure, and individuals who are infected with an STD are more likely to acquire HIV through sexual exposure from an HIV-positive partner (Centers for Disease Control and Prevention 2007). HIV weakens the immune system, ultimately leading to death when the immune system is so weakened it is unable to fight off infections and cancers (Centers for Disease Control and Prevention 2010a). STDs can also have negative interpersonal and social consequences due to the stigma surrounding some STDs.

The majority of STDs are either bacterial or viral (Holmes et al. 2008). In general, bacterial STDs can be cured with antibiotics, although some STDs are becoming resistant to many classes of antibiotics that had previously successfully treated the disease (Centers for Disease Control and Prevention 2010b). Viral STDs have no cure, although there are medications that can help to manage outbreaks or viral load. STDs such as HPV may sometimes be cleared from the body with no treatment (Centers for Disease Control and Prevention 2010c).

An individual's likelihood of acquiring an STD is based on his or her sexual behavior and the risk of transmission per sexual act with an infected partner. Sexual behaviors that affect the likelihood of acquiring an STD include the number of sexual partners, the number of unprotected sexual acts, and the types of unprotected sexual acts. The risk of transmission per sexual act depends in part on the pathogen as well as the individual's biology. Different pathogens are associated with different risks of transmission per sexual act. Biological factors associated with transmission risk include the individual's immune response and the mucosal surface area exposed during the sexual act; women, for example, are more likely than men to be infected with an STD through vaginal intercourse because women have a larger mucosal surface area that is exposed during vaginal intercourse, and young women are more likely to acquire an STD because they have an immature cervix. STD risk is also affected by the STD prevalence in an individual's sexual network, which is influenced by the rate of sexual partner change, partner concurrency (i.e., multiple, overlapping sexual partnerships), and degree of disassortative mixing (i.e., sexual partners who are dissimilar in certain characteristics, such as age or sexual activity Garnett 2008).

To date, some types of HPV and some types of hepatitis are the only STDs that can be prevented through vaccination. Other medical strategies are

currently being developed for STD prevention, such as vaginal microbicides and preexposure prophylaxis. STDs can also be prevented through behavioral change. Abstaining from sexual contact is the only certain way to prevent most STDs. However, other behavioral strategies including correct and consistent condom use, engaging in sexual activity only with one partner who is not infected with any STD and who has no other sexual partners, and having fewer sexual partners will reduce the likelihood of acquiring an STD.

Behavioral medicine can play a large role in STD prevention. Behavioral interventions can promote sexual risk reduction behavior, by encouraging individuals to use condoms consistently and correctly for all sexual activity, be in a mutually monogamous sexual relationship with an uninfected partner, or adopt other strategies that will reduce the risk of contracting an STD. Behavioral interventions have been shown to be effective in reducing sexual risk behavior and STDs in a variety of populations (Crepaz et al. 2007, 2009; Johnson et al. 2006). Behavioral medicine can also play a role in encouraging the adoption of biomedical strategies. For example, behavioral medicine strategies could be used to encourage STD and HIV testing, vaccine acceptance, the completion of medications for curable STDs and adherence to medications for viral STDs, and male circumcision, which may reduce the spread of STDs and HIV.

Cross-References

► Sexual Risk Behavior

References and Readings

Centers for Disease Control and Prevention. (2007). *CDC fact sheet: The role of STD prevention and treatment in HIV prevention*. Retrieved 6 Jan 2011 from http://cdc.gov/std/HIV/stds-and-hiv-fact-sheet.pdf

Centers for Disease Control and Prevention. (2009). *Sexually transmitted disease surveillance, 2008*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, Division of STD Prevention.

Centers for Disease Control and Prevention. (2010a). *Basic information about HIV and AIDS*. Retrieved 6 Jan 2011 from http://www.cdc.gov/hiv/topics/basic/index.html

Centers for Disease Control and Prevention. (2010b). *CDC fact sheet: Basic information about antibiotic-resistant gonorrhea (ARG)*. Retrieved 6 Jan 2011 from http://cdc.gov/std/Gonorrhea/arg/basic.htm

Centers for Disease Control and Prevention. (2010c). *CDC fact sheet: Genital HPV*. Retrieved 6 Jan 2011 from http://cdc.gov/std/hpv/hpv-fact-sheet-press.pdf

Centers for Disease Control and Prevention. (2010d). *CDC fact sheets: Sexually transmitted diseases*. Retrieved 6 Jan 2011 from http://cdc.gov/std/healthcomm/fact_sheets.htm

Crepaz, N., Horn, A. K., Rama, S. M., Griffin, T., Deluca, J. B., Mullins, M. M., et al. (2007). The efficacy of behavioral interventions in reducing HIV risk sex behaviors and incident sexually transmitted disease in Black and Hispanic sexually transmitted disease clinic patients in the United States: A meta-analytic review. *Sexually Transmitted Diseases, 34*, 319–332.

Crepaz, N., Marshall, K. J., Aupont, L. W., Jacobs, E. D., Mizuno, Y., Kay, L. S., et al. (2009). The efficacy of HIV/STI behavioral interventions for African American females in the United States: A meta-analysis. *American Journal of Public Health, 99*, 2069–2078.

Garnett, G. P. (2008). The transmission dynamics of sexually transmitted infections. In K. K. Holmes, P. F. Sparling, W. E. Stamm, P. Piot, J. N. Wasserheit, L. Corey, et al. (Eds.), *Sexually transmitted diseases* (4th ed., pp. 27–40). New York: McGraw Hill.

Holmes, K. K., Sparling, P. F., Stamm, W. E., Piot, P., Wasserheit, J. N., et al. (2008). Introduction and overview. In K. K. Holmes, P. F. Sparling, W. E. Stamm, P. Piot, J. N. Wasserheit, L. Corey, et al. (Eds.), *Sexually transmitted diseases* (4th ed., pp. xvii–xxv). New York: McGraw Hill.

Johnson, B. T., Carey, M. P., Chaudoir, S. R., & Reid, A. E. (2006). Sexual risk reduction for persons living with HIV: Research synthesis of randomized controlled trials, 1993 to 2004. *Journal of Acquired Immune Deficiency Syndromes, 41*, 642–650.

World Health Organization. *Health topics: Sexually transmitted infections*. Retrieved 6 Jan 2011 from http://www.who.int/topics/sexually_transmitted_infections/en/

Sexually Transmitted Infections

► Sexually Transmitted Diseases (STDs)

SF-36

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Synonyms

Short form 36

Definition

The SF-36 is a 36-item self-report measure of health-related quality of life. It has eight subscales measuring different domains of health-related quality of life: physical functioning (PF), role-physical (RP), bodily pain (BP), general health (GH), vitality (VT), social functioning (SF), role-emotional (RE), and mental health (MH). Two component scores are derived from the eight subscales: a physical health component score and a mental health component score. The SF-36 also includes a single item that assesses perceived change in health status over the past year. Higher scores on all subscales represent better health and functioning. From its development to 2011, more than 16,000 articles were published using the SF-36. SF-36 is also known as the Short Form 36 Health Survey Questionnaire.

Description

Development

Ware and colleagues (Stewart and Ware 1992; Ware 1988, 1990) developed the SF-36 from the Medical Outcomes Study, a study of the health, well-being, and functioning of randomly selected patients seen by randomly selected physicians and other medical providers in three large metropolitan areas. Items were chosen for the SF-36 because they were items commonly used in other health surveys and the domains were ones that seemed to be commonly affected by differing health and disease states. After 10 years of use, the SF-36 was revised to address wording and response choice categories. The SF-36 version 2 (SF-36v2) is the current version (Ware et al. 2007).

Health Domain Scales

Physical Functioning (PF). The PF scale is a 10-item measure of physical limitation in a range of activities from vigorous exercise to performing self-care activities.

Role-Physical (RP). The RP scale contains four items and measures limitations in various roles, including work and daily activities.

Bodily Pain (BP). The BP scale has two items that measure body pain intensity and the extent to which pain interferes with daily activities.

General Health (GH). The five-item GH scale measures overall self-rated health.

Vitality (VT). The VT scale has four items that measure vitality, energy level, and fatigue and is meant to be a measure of subjective well-being.

Social Functioning (SF). The SF scale includes two items that measure the impact of physical and mental health on social functioning.

Role-Emotional (RE). The RE scale measures role limitations due to mental health difficulties with three items, including amount of time spent on work or other activities, amount of work accomplished, and the care with which work is performed.

Mental Health (MH). The MH scale has five items that measure anxiety, depression, loss of behavioral/emotional control, and psychological well-being.

Component Summary Scales

Component summaries were developed to reduce the number of scores derived from this measure from 8 to 2. They also have the advantages of having smaller confidence intervals than the health domain scales and limiting floor and ceiling effects. The Physical Component Summary (PCS) combines items from the PF, RP, BP, and GH scales, and the Mental Health Component Summary (MCS) combines items from the VT, SF, RE,

and MH scales. Each provides one score to assess physical and mental health, respectively.

Reliability

Estimates for internal consistency reliability are very good for all subscales. The two component summary scores show evidence for very high internal consistency ($\alpha = .95$ and $\alpha = .93$ for the PCS and MCS, respectively). Internal consistency estimates for the health domain scales are also high, ranging from $\alpha = .83$ (GH) to $\alpha = .95$ (RP) (Ware et al. 2007). Evidence suggests that test-retest reliability for the SF-36 over a 3-week interval is very good, with estimates of .94 and .81 for the PCS and MCS scales, respectively (Ware et al. 1995).

Validity

The SF-36 has been widely used in health research, and the user manual (Ware et al. 2007) provides a comprehensive list of studies offering evidence for the scales' construct validity. The content of the SF-36 survey was compared with other well-known health surveys to establish content validity (cf., Ware et al. 1994, for a list of references).

Options

Along with the 36-item SF-36, the shorter 12-item (SF-12) and 8-item (SF-8) versions of the SF are also available. Both shorter versions offer scores on all eight health domains and the two component summary scores. Versions 1 and 2 of both the SF-36 and the SF-12 are available for use (there is only one version of the SF-8). All forms are available in the standard 4-week recall and the acute 1-week recall versions. Additionally, the SF-8 is available in a 24-h recall version.

Administration

The survey is designed for adults 18 and over and can be given in a self-report paper/pencil format or in an interview format. The SF-36 and its other forms are available for licensure from QualityMetric Incorporated (www.qualitymetric.com).

Cross-References

▶ Health-Related Quality of Life

References and Readings

Quality Metric Incorporated. (2011). http://www.qualitymetric.com/

Stewart, A. L., & Ware, J. E., Jr. (Eds.). (1992). *Measuring functioning and well-being: The medical outcomes study approach*. Durham: Duke University Press.

Ware, J. E., Jr. (1988). *How to score the revised MOS short-form health scales*. Boston: Institute for the Improvement of Medical Care and Health, New England Medical Center.

Ware, J. E., Jr. (1990). Measuring patient function and well-being: Some lessons from the medical outcomes study. In K. A. Heitgoff & K. N. Lohr (Eds.), *Effectiveness and outcomes in health care: Proceedings of an invitational conference by the Institute of Medicine Division of Health Care Services* (pp. 107–119). Washington, DC: National Academy Press.

Ware, J. E., Jr., Kosinski, M., DeBrota, D. J., Andrejasich, C. M., & Bradt, E. W. (1995, October). *Comparison of patient responses to SF-36 Health Surveys that are self-administered, interview administered by telephone, and computer-administered by telephone*. Paper presented at the Eastern Regional Meeting of the American Federation for Clinical Research, New York, NY.

Ware, J. E., Jr., Gandek, B., & The IQOLA Project Group. (1994). The SF-36 health survey: Development and use in mental health research and the IQOLA project. *International Journal of Mental Health, 23*, 49–73.

Ware, J. E., Jr., Kosinski, M., Bjorner, J. B., Turner-Bowker, D. M., Gandek, B., & Meruish, M. E. (2007). *User's manual for the SF-36v2 health survey* (2nd ed.). Lincoln: Quality Metric Incorporated.

Short Form 36

▶ SF-36

Shortness of Breath

▶ Dyspnea

Sick Headache

▶ Migraine Headache

Sickness Absence

► Return to Work

Sickness Behavior

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Synonyms

Cytokine-induced depression; Inflammation-associated depression

Definition

Sickness behavior is a coordinated set of adaptive behavioral changes that occur in physically ill animals and humans during the course of infection. These behaviors include lethargy, depressed mood, reduced social exploration, loss of appetite, sleepiness, hyperalgesia, and, at times, confusion. This set of behaviors often accompanies fever and is considered a motivational state responsible for reorganizing an ill individual's perceptions and actions to enable better coping with infection (Dantzer et al. 2008).

Description

Sickness behavior is a normal response to infection and is characterized by endocrine, autonomic, and behavioral changes triggered by soluble proteins produced at the site of infection. Activated immune cells, such as macrophages and dendritic cells, release biochemical mediators called pro-inflammatory cytokines, such as interleukin (IL)-1, IL-6, and tumor necrosis factor (TNF)-alpha, which coordinate the local and systemic inflammatory response during active infection. These inflammatory mediators, in turn, act on the brain facilitating behavioral changes associated with sickness.

Much of the evidence supporting a link between pro-inflammatory cytokines and sickness behaviors comes from experimental studies in animals and humans. Peripheral and central administration of IL-1 and TNF-alpha in healthy laboratory animals induces fever and behavioral symptoms of sickness, including depressed activity, decreased food intake, and a curled posture. Sexual behavior is similarly reduced, particularly among females. IL-1 receptor antagonist (IL-1RA) blocks the biological effects of IL-1 when co-administered at 100- to 1000-fold excess dose with IL-1. Treatment with IL-1RA abrogates the depressing effect on social behavior but not food-motivated behavior, suggesting that IL-1 is a key mechanism in social function (Bluthe et al. 1992). Similar effects are seen when IL-1RA is injected directly into the brain. Time course studies of the behavioral effects of IL-1 in animals show changes in social exploration gradually develop within 2 h of peripheral administration whereas changes in food-motivated behavior reach a maximum by 1 h following treatment. Interestingly, IL-6 administered systemically or centrally has no behavioral effects despite inducing a fever response. That said, IL-6 does have the capacity to potentiate the effects of subthreshold dose of IL-1 administration suggesting that IL-6 may be behaviorally active only in the context of other pro-inflammatory mediators.

In humans, administration of endotoxin, a component of the outer membrane of Gram-negative bacteria, leads to systemic elevations in pro-inflammatory cytokines. This stimulus has been shown to cause participants to experience flu-like symptoms (e.g., fever, chills) as well as fatigue and depressed affect (reviewed in DellaGioia and Hannestad 2010). Moreover, a recent study demonstrated that subjects exposed experimentally to endotoxin led to increased self-reported levels of depressed mood and reduced activity in the ventral striatum in response to reward cues (Eisenberger et al. 2010), which is consistent with anhedonia.

There is substantial overlap between the behavioral components of sickness behavior and major depression in humans. As such, pro-

inflammatory cytokines are proposed to participate in the pathophysiology of depression and potentially account for the high prevalence of depression among the medically ill (Smith 1991; Raison et al. 2006; Dantzer et al. 2008). Patients treated with immune-activating medications, such as IFN-a therapy prescribed for patients suffering with Hepatitis C or certain cancers, show elevated rates of depression compared to patients undergoing alternative therapies (Raison et al. 2006). Indeed, patients undergoing IFN-a therapy tend to experience depressive symptoms coupled with anxiety and irritability over a background of neurovegetative sickness–like symptoms, including sleep disorders, fatigue, and decreased appetite. While the mood disturbances generally occur between 4 and 12 weeks of treatment, the neurovegetative symptoms occur more rapidly, within the first 2 weeks of treatment.

Significant research efforts have focused on the neurochemical effects of inflammation that underlie sickness behavior and related depressive symptoms. Experimental animal data demonstrates that pro-inflammatory cytokines enhance indoleamine 2,3 dioxygenase (IDO) that peaks 24-h after endotoxin administration. This increase in IDO leads to a decrease in tryptophan (TRP), an essential amino acid that is actively transported into the brain for the synthesis of serotonin. IDO also leads to a decrease in kynurenine (KYN) and other tryptophan-related metabolites. Animals pretreated with a potent anti-inflammatory agent that blocks pro-inflammatory cytokines in the periphery and in the brain show significant reductions in both sickness and depressive behaviors. In contrast, animals treated with an inhibitor of IDO show a reduction in depressive behaviors but not neurovegetative symptoms, providing important evidence for the role of tryptophan metabolism in cytokine-induced depression (Dantzer et al. 2008). It is anticipated that this research will have important implications for effective treatment of inflammation-related depression in humans.

Cross-References

- ▶ Depression: Symptoms
- ▶ Illness Behavior
- ▶ Inflammation
- ▶ Psychoneuroimmunology

References and Further Readings

Bluthe, R. M., Dantzer, R., & Kelley, K. W. (1992). Effects of interleukin-1 receptor antagonist on the behavioral effects of lipopolysaccharide in rat. *Brain Research, 573*, 318–320.

Dantzer, R., Bluthe, R. M., Castanon, N., Kelley, K. W., Konsman, J. P., Laye, S., et al. (2007). Cytokines, sickness behavior, and depression. In R. Ader (Ed.), *Psychoneuroimmunology* (4th ed., pp. 281–318). New York: Academic Press.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Neuroscience Reviews, 9*, 46–56.

DellaGioia, N., & Hannestad, J. (2010). A critical review of human endotoxin administration as an experimental paradigm of depression. *Neuroscience and Biobehavioral Reviews, 34*, 130–143.

Eisenberger, N. I., Berkman, E. T., Inagaki, T. K., Rameson, L. T., Mashal, N. M., & Irwin, M. R. (2010). Inflammation-induced anhedonia: Endotoxin reduces ventral striatum responses to reward. *Biological Psychiatry, 15*, 748–754.

Miller, A. H., Maletic, V., & Raison, C. L. (2009). Inflammation and its discontents: The role of cytokines in the pathophysiology of major depression. *Biological Psychiatry, 65*, 732–741.

Raison, C. L., Capuron, L., & Miller, A. H. (2006). Cytokines sing the blues: Inflammation and pathogenesis of depression. *Trends in Immunology, 27*, 24–31.

Smith, R. S. (1991). The macrophage theory of depression. *Medical Hypotheses, 35*, 298–306.

Siegrist, Johannes

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Johannes Siegrist was born in Zofingen, Switzerland, on August 6, 1943. His nationality is Swiss, and he is married to Karin and has two

daughters. Siegrist studied Sociology, Social Psychology, Philosophy, and History at the Universities of Basel (Switzerland) and Freiburg i.Br. (Germany). He received his M.A. (1967) and his Ph.D. (1969) in Sociology at the University of Freiburg. After postdoctoral training at the Universities of Ulm and Freiburg, he accomplished his habilitation in Sociology at the University of Freiburg (1973). In 1973, he was appointed as Professor of Medical Sociology at the Faculty of Medicine, University of Marburg (Germany), where he served until 1992, interrupted by Visiting Professorships at the Institute for Advanced Studies in Vienna (Austria) and at the Johns Hopkins Bloomberg School of Public Health in Baltimore, USA. In 1992, Siegrist was appointed as Professor of Medical Sociology and Director of the Department of Medical Sociology at the Faculty of Medicine, Heinrich Heine University of Dusseldorf, Germany, and as Director of the Postgraduate Training Program of Public Health at the same university. After his retirement in 2012, he was granted a Senior Professorship at this university to continue his research activities (Fig. 1).

Siegrist, Johannes, Fig. 1 Johannes Siegrist

Siegrist has been President of the International Society of Behavioral Medicine (ISBM; 1996–1998), President of the European Society of Health and Medical Sociology (1990–1992), and Director of the European Science Foundation Program on Social Variations in Health Expectancy in Europe (1999–2003). He was Chair of the Section "Behavioral Sciences" of Academia Europaea (2004–2012), member of the Expert Panel of the German Research Foundation (2006–2010), and member of a Scientific Committee and two working groups of the German Academy of Sciences Leopoldina (since 2011).

He has been a Task Group Leader to the Marmot Review (Strategic Review of Health Inequalities in England post-2010) for the British Government, with a focus on work and health. In this same function, he has coordinated and edited a report on employment and working conditions in the context of the "Review of Social Determinants of Health and the Health Divide in the WHO European Region," commissioned by the WHO European Office in 2011. Since 2015, he is a member of the Advisory Board of OECD on guidelines for measuring the quality of working environments and a lead author of the report on social progress prepared by the International Panel for Social Progress (IPSP).

Siegrist served as Associate Editor or Advisory Board Member of several international journals (e.g., *International Journal of Behavioral Medicine*, *Social Science & Medicine*, *Social Psychiatry and Psychiatric Epidemiology*, *Work & Stress*, *European Journal of Public Health*, *Scandinavian Journal of Work*, *Environment and Health*).

The awards he received include Honorary Member of the European Society of Health and Medical Sociology where he also received the Research Award, Member of Academia Europaea (London), and Corresponding Member of the Heidelberg Academy of Sciences. He received the Salomon Neumann Award of the German Society of Social Medicine and Prevention, the Hans Roemer Award of the German College of Psychosomatic Medicine, and the Belle van Zuylen Chair at the University of Utrecht, the Netherlands.

Major Accomplishments

Siegrist's major contribution to scientific research concerns the development and test of a theoretical model of an adverse psychosocial work environment, with the aim of explaining stress-related disorders, termed "effort-reward imbalance" (ERI). The model posits that failed reciprocity of effort spent and rewards received at work ("high cost-low gain") elicits strong negative emotions and psychobiological stress responses with adverse long-term effects on health. Starting from cross-sectional and longitudinal epidemiological research in the late 1970s and early 1980s, together with collaborators Ingbert Weber, Karin Siegrist, Richard Peter, and others at Marburg University, he systematically elaborated and expanded research on the ERI model in a network of national and international scientific collaboration. The questionnaire measuring the model has been incorporated in a number of epidemiological studies (e.g., Whitehall II, GAZEL, CONSTANCES, German Socioeconomic Panel, German National Cohort Study) and was successfully applied in other sociocultural contexts (e.g., Japan) and in rapidly developing societies (e.g., China, Brazil).

Evidence from prospective cohort studies indicates that continued experience of failed reciprocity in terms of the ERI model is associated with significantly elevated odds of stress-related disorders. Most robust results are available on depression and coronary heart disease. Additionally, findings point to elevated risks of alcohol dependence, type 2 diabetes, reduced health functioning, sickness absence, and disability. This epidemiological evidence was supplemented by experiments and "naturalistic" studies (e.g., ambulatory blood pressure and heart rate monitoring), where reduced immune function, enhanced autonomic activity, and altered release patterns of stress hormones were linked to ERI, often in a dose-response relationship. Siegrist was also involved in some intervention studies where measures of reducing ERI at work were followed by improvements of well-being and mental health.

Siegrist applied the ERI model to other types of contractual social exchange, e.g., voluntary work, marital, or parent-child relations. Available results support the notion that failed reciprocity in core social roles exerts negative effects on health and well-being, suggesting a basic link between perceived injustice of effortful exchange and the development of stress-related disorders in humans. Siegrist has expanded this research with a focus on retirement, volunteering, and healthy aging, together with Morten Wahrendorf and other colleagues, in the frame of the Survey of Health, Ageing and Retirement in Europe (SHARE) and additional longitudinal investigations on aging populations. As a crosscutting topic of his long-lasting research career, Siegrist has put special emphasis on explaining and reducing avoidable social inequalities in health, both as a scientist and as an advocate for different stakeholders where he has proposed evidence-based policy recommendations for improving quality of work and employment and for reducing the burden of stress-related disease.

Cross-References

- ▶ International Society of Behavioral Medicine
- ▶ Occupational Health

References and Further Reading

Siegrist, J., & Marmot, M. (Eds.). (2006). *Social inequalities in health: New evidence and policy implications*. Oxford: Oxford University Press.

Siegrist, J., & Wahrendorf, M. (Eds.). (2016). *Work stress and health in a globalized economy: The model of effort-reward imbalance*. Dordrecht: Springer.

Siegrist, J., Starke, D., Chandola, T., Godin, I., Marmot, M., Niedhammer, I., & Peter, R. (2004). The measurement of effort-reward imbalance at work. European comparisons. *Social Science & Medicine, 58*, 1483–1499.

Simulation Games

- ▶ Health Gaming

Singing and Health

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Synonyms

Choir; Choral singing; Group singing; Vocal group

Definition

Singing in health contexts typically involves participants with one or more health condition(s) gathering to sing together at weekly rehearsals and sometimes performing within a hospital or health service or in the community. Sessions commonly include a series of warm-up exercises, learning new songs as a group, and singing songs already in the singers' repertoire. Singing groups may be led by musicians, music educators, music therapists, or community musicians. Typically one or more health professionals are in attendance to support participants who require it. Some health choirs are for patients only while others feature patients and staff, or patients and carers, singing together.

Description

Examples of single condition choirs include the "Sing to Live" choir for people affected by breast cancer in Illinois; the "Brainwaves" and "Stroke a Chord" choirs in Australia for adults who have experienced a stroke; "Sing Your Lungs Out" choir in New Zealand and "Singing for Better Breathing" choirs in the UK for people who have chronic obstructive pulmonary disease; and "Remini-Sing" choir in Australia and "Singing for the Brain" in the UK for people with dementia and their carers. Other choirs include singers with a variety of diagnoses, for instance, the "Choir of Hard Knocks" in Australia comprises adults who are socially marginalized as a result of their mental illness, addiction, neurological condition, disability, or other ongoing health condition.

The purpose of health choirs is to improve the health and wellbeing of participants, rather than merely to keep patients occupied or, at the other extreme, to achieve an elite level of musical performance. These health aims may be met through the physical demands of standing, moving, and controlling the breath during regular singing rehearsals (e.g., Skingley et al. 2018) and the cognitive demands of listening, producing the correct sounds with the correct timing, learning and recalling lyrics and melodies, and coordinating movements such as swaying, tapping, and clicking while singing. The wellbeing aims may be met through the social bonding effects of group singing (e.g., Pearce et al. 2015) as well as the uplifting effect group singing has on emotions (e.g., Dingle et al. 2017). The social and emotional effects of choir singing may be particularly important for individuals affected by chronic health conditions whose ability to socialize may be adversely affected as a consequence of their health condition and its treatment. Indeed, a cross-national survey study of 1779 choristers revealed that singers perceived the following health benefits: social connection, physical and physiological benefits (specifically respiratory health), cognitive stimulation, mental health, enjoyment, and transcendence (Moss et al. 2018).

Emerging research shows that group singing is related to improved health and wellbeing among adults affected by cancers. For instance, one study examined the impact of singing on mood, stress, and immune response in three populations affected by cancer: carers ($n = 72$), bereaved carers ($n = 66$), and patients ($n = 55$). Assessments taken before and after 1 h of singing showed that singing was associated with significant reductions in negative emotions and increases in positive emotions, accompanied by significant increases in a majority of measured cytokines (substances secreted by cells as part of an immune response). Furthermore, singing was

associated with reductions in cortisol, beta-endorphin, and oxytocin levels. These positive effects of singing were found in all three groups (Fancourt et al. 2016). In a Welsh study with 816 participants, a sub-sample of 203 completed initial, 3-month and 6-month questionnaires. Over time, measures of vitality and overall mental health improved, while levels of anxiety reduced significantly in both patients and non-patients (Reagon et al. 2017).

In the area of respiratory health, a review of six studies reported that singing has the potential to improve health-related quality of life, particularly related to physical health, and levels of anxiety without causing significant side effects. Qualitative data indicate that singing is an enjoyable experience for patients, who consistently report that it helps them to cope with their condition better (Lewis et al. 2016).

In relation to cognitive health, choir singing has been found to improve measures of cognitive functioning and wellbeing among healthy older adults (e.g., Coulton et al. 2015; Dingle et al. 2018). Much research is currently focusing on the potential benefits of choir singing for people with dementias. For instance, a Finnish study recruited 89 dyads (people with early dementia and their caregivers) and randomized them to a 10-week singing coaching group, a 10-week music listening coaching group, or a usual care control group (Särkämö et al. 2013). Compared with usual care, both weekly singing and music listening improved mood, orientation, and remote episodic memory and to a lesser extent, also attention and executive function and general cognition. Singing also enhanced short-term and working memory and caregiver wellbeing, whereas music listening had a positive effect on quality of life.

Turning to mental health, a systematic review of studies of choir singing for adults experiencing mental health conditions found that the results of seven longitudinal studies showed that while people with mental health conditions participated in choir singing, their mental health and wellbeing significantly improved with moderate to large effect sizes (Williams et al. 2018). Often physical health conditions were also featured in these samples, although changes in physical health measures were less frequently reported. Moreover, six qualitative studies based on interviews with choir participants yielded converging themes, indicating that group singing can provide enjoyment, improve emotional states, develop a sense of belonging, and enhance self-confidence in participants.

Taken together, this body of research on choir singing shows that it is associated with improvements in biological measures such as improved immune functioning and decreased stress hormone levels; improved lung functioning among people with respiratory conditions; improved cognitive performance in older adults; and improved self-reported mood, mental health, and wellbeing. Often these benefits extend to family members and carers who are also affected by the health condition and its treatment.

Cross-References

▶ Music and Health

References and Further Reading

Coulton, S., Clift, S., Skingley, A., & Rodriguez, J. (2015). Effectiveness and cost-effectiveness of community singing on mental health-related quality of life of older people: Randomised controlled trial. *The British Journal of Psychiatry, 207*(3), 250–255. https://doi.org/10.1192/bjp.bp.113.129908.

Dingle, G. A., Williams, E., Jetten, J., & Welch, J. (2017). Choir singing and creative writing enhance emotion regulation in adults with chronic mental health conditions. *British Journal of Clinical Psychology, 56*(4), 443–457. https://doi.org/10.1111/bjc.12149.

Dingle, G., Ellem, R., Davidson, R., Haslam, C., Clift, S., Humby, M., Stathis, A., & Williams, E. (2018). *Live wires music program connects and aids cognitive performance of older adults*. Paper presented at the Australian Association for cognitive behaviour therapy national conference, Brisbane, 25–27 Oct 2018.

Fancourt, D., Williamon, A., Carvalho, L. A., Steptoe, A., Dow, R., & Lewis, I. (2016). Singing modulates mood, stress, cortisol, cytokine and neuropeptide activity in cancer patients and carers. *eCancer, 10*, 631. https://doi.org/10.3332/ecancer.2016.631.

Lewis, A., Cave, P., Stern, M., Welch, L., Taylor, K., Russell, J., Doyle, A., Russell, A., McKee, H., Clift, S., Bott, J., & Hopkinson, N. S. (2016). Singing

for lung health – A systematic review of the literature and consensus statement. *NPJ Primary Care Respiratory Medicine, 26*, 16080. https://doi.org/10.1038/npjpcrm.2016.80.

Moss, H., Lynch, J., & O'Donoghue, J. (2018). Exploring the perceived health benefits of singing in a choir: An international cross-sectional mixed-methods study. *Perspectives in Public Health, 138*(3), 160–168. https://doi.org/10.1177/1757913917739652.

Pearce, E., Launay, J., & Dunbar, R. I. M. (2015). The ice-breaker effect: Singing mediates fast social bonding. *Royal Society Open Science, 2*, 150221. https://doi.org/10.1098/rsos.150221.

Reagon, C., Gale, N., Dow, R., Lewis, I., & van Duersen, R. (2017). Choir singing and health status in people affected by cancer. *European Journal of Cancer Care, 26*, e12568. https://doi.org/10.1111/ecc.12568.

Särkämö, T., Tervaniemi, M., Laitinen, S., Numminen, A., Kurki, M., Johnson, J. K., & Rantanen, P. (2013). Cognitive, emotional, and social benefits of regular musical activities in early dementia: Randomized controlled study. *The Gerontologist, 54*(4), 634–650. https://doi.org/10.1093/geront/gnt100.

Skingley, A., Clift, S., Hurley, S., Price, S., et al. (2018). Community singing groups for people with chronic obstructive pulmonary disease: Participant perspectives. *Perspectives in Public Health, 133*(1), 66–75. https://doi.org/10.1177/1757913917740930.

Williams, E., Dingle, G., & Clift, S. (2018). A systematic review of mental health and wellbeing outcomes of group singing for adults with a mental health condition. *European Journal of Public Health*. https://doi.org/10.1093/eurpub/cky115. Accepted 26 May 2018.

Single Nucleotide Polymorphism (SNP)

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Synonyms

SNP (pronounced "snip")

Definition

The term "single nucleotide polymorphism" contains two defining criteria. First, it refers to a single nucleotide, i.e., an individual base pair, that can differ between individuals. Second, the word polymorphism indicates that a particular nucleotide change of interest is shared by at least 1% of the population.

SNPs occur when one base pair replaces another base pair in a point mutation (see DNA entry for discussion of bases). For example, an A-T pairing may be replaced by a G-C pairing. Such a mutation does not typically harm the organism.

Cross-References

▶ DNA
▶ Polymorphism

References and Further Reading

Britannica. (2009). *The Britannicaguide to genetics (Introduction by Steve Jones)*. Philadelphia: Running Press.

Single Subject

▶ Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale

Single-Case Experimental, or N of 1 Clinical Trials

▶ Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale

Situational Responsiveness

▶ Job Performance

Skeletal Muscle Atrophy

► Sarcopenia

Skin Cancer Prevention: Sun Protection, Sun Safety, Sunscreen Use

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Definition

Skin cancer is the most commonly diagnosed cancer in the United States, with more than one million Americans diagnosed with skin cancer each year. The incidence of skin cancer has increased dramatically worldwide in the last decade. Both main types of skin cancer – malignant melanoma and non-melanoma skin cancer (NMSC) – are now significant public health concerns. While skin cancer rates are increasing, it is considered one of the most preventable types of cancer.

The greatest risk factor for skin cancer is exposure to ultraviolet radiation, or UV radiation, which comes mainly from the sun. Behavioral recommendations for primary prevention of skin cancer include: limit time spent in the sun, avoid the sun during peak hours (10 a.m. to 4 p.m.), use sunscreen with a sun protection factor (SPF) of 15 or higher when outside, wear protective clothing (hats, shirts, pants) and sunglasses, seek shade when outdoors, and avoid sunburn. These behaviors, if consistently practiced, can help prevent all forms of skin cancer. There is some concern that using sunscreen will lead people who are trying to get a suntan to stay in the sun for a longer time, so another recommendation for prevention is not to intentionally bake in the sun or seek a tan.

Additional, important recommendations for behaviors to prevent skin cancer and related morbidity and mortality include performing regular skin self-examination and seeking professional evaluation of suspicious skin changes. Further, avoidance of indoor tanning and the use of tanning salons and tanning beds (also called "solaria") are strongly recommended.

An understanding of patterns of behavior can help to guide efforts to prevent skin cancer. More people take precautions at the beach or on vacation than when taking outdoor recreation. Parents are more likely to protect their children than themselves. Children are more often protected from UV radiation if their parents also protect themselves. Adolescents seem especially resistant to advice about skin cancer prevention and minimizing sun exposure.

Most skin cancer prevention interventions reported in the literature are directed at the general population through school-based curricula, multicomponent community programs, or media campaigns, and some recent trials have targeted people with high sun exposure at work or during outdoor recreation. Children, adolescents, and adults at high risk are important audiences for skin cancer prevention.

This chapter provides an overview of skin cancer prevention for the general population and groups at increased risk due to genetic or environmental exposures. The reference sources include evidence reviews, key research articles reporting on well-designed studies, and works addressing issues in measurement and methodology for skin cancer prevention research and evaluation.

Description

Evidence Reviews

An extensive evidence review of strategies to prevent skin cancer was undertaken by the Task Force on Community Preventive Services, and the results and recommendations were published in Saraiya et al. (2004). This report presents the results of systematic reviews of the effectiveness of interventions to prevent skin cancer by reducing exposure to ultraviolet radiation (UVR). The Task Force on Community Preventive Services

found that education and policy approaches were effective when implemented in primary schools and in recreational or tourism settings but found insufficient evidence to determine effectiveness in other settings. This evidence review is currently being updated to reflect the continuing growth of the scientific literature on behavioral interventions to prevent skin cancer during the past decade.

Comprehensive Community Programs Including Mass Media

There is a long history of comprehensive, multicomponent community skin cancer prevention programs, especially in Australia, where skin cancer is highly prevalent. These programs include mass media and communication campaigns as an integral part of these community programs.

Two related sun protection programs have been conducted in Australia for more than 20 years: Slip! Slop! Slap! from 1980 to 1988 and SunSmart from 1988 to the present (Montague et al. 2001). These programs have played an important role in changing the whole society's approach to the sun and have resulted in marked reductions in sun exposure. An examination of trends in behavioral risk factors for skin cancer over 15 years was examined in an Australian population exposed to the SunSmart program including SunSmart television advertising. Higher exposure to SunSmart advertising in the weeks before the interview increased preferences for not tanning, hat and sunscreen use, and greater clothing protection. These results indicate that sustained multicomponent programs with media campaigns can both prompt and reinforce skin cancer preventive behaviors.

Interventions in Schools

The most often studied settings for skin cancer prevention programs are schools, and there is good evidence that educational and policy interventions can be effective in primary schools (Saraiya et al. 2004). Of the many reported studies, a few are particularly well designed, carefully described, have long follow-up periods, and/or use objective outcome measures. The Kidskin intervention trial in Western Australia is particularly noteworthy and had a 6-year follow-up period. The "Kidskin" study involved three groups: control, "moderate," and "high" intervention. Results showed that children in the intervention groups – especially the "high" group – reported less sun exposure and spent less time outdoors in the middle of the day. There was little difference between groups in the wearing of hats or sunscreen (Milne et al. 2001). Children in the intervention groups – especially the high group – were less tanned at the end of the summer; this effect was greater for the back than for the forearms. There was also a smaller increase in the number of nevi (or moles) on the backs of children in the intervention groups (English et al. 2005). Further, the program had a positive effect on hat wearing on the playground, especially in the "high" intervention groups, but did not change children's use of shade at lunchtime (Giles-Corti et al. 2004).

Outdoor Workers

Outdoor workers are at high risk for skin cancer because they receive regular and significant solar UVR exposure. In a well-designed study to reduce UVR exposure among ski instructors, greater program implementation was associated with less sunburn. In an intervention for US Postal Service workers, regular sunscreen and hat use were higher among the intervention group than among the control group after 3 months and at 3-year follow-up (Mayer et al. 2007, 2009).

Recreation Settings

Intense and prolonged sun exposure often occurs during outdoor recreation activities. High UVR exposure, often with minimal clothing, tends to occur at beach and swimming pool settings. Other outdoor recreation settings include camps, zoos, and parks. Large and well-designed studies of skin cancer prevention in these locations have been reported. Effective skin cancer prevention programs for children have been evaluated in swimming pool settings (Glanz et al. 2002; Glanz et al. 2005) and for beachgoers at Northeastern (Weinstock et al. 2002) and Midwestern (Pagoto et al. 2003) beaches as well as at zoos (Mayer et al. 2001).

High-Risk Groups

Targeting skin cancer prevention to people at high risk may result in greater effects of preventive strategies and an efficient public health strategy. Risk factors for skin cancer include age, sun-sensitive phenotypes, excess sun exposure, family history, personal history of skin cancer or precancerous lesions, and some other medical conditions. There is a need to develop low-cost, effective interventions to improve skin cancer prevention and early detection behaviors among a broader population of persons at moderate and high risk. (Geller et al. 2006) and Glanz, Schoenfeld, and Steffen (2010) describe studies of effective tailored interventions that specifically target individuals at high risk, either siblings of melanoma patients or adults determined to be at moderate or high risk for skin cancer. These studies focused on both prevention and skin examinations. A study of a group of people who have tested positive in genetic testing for skin cancer-related mutations and found that positive genetic test results led to greater intentions to obtain total body skin examinations and adhere to skin self-examination recommendations (Aspinwall et al. 2008).

Screening and Early Detection

Screening for skin cancer through health-care-provider skin exams and skin self-examination has the potential to help detect skin cancers at an earlier stage (i.e., when they are thinner) so that they are more curable and less serious. Although there has not been a large randomized trial of skin screening in the United States, an Australian trial reported by Aitken et al. (2006) provides promising evidence of the impact of skin screening and how it can be successfully implemented. A randomized trial was conducted to determine whether a multicomponent intervention can increase total skin self-examination (TSSE) performance. Participants received instructional materials, a video, and a brief counseling session and a brief follow-up phone call and tailored feedback letters. Results showed that the intervention group increased TSSE performance in the intervention group compared to the control group (Weinstock et al. 2007). A follow-up article aimed to identify the most important Check-It-Out intervention components for promoting TSSE. Results showed that watching the video, using the hand mirror, shower card, American Cancer Society brochure, sample photographs, and finding the health educator helpful were associated with performing TSSE at 2 months, 12 months, or both. The studies of high-risk groups reported by Geller et al. (2006) and Glanz et al. (2010) also targeted behavioral outcomes of skin self-examination and thorough examination of all moles.

Measurement and Methodology

Advances in skin cancer prevention depend on good quality research and ideally different intervention studies that can be compared to understand the impact of various strategies. Most skin cancer prevention studies uses verbal reports, or self-report, to measure habitual sun exposure and solar protection behaviors. Despite the well-known limitations of verbal reports of behavior, these measures are the most practical for use in both population surveillance and descriptive and intervention research (Glanz and Mayer 2005). Therefore, the comparability of assessments across population-based surveys and outcome measures used in intervention research is important, and a core set of measures was recently published by a diverse group of investigators in the field (Glanz et al. 2008). In addition, because it is important to continue to build a research tool kit for measures other than surveys, including objective biological measures and observational measures, recent research to validate self-reports of sunscreen use (Glanz et al. 2009) and other behaviors is of particular importance to the field.

Conclusion

Skin cancer prevention interventions have demonstrated modest success, with the majority of programs being conducted in school settings. It is believed that the ideal intervention strategies for reducing exposure to ultraviolet radiation (UVR) exposure are coordinated, sustained,

community-wide approaches that combine education, mass media, and environmental and structural changes. Interventions within specific organizational settings such as schools, health care, recreation programs, and workplaces provide useful ways to reach important audiences like children and are suitable venues for structural supports such as environmental and policy change that complement educational efforts. It is generally agreed that environmental and structural changes also need to be part of successful skin cancer prevention efforts. Advances in measurement and methods in skin cancer prevention research will contribute to future efforts to address this important and widespread health and behavior problem.

Cross-References

► Cancer Prevention

References and Readings

Aitken, J. F., Janda, M., Elwood, M., Youl, P. H., Ring, I. T., & Lowe, J. B. (2006). Clinical outcomes from skin screening clinics within a community-based melanoma screening program. *Journal of the American Academy of Dermatology, 54*, 105–114.

Aspinwall, L. G., Leaf, S. L., Dola, E. R., Kohlmann, W., & Leachman, S. A. (2008). CDKN2A/p16 genetic test reporting improves early detection intentions and practices in high-risk melanoma families. *Cancer Epidemiology, Biomarkers & Prevention, 17*, 1510–1519.

English, D. R., Milne, E., Jacoby, P., Giles-Corti, B., Cross, D., & Johnston, R. (2005). The effect of a school-based sun protection intervention on the development of melanocytic nevi in children: 6-year follow-up. *Cancer Epidemiology, Biomarkers & Prevention, 14*, 977–980.

Geller, A. C., Emmons, K. M., Brooks, D. R., Powers, C., Zhang, Z., Koh, H. K., Heeren, T., Sober, A. J., Li, F., & Gilchrest, B. A. (2006). A randomized trial to improve early detection and prevention practices among siblings of melanoma patients. *Cancer, 107*, 806–814.

Giles-Corti, B., English, D., Costa, C., Milne, E., Cross, D., & Johnston, R. (2004). Creating SunSmart schools. *Health Education Research, 19*, 98–109.

Glanz, K., & Mayer, J. A. (2005). Reducing UVR exposure to prevent skin cancer: Methodology and measurement. *American Journal of Preventive Medicine, 29*, 131–142.

Glanz, K., Geller, A. C., Shigaki, D., Maddock, J., & Isnec, M. R. (2002). A randomized trial of skin cancer prevention in aquatic settings: The Pool Cool program. *Health Psychology, 21*, 579–587.

Glanz, K., Steffen, A., Elliott, T., & O'Riordan, D. (2005). Diffusion of an effective skin cancer prevention program: Design, theoretical foundations, and first-year implementation. *Health Psychology, 24*, 477–487.

Glanz, K., Yaroch, A. L., Dancel, M., Saraiya, M., Crane, L. A., Buller, D. B., et al. (2008). Measures of sun exposure and sun protection practices for behavioral and epidemiologic research. *Archives of Dermatology, 144*, 217–222.

Glanz, K., McCarty, F., Nehl, E. J., O'Riordan, D. L., Gies, P., Bundy, L., et al. (2009). Validity of self-reported sunscreen use by parents, children and lifeguards. *American Journal of Preventive Medicine, 36*, 63–69.

Glanz, K., Schoenfeld, E. R., & Steffen, A. (2010). Randomized trial of tailored skin cancer prevention messages for adults: Project SCAPE. *American Journal of Public Health, 100*, 735–741.

Mayer, J. A., Lewis, E. C., Eckhardt, L., Slymen, D., Belch, G., Elder, J., et al. (2001). Promoting sun safety among zoo visitors. *Preventive Medicine, 33*, 162–169.

Mayer, J. A., Slymen, D. J., Clapp, E. J., Pichon, L. C., Eckhardt, L., Eichenfield, L. F., et al. (2007). Promoting sun safety among US postal service letter carriers: Impact of a 2-year intervention. *American Journal of Public Health, 97*, 559–565.

Mayer, J. A., Slymen, D. J., Clapp, E. J., Pichon, L. C., Elder, J. P., Sallis, J. F., et al. (2009). Long-term maintenance of a successful occupational sun safety intervention. *Archives of Dermatology, 145*, 88–89.

Milne, E., English, D. R., Johnston, R., Cross, D., Borland, R., Giles-Corti, B., et al. (2001). Reduced sun exposure and tanning in children after 2 years of a school-based intervention (Australia). *Cancer Causes and Control, 12*, 387–393.

Montague, M., Borland, R., & Sinclair, C. (2001). Slip! Slop! Slap! and SunSmart, 1980-2000: Skin cancer control and 20 years of population-based campaigning. *Health Education & Behavior, 28*, 290–305.

Pagoto, S., McChargue, D., & Fuqua, R. (2003). Effects of a multicomponent intervention on motivation and sun protection behaviors among midwestern beachgoers. *Health Psychology, 22*, 429–433.

Saraiya, M., Glanz, K., Briss, P. A., Nichols, P., White, C., Das, D., et al. (2004). Interventions to prevent skin cancer by reducing exposure to ultraviolet radiation – a systematic review. *American Journal of Preventive Medicine, 27*, 422–466.

Weinstock, M. A., Rossi, J. S., Redding, C. A., & Maddock, J. E. (2002). Randomized controlled community trial of the efficacy of a multicomponent stage-matched intervention to increase sun protection among beachgoers. *Preventive Medicine, 35*, 584–592.

Weinstock, M. A., Risica, P. M., Martin, R. A., Rakowski, W., Dubé, C., Berwick, M., et al. (2007). Melanoma early detection with thorough skin self-examination: The "check it out" randomized trial. *American Journal of Preventive Medicine, 32*, 517–524.

SLC6A4 (Solute Carrier Family 6, Member 4)

▶ Serotonin Transporter Gene

Sleep

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Definition

Sleep is a complex reversible neurobiological state characterized by closed eyes, behavioral quiescence, and perceptual disengagement from one's surroundings.

Description

Healthy adults cycle between two types of sleep during the typical nocturnal sleep period: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. When healthy adults fall asleep, they enter NREM sleep and usually move from lighter stages of sleep (e.g., Stages N1 and N2) to deeper sleep (e.g., Stage N3) before entering their first REM sleep period. The terms "light" and "deep" sleep refer to the ease with which one can be awakened from sleep and become fully oriented to one's surroundings. The descent from light into deep NREM sleep is characterized by decreasing inputs from external stimuli, a slowing of catabolic processes, and an increase in parasympathetic nervous system activity. In contrast, REM sleep is characterized by autonomic instability and active mental activity. In healthy adults, individual NREM-REM cycles generally last approximately 90 min, although the duration of sleep cycles varies across individuals. During the first third of the night, NREM sleep is more prevalent, whereas REM sleep becomes more prevalent during the last third of the night.

Sleep can be characterized along multiple dimensions. Here we focus on four dimensions of sleep that have been most widely evaluated in relation to health and functioning; these include sleep *duration*, *continuity*, *architecture*, and *quality*. It is important to recognize that each of these dimensions of sleep changes across the life span, from infancy through old age and may, additionally, be moderated by sex, race/ethnicity, and mental and physical health conditions (Carrier et al. 2001; Carskadon and Dement 2005; Hall et al. 2009; Ohayon et al. 2004).

Sleep Duration

The two most commonly assessed indices of sleep duration include "time in bed" and "total sleep time." Operationally, time in bed (TIB) may be defined as total hours elapsed between getting into bed to go to sleep at night ("good night time") and waking up in the morning ("good morning time"). Total sleep time (TST) may be operationalized as time in bed minus the amount of time needed to fall asleep ("sleep latency") and amount of time spent awake during the night ("wakefulness after sleep onset").

Sleep duration is one of the most widely studied dimensions of sleep in relation to health and functioning (see entry on ▶ "Sleep Duration, ▶ Sleep and Health"). For the most part, this literature has documented robust associations among sleep duration extremes (generally, <6 h or >8 h) and indices of morbidity and mortality. One meta-analysis of 23 studies reported pooled relative risk (RR) values of 1.10 (95% CI = 1.06–1.15) and 1.23 (95% CI = 1.17–1.30) for all-cause mortality and short and long sleep duration, respectively (Gallicchio and Kalesan 2009).

It must be noted, however, that studies using objective measures (actigraphy, PSG) of sleep duration and health outcomes are lacking. This issue is especially important given discrepancies between self-reported and objective indices of sleep duration, which may be confounded by other risk factors for morbidity and mortality such as age, sex, race, BMI, and comorbidities. Nor do measures of sleep duration differentiate

between individuals with or without primary sleep disorders such as sleep apnea and insomnia, which have been widely linked to health and functioning (Boivin 2000; Somers 2005).

Sleep Continuity

Measures of sleep continuity focus on one's ability to initiate and maintain sleep (see ▶ "Sleep Continuity, ▶ Sleep Fragmentation" entries). *Sleep latency* refers to the amount of time it takes to fall asleep (e.g., minutes from "good night time" to onset of sleep), whereas *wakefulness after sleep onset* (WASO) refers to the total amount of wakefulness during the sleep period (e.g., minutes of wakefulness between sleep onset and "good morning time"). *Sleep efficiency* is a proportional sleep continuity measure which refers to the percentage of time in bed spent asleep. Although operational definitions may differ across laboratories, sleep efficiency is commonly calculated as follows: (time spent asleep/time in bed) × 100.

Compared to sleep duration, fewer population-based studies have evaluated relationships among sleep continuity and indices of health and functioning. Several studies have linked self-reported sleep continuity disturbances with incident Type2 diabetes and cardiovascular disease (as reviewed by Mezick et al. under review). Although few in number, other studies have reported significant cross-sectional associations between objectively assessed sleep continuity disturbances and health outcomes including obesity, increased blood pressure, increased inflammation, decreased circulating natural killer cell numbers, and the metabolic syndrome (Hall et al. 1998; Knutson et al. 2009; Mills et al. 2007). In their longitudinal study of sleep and all-cause mortality in healthy older adults, Dew and colleagues reported that participants with PSG-assessed sleep latencies of greater than 30 min were at 2.14 times greater risk of death (95% CI = 1.25–3.6) compared to those who fell asleep in less than 30 min, after adjusting for age, medical burden, and other relevant covariates (Dew et al. 2003).

Emerging evidence based on experimental models of sleep fragmentation suggests that endocrine, immune, metabolic, and autonomic mechanisms may be important pathways through which sleep continuity disturbances influence health and functioning (Bonnet and Arand 2003; Janackova and Sforza 2008; Redwine et al. 2003; Tartar et al. 2009). In terms of its relevance to behavioral medicine and health, sleep continuity appears to be exquisitely sensitive to psychological and social factors such as stress, loneliness, relationship quality, and socioeconomic status Akerstedt et al. 2002; Cacioppo et al. 2002; Cartwright and Wood 1991; Friedman et al. 2005; Hall et al. 2008).

Sleep Architecture

Sleep architecture refers to the pattern or distribution of visually scored NREM and REM sleep stages as well as quantitative measures derived from power spectral analysis of the EEG (see "▶ Sleep Architecture" entry). Within NREM sleep, measures of sleep architecture include stages N1-N3. Lighter stages of sleep are characterized by low-amplitude, fast-frequency EEG activity whereas deeper stages of sleep are characterized by high-amplitude, low-frequency EEG activity generated by rhythmic oscillations of thalamic and cortical neurons (see Jones 2005).

Patients with medical disorders including cardiovascular and kidney disease, diabetes, and cancer exhibit lighter sleep architecture profiles compared to healthy individuals (e.g., Jauch-Chara et al. 2008; Ranjbaran et al. 2007). Yet, these studies do not indicate whether sleep architecture profiles were a contributing cause or consequence of disease. Both possibilities are plausible given experimental evidence of bidirectional relationships among components of sleep architecture and physiological processes important to health and functioning including metabolic, endocrine, autonomic, and immune mechanisms (e.g., Hall et al. 2004; Opp 2006; Rasch et al. 2007). The longitudinal study of sleep and mortality by Dew et al. (2003) is the only published study, to date, that has evaluated

relationships among measures of sleep architecture and indices of morbidity or mortality. In this study, risk for mortality was significantly higher in individuals with extreme amounts of REM sleep (upper and lower 15th percentile of the sample distribution); the visually scored slow-wave sleep percentage was also modestly associated with survival time.

Experimental manipulation of sleep architecture, although technically complex, may be an especially promising approach to disentangling cause and effect and evaluating cellular and molecular mechanisms through which sleep architecture affects and is affected by health. Quantitative analysis of the EEG, which shows trait-like characteristics, may hold promise for identifying sleep phenotypes that confer vulnerability to or resilience against disease (e.g., Tucker et al. 2007). This latter point may be especially relevant to behavioral medicine models of disease given that decreased slow-wave sleep and increased EEG spectral power in the fast-frequency beta band have been linked with symptoms of stress and a variety of chronic stressors including job strain, marital dissolution, and bereavement (Cartwright and Wood 1991; Hall et al. 1997; Kecklund and Akerstedt 2004).

Sleep Quality

Sleep quality generally refers to subjective perceptions about one's sleep. The Pittsburgh Sleep Quality Index (PSQI), which is the most widely used self-report sleep instrument and has been translated into over 30 languages, is an example of a "multiple-indicator" measure of sleep quality (Buysse et al. 1989). The PSQI includes 18 retrospective questions about one's sleep over the past month. These questions are used to derive seven subscales (sleep duration, sleep latency, sleep efficiency, sleep disturbance, daytime dysfunction, use of medications for sleep, and overall sleep quality), each of which has a range of 0–3. These subscales may be summed to generate a global measure of subjective sleep quality with a range of 0–21; higher values reflect greater subjective sleep complaints.

In a community-based study of midlife adults without clinical cardiovascular disease, Jennings and colleagues reported that higher PSQI-assessed sleep quality complaints were associated with increased prevalence of the metabolic syndrome (Jennings et al. 2007). Other cross-sectional studies have reported greater subjective sleep quality complaints in patients with hypertension, diabetes, kidney disease, polycystic ovary syndrome, and cancer compared to age- and sex-matched healthy controls (e.g., Liu et al. 2009; Sabbatini et al. 2008; Tasali et al. 2006). Subjective sleep quality complaints may be a consequence of disease. It may also indirectly impact health via health behavior pathways. For instance, subjective perceptions that one's sleep is not sound or restorative may lead to increased daytime caffeine use and increased use of alcohol prior to sleep which, in turn, may negatively impact health and functioning.

Summary

Converging evidence suggests numerous links between specific dimensions of sleep and important indices of health and functioning. The two most prevalent sleep disorders, insomnia and sleep apnea, too have been prospectively linked to adverse health outcomes (see entries for "► Obstructive Sleep Apnea"). Yet, little is understood about *how* specific dimensions of sleep or sleep disorders may confer vulnerability or resilience to disease. Identification of the cellular and molecular pathways through which sleep affects and is affected by health is critical to advancing our understanding of the sleep-health relationship in the context of behavioral medicine.

Cross-References

► Coffee Drinking, Effects of Caffeine
► Sleep Architecture
► Sleep Continuity
► Sleep Duration
► Sleep Quality

References and Further Readings

Akerstedt, T., Knutsson, A., Westerholm, P., Theorell, T., Alfredsson, L., Kecklund, G., et al. (2002). Sleep disturbances, work stress and work hours: A cross-sectional study. *Journal of Psychosomatic Research, 53*, 741–748.

Boivin, D. B. (2000). Influence of sleep-wake and circadian rhythm disturbances in psychiatric disorders. *Journal of Psychiatry & Neuroscience, 25*, 446–458.

Bonnet, M. H., & Arand, D. L. (2003). Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep Medicine Reviews, 7*, 297–310.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213.

Cacioppo, J. T., Hawkley, L. C., Berntson, G. G., Ernst, J. M., Gibbs, A. C., Stickgold, R., et al. (2002). Do lonely days invade the nights? Potential social modulation of sleep efficiency. *Psychological Science, 13*, 384–387.

Carrier, J., Land, S., Buysse, D. J., Kupfer, D. J., & Monk, T. H. (2001). The effects of age and gender on sleep EEG power spectral density in the middle years of life (aged 20–60 years old). *Psychophysiology, 38*, 232–242.

Carskadon, M. A., & Dement, W. C. (2005). Normal human sleep: an overview. In M. H. Kryger, T. Roth, & T. Dement (Eds.), *Principles and practice of sleep medicine* (pp. 13–23). Philadelphia: Elsevier/Saunders.

Cartwright, R. D., & Wood, E. (1991). Adjustment disorders of sleep: The sleep effects of a major stressful event and its resolution. *Psychiatry Research, 39*, 199–209.

Dew, M. A., Hoch, C. C., Buysse, D. J., Monk, T. H., Begley, A. E., Houck, P. R., et al. (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. *Psychosomatic Medicine, 65*, 63–73.

Friedman, E. M., Hayney, M. S., Love, G. D., Urry, H. L., Rosenkranz, M. A., Davidson, R. J., et al. (2005). Social relationships, sleep quality, and interleukin-6 in aging women. *Proceedings of the National Academy of Sciences U S A, 102*, 18757–18762.

Gallicchio, L., & Kalesan, B. (2009). Sleep duration and mortality: A systematic review and meta-analysis. *Journal of Sleep Research, 18*, 148–158.

Hall, M., Buysse, D. J., Dew, M. A., Prigerson, H. G., Kupfer, D. J., & Reynolds, C. F. (1997). Intrusive thoughts and avoidance behaviors are associated with sleep disturbances in bereavement-related depression. *Depression and Anxiety, 6*, 106–112.

Hall, M., Baum, A., Buysse, D. J., Prigerson, H. G., Kupfer, D. J., Reynolds, C. F., et al. (1998). Sleep as a mediator of the stress-immune relationship. *Psychosomatic Medicine, 60*, 48–51.

Hall, M., Vasko, R., Buysse, D. J., Ombao, H., Chen, Q., Cashmere, J. D., et al. (2004). Acute stress affects heart rate variability during sleep. *Psychosomatic Medicine, 66*, 56–62.

Hall, M., Buysse, D. J., Nofzinger, E. A., Reynolds, C. F., & Monk, T. H. (2008). Financial strain is a significant correlate of sleep continuity disturbances in late-life. *Biological Psychology, 77*, 217–222.

Hall, M., Matthews, K. A., Kravitz, H. K., Gold, E. B., Buysse, D. J., Bromberger, J. T., et al. (2009). Race and financial strain are independent correlates of sleep in mid-life women: The SWAN sleep study. *Sleep, 32*, 73–82.

Janackova, S., & Sforza, E. (2008). Neurobiology of sleep fragmentation: Cortical and autonomic markers of sleep disorders. *Current Pharmaceutical Design, 14*, 3474–3480.

Jauch-Chara, K., Schmid, S. M., Hallschmid, M., Born, J., & Schultes, B. (2008). Altered neuroendocrine sleep architecture in patients with type 1 diabetes. *Diabetes Care, 31*, 1183–1188.

Jennings, J. R., Muldoon, M., Hall, M., Buysse, D. J., & Manuck, S. B. (2007). Self-reported sleep quality is associated with the metabolic syndrome. *Sleep, 30*, 219–223.

Jones, B. E. (2005). Basic mechanisms of sleep-wake states. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (pp. 136–153). Philadelphia: Elsevier/Saunders.

Kecklund, G., & Akerstedt, T. (2004). Apprehension of the subsequent working day is associated with a low amount of slow wave sleep. *Biological Psychology, 66*, 169–176.

Knutson, K. L., Van Cauter, E., Rathouz, P. J., Yan, L. L., Hulley, S. B., Liu, K., et al. (2009). Association between sleep and blood pressure in midlife: The CARDIA sleep study. *Archives of Internal Medicine, 169*, 1055–1061.

Liu, L., Fiorentino, L., Natarajan, L., Parker, B. A., Mills, P. J., Sadler, G. R., et al. (2009). Pre-treatment symptom cluster in breast cancer patients is associated with worse sleep, fatigue and depression during chemotherapy. *Psycho-Oncology, 18*, 187–194.

Mills, P. J., von Kanel, R., Norman, D., Natarajan, L., Ziegler, M. G., Dimsdale, J. E., et al. (2007). Inflammation and sleep in healthy individuals. *Sleep, 30*, 729–735.

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.

Opp, M. R. (2006). Sleep and psychoneuroimmunology. *Neurologic Clinics, 24*, 493–506.

Ranjbaran, Z., Keefer, L., Stepanski, E., Farhadi, A., & Keshavarzian, A. (2007). The relevance of sleep abnormalities to chronic inflammatory conditions. *Inflammation Research, 56*, 1–7.

Rasch, B., Dodt, C., Moelle, M., & Born, J. (2007). Sleep-stage-specific regulation of plasma catecholamine concentration. *Psychoneuroendocrinology, 32*, 884–891.

Redwine, L., Dang, J., Hall, M., & Irwin, M. (2003). Disordered sleep, nocturnal cytokines, and immunity in alcoholics. *Psychosomatic Medicine, 65*, 75–85.
Sabbatini, M., Pisani, A., Crispo, A., Nappi, R., Gallo, R., Cianciaruso, B., et al. (2008). Renal transplantation and sleep: A new life is not enough. *Journal of Nephrology, 21*(Suppl 13), S97–S101.
Somers, V. K. (2005). Sleep: A new cardiovascular frontier. *The New England Journal of Medicine, 353*, 2070–2073.
Tartar, J. L., Ward, C. P., Cordeira, J. W., Legare, S. L., Blanchette, A. J., McCarley, R. W., & Strecker, R. E. (2009). Experimental sleep fragmentation and sleep deprivation in rats increases exploration in an open field test of anxiety while increasing plasma corticosterone levels. *Behavioural Brain Research, 197*, 450–453.
Tasali, E., Van Cauter, E., & Ehrmann, D. A. (2006). Relationships between sleep disordered breathing and glucose metabolism in polycystic ovary syndrome. *The Journal of Clinical Endocrinology and Metabolism, 91*, 36–42.
Tucker, A. M., Dinges, D. F., & Van Dongen, H. P. (2007). Trait interindividual differences in the sleep physiology of healthy young adults. *Journal of Sleep Research, 16*, 170–180.

Sleep and Health

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Synonyms

Sleep deprivation

Definition

Sleep is defined as a reversible state of perceptual disengagement and unresponsiveness to the external environment. Sleep is a complex physiological and behavioral process that is part of every individual's life and a critical determinant of physical and mental health.

Description

It is generally accepted that 7–8 h is the optimal amount of sleep needed per night for adequate daytime functioning and to reduce the risk of developing serious medical conditions. However, many Americans sleep less than 7 h per night and many report sleep difficulties. The percentage of men and women reporting sleeping less than 6 h per night has increased significantly over the last 20 years. Broad societal changes, including longer work hours, shift work, later night life, increased dependence on technology, and a current mindset of "if you snooze, you lose" have contributed to the increases in sleep loss among adults. Sleep loss increases the risk and incidence of diseases that may ultimately result in death. Many of the studies examining sleep duration and adverse health outcomes have found a U-shaped relationship suggesting that too little sleep and too much sleep is detrimental to health. And, between 7 and 8 h of sleep appears to be associated with reduced health risk.

Sleep and Mortality

Growing evidence over the last few decades suggests that progressively shorter (<7 h per night) or longer (>8 h per night) sleep duration is associated with a greater risk of mortality (Cappuccio et al. 2010; Kripke et al. 2002). The mechanisms that underlie these associations are not fully understood. Potential adverse physiologic effects of short sleep duration may contribute to negative health outcomes like cardiovascular disease, diabetes, and obesity, all of which are associated with increased mortality risk. Sleep restriction has been shown to impair glucose tolerance, increase evening cortisol levels, alter sympathetic nervous system activity, reduce leptin levels and increase levels of ghrelin, and increase inflammatory markers. The mechanisms linking long sleep duration with mortality is unknown and may be explained by underlying confounders such as depression, low socioeconomic status, undiagnosed

medical disease, poor physical health, and less physical activity.

Sleep and Cardiovascular Disease

Sleeping less than 7 h per night has been found to increase the risk of developing high blood pressure (i.e., hypertension) and elevate blood pressure in those with existing hypertension (Calhoun and Harding 2010). Long sleep duration (≥9 h per night) may also increase the risk of hypertension. The effect of insufficient sleep on blood pressure may help to explain the relationship between poor sleep and cardiovascular disease and stroke. Researchers have found both short (<7 h per night) and long (>8 h per night) sleep durations are associated with a greater risk of developing or dying from coronary heart disease and stroke (Cappuccio et al. 2011).

There is also growing evidence of a connection between ▶ "Sleep Apnea" and cardiovascular disease. People with sleep apnea have frequent awakenings at night as a result of repetitive pauses in breathing. This sleep fragmentation and reoccurring drops in oxygen levels in the blood called hypoxemia cause increases in blood pressure during the night that can persist during the daytime and over time can lead to hypertension. People with sleep apnea have an increased risk of developing coronary heart disease, stroke, and heart failure. In a 10-year study, severe sleep apnea was associated with an increased risk of fatal and nonfatal cardiovascular events.

Sleep and Obesity

Numerous studies have reported a link between sleep duration and obesity. For example, researchers have shown that people who sleep less than 6 h per night or more than 8 h per night are more likely to have a higher body mass index and that people who sleep 8 h have the lowest BMI (Cappuccio et al. 2008). Several pathways have been identified that could mediate the relationship between short sleep and increased risk of obesity: alterations in glucose metabolism, appetite control, and energy expenditure. Sleep loss impacts hormones that regulate glucose processing and appetite (Taheri et al. 2004). After sleep loss, the body's tissues are less responsive to insulin (i.e., insulin resistance), a hormone secreted by the pancreas that regulates the level of glucose in the body. As a result, glucose levels in the blood remain high, making it more difficult for the body to use stored fat for energy. Sleep loss also impacts hormones involved in appetite regulation causing people to eat even when they have had an adequate number of calories. Specifically, short sleep lowers levels of leptin, a "full signal" hormone, and increases levels of ghrelin, an appetite stimulant hormone. One night of sleep loss can decrease energy expenditure (i.e., calories burned) during rest.

Sleep and Diabetes

Numerous epidemiological studies have found that short (≤6 h per night) and long (≥9 h per night) sleep durations are associated with an increased risk of developing type 2 diabetes and impaired glucose tolerance, a precursor to diabetes (Knutson and Van Cauter 2008). Insulin resistance associated with sleep loss can over time compromise the ability of β-cells in the pancreas to release insulin, causing higher than normal levels of glucose in the blood which can lead to type 2 diabetes. In a study of healthy adults, restricting sleep to 4 h per night for six nights led to impaired glucose tolerance.

In addition, sleep apnea is a risk factor for developing type 2 diabetes. Several potential mechanisms explaining how sleep apnea may alter glucose metabolism have been proposed. Sleep fragmentation and hypoxemia associated with sleep apnea can alter autonomic and neuroendocrine function, increase release of inflammatory cytokines, and induce adipokines and thus play a role in altering glucose metabolism.

Sleep and Cancer

Emerging evidence suggests that sleep duration may increase risks of several types of cancer. The first investigation of the association between sleep and breast cancer found women with longer sleep durations (≥9 h per night) had a decreased risk of breast cancer. Subsequent studies have found mixed results with some studies finding an inverse relationship between short sleep duration and risk of breast cancer and one study found no association. Short sleep duration was associated with an increased risk of colorectal cancer in patients undergoing colonoscopy screening and an increased risk of prostate cancer. Epidemiological studies have reported a significantly increased risk of developing a number of malignancies including breast, colon, prostate, and endometrial cancer in night-shift workers. Nocturnal melatonin suppression due to decreased sleep duration or light exposure at night in the case of shift workers may alter the oncostic action of melatonin (Blask 2009).

Sleep and the Immune System

The relationship between sleep and immunity is reciprocal such that infections increase sleep duration and sleep deprivation negatively impacts immune function. Experimental studies have shown that sleep deprivation results in suppression in natural killer cell activity, reductions in interleukin-2 production, increased circulation of pro-inflammatory cytokines such as IL-6, tumor necrosis factor (TNF) α, and reduction of anti-inflammatory cytokines (Bryant et al. 2004). Sleep is needed for optimal resistance to infection. Sleep restriction in healthy adults has been shown to reduce antibody production response to the influenza vaccination. One night of sleep deprivation reduced the formation of specific antibodies to hepatitis A antigens after vaccination. Short sleep duration in the weeks preceding exposure to rhinovirus increased the susceptibility to developing a cold.

Sleep and Neurocognitive Function

It has been established that sleep deprivation impairs cognitive and motor performance (Durmer and Dinges 2005). However, some people are more vulnerable to the effects of sleep loss than others. Sleep deprivation studies have found a wide range of effects on cognitive function including decrements in attention especially vigilance, working and long-term memory, decision making, response inhibition, processing speed, and reasoning (Lim and Dinges 2010). Sleep deprivation results in impairments in psychomotor performance that are comparable to those induced by alcohol consumption at or above the legal limit (Williamson and Feyer 2000). Sleep loss increases the risk of traffic accidents as demonstrated by poor performance on driving simulators after sleep deprivation. In addition to an increased risk for motor vehicle crashes, sleep deprivation and related neurocognitive impairments are associated with work-related injuries and fatal accidents. Sleep apnea is also associated with deficits in cognitive function and accounts for a significant proportion of motor vehicle accidents.

Sleep and Mental Health

Numerous studies have shown a high rate of comorbidity between sleep complaints (e.g., insomnia) and psychiatric disorders, especially mood and anxiety disorders (Staner 2010). This relationship goes beyond mere co-occurrence and is bidirectional since insomnia contributes to the development or exacerbation of depression and anxiety disorders, and affective disorders and their treatments contribute to insomnia (Neckelmann et al. 2007; Sateia 2009). Nondepressed individuals with insomnia have a two times greater risk for developing depression than individuals without sleep difficulties. Sleep problems affect outcomes for patients with depression. Studies report that depressed patients who continue to experience insomnia are at greater risk of relapse and recurrence of depression and risk of suicide.

Conclusions

Sleep loss is a growing public health problem worldwide. The health consequences of a sleepy society are enormous and have a significant economic impact with billions of dollars spent on direct medical costs associated with morbidities and sleep-related injuries and accidents. Sleep deprivation can alter biological processes underlying cardiovascular, metabolic, and immune function. Cumulative long-term effects of sleep loss have been associated with a number of serious health consequences, including cardiovascular disease, obesity, cancer, and type 2 diabetes. Sleep is not a luxury and is as important for health as other health-promoting behaviors like diet and exercise. Public awareness is needed to emphasize and reinforce the essentialness of sleep for health.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Glucose: Levels, Control, Intolerance, and Metabolism
- ▶ Hypertension
- ▶ Immune Function
- ▶ Insomnia
- ▶ Insulin
- ▶ Sleep
- ▶ Sleep Apnea
- ▶ Sleep Duration
- ▶ Type 2 Diabetes

References and Further Readings

Blask, D. E. (2009). Melatonin, sleep disturbance and cancer risk. *Sleep Medicine Reviews, 13*(4), 257–264.

Bryant, P. A., Trinder, J., & Curtis, N. (2004). Sick and tired: Does sleep have a vital role in the immune system? *Nature Reviews Immunology, 4*, 457–467.

Calhoun, D. A., & Harding, S. M. (2010). Sleep and hypertension. *Chest, 138*(2), 434–443.

Cappuccio, F. P., Taggart, F. M., Kandala, N. B., & Currie, A. (2008). Meta-analysis of short sleep duration and obesity in children and adults. *Sleep, 31*(5), 619–626.

Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Sleep duration and all-cause mortality: A systematic review and meta-analysis of prospective studies. *Sleep, 33*(5), 585–592.

Cappuccio, F. P., Cooper, D., D'Elia, L., Strazzullo, P., & Miller, M. A. (2011). Sleep duration predicts cardiovascular outcomes: A systematic review and meta-analysis of prospective studies. *European Heart Journal, 32*(12), 1484–1492.

Durmer, J. S., & Dinges, D. F. (2005). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology, 25*(2), 117–129.

Knutson, K. L., & Van Cauter, E. (2008). Associations between sleep loss and increased risk of obesity and diabetes. *Annals of the New York Academy of Sciences, 1129*(1), 287–304.

Kripke, D. F., Garfinkel, L., Wingard, D. L., Klauber, M. R., & Marler, M. R. (2002). Mortality associated with sleep duration and insomnia. *Archives of General Psychiatry, 59*(2), 131–136.

Lim, J., & Dinges, D. F. (2010). A meta-analysis of the impact of short-term sleep deprivation on cognitive variables. *Psychological Bulletin, 136*(3), 375–389.

Neckelmann, D., Mykletun, A., & Dahl, A. A. (2007). Chronic insomnia as a risk factor for developing anxiety and depression. *Sleep, 30*(7), 873–880.

Sateia, M. J. (2009). Update on sleep and psychiatric disorders. *Chest, 135*(5), 1370–1379.

Staner, L. (2010). Comorbidity of insomnia and depression. *Sleep Medicine Reviews, 14*(1), 35–46.

Taheri, S., Lin, L., Austin, D., Young, T., & Mignot, E. (2004). Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Medicine, 1*(3), e62.

Williamson, A. M., & Feyer, A. M. (2000). Moderate sleep deprivation produces impairments in cognitive and motor performance equivalent to legally prescribed levels of alcohol intoxication. *Occupational and Environmental Medicine, 57*(10), 649–655.

Sleep Apnea

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Synonyms

Obstructive sleep apnea; Sleep-disordered breathing

Definition

Sleep apnea is a common sleep disorder characterized by repetitive pauses in breathing or very shallow breaths during sleep (Strollo and Rogers 1996; Young et al. 2002). Pauses in breathing, known as apneas, and shallow breathing events, called hypopneas, can last a few seconds to minutes and can occur multiple times during the night. The termination of apneas and hypopneas is associated with a transient arousal from sleep. Sleep disruption due to frequent arousals may lead to excessive daytime sleepiness or fatigue. Loud snoring, witnessed breathing interruptions, and excessive daytime sleepiness are the most common signs of sleep apnea. Obstructive sleep apnea (OSA) occurs when the airway collapses or is blocked during sleep. Central sleep apnea results from temporary loss of ventilatory effort lasting at least 10 seconds and can co-occur with OSA. An evaluation for sleep apnea entails an assessment for signs and symptoms and a detailed craniofacial examination followed by a full night of in-laboratory or portable polysomnography to confirm diagnosis (Epstein et al. 2009). Sleep apnea requires long-term management. Positive airway pressure therapy, oral appliances, and surgery are the most common treatments for sleep apnea, but other behavioral interventions such as weight loss, smoking cessation, body position, and alcohol and sedative cessation, can be useful adjuncts to conventional therapies (Epstein et al. 2009). Untreated sleep apnea can result in a number of negative consequences, including excessive daytime sleepiness and fatigue, psychological symptoms, cognitive and performance impairments, and increased risk for cardiovascular and cerebrovascular disease (Bradley and Floras 2009; Sateia 2003). Decreased vigilance or falling asleep at the wheel increases the risk of motor vehicle crashes in individuals with sleep apnea (Ellen et al. 2006).

Cross-References

- ▶ Polysomnography
- ▶ Sleep Fragmentation

References and Further Readings

Bradley, T. D., & Floras, J. S. (2009). Obstructive sleep apnoea and its cardiovascular consequences. *The Lancet, 373*(9657), 82–93.

Ellen, R. L. B., Marshall, S. C., Palayew, M., Molnar, F. J., Wilson, K. G., & Man-Son-Hing, M. (2006). Systematic review of motor vehicle crash risk in persons with sleep apnea. *Journal of Clinical Sleep Medicine, 2*(2), 193–200.

Epstein, L. J., Kristo, D., Strollo, P. J., Friedman, N., Malhotra, A., Patil, S. P., et al. (2009). Clinical guideline for the evaluation, management, and long-term care of obstructive sleep apnea in adults. *Journal of Clinical Sleep Medicine, 5*(3), 263–276.

Sateia, M. J. (2003). Neuropsychological impairment and quality of life in obstructive sleep apnea. *Clinics in Chest Medicine, 24*(4), 249–259.

Strollo, P. J., & Rogers, R. M. (1996). Obstructive sleep apnea. *The New England Journal of Medicine, 334*(2), 99–104.

Young, T., Peppard, P. E., & Gottlieb, D. J. (2002). Epidemiology of obstructive sleep apnea: A population health perspective. *American Journal of Respiratory and Critical Care Medicine, 165*(9), 1217–1239.

Sleep Architecture

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Definition

Sleep Architecture is the visual representation of the way sleep stages are organized throughout a polysomnographically recorded sleep interval. Sleep Architecture is displayed on a hypnogram plot.

Description

Sleep can be measured with polysomnography (PSG), which consists of multiple measures that include, but are not limited to, electroencephalography (EEG), electrooculography (EOG), and electromyography (EMG) (see for a review Keenan and Hirshkowitz 2011). PSG literally translates to "many" (poly) "sleep" (somno) "writings" (graphy). When sleep is measured

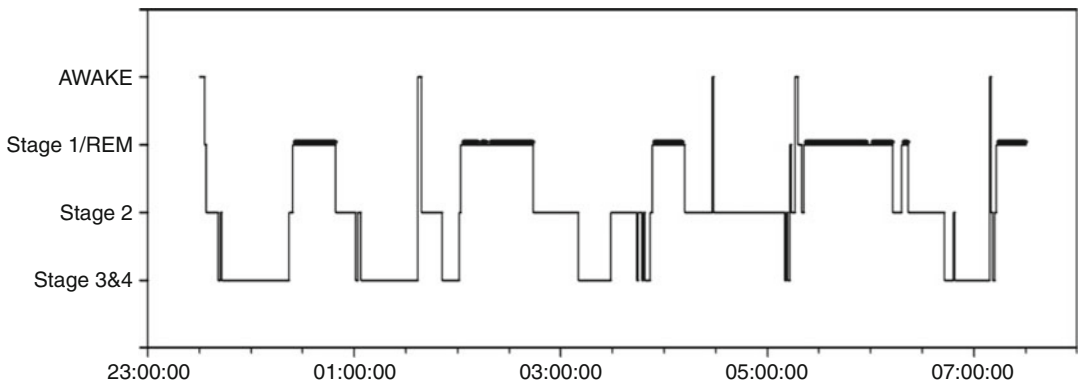
with PSG, the measured signals are typically sectioned into 30-s intervals consecutively throughout the entire PSG recording period. These 30-s intervals are termed "epochs." Within each epoch, the PSG-measured signals (from EEG, EOG, and EMG) are cumulatively used to differentiate sleep from wake, and to further classify sleep into different categories that are known as sleep stages.

According to the 2007 American Academy of Sleep Medicine standard practice parameters, sleep can be classified into four stages that include N1, N2, N3, and Rapid Eye Movement (REM) sleep (Iber et al. 2007). The PSG signals are used to identify each individual epoch as a particular sleep stage. The act of using PSG signals to identify each epoch as a particular sleep stage is known as sleep stage scoring. Sleep stage scoring is completed by a trained technician who visually interprets the PSG signals in accordance with standard practice parameters.

Once the entire PSG sleep study is scored, the study is summarized into a clinical report. The clinical report describes the sleep parameters measured (e.g., EEG, EOG, EMG, airflow parameters), sleep scoring data (e.g., lights out clock time, lights on clock time, total sleep time), arousal events (e.g., number of arousals, arousal index), respiratory events (e.g., apnea index, hypopnea index), cardiac events (e.g., average heart rate during sleep), movement events (e.g., number of periodic limb movements during sleep), and summary statements (e.g., findings related to sleep diagnoses, behavioral observations, sleep hypnogram). Of particular relevance to sleep architecture is the sleep hypnogram.

The sleep hypnogram is a summary figure that visually displays the scored wake and sleep stages as they occurred throughout the entire PSG recording period. The sleep hypnogram is formatted with time throughout the entire PSG sleep study as a continuous variable on the X-axis, and the visually scored wake and sleep stages as categorical variables on the Y-axis. The categorical wake and sleep stages are positioned with REM closest to the intersection with the X-axis, followed by N3, N2, N1, and wake in an upward ascending order; however, the order and format of these stages can vary per laboratory (e.g., Fig. 1).

As time progresses throughout the sleep monitoring period, the person being monitored naturally enters and exits the wake and sleep stages that are indicated on the Y-axis. The time spent in a particular stage is represented by a horizontal line adjacent to the respective stage, and the length of that horizontal line reflects the time spent in that particular stage as it corresponds to the "real time" for which the stage occurred – indicated on the X-axis. During a stage transition, the horizontal line turns 90° (right angle) positive or negative to become vertical and adjacent to the newly entered stage – as indicated on the Y-axis; then the line turns 90° (right angle) once again to reach a horizontal position with a length that represents the time spent in that particular stage – indicated on



Sleep Architecture, Fig. 1 Sleep hypnogram and sleep architecture during a 20-year-old female's overnight sleep monitoring period

the X-axis. As the different wake and sleep stages fluctuate throughout the night, the hypnogram line appears at different horizontal levels (stage-dependent [Y-axis]), at different lengths on each level (time-dependent [X-axis]); and as the levels shift (stage shift), the horizontal line is connected to the previous and subsequent horizontal level by a vertical line (e.g., elbow connector).

Consequently, the wake and sleep stages represented on a sleep hypnogram resemble the back drop of a city skyline, with continuous geometric cubes and rectangles of different heights and widths that appear to continuously penetrate and retreat from the skyline. The figurative reference to a city skyline has been generally accepted, and has literally generated the term "sleep architecture." Thus, sleep architecture is the visual representation of wake and sleep stages that occurred throughout a polysomnographically recorded sleep interval, and is displayed on a hypnogram plot.

The visual structure of sleep architecture is dependent upon the sleep stage, and the time in the particular sleep stage. Typically, sleep is entered through N1, followed by N2, followed by N3, and then followed by REM – this progressive series is termed a "sleep cycle." The typical sleep cycle occurs throughout sleep at approximately 50-min intervals among infants (Grigg-Damberger et al. 2007) and approximately 90–110-min intervals among adults (Iber et al. 2007). The time spent in the different stages changes throughout the night. The first portion of the night primarily consists of non-REM sleep (i.e., N1, N2, and N3), and the second portion of the night primarily consists of REM sleep.

The sleep architecture displayed on the hypnogram from a normative adult PSG sleep study will display cumulatively longer horizontal lines adjacent to N1, N2, and N3 during the first portion of the night relative to the second portion, and will conversely display cumulatively longer horizontal lines adjacent to REM during the second portion of the night relative to the first portion. A sleep hypnogram will display the sleep stage cyclicity, as well as changes in time spent in particular stages as they occur throughout the recording period – this differs across nights. Since the time spent in different sleep stages generally changes throughout the lifespan (Ohayon et al. 2004), the percentage of time spent in each sleep stage, as reflected in the histogram, can differ by to the age of the person assessed.

Cross-References

- ▶ Non-REM Sleep
- ▶ Polysomnography
- ▶ REM Sleep
- ▶ Sleep

References and Further Readings

Grigg-Damberger, M., Gozal, D., Marcus, C. L., Quan, S. F., Rosen, C. L., Chervin, R. D., et al. (2007). The visual scoring of sleep and arousals in infants and children. *Journal of Clinical Sleep Medicine, 3*, 201–240.

Iber, C., Ancoli-Israel, S., Chesson, A., Quan, S. F., & American Academy of Sleep Medicine. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications* (1st ed.). Westchester: American Academy of Sleep Medicine.

Keenan, S., & Hirshkowitz, M. (2011). Monitoring and staging human sleep. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 1602–1609). St. Louis: Elsevier.

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.

Sleep Continuity

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Synonyms

Sleep efficiency; Sleep fragmentation; Sleep maintenance

Definition

Sleep continuity refers to the amount and distribution of sleep versus wakefulness in a given

sleep period; it includes both sleep initiation and sleep maintenance (Hall et al. in press). Specific indices of sleep continuity may include latency to sleep onset, number of awakenings after sleep onset, total time of wakefulness after sleep onset, and overall sleep efficiency. Sleep continuity is most often assessed using self-report questionnaires (i.e., retrospective reports, morning diaries or logs), wrist actigraphy, or polysomnography.

Sleep continuity declines as part of the normal aging process (Ohayon et al. 2004). Disruptions in sleep continuity are typical among individuals with insomnia and are also commonly reported by or observed in those with mood disorders, anxiety disorders, and substance disorders. Many medical conditions and treatments are also related to disrupted sleep continuity; some of the most common examples include sleep apnea, chronic pain, asthma and respiratory conditions, chronic renal disease, infectious diseases, and cancer. Decreased sleep continuity has been associated with increased inflammatory markers, susceptibility to infection, elevated blood pressure, obesity, presence of metabolic syndrome, diabetes, and cardiovascular disease. Several studies have reported a link between decreased sleep continuity and incident diabetes or incident cardiovascular disease.

Cross-References

► Sleep Fragmentation

References and Further Readings

Hall, M., Greeson, J., & Mezick, E. (in press). Sleep as a biobehavioral risk factor for cardiovascular disease. In S. R. Waldstein, W. J. Kop, & L. I. Katzel (Eds.), *Handbook of cardiovascular behavioral medicine*. New York: Springer.

Ohayon, M. M., Carkadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.

Sleep Curtailment

► Sleep Restriction

Sleep Debt

► Sleep Restriction

Sleep Deprivation

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Synonyms

Sleep deprived

Definition

Sleep deprivation generally refers to the total loss of sleep due to experimental manipulations or circumstance.

Description

Sleep deprivation refers to the total loss of sleep. The term is generally used in the context of experimental manipulations which keep individuals (or experimental animals) awake throughout their usual sleep period. Sleep deprivation also occurs in naturalistic conditions such as when a student stays awake all night to study for an exam. Experimental and observational sleep deprivation studies have been used to evaluate the cognitive, behavioral, emotional, and physiological consequences of sleep loss.

Experimental sleep deprivation studies in humans generally deprive participants of 1–3 nights of sleep, resulting in 24–72 h of wakefulness. Experimental animal models can extend sleep deprivation for much longer periods of time (e.g., 2 weeks). In both kinds of studies,

electroencephalographic (EEG) monitoring is used to ensure wakefulness, usually through interactions with study staff or procedural manipulations. For example, the classical "disk-over-water" method pioneered by Dr. Allan Rechtschaffen at the University of Chicago involved placing two animals on a rotating disk suspended above water (Rechtschaffen and Bergmann 1995). A barrier divided the disk into half, with the experimental animal on one side of the disk and the yoked animal on the other. When the experimental animal showed EEG signs of sleep, the disk would rotate. If the animal did not wake up, they would fall into the water when they reached the barrier. Through conditioning, the experimental animal would learn to wake up as soon as the disk started to rotate. The yoked animal, on the other hand, would learn to sleep when the disk was *not* rotating. This elegant design allowed experimenters to tease apart the effects of movement and stress associated with older forms of sleep deprivation (e.g., placing the animal on a continuous running wheel with no option for escape) from the effects of sleep deprivation. In human studies, the constant routine protocol, which involves keeping participants in a recumbent position for the duration of the protocol, is used to control for the influence of extraneous factors such as increased movement on study outcomes.

Although individuals may feel that sleep deprivation has no adverse effects on them, experimental evidence suggests that perceptions of resiliency to sleep loss are unfounded. The "disconnect" between subjective perceptions of sleepiness and objective indices of health and functioning in humans has been systematically documented by Dr. David Dinges and his colleagues at the University of California (see Lim and Dinges 2008). More recently, others have demonstrated that one night of total sleep deprivation in healthy young adults is associated with increased blood pressure and amydgala reactivity to negative emotional stimuli (Franzen et al. 2011; Yoo et al. 2007). As a complement to studies in humans, animal models, which allow exquisite control over sleep and wakefulness, have begun to elucidate the influence of sleep deprivation on gene expression, molecular signaling, and synaptic plasticity (e.g., Seugnet et al. 2011; Wang et al. 2010). Taken as a whole, these studies demonstrate that sleep is essential to health and functioning across species.

Cross-References

- ▶ Gene Expression
- ▶ Sleep
- ▶ Sleep and Health

References and Further Readings

Franzen, P. L., Gianaros, P. J., Marsland, A. L., Hall, M., Siegle, G. J., Dahl, R. E., et al. (2011). Cardiovascular reactivity to acute psychological stress following sleep deprivation. *Psychosomatic Medicine, 73*(8), 679–682.

Lim, J., & Dinges, D. F. (2008). Sleep deprivation and vigilant attention. *Annals of the New York Academy of Sciences, 1129*, 305–322.

Rechtschaffen, A., & Bergmann, B. M. (1995). Sleep deprivation in the rat by the disk-over-water method. *Behavioural Brain Research, 69*(1–2), 55–63.

Seugnet, L., Suzuki, Y., Donlea, J. M., Gottschalk, L., & Shaw, P. J. (2011). Sleep deprivation during early-adult development results in long-lasting learning deficits in adult *Drosophila*. *Sleep, 34*(2), 137–146.

Wang, H., Liu, Y., Briesemann, M., & Yan, J. (2010). Computational analysis of gene regulation in animal sleep deprivation. *Physiological Genomics, 42*(3), 427–436.

Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep: A prefrontal amygdala disconnect. *Current Biology, 17*, R877–R878.

Sleep Deprived

- ▶ Sleep Deprivation

Sleep Duration

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Synonyms

Total sleep time

Definition

Sleep duration typically refers to the total amount of sleep obtained, either during the nocturnal sleep episode or across the 24-h period.

Description

Measurement

Sleep duration can be measured via questionnaire, diary, actigraphy, or polysomnography. In population-based epidemiologic studies, single-item self-report measures of sleep duration have often been utilized (e.g., *How many hours of sleep do you obtain on a typical night?*). In clinical and research settings, sleep diaries, actigraphy, and polysomnography provide assessments of sleep duration. Sleep diaries involve the subjective report of sleep duration, typically assessed daily for a minimum of 1 week. Actigraphy provides an objective estimate of sleep/wake status from the detection of bodily movement, whereas polysomnography measures sleep duration through the assessment of multiple physiological signals, including brain, eye, and muscle activity. Although typically correlated, large discrepancies are often noted between subjective and objective measures of sleep duration; in most populations, self-reported sleep duration is overestimated compared to objective measurement (Matthews et al. 2018).

Sleep Duration Across the Lifespan

Sleep is regulated by a complex interaction of homeostatic and circadian factors. The homeostatic process reflects the need for sleep, accumulating during sustained wakefulness and dissipating during sleep. The circadian process, driven by outputs of the circadian pacemaker, promotes wakefulness during the day and evening and promotes sleep during the night, with the peak sleep-promoting signal during the early morning. Thus, circadian signals oppose the rise of homeostatic sleep pressure during the day, allowing for uninterrupted daytime wakefulness. Both circadian and homeostatic processes promote sleep onset, with circadian sleep-promoting signals facilitating continued consolidated sleep despite the gradual dissipation of homeostatic sleep pressure over the course of the night. Aging results in changes in the homeostatic and circadian regulation of sleep, with a phase advance of the circadian wake-promoting signal (i.e., increased signal for wakefulness in the morning but decreased in the evening) and reduced homeostatic drive for sleep (Dijk et al. 1999). As a result, sleep in older adults is commonly phase advanced, of shorter duration, and with greater fragmentation compared to younger adults.

In general, sleep duration decreases from infancy through old age. Sleep duration (per 24 h) averages >13 h during infancy, gradually declining to 8–9 h during late adolescence (Williams et al. 2013). Normative sleep duration then declines throughout adulthood to approximately 6.5 h at 60 years, at which point sleep duration tends to stabilize (Ohayon et al. 2004). However, these average values do not indicate whether sleep need is being met, particularly during development. For instance, although weekday sleep duration decreases throughout childhood and adolescence, weekend sleep duration remains similar from age 5 to 16. This pattern suggests a greater influence of environmental factors (e.g., school schedules) than biological or maturational influences on sleep duration during childhood and adolescence.

Whether sleep duration has changed dramatically over recent decades is controversial. Whereas some studies have documented significant decreases in sleep duration and increased prevalence of short sleep over the past 30–50 years (Ford et al. 2015), others have presented evidence that longitudinal changes in sleep duration have been minimal to nonexistent (Bin et al. 2013; Youngstedt et al. 2016). Moreover, recent work suggests that average adult sleep duration has minimally increased (~1 min/year) over the past 15 years (Basner and Dinges 2018). Other studies estimate that the prevalence of short and long sleep duration (i.e., the extremes of sleep duration that are most strongly associated with health risk) has not appreciably changed over ~30 years (Bonke 2015).

Sleep Duration and Health

Sleep is an essential behavior for memory consolidation, development, and restoration of nervous, immune, skeletal, and muscular systems. Consequently, the amount of sleep obtained has a significant influence on one's health and functioning. Sleep duration is at least partly determined by genetic influences, so interindividual differences in sleep duration are to be expected. Nevertheless, a significant association between sleep duration and health has been consistently documented in epidemiologic research.

The association between sleep duration and health typically has a U-shaped distribution, with the lowest risk associated adverse health outcomes typically observed with a sleep duration of ~7 h for adults (Itani et al. 2017; Jike et al. 2018). Both short and long sleep have been associated with increased risk of mortality and numerous morbidities, including cardiovascular disease (e.g., atherosclerosis), metabolic dysfunction (e.g., type 2 diabetes, obesity), and cognitive impairment. However, the most marked morbidity and mortality risks have been associated with extreme short and long sleep (<5 and >9 h, respectively).

The possible mechanisms by which sleep duration affects health likely differ between short and long sleep duration. Most research has focused on how short sleep may affect health, with blunted hormonal control of appetite, increased inflammatory levels, alterations in hypothalamic-pituitary-adrenal axis and sympathoadrenal activity, and decreased immunity to pathogens observed following short-term experimental sleep restriction (Grandner et al. 2010). Plausible mechanisms linking long sleep duration to excess morbidity and mortality are less clear; however, fatigue, impaired immunity, reduced photoperiod length, and underlying disease have been postulated (Youngstedt and Kripke 2004). However, short and long sleep share some possible mechanisms. Both short and long sleep duration are more common among with those with low socioeconomic status and racial/ethnic minorities. Moreover, sleep complaints and poor health behaviors (e.g., smoking, alcohol, physical inactivity) are more prevalent in short and long sleepers in comparison to those with normal sleep duration.

Sleep Duration Recommendations

In recognition of the growing evidence base on the importance of adequate sleep duration for optimal health, multiple professional organizations have published consensus sleep duration recommendations for children and adults. The American Academy of Sleep Medicine (AASM) recognized that daily sleep need differs across development as they recommended, for example, 12–16 h for infants and 8–10 h for teenagers (Paruthi et al. 2016). For adults, the AASM (along with the Sleep Research Society) recommended sleep duration ≥ 7 h to promote ideal health (Watson et al. 2015). Recommendations from the American Thoracic Society and National Sleep Foundation were similar to those from the AASM (Mukherjee et al. 2015; Hirshkowitz et al. 2015).

Cross-References

- ▶ Actigraphy (Wrist, for Measuring Rest/Activity Patterns and Sleep)
- ▶ Polysomnography
- ▶ Sleep
- ▶ Sleep and Health

- ▶ Sleep Deprivation
- ▶ Sleep Quality
- ▶ Sleep Restriction

References and Further Reading

Basner, M., & Dinges, D. F. (2018). Sleep duration in the United States 2003–2016: First signs of success in the fight against sleep deficiency? *Sleep, 41*, zsy012.

Bin, Y. S., Marshall, N. S., & Glozier, N. (2013). Sleeping at the limits: The changing prevalence of short and long sleep durations in 10 countries. *American Journal of Epidemiology, 177*, 826–833.

Bonke, J. (2015). Trends in short and long sleep in Denmark from 1964 to 2009, and the associations with employment, SES (socioeconomic status) and BMI. *Sleep Medicine, 16*, 385–390.

Dijk, D. J., Duffy, J. F., Riel, E., Shanahan, T. L., & Czeisler, C. A. (1999). Ageing and the circadian and homeostatic regulation of human sleep during forced desynchrony of rest, melatonin and temperature rhythms. *Journal of Physiology, 516*, 611–627.

Ford, E. S., Cunningham, T. J., & Croft, J. B. (2015). Trends in self-reported sleep duration among US adults from 1985 to 2012. *Sleep, 38*, 829–832.

Grandner, M. A., Hale, L., Moore, M., & Patel, N. P. (2010). Mortality associated with short sleep duration: The evidence, the possible mechanisms, and the future. *Sleep Medicine Reviews, 14*, 191–203.

Hirshkowitz, M., Whiton, K., Albert, S. M., Alessi, C., Bruni, O., DonCarlos, L., … Ware, J. C. (2015). National Sleep Foundation's updated sleep duration recommendations: Final report. *Sleep Health, 1,* 233–243.

Itani, O., Jike, M., Watanabe, N., & Kaneita, Y. (2017). Short sleep duration and health outcomes: A systematic review, meta-analysis, and meta-regression. *Sleep Medicine, 32*, 246–256.

Jike, M., Itani, O., Watanabe, N., Buysse, D. J., & Kaneita, Y. (2018). Long sleep duration and health outcomes: A systematic review, meta-analysis and meta-regression. *Sleep Medicine Reviews, 39*, 25–36.

Matthews, K. A., Patel, S. R., Pantesco, E. J., Buysse, D. J., Kamarck, T. W., Lee, L., & Hall, M. H. (2018). Similarities and differences in estimates of sleep duration by polysomnography, actigraphy, diary, and self-reported habitual sleep in a community sample. *Sleep Health, 4*, 96–103.

Mukherjee, S., Patel, S. R., Kales, S. N., Ayas, N. T., Strohl, K. P., Gozal, D., & Malhotra, A. (2015). An official American Thoracic Society statement: The importance of healthy sleep. Recommendations and future priorities. *American Journal of Respiratory and Critical Care Medicine, 191*, 1450–1458.

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.

Paruthi, S., Brooks, L. J., D'Ambrosio, C., Hall, W. A., Kotagal, S., Lloyd, R. M., … Wise, M. S. (2016). Recommended amount of sleep for pediatric populations: A consensus statement of the American Academy of sleep medicine. *Journal of Clinical Sleep Medicine, 12,* 785–786.

Watson, N. F., Badr, M. S., Belenky, G., Bliwise, D. L., Buxton, O. M., Buysse, D., … Tasali, E. (2015). Recommended amount of sleep for a healthy adult: A joint consensus statement of the American Academy of sleep medicine and Sleep Research Society. *Sleep, 38,* 843–844.

Williams, J. A., Zimmerman, F. J., & Bell, J. F. (2013). Norms and trends of sleep time among US children and adolescents. *JAMA Pediatrics, 167*, 55–60.

Youngstedt, S. D., Goff, E. E., Reynolds, A. M., Kripke, D. F., Irwin, M. R., Bootzin, R. R., … Jean-Louis, G. (2016). Has adult sleep duration declined over the last 50+ years? *Sleep Medicine Reviews, 28,* 69–85.

Youngstedt, S. D., & Kripke, D. F. (2004). Long sleep and mortality: Rationale for sleep restriction. *Sleep Medicine Reviews, 8*, 159–174.

Sleep Efficiency

- ▶ Sleep Continuity
- ▶ Sleep Fragmentation

Sleep Fragmentation

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Synonyms

Sleep continuity; Sleep efficiency; Sleep maintenance

Definition

Sleep fragmentation typically refers to brief arousals that occur during a sleep period. The American Sleep Disorders Association (1992) defines an arousal as an abrupt shift in

electroencephalographic (EEG) frequency (suggestive of an awake state) which is 3 s or greater in duration and which occurs after at least 10 consecutive seconds of sleep. A number of other definitions of arousal have been published or suggested since that time, with some recommending that elements or physiological responses other than EEG frequency be taken into account (e.g., autonomic activation without cortical involvement) (Janackova and Sforza 2008). When assessed with actigraphy, sleep fragmentation may refer to the amount of movement or restlessness in a sleep period. For example, actigraph software programs use algorithms to calculate a sleep fragmentation index, based on the number or proportion of total sleep epochs characterized by movement. When used in a more general sense, sleep fragmentation may also refer to the overall amount and distribution of wakefulness in a sleep period and can be considered the inverse of sleep continuity. For example, some authors have used the term "sleep fragmentation" to describe parameters such as wakefulness after sleep onset and sleep efficiency.

Elevated sleep fragmentation occurs in those with sleep apnea and may correlate with daytime sleepiness. Increased sleep fragmentation as assessed by actigraphy has been associated with a number of physical and psychiatric health outcomes, as well as deficits in neurobehavioral performance. Experimental models of sleep fragmentation, which typically disrupt sleep briefly using auditory, mechanical, or other stimuli, have been used to examine the neurophysiologic, cognitive, and behavioral consequences of fragmented sleep.

Cross-References

- ▶ Sleep Apnea
- ▶ Sleep Continuity

References and Further Readings

American Sleep Disorders Association Atlas Task Force. (1992). EEG arousals: Scoring rules and examples. *Sleep, 15*, 173–184.

Bonnet, M. H., & Arand, D. L. (2003). Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep Medicine Reviews, 7*, 297–310.

Janackova, S., & Sforza, E. (2008). Neurobiology of sleep fragmentation: Cortical and autonomic markers of sleep disorders. *Current Pharmaceutical Design, 14*, 3474–3480.

Sleep Maintenance

- ▶ Sleep Continuity
- ▶ Sleep Fragmentation

Sleep Quality

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Synonyms

Sleep refreshment; Sleep satisfaction

Definition

Sleep quality is defined as one's satisfaction of the sleep experience, integrating aspects of sleep initiation, sleep maintenance, sleep quantity, and refreshment upon awakening.

Description

Sleep quality is widely recognized as a vital contributor to optimal health and functioning. Yet, despite its common usage, "sleep quality" is a term without a clear definition or operationalization (Ohayon et al. 2017).

Measurement

Measurement of sleep quality is difficult due to its imprecise definition. The construct of sleep quality likely incorporates aspects of sleep quantity,

sleep continuity, the feeling of refreshment upon awakening, and daytime sleepiness (Harvey et al. 2008). Many of these aspects cannot be easily measured in an objective fashion. Thus, sleep quality is often assessed with self-reported measures.

Sleep diaries, which track sleep on a daily basis, often assess sleep quality by incorporating a Likert-type rating scale (e.g., 1: *very poor sleep quality*; 5: *very good sleep quality*) or a visual analogue scale (with anchors of *very poor* and *very good* sleep quality) into the diary. Questionnaires, on the other hand, assess sleep quality through the measurement of different domains of the sleep experience. The most widely used questionnaire, the Pittsburgh Sleep Quality Index, is an 18-item instrument with seven components: sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, sleeping medication use, and daytime dysfunction (Buysse et al. 1989). Another common measure, the Medical Outcomes Study Sleep Scale, assesses six different aspects of sleep: sleep disturbance, sleep adequacy, daytime sleepiness, snoring, awakening with shortness of breath or headache, and sleep quantity (Hays et al. 2005). Scores on both of these measures have been shown to identify individuals who characterize their sleep as being poor in quality.

An alternative approach to measuring sleep quality involves measuring self-reported or objectively recorded sleep parameters related to the magnitude of nocturnal wakefulness, such as sleep onset latency, total wakefulness, sleep efficiency, number of awakenings, or arousals. Moreover, measures of sleep "depth" derived from polysomnography, such as the amount of N1 sleep or N3 sleep (sometimes referred to as "light" and "deep" sleep, respectively), have sometimes been used to characterize sleep quality. However, these parameters often fail to completely capture the essence of sleep quality. In particular, sleep quality should not be considered to be synonymous with the amount of sleep obtained. For example, in comparison to those who report a sleep duration of 7–8 h, poor sleep quality is more prevalent with short and long sleep.

Correlates with Sleep Parameters

Across multiple studies, objective sleep parameters have exhibited only minimal concordance with sleep quality, with sleep efficiency, wakefulness after sleep onset, and total sleep time typically showing the strongest, yet still modest, correlations (Kaplan et al. 2017a, b). While some studies have found that the spectral density of the sleep electroencephalographic (EEG) content correlated with sleep quality (Krystal and Edinger 2008), others have failed to observe a strong link (Gabryelska et al. 2019; Kaplan et al. 2017a). Overall, these findings suggest that sleep quality is a multifaceted construct that cannot be captured by a single surrogate sleep parameter or objective surrogate markers.

Sleep Quality and Health

Poor sleep quality is widespread in modern society. Approximately one-third of adults complain of poor sleep quality, though in most studies, prevalence estimates are based upon insomnia-related symptoms. Poor sleep quality has been associated with increasing age, low socioeconomic status, poor general health, psychological distress, and poor lifestyle behaviors (e.g., high caffeine use, sedentary lifestyle, smoking).

How poor sleep quality may influence future morbidity or mortality risk is less clear. Epidemiologic studies have found that poor sleep quality is predictive of increased risk of metabolic dysfunction and mortality risk (Cappuccio et al. 2010; Kojima et al. 2000). However, most studies have focused on sleep duration rather than sleep quality, perhaps due to the difficulty in concisely defining sleep quality. Interestingly, in studies that have concurrently assessed the health risks of sleep duration and sleep quality, the strongest risk is associated with those reporting poor sleep quality and inadequate sleep duration (Vgontzas et al. 2013), with similar (or greater, in some cases) risk associated with poor sleep quality relative to extreme sleep duration (Bin 2016).

Cross-References

► Actigraphy (Wrist, for Measuring Rest/Activity Patterns and Sleep)

- ▶ Insomnia
- ▶ Polysomnography
- ▶ Sleep
- ▶ Sleep and Health
- ▶ Sleep Architecture

References and Further Reading

Bin, Y. S. (2016). Is sleep quality more important than sleep duration for public health? *Sleep, 39*, 1629–1630.

Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213.

Cappuccio, F. P., D'Elia, L., Strazzullo, P., & Miller, M. A. (2010). Quantity and quality of sleep and incidence of type 2 diabetes: A systematic review and meta-analysis. *Diabetes Care, 33*, 414–420.

Gabryelska, A., Feige, B., Riemann, D., Spiegelhalder, K., Johann, A., Białasiewicz, P., & Hertenstein, E. (2019). Can spectral power predict subjective sleep quality in healthy individuals? *Journal of Sleep Research, 28*, e12848.

Harvey, A. G., Stinson, K., Whitaker, K. L., Moskovitz, D., & Virk, H. (2008). The subjective meaning of sleep quality: A comparison of individuals with and without insomnia. *Sleep, 31*, 383–393.

Hays, R. D., Martin, S. A., Sesti, A. M., & Spritzer, K. L. (2005). Psychometric properties of the medical outcomes study sleep measure. *Sleep Medicine, 6*, 41–44.

Kaplan, K. A., Hardas, P. P., Redline, S., & Zeitzer, J. M. (2017a). Correlates of sleep quality in midlife and beyond: A machine learning analysis. *Sleep Medicine, 34*, 162–167.

Kaplan, K. A., Hirshman, J., Hernandez, B., Stefanick, M. L., Hoffman, A. R., Redline, S., … Zeitzer, J. M. (2017b). When a gold standard isn't so golden: Lack of prediction of subjective sleep quality from sleep polysomnography. *Biological Psychology, 123,* 37–46.

Kojima, M., Wakai, K., Kawamura, T., Tamakoshi, A., Aoki, R., Lin, Y., … Ohno, Y. (2000). Sleep patterns and total mortality: A 12-year follow-up study in Japan. *Journal of Epidemiology, 10*, 87–93.

Krystal, A. D., & Edinger, J. D. (2008). Measuring sleep quality. *Sleep Medicine, 9*, S10–S17.

Ohayon M., Wickwire E. M., Hirshkowitz M., Albert S. M., Avidan A., Daly F. J., … Vitiello, M. V. (2017). National Sleep Foundation's sleep quality recommendations: First report. *Sleep Health, 3,* 6–19.

Vgontzas, A. N., Fernandez-Mendoza, J., Liao, D., & Bixler, E. O. (2013). Insomnia with objective short sleep duration: The most biologically severe phenotype of the disorder. *Sleep Medicine Reviews, 17*, 241–254.

Sleep Refreshment

- ▶ Sleep Quality

Sleep Restriction

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Synonyms

Partial sleep deprivation; Sleep curtailment; Sleep debt

Definition

Sleep restriction generally refers to situational or experimentally induced reductions in overall sleep duration.

Description

Sleep restriction refers to the partial loss of sleep. The term is generally used in the context of experimental manipulations which keep individuals (or experimental animals) awake for some portion of their usual sleep period. Sleep restriction also occurs in naturalistic conditions such as when new parents have to wake repeatedly to care for their newborn child or when an individual purposefully reduces their sleep time in order to meet the competing demands of work, family, or other obligations. Importantly, sleep restriction differs from insomnia in that sleep-restricted individuals do not have an adequate opportunity to sleep whereas individuals with insomnia have sleep difficulties despite adequate opportunity to sleep (see ▶ "Insomnia" entry). Experimental and observational sleep restriction studies have been used to

evaluate the cognitive, behavioral, emotional, and physiological consequences of sleep restriction, including the loss of specific components of sleep (e.g., slow-wave sleep).

Dr. Eve Van Cauter and her colleagues at the University of Chicago have been instrumental in highlighting the public health implications of partial sleep restriction, or sleep "curtailment" across the life span (Hanlon and Van Cauter 2011). In a series of carefully controlled studies in healthy young adults, Van Cauter and colleagues demonstrated that sleep restriction is associated with metabolic and endocrine alterations that underlie glucose tolerance, insulin sensitivity, and weight gain. These results are mirrored in epidemiologic studies of obesity and diabetes risk in children and adults (Hanlon and Van Cauter 2011). Other consequences of experimental sleep restriction include decrements in working memory, alterations in inflammatory markers, and decreased testosterone levels in males (Casement et al. 2006; Irwin et al. 2006; Leproult and Van Cauter 2011; Prather et al. 2009). Experimental laboratory studies have also begun to selectively deprive experimental subjects of specific sleep stages to better understand their function. For instance, animal models have been used to identify the neurophysiological mechanisms through which REM sleep regulates learning and memory (Poe et al. 2010). In the only study of its kind conducted to date, Tasali et al. (2008) demonstrated that selective suppression of slow-wave sleep in healthy, lean adults resulted in marked decreases in insulin sensitivity. Importantly, these effects were independent of sleep duration.

Societal trends suggest that large numbers of adults are chronically sleep-restricted. During the work week, they may build up a sleep "debt" and perceive that this debt may be "paid" on non-work nights. Epidemiologic evidence that documents the buildup of sleep debt during the work week and its payment on non-work nights is lacking. Moreover, experimental evidence suggests that multiple, long recovery nights may be necessary to "repay" sleep debt induced by chronic partial sleep restriction (Banks et al. 2010). Although the systematic evaluation of the impact of sleep restriction on indices of health and functioning is in its infancy, the epidemiological and experimental data amassed thus far supports the belief that this is an important public health concern.

References and Further Readings

Banks, S., Van Dongen, H. P., Maislin, G., & Dinges, D. F. (2010). Neurobehavioral dynamics following chronic sleep restriction: dose-response effects of one night for recovery. *Sleep, 33*, 1013–1026.

Casement, M. D., Broussard, J. L., Mullington, J. M., & Press, D. Z. (2006). The contribution of sleep to improvements in working memory scanning speed: A study of prolonged sleep restriction. *Biological Psychology, 72*(2), 208–212.

Hanlon, E. C., & Van Cauter, E. (2011). Quantification of sleep behavior and of its impact on the cross-talk between the brain and peripheral metabolism. *Proceedings of the National Academy of Sciences of the United States of America, 108*(Suppl 3), 15609–15616.

Irwin, M. R., Wang, M., Campomayor, C. O., Collado-Hidalgo, A., & Cole, S. (2006). Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. *Archives of Internal Medicine, 166*(16), 1756–1762.

Leproult, R., & Van Cauter, E. (2011). Effect of 1 week of sleep restriction on testosterone levels in young healthy men. *JAMA: The Journal of the American Medical Association, 305*(21), 2173–2174.

Poe, G. R., Walsh, C. M., & Bjorness, T. E. (2010). Cognitive neuroscience of sleep. *Progress in Brain Research, 185*, 1–19.

Prather, A. A., Marsland, A. L., Hall, M., Neumann, S. A., Muldoon, M. F., & Manuck, S. B. (2009). Normative variation in self-reported sleep quality and sleep debt is associated with stimulated pro-inflammatory cytokine production. *Biological Psychology, 82*(1), 12–17.

Spiegel, K., Tasali, E., Penev, P., & Van Cauter, E. (2004). Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Annals of Internal Medicine, 141*(11), 846–850.

Tasali, E., Leproult, R., Ehrmann, D. A., & Van Cauter, E. (2008). Slow-wave sleep and the risk of type 2 diabetes in humans. *Proceedings of the National Academy of Sciences of the United States of America, 105*(3), 1044–1049.

Sleep Satisfaction

► Sleep Quality

Sleep Stages 1, 2, 3, and 4

▶ Non-REM Sleep

Sleep Stages 3 and 4

▶ Slow-Wave Sleep

Sleep Study

▶ Polysomnography

Sleep-Disordered Breathing

▶ Sleep Apnea

Slim Disease

▶ Cachexia (Wasting Syndrome)

Slow-Wave Sleep

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Synonyms

Deep sleep; Delta sleep; N3; Sleep stages 3 and 4

Definition

Sleep can be measured with polysomnography (PSG) (see review, Keenan and Hirshkowitz 2011). Polysomnographically measured sleep behaviors can be classified into distinct categories. Slow-wave sleep (SWS) is a distinct sleep behavior classification. SWS is also known as stage N3 sleep as defined by the 2007 American Academy of Sleep Medicine sleep scoring manual (Iber et al. 2007). SWS is otherwise known as the combination of sleep stages 3 and 4 according to the Rechtschaffen and Kales "classic criteria" (Rechtschaffen and Kales 1968). Other common terms used to describe SWS are "delta sleep" and "deep sleep."

A characteristic component of SWS is the organized pattern of neurological activity that is emitted from the brain as measured by electroencephalography. A component of the organized pattern of neurological activity is the presence of slow waves, otherwise known as delta waves. Slow waves have high amplitude (>75 V) and low frequency (0.5–2 Hz). When slow waves occur during sleep, and present within 20% or more of an epoch, that epoch is classified as SWS, or N3.

Although the true function of sleep and particularly SWS is unknown (e.g., Rector et al. 2009; Siegel 2009), to date the primary explanation is that SWS reflects the homeostatic sleep drive, or one's escalating need for sleep with increasing time awake. This relation is exemplified when One's time spent in SWS increases relative to their time previously spent awake (Bersagliere and Achermann 2010). Thus, SWS is described as the "restorative component" of sleep. Common parasomnias that can occur during SWS are sleep walking and night terrors.

Human Development: Due to developmental changes in infant sleep patterns, typically SWS can be scored by 4–4.5 months post-term (Grigg-Damberger et al. 2007). The percentage of SWS obtained is highest during early-life and decreases from early childhood through old age (Ohayon et al. 2004).

Cross-References

▶ Non-REM Sleep
▶ Polysomnography
▶ REM Sleep

- ▶ Sleep
- ▶ Sleep Architecture

References and Further Readings

Bersagliere, A., & Achermann, P. (2010). Slow oscillations in human non-rapid eye movement sleep electroencephalogram: Effects of increased sleep pressure. *Journal of Sleep Research, 19*, 228–237.

Grigg-Damberger, M., Gozal, D., Marcus, C. L., Quan, S. F., Rosen, C. L., Chervin, R. D., et al. (2007). The visual scoring of sleep and arousals in infants and children. *Journal of Clinical Sleep Medicine, 3*, 201–240.

Iber, C., Ancoli-Israel, S., Chesson, A., Quan, S. F., & for the American Academy of Sleep Medicine. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications* (1st ed.). Westchester: American Academy of Sleep Medicine.

Keenan, S., & Hirshkowitz, M. (2011). Monitoring and staging human sleep. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed., pp. 1602–1609). St. Louis: Elsevier.

Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: Developing normative sleep values across the human lifespan. *Sleep, 27*, 1255–1273.

Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized, techniques and scoring system for sleep stages in human subjects (NIH Publication No. 204)*. Washington DC: US Government Printing Office.

Rector, D. M., Schei, J. L., Van Dongen, H. P., Belenky, G., & Krueger, J. M. (2009). Physiological markers of local sleep. *The European Journal of Neuroscience, 29*, 1771–1778.

Siegel, J. M. (2009). Sleep viewed as a state of adaptive inactivity. *Nature Reviews: Neuroscience, 10*, 747–753.

Small-N

- ▶ Outcome for the Single Case: Random Control Index, Single Subject Experimental Design, and Goal Attainment Scale

Smokeless Tobacco

- ▶ Tobacco Control

Smoking

- ▶ Lifestyle Changes
- ▶ Nicotine
- ▶ Tobacco Control

Smoking and Health

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Synonyms

Cigarette smoking and health; Health consequences of smoking; Smoking and health effects; Tobacco smoking and health

Definition

Health represents a physical, mental, and emotional state of well-being. Health is not simply the absence of disease or sickness but can be defined as a state of optimal wellness.

Cigarette smoking is the act of inhaling smoke from burning tobacco. Cigarette smoking is directly related to a decline in health and various associated health outcomes. There is no safe level of smoking; therefore, for optimal health, tobacco use should be avoided.

Description

The adverse health effects of smoking have been well documented. Each year, smoking causes more deaths than murders, suicides, HIV, drug and alcohol use, and auto accidents combined (Centers for Disease Control and Prevention [CDC] 2009). In the United States, cigarette smoking is responsible for over 443,000 deaths, $96 billion in medical expenditures, and 5.1 million years of potential life lost annually (CDC

2009). In fact, 1 out of 5 Americans will die prematurely from the effects of smoking. Tobacco use is responsible for five to six million deaths per year worldwide (Jha 2009). This makes cigarette smoking the single largest preventable cause of disease and death.

Cigarette smoking causes much of its damage to the body through the inhaled smoke (U.S. Department of Health and Human Services et al. 2010). Cigarettes contain over 7,000 chemical compounds, many of which are toxic and/or carcinogenic. It is the inhalation of these chemicals that leads to increased risks for heart disease, cancer, and stroke (USDHHS 2004). Heart disease is the leading cause of death in the USA and particularly among smokers. Smoking cigarettes causes the heart to work harder by raising heart rate and blood pressure (Erhardt 2009). Additionally, poisonous gases such as carbon monoxide limit the amount of oxygen carried in the blood. This results in a two- to four-fold increased risk of heart attack and stroke (USDHHS 2004). Smoking also causes lung disease such as chronic obstructive pulmonary disease (COPD), which includes emphysema and chronic bronchitis (U.S. Department of Health & Human Services et al. 2010). Indeed, smoking causes about 90% of all deaths from chronic airway obstruction (Forey et al. 2011). There is a well-established link between smoking and multiple cancers including the lung, mouth, throat, stomach, uterus, esophagus, cervix, bladder, and acute myeloid leukemia (USDHHS 2004). Nearly one third of all cancer deaths can be linked directly to cigarette smoking (CDC 2010). People who suffer from chronic diseases such as diabetes and HIV are especially vulnerable to the negative effects of tobacco use. Indeed, smokers with a chronic disease may experience increased complications, longer hospitalizations, interactions with medications, and increased risk of death (CDC 2010).

Smoking also takes a toll on mental health and overall well-being. There is a robust relationship between cigarette smoking and perceived stress (Kassel et al. 2003). Smokers tend to report greater stress levels compared to nonsmokers (Cohen and Williamson 1988). And although smokers believe that cigarette smoking provides stress relief, research suggests that smoking might cause additional stress (Heishman 1999). Smoking is also associated with increased risk of affective disorders, such as anxiety and depression. Smokers with mental health disorders report substantially greater symptom burden and functional disability compared to nonsmokers (Covey 1998; McCloughen 2003; Morissette 2007). Smoking also reduces the effectiveness of a number of medications used to help manage the symptoms of depression and schizophrenia (Goff 1992).

Smoking also affects health in other ways. Compared to nonsmokers, smokers have increased risks of blindness, periodontal disease, deafness, sexual dysfunction, sleeping difficulties, headaches, and premature aging, including wrinkles and damage to the skin (USDHHS 2010). Among men, smoking causes an increased risk of erectile dysfunction (Tengs 2001). Women who smoke have increased risk of hip fractures, infertility, and premature menopause (USDHHS 2001).

Smoking not only affects the health of smokers but also impacts individuals around them (USDHHS 2006). Nonsmokers who are exposed to tobacco smoke inhale many of the same toxins and cancer-causing substances as smokers. Secondhand smoke (or passive smoking) is responsible for numerous health problems among adults, including an increased risk of heart disease and cancer (USDHHS 2006). Women who smoke during pregnancy risk passing on the toxins from cigarettes to their babies. Such exposure leads to increased risk for premature labor, low birth weight, and birth defects (USDHHS 2006). Children and infants also suffer from secondhand smoke exposure including asthma attacks, ear infections, respiratory illness, and sudden infant death syndrome (USDHHS 2006).

There are many immediate and long-term health benefits of smoking cessation (USDHHS 2004). After the last cigarette, a person's heart rate and blood carbon monoxide level drop to normal within hours. Lung function and capacity improves within days. The excess risk of coronary heart disease, cancers of the lung and mouth, and

stroke reduce to that of a nonsmoker 1–15 years after quitting smoking (USDHHS 2004). Quitting smoking at any age and despite any existing medical conditions can help improve overall health.

Cross-References

► Smoking Cessation

References and Further Readings

Centers for Disease Control and Prevention. (2009). Annual smoking-attributable mortality, years of potential life lost, and productivity losses-United States, 2000–2004. *Morbidity and Mortality Weekly Report, 58*(2), 29–33. Retrieved from http://www.cdc.gov/mmwr/

Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. (2010). *Cancer statistics 2010*. Retrieved from http://www.cdc.gov/Features/

Cohen, S., & Williamson, G. (1988). Perceived stress in a probability sample of the United States. In S. Spacapan & S. Oskamp (Eds.), *The social psychology of health: Claremont symposium on applied social psychology* (pp. 31–68). Newbury Park: SAGE.

Covey, L. L. S. C. (1998). Cigarette smoking and major depression. *Journal of Addictive Diseases, 17*(1), 35–46.

Erhardt, L. (2009). Cigarette smoking: An undertreated risk factor for cardiovascular disease. *Atherosclerosis, 205*(1), 23–32. https://doi.org/10.1016/j.atherosclerosis.2009.01.007.

Forey, B., Thornton, A., & Lee, P. (2011). Systematic review with meta-analysis of the epidemiological evidence relating smoking to COPD, chronic bronchitis and emphysema. *BMC Pulmonary Medicine, 11*(1), 36.

Goff, D. C. (1992). Cigarette smoking in schizophrenia: Relationship to psychopathology and medication side effects. *The American Journal of Psychiatry, 149*(9), 1189–1194.

Heishman, S. J. (1999). Behavioral and cognitive effects of smoking: Relationship to nicotine addiction. *Nicotine & Tobacco Research, 1*(2), S143–S147. https://doi.org/10.1080/14622299050011971.

Jha, P. (2009). Avoidable global cancer deaths and total deaths from smoking. *Nature Reviews Cancer, 9*(9), 655–664. https://doi.org/10.1038/nrc2703.

Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin, 129*, 270–304. https://doi.org/10.1037/0033-2909.129.2.270.

McCloughen, A. A. (2003). The association between schizophrenia and cigarette smoking: A review of the literature and implications for mental health nursing practice. *International Journal of Mental Health Nursing, 12*(2), 119–129.

Morissette, S. S. B. (2007). Anxiety, anxiety disorders, tobacco use, and nicotine: A critical review of interrelationships. *Psychological Bulletin, 133*(2), 245–272.

Tengs, T. O. (2001). The link between smoking and impotence: Two decades of evidence. *Preventive Medicine, 32*(6), 447.

U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. (2006). *The health consequences of involuntary exposure to tobacco smoke: A report of the surgeon general*. Atlanta: Author.

U.S. Department of Health and Human Services, Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. (2004). *The health consequences of smoking: A report of the surgeon general*. Atlanta: Author.

U.S. Department of Health and Human Services, Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion. (2010). *How tobacco smoke causes disease: The biology and behavioral basis for smoking-attributable disease: A report of the surgeon general*. Atlanta: Author.

U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General. (2001). *Women and smoking: A report of the surgeon general*. Rockville: Author.

Smoking and Health Effects

► Smoking and Health

Smoking Behavior

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Synonyms

Cigarette smoking; Smoking habits; Smoking topography; Tobacco use

Definition

Smoking behaviors are actions taken by a person that are associated with the burning and inhalation of a substance. Smoking behavior is multifaceted and includes the actual act of smoking, puffing style, depth of inhalation, and rate and frequency of smoking.

Description

The act of smoking consists of several behaviors and is usually applied to tobacco/cigarettes. A smoker is defined as a person who has a lifetime history of smoking 100 cigarettes or more with current smoking on some days or every day. Most people experiment with smoking during adolescence and do not intend to become regular, addicted, or dependent smokers. Environmental or social factors (e.g., peer pressure) often play a role in smoking initiation. Over time, smoking behavior can become a pattern (i.e., habit) and tolerance develops. The next step is the development of dependence, which is indicated by both tolerance and withdrawal symptoms during periods of abstinence from smoking. Finally, people maintain the compulsive (e.g., addictive) behavior largely because of nicotine dependence.

Smoking behavior is based on individual differences. Smoking topography is defined as the unique manner in which an individual smokes a cigarette. In particular, topography includes the quantity of puffs per cigarette, puff volume, velocity, and duration (Scherer 1999). Each of these behaviors is a component of how a cigarette is smoked. On average, smokers ingest less than 1.5 mg of nicotine per cigarette (Jarvis et al. 2001; United States Federal Trade Committee 2000). This amount varies depending on cigarette brand and how an individual smokes a cigarette (Djordjevic 2000). Dependent smokers seek to regulate the amount of nicotine they receive to maintain the desired physical and emotional state and to avoid withdrawal. In general, a smoker takes about 10 puffs per cigarette (Scherer 1999). A smoker can consciously or unconsciously control nicotine intake by the time taken in between puffs (referred to as puff frequency or puff interval). Smokers may also block the ventilation holes in filtered cigarettes to increase nicotine delivery. The depths of inhalation, duration of the puff, and the amount of smoke in a puff (volume) are all characteristics of smoking behavior that impact exposure to the chemicals and toxins in cigarettes and the subsequent damage to the body (Scherer 1999).

The frequency of daily smoking is another aspect of smoking behavior. The Centers for Disease Control [CDC] (2005) reported that 83% of all smokers are daily users. Daily smokers average 20 cigarettes per day (CDC 2005). Intermittent or light smokers do not smoke daily and are thought to be less nicotine dependent. The time to the first cigarette of the day and the frequency of daily smoking are indicators of nicotine dependence. Smoking immediately after waking is one of the best predictors of nicotine dependence. In addition, smoking most of one's cigarettes during the first 2 h of the day is suggestive of dependence (Heatherton 1989). A greater frequency of daily smoking may be related to difficulty quitting smoking.

Smoking behavior is variable and can change depending on the circumstance or day. For instance, puffs per cigarette may decrease while watching television or may increase while listening to another person talking. The number or duration of puffs may also change according to emotional state. Research has found that increased smoking often occurs during times of personal crisis and stressors (Shiffman et al. 1996). Many smokers report increased smoking in social situations such as being in the company of other smokers or while attending parties. Smokers also vary in their handling of cigarettes; some may hold the cigarette in their mouth, while others allow them to burn in ashtrays or between their fingers.

Smoking behavior is complex. Learning models suggest that smoking behavior is maintained by operant conditioning, including positive and negative reinforcement, and classical conditioning, through the repeated pairing of

smoking to various physical and emotional states (Wilker 1973). Such learning makes smoking cessation a formidable challenge for most smokers (Patten and Martin 1996).

Cross-References

- ▶ Behavior Change
- ▶ Smoking Cessation
- ▶ Smoking Topography

References and Further Reading

Centers for Disease Control and Prevention. (2005). Cigarette smoking among adults – United States, 2004. *Morbidity and Mortality Weekly Report, 54*(44), 1121–1124. Retrieved 23 November 2011, from http://www.cdc.gov/mmwr/

Djordjevic, M. M. V. (2000). Doses of nicotine and lung carcinogens delivered to cigarette smokers. *Journal of the National Cancer Institute, 92*(2), 106–111.

Heatherton, T. T. F. (1989). Measuring the heaviness of smoking: Using self-reported time to the first cigarette of the day and number of cigarettes smoked per day. *British Journal of Addiction, 84*(7), 791–800.

Jarvis, M. J., Boreham, R., Primatesta, P., Feyerabend, C., & Bryant, A. (2001). Nicotine yield from machine-smoked cigarettes and nicotine intakes in smokers: Evidence from a representative population survey. *Journal of the National Cancer Institute, 93*(2), 134–138. https://doi.org/10.1093/jnci/93.2.134.

Patten, C., & Martin, J. (1996). Does nicotine withdrawal affect smoking cessation? Clinical and theoretical issues. *Annals of Behavioral Medicine, 18*(3), 190–200. https://doi.org/10.1007/BF02883397.

Scherer, G. G. (1999). Smoking behaviour and compensation: A review of the literature. *Psychopharmacology, 145*(1), 1–20.

Shiffman, S., Paty, J., Gnys, M., Kassel, J., & Hickcox, M. (1996). First lapses to smoking: Within-subjects analysis of real-time reports. *Journal of Consulting and Clinical Psychology, 64*(2), 366–379. https://doi.org/10.1037/0022-006X.64.2.366.

United States Federal Trade Committee. (2000). *"Tar," nicotine, and carbon monoxide of the smoke of 1294 varieties of domestic cigarettes for the year 1998*. Retrieved 23 November 2011, from http://www.ftc.gov/reports/tobacco/1998tar%26nicotinereport.pdf

Wilker, A. (1973). Dynamics of drug dependence: Implications of a conditioning theory for research and treatment. *Archives of General Psychiatry, 28*, 611.

Smoking Cessation

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Synonyms

Cigarette smoking cessation; Quit smoking; Stop smoking; Tobacco cessation; Tobacco smoking cessation

Definition

Tobacco smoking is defined as the practice of burning and inhaling tobacco. The combustion from the burning allows the nicotine, tar, and other chemicals and toxins to be absorbed through the lungs. Cigarette smoking is the most prevalent form of consuming tobacco. Most national surveys define a current smoker as having smoked at least 100 (5 packs) cigarettes in their lifetime and currently smokes on at least some days.

Cessation refers to a halting or stopping. Smoking cessation refers to the stopping of cigarette use. Smoking cessation may refer to choosing to stop smoking deliberately or become abstinent due to external and/or environmental factors leading to stopping or quitting smoking.

Description

Smoking cessation is the single most important health behavior change a person can make. Since the 1964 Surgeon General's Report concluded that smoking causes cancer, about 50 million people have successfully quit smoking. Approximately 69% of current smokers state that they want to stop, and 52% have made an attempt to quit smoking in the past year (Centers for Disease Control & Prevention [CDC] 2011).

Smoking cessation has major health benefits. The most common causes of death in the United States are cardiovascular disease, cancer, cerebrovascular accidents, and chronic lower respiratory diseases (Kochanek et al. 2011); smoking cessation has been associated with a reduced risk of dying from these diseases (U.S. Department of Health & Human Services [USDHHS] 1990). The benefits of smoking cessation are greater the earlier one quits, though the benefits of quitting can be experienced even after an extended smoking history. For example, after 15 years of smoking cessation, the excess risk of heart disease for a former smoker is equivalent to a never-smoker. With the increasing duration of cessation, the overall rate of cancer mortality approaches that of nonsmokers (USDHHS).

There are several evidence-based smoking cessation methods. Behavioral interventions include brief physician advice to quit, self-help materials, telephone-based interventions, internet-based counseling and support groups, and group and individual counseling (Fiore et al. 2008). Brief physician advice to quit smoking has been demonstrated to increase the likelihood of cessation by 30%. Self-help cessation interventions increase the likelihood of cessation by 20%. Telephone-based interventions increase the likelihood of cessation by 20%, though tobacco quitlines have increased the odds of cessation by up to 60%. Group and individual counseling increase the likelihood of cessation by 30% and 70%, respectively. The U.S. Food and Drug Administration (FDA) has approved seven smoking cessation pharmacotherapies. These include varenicline (marketed as Chantix in the United States and Champix in Canada and Europe), bupropion (marketed as Zyban), transdermal nicotine patches, nicotine gum, nicotine lozenges, nicotine nasal spray, and the nicotine inhaler. Nicotine patches, gum, and lozenges are available in the USA over the counter; varenicline, bupropion, nicotine nasal spray, and the nicotine inhaler require a physician's prescription. Use of nicotine replacement therapies or bupropion doubles the chances of smoking cessation, while recent evidence suggests that varenicline can triple the likelihood of cessation. Using more than one method when trying to quit (e.g., combination of nicotine replacement with counseling or a telephone quitline) further increases the likelihood of cessation, and the likelihood of cessation increases with the number of formats utilized.

There are also smoking cessation methods that are not based on empirical evidence. Indeed, the most common method of attempting to quit is unassisted quitting (going "cold turkey"). However, approximately 90% of unassisted quit attempts fail. Complementary and alternative methods such as acupuncture, acupressure, laser therapy, and hypnotherapy have been evaluated as smoking cessation methods. However, these methods are not more effective than a placebo (Barnes et al. 2010; White et al. 2011).

Public health policy and campaigns have been instrumental in decreasing the prevalence of smoking in the USA. These policies have restricted indoor smoking via clear indoor air acts (e.g., restaurants, airplanes, and workplaces) in most states and have increased taxes on cigarettes. Public health campaigns have utilized mostly media formats to increase knowledge about the dangers of smoking.

Smoking cessation can result in temporary discomfort, known as nicotine withdrawal. When a person quits smoking, they can enter into nicotine withdrawal within 30 min. The peak of withdrawal occurs within 48–72 h for dependent smokers and usually lasts 1–2 weeks if cessation is maintained. Symptoms of nicotine withdrawal include changes in mood (e.g., irritability, anger, depression, sadness, anxiety, and nervousness), desire or craving to smoke, difficulty concentrating, increased appetite, weight gain, insomnia, restlessness, impatience, constipation, dizziness, coughing, and dreaming or nightmares. The use of FDA-approved cessation aids can minimize withdrawal symptoms.

Smoking cessation is greatly encouraged because of the multiple health benefits that follow. Since the dangers of smoking have been identified, the prevalence has declined and leveled off at 20%. Cigarette smoking is the leading cause of preventable death, disease, and disability in the

USA. Multiple methods to achieve smoking cessation exist, though some of these methods have a greater likelihood of success.

Cross-References

- ▶ Cancer and Smoking
- ▶ Lifestyle Changes
- ▶ Smoking and Health
- ▶ Tobacco Control

References and Further Readings

Barnes, J., Dong, C. Y., McRobbie, H., Walker, N., Mehta, M., & Stead, L. F. (2010). Hypnotherapy for smoking cessation. *Cochrane Database of Systematic Reviews*. https://doi.org/10.1002/14651858.CD001008.pub2.

Centers for Disease Control and Prevention. (2011). Quitting smoking among adults-United States, 2001–2010. *Morbidity and Mortality Weekly Report, 60*(44), 1513–1519.

Fiore, M. C., Jaén, C. R., Baker, T. B., Bailey, W. C., Benowitz, N. L., Curry, S. J., et al. (2008). *Clinical practice guideline: Treating tobacco use and dependence*. Rockville: U.S. Department of Health and Human Services. Public Health Service.

Kochanek, K. D., Xu, J., Murphy, S. L., Miniño, A. M., & Kung, H.-C. (2011). Deaths: Preliminary data for 2009. *National Vital Statistics Reports, 59*(4), 1–51.

U.S. Department of Health and Human Services. (1990). *The health benefits of smoking cessation: A report of the surgeon general*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

United States Department of Health and Human Services (USDHHS). (1982). *The health consequences of smoking: Cancer: A report of the surgeon general*. Rockville: USDHHS.

White, A. R., Rampes, H., Liu, J. P., Stead, L. F., & Campbell, J. (2011). Acupuncture and related interventions for smoking cessation. *Cochrane Database of Systematic Reviews, 1*, CD000009. https://doi.org/10.1002/14651858.CD000009.pub3.

Smoking Habits

- ▶ Smoking Behavior

Smoking Prevention

- ▶ Tobacco Control

Smoking Prevention Policies and Programs

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Synonyms

Tobacco control

Definition

Since up to 80% of smoking initiation has been shown to occur in adolescence, individual and group-level smoking prevention interventions have focused on youth to avert addiction and the long-term health consequences of smoking. A focus of policy efforts for youth prevention has been restricting youth access to tobacco products at the point of purchase. Effective interventions with broader reach to reduce tobacco product initiation among adolescents and adults include policies that increase the unit price of tobacco products and mass media campaigns when combined and coordinated with other interventions.

Description

From 2002 to 2010, the prevalence of past month tobacco use in the United States among those aged 12 and above declined from 30.4% to 27.4%, with stalled reductions in prevalence in the later years. Currently, 23.0% of the population aged 12 or

older smokes cigarettes. Patterns in current tobacco and cigarette use and experimentation with smoking cigarettes were similar for middle school and high school students; while overall prevalence has reached a historic low, no overall declines were noted in youth tobacco use for the 2006–2009 period. Among youth, cigarette smoking is more prevalent among whites than blacks and slightly higher among males than females.

Young people are particularly vulnerable to tobacco addiction due to the effect of nicotine on the reward pathways in the developing brain (from in utero to young adulthood). Additionally, some people are at much greater risk of tobacco use due to genetics, poverty, abuse, neglect, trauma, and other comorbid psychological or cognitive factors such as mood disorders, behavioral, conduct, and attention problems. Beyond the individual, there are numerous variables that affect tobacco use, including adult, household and peer behavior, advertising and promotion, availability and price of tobacco products, poverty, unemployment, neighborhood, community, and cultural norms.

Comprehensive national tobacco strategies use a combination of methods to achieve four main goals: (1) to prevent initiation among youth and young adults, (2) to promote quitting among adults and youth, (3) to eliminate exposure to secondhand smoke, and (4) to identify and eliminate tobacco-related disparities among population groups. Since up to 80% of smoking initiation has been shown to occur in adolescence, individual and group-level smoking prevention interventions have focused on youth to avert addiction and the long-term health consequences of smoking. A focus of policy efforts for youth prevention has been restricting youth access to tobacco products at the point of purchase. Recent data indicates increasing initiation in young adults in the USA, possibly due to targeted marketing efforts by the tobacco industry, and signals the need for extended prevention efforts beyond youth. Effective interventions with broader reach to reduce tobacco product initiation among adolescents and adults include policies that increase the unit price of tobacco products and mass media campaigns when combined and coordinated with other interventions.

Individual and School-Based Programs for Youth

For many years, smoking prevention efforts for adolescents were conducted primarily through school-based programs, which have been shown to have positive short-term effects, but little effect on long-term prevention. Early school-based tobacco curricula focused on social influences, training youth to resist social pressures to use tobacco; studies of these interventions have shown that social influences programming alone is not effective in reducing long-term initiation of smoking. More recent school-based programs have achieved greater success by addressing multiple determinants of tobacco use, including communication skills, coping, personal and social competence, and physical consequences of smoking.

Restricting Youth Access to Tobacco Products

Youth access laws prohibit retailers from selling tobacco products to youth under the age of 18. These laws require enforcement to ensure compliance by the many types of retailers selling tobacco products – from street vendors to convenience stores and online distributors. Adolescent access to cigarette vending machines, low enforcement of these laws, and difficulties in confirming age at purchase in online transactions are all barriers to the success of these laws. Studies indicate that youth access laws can slow increases in adolescent smoking and reduce both smoking prevalence and cigarette consumption, but a very high level of retailer compliance is necessary before these changes occur.

Interventions to Increase the Price of Tobacco Products

Tobacco taxation has been hailed as the most effective intervention to reduce demand for and consumption of tobacco products. Increases in tobacco taxes result in increased cigarette prices, and price has been shown to be a key factor in determining both smoking initiation and cigarette consumption among adults and adolescents.

Price elasticity of demand is defined as the percentage change in consumption of a product following a 1% increase in price. Among US adults, estimates of the price elasticity of cigarette

demand typically fall between −0.3 and −0.5, relating to a 3% or 5% decrease in consumption, respectively, for a 10% increase in price. Adolescents have been shown to be almost three times more sensitive to cigarette price increases than adults for several reasons, including the following: First, adolescents have been posited to be less addicted to nicotine and more able to reduce or quit smoking following cigarette price increases. Second, adolescents typically have a lower income and spend a larger fraction of their disposable income on cigarettes than adults. Third, adolescents are also likely to be present-oriented and may not be willing to spend the additional money on cigarettes at the expense of other activities. Reductions in adolescent smoking following tobacco tax interventions may also result from fewer smoking peers.

Mass Media Campaigns to Prevent Tobacco Use

Tobacco marketing includes all efforts of the industry to promote tobacco products, and industry marketing has been linked to a variety of smoking behaviors among both adults and youth, notably youth tobacco initiation. Countermarketing is a strategy used by public health agencies to protect individuals who may be susceptible to the influence of tobacco industry marketing – particularly youth – by responding directly to that marketing. Prior to the end of 1999, major statewide comprehensive tobacco control programs in California, Massachusetts, Arizona, Oregon, and Florida included countermarketing media campaigns which were shown to reduce adult and youth smoking; results from evaluations of these interventions indicated that these campaigns had more influence on smoking behavior in younger compared to older adolescents. The 1998 Tobacco Master Settlement Agreement (MSA) in the United States included provisions for reducing youth access to tobacco products and restricted marketing in venues or media attended by youth. Funds were also dedicated from the MSA to create a foundation to develop and deliver national anti-tobacco messages. The truth® campaign, a national tobacco countermarketing effort launched in 2000 by the American Legacy Foundation, has repeatedly been shown to be effective in reducing smoking among adolescents, specifically younger adolescents. Around the same time, tobacco companies developed youth smoking prevention media campaigns which have been shown to be ineffective, and in the case of parent-targeted advertising, to reduce perceptions of smoking-related harm and to increase approval of smoking and intention to smoke among older adolescents.

Future Directions in Smoking Prevention

Passage of the Family Smoking Prevention and Tobacco Control Act in 2009 gave the Food and Drug Administration (FDA) authority to regulate tobacco products and their marketing to protect the public health. One of FDA's first actions was to ban candy flavorings in cigarettes shown to be appealing to youth in order to reduce youth smoking initiation. Other possible regulatory actions aimed at tobacco use prevention include banning menthol in cigarettes, reducing the amount of nicotine in cigarettes to a non-addictive level, and introducing large, graphic warning labels on health effects of tobacco use to cigarette and smokeless tobacco packaging. The national scale of regulatory action through the FDA has the potential to dramatically influence population-level tobacco use and must be complemented by tobacco control efforts at the community, local, and state levels to achieve the maximum impact on prevention.

Summary

Comprehensive tobacco control programs have taken a community-based approach to smoking prevention, using a combination of school-based, policy, educational, and mass media interventions to change the social environment and conditions related to smoking behavior with positive results. Reviews of community-based programs show that coordinated multicomponent interventions reduce smoking among young people more than single strategies alone and that the level of program funding and implementation is critical to the success of these interventions. A population-based health promotion approach that includes media, education, screening, interventions in community settings and policy can provide avenues to address tobacco use more comprehensively. Coordination

of policy and program efforts from the local to the national level is needed to enhance the effectiveness of tobacco use prevention interventions.

Cross-References

- ▶ Smoking and Health
- ▶ Smoking Behavior
- ▶ Smoking Cessation
- ▶ Tobacco Cessation
- ▶ Tobacco Use

References and Further Readings

American Legacy Foundation. *Youth smoking prevention mass media campaign*. Retrieved from http://www.thetruth.com/

Center for Tobacco Products, Food and Drug Administration. Retrieved from http://www.fda.gov/tobaccoproducts/default.htm

Centers for Disease Control and Prevention. (2007). *Best practices for comprehensive tobacco control programs-2007*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. Retrieved from http://www.cdc.gov/tobacco/stateandcommunity/best_practices/index.htm

Farrelly, M. C., Davis, K. C., Haviland, M. L., Messeri, P., & Healton, C. G. (2005). Evidence of a dose–response relationship between "truth" antismoking ads and youth smoking prevalence. *American Journal of Public Health, 95*(3), 425–431.

Institute of Medicine. (2007). *Ending the tobacco problem: A blueprint for the nation*. Washington, DC: National Academies Press. Retrieved from http://www.iom.edu/Reports/2007/Ending-the-Tobacco-Problem-A-Blueprint-for-the-Nation.aspx

Substance Abuse and Mental Health Services Administration. (2011). *Results from the 2010 National Survey on drug use and health: Summary of national findings*. NSDUH Series H-41, HHS Publication No. (SMA) 11-4658. Rockville: Substance Abuse and Mental Health Services Administration. Retrieved from http://www.samhsa.gov/data/NSDUH/2k10Results/Web/PDFW/2k10Results.pdf

Task Force on Community Preventive Services. (2005). Tobacco. In: S. Zaza, P. A. Briss, & K. W. Harris (Eds.), *The guide to community preventive services: What works to promote health?* (pp. 3–79). Atlanta: Oxford University Press. Retrieved from http://www.thecommunityguide.org/tobacco/index.html

Smoking Profile

▶ Smoking Topography

Smoking Topography

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Synonyms

Puff topography; Smoking behavior; Smoking profile

Definition

Smoking topography is a representation of the physical characteristics of smoking behavior, such as puff count, puff volume, average flow, puff duration, and interpuff interval.

Description

An individual's interaction with a substance (e.g., tobacco, marijuana) or object (e.g., pipe, e-cigarette) used for smoking is highly complex, multifaceted, and distinct. Smoking topography attributes have been found to be a function of sex, personality, stress level, nicotine yield, cigarette type (i.e., menthol versus non-menthol), ethnicity, and body mass index.

An array of subjective and objective methods exist to measure smoking topography, each differing in accuracy, precision, and feasibility. These include observation, self-report, cigarette weighing, expired carbon monoxide breath levels, and biomarkers (e.g., nicotine, cotinine, or thiocyanate). Specialized instruments (e.g., pressure transducers, flowmeters, and puff analyzers)

provide an unrestricted assessment of smoking topography.

Smoking topography has been examined across various disciplines and populations, such as nicotine metabolism, smoking cessation, exercise, adolescents, and individuals with schizophrenia. There is an impetus to further study smoking topography as it is reflective of the rewarding and reinforcing effects of addictive substances, compensation, harm reduction efforts, and exposure to harmful elements and potential carcinogens.

Cross-References

- ▶ Cancer and Smoking
- ▶ Cessation Intervention (Smoking or Tobacco)
- ▶ Nicotine
- ▶ Smoking and Health
- ▶ Smoking Behavior
- ▶ Smoking Cessation

References and Further Readings

Benowitz, N. L. (2001). Compensatory smoking of low-yield cigarettes. In National Cancer Institute (Ed.), *Risks associated with smoking cigarettes with low machine-measured yields of tar and nicotine* (pp. 39–64). Bethesda: US Department of Health and Human Services, National Institutes of Health, National Cancer Institute.

De Jesus, S., Hsin, A., Faulkner, G., & Prapavessis, H. (2015). A systematic review and analysis of data reduction techniques for the CReSS smoking topography device. *Journal of Smoking Cessation, 10*, 12–28. https://doi.org/10.1017/jsc.2013.31.

Shahab, L., West, R., & McNeill, A. (2008). The feasibility of measuring puffing behaviour in roll-your-own cigarette smokers. *Tobacco Control, 17*(Suppl I), i17–i23. https://doi.org/10.1136/tc.2007.021824.

SNP (Pronounced "Snip")

- ▶ Single Nucleotide Polymorphism (SNP)

Social Behavior

- ▶ Interpersonal Circumplex

Social Capital and Health

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Social capital has no direct synonyms. Social cohesion is a broader concept which is *not* directly synonymous with social capital. Social cohesion is a combination of absence of latent social conflict, e.g., in the form of social and economic inequality, ethnic tensions or other forms of polarization, and the presence of strong social bonds measured by levels of trust and reciprocity (i.e., social capital), a strong "civil society" which bridge social divisions, and institutions of conflict management (e.g., a responsive democracy and an independent judiciary) (Kawachi and Berkman 2000).

Definition

The definition of social capital varies by author and academic tradition. Robert Putnam (political science) defines social capital as "features of social organization, such as trust, norms, and networks that can improve the efficiency of society by facilitating coordinated actions" (Putnam 1993). According to James Coleman (sociology), social capital is ". . .a variety of different entities (that) facilitate certain actions of individuals who are within a structure" (Coleman 1990), while Pierre Bourdieu (sociology) defines it as ". . .the sum of the resources, actual or virtual, that accrue to an individual or a group by virtue of possessing a durable network" (Bourdieu and Wacquant 1992). The definitions of Putnam, pertaining to the population level of social capital, and Coleman, pertaining to social networks of

individuals, have been more commonly used in behavioral medicine and public health than that of Bourdieu, which is closer to the individual level, is less distinct in relation to social support, and also concerns the power structure within social groups and structures. Health as a concept is defined elsewhere.

Description

The social capital concept was first used in 1916 but originates in its modern context in the fields of sociology and political science. Social capital concerns the characteristics of interaction between actors such as individuals, social groups, and organizations rather than the characteristics of actors per se. Social capital includes interpersonal trust, trust in institutions, reciprocity, civic participation, social participation, and social networks which increase cooperation and decrease transaction costs in society.

The first articles concerning social capital and health appeared in international medical and public health journals in the mid-1990s with a fast increase in the number of publications after 1996–1998. From approximately 40 international journal publications in 2001 (Macinko and Starfield 2001), the number of journal publications has increased to 1,179 in 2011 in PubMed only (December 19, 2011). Already in 2001, Macinko and Starfield identified four levels of analysis of social capital and health: the macro (countries, regions), meso (neighborhoods, municipalities), micro (social networks), and psychological (trust) levels (Macinko and Starfield 2001). Putnam's definition of social capital, which includes trust despite Putnam's macro and meso perspectives because the level of trust may be regarded as a trait of populations and countries just as well as a cognitive trait of the individual, has, together with Coleman's definition, had the strongest impact in the behavioral medicine and public health literature, although other authors such as Bourdieu and Portes are also referred to and their definitions also used in empirical studies.

Although Putnam had earlier stated that the area of health and public health was probably a research area not particularly strongly associated with and affected by social capital (Putnam 1993), he 7 years later contended, based on the dramatic increase of results of empirical studies, that health and public health was probably one of the most important areas associated with and affected by social capital (Putnam 2000). Kawachi et al. (1999) suggested four main causal pathways by which social capital may affect health: direct psychological and psychosocial stress pathways, social norms and values which foster health-related behaviors, access to health care and amenities through social networks and contacts, and crime, particularly violent crime (Kawachi et al. 1999).

Kawachi et al. (1997) found in an early ecological study that low levels of social capital were associated with a higher total mortality rate and higher rates of a wide range of major causes of death, including coronary heart disease (CHD), cerebrovascular disease, unintentional injury, and infant mortality (Kawachi et al. 1997). Later epidemiological, ecological, and individual level studies have, just to give some examples, revealed significant associations between low levels of social capital and higher age-adjusted mortality rates, shorter life expectancy, higher mortality and violent crime rates, higher coronary heart disease morbidity and mortality, low birth weight rates, higher incidence rates of a variety of sexually transmitted diseases, low life satisfaction, less happiness, poor self-rated health, poor mental health (measured with the GHQ12 index), poor physical and mental health measured by SF-12, depression, psychiatric morbidity, higher suicide rates, worse chronic conditions, functional limitations (Islam et al. 2006), and health-related behaviors such as lower levels of leisure time physical activity (Lindström et al. 2001). Some studies have investigated associations between vertical social capital, i.e., trust and participation across a well-defined power gradient, and health (Sundquist et al. 2006). Multilevel studies, which mostly include the individual level and one or more contextual levels of analysis, have e.g. shown that low social capital is associated with poor self-rated health and violent crime

(Islam et al. 2006). Multilevel analyses have also demonstrated significant associations between social capital and access to health care (Lindström et al. 2006). Multilevel analyses have increasingly been conducted in order to disentangle the associations between contextual level social capital and individual level health in order to avoid the "ecological fallacy," which denotes the risk of drawing erroneous conclusions concerning individual health from observations of ecological data, and the "individualist fallacy," which denotes the risk of missing associations and effects of neighborhood or other forms of contextual social capital on individual health.

Since social capital is a characteristic of social relations, trust, and cooperation between individuals, groups, and organizations rather than of individuals, one important issue related to social capital and health is to define relevant social contexts for the analysis of the association and impact of contextual social capital on health and health-related phenomena such as health-related behaviors and access to health care and amenities. Most multilevel studies include neighborhoods or other geographic entities as second, third, and so forth levels of analysis. However, one question concerns whether geographic entities are the most relevant social contexts. This discussion is highly relevant because many multilevel studies have shown small or moderate associations between neighborhood and geographic social capital and health of individuals within single countries, while the statistical associations between social capital and health have been stronger and more consistent in individual level studies. Recently, some prospective cohort studies have investigated workplace social capital and found significant effects on, e.g., depression (Kouvanonen et al. 2008). In studies of social capital and school children and adolescents, the family and schools are relevant social contexts in addition to neighborhoods. Putnam suggested already in 2000 that the Internet would become a relevant area for studies of social capital and social networks (Putnam 2000).

Most studies of social capital and health are cross-sectional, i.e., they measure all factors at the same point in time which makes causal inference formally impossible. In many instances, causality may go in both directions. Social capital may, e.g., cause poor mental health, but mental health may also affect social capital in the forms of social participation in social networks, civic participation, and feelings of trust and reciprocity. A few longitudinal studies with panel data including three waves of observations or more have been conducted in recent years (Giordano and Lindström 2011).

Social capital is not always associated with better health. Szreter and Woolcock developed the concepts of bonding, bridging, and linking social capital. Bonding social capital denotes "trusting and cooperative relations between members of a network who see themselves as being similar in terms of their shared social identity," while bridging social capital refers to "relations of respect and mutuality between people who know they are not alike in some sociodemographic (or social identity) sense (differing by age, ethnic group, class, etc.)" (Szreter and Woolcock 2004). While many social networks, associations, and organizations, e.g., youth organizations, sports clubs, and labor unions, manage to combine bonding and bridging social capital, some other, e.g., criminal networks, do not. This phenomenon of exclusion of outsiders and the rest of society is sometimes referred to as "the dark side of social capital," and its effects on health may be detrimental. Linking social capital refers to "norms of respect and networks of trusting relationships between people who are interacting across explicit, formal, or institutionalized power or authority gradients in society" (Szreter and Woolcock 2004).

Critique against the research concerning social capital and health has been expressed by the so-called neo-materialists, who claim that it obscures underlying ideological, political, administrative, and economic determinants of health inequalities and other public health issues. The neo-materialists emphasize the importance of active governments, active welfare politics, and economic preconditions for the realization of public health programs (Navarro 2004).

Cross-References

- ▶ Cross-Sectional Study
- ▶ Longitudinal Study
- ▶ Multilevel Analysis
- ▶ Social Cohesion
- ▶ Social Support

References and Further Readings

Bourdieu, P., & Wacquant, L. (1992). *Invitation to reflexive sociology*. Chicago: University of Chicago Press.

Coleman, J. S. (1990). *Foundations of social theory*. Princeton: Harvard University Press.

Giordano, G. N., & Lindström, M. (2011). Social capital and change in psychological health over time. *Social Science and Medicine, 72*, 1219–1227.

Islam, K., Merlo, J., Kawachi, I., Lindström, M., & Gerdtham, U. (2006). Social capital and health: Does egalitarianism matter? A literature review. *International Journal for Equity in Health, 5*, 3.

Kawachi, I., & Berkman, L. (2000). Social cohesion, social capital, and health. In L. Berkman & I. Kawachi (Eds.), *Social epidemiology* (pp. 174–190). Oxford: Oxford University Press.

Kawachi, I., Kennedy, B. P., Lochner, K., & Prothrow-Stith, D. (1997). Social capital, income inequality, and mortality. *American Journal of Public Health, 87*, 1491–1498.

Kawachi, I., Kennedy, B. P., & Wilkinson, R. G. (1999). Social capital and self-rated health: A contextual analysis. *American Journal of Public Health, 89*, 1187–1193.

Kouvanonen, A., Oksanen, T., Vahtera, J., Stafford, M., Wilkinson, R., Schneider, J., et al. (2008). Low workplace social capital as a predictor of depression. The Finnish public sector study. *American Journal of Epidemiology, 167*(10), 1143–1151.

Lindström, M., Hanson, B. S., & Östergren, P. O. (2001). Socioeconomic differences in leisure-time physical activity: The role of social participation and social capital in shaping health related behaviour. *Social Science and Medicine, 52*, 441–451.

Lindström, M., Axén, E., Lindström, C., Beckman, A., Moghaddassi, M., & Merlo, J. (2006). Social capital and access to a regular doctor: A multilevel analysis in southern Sweden. *Health Policy, 79*, 153–164.

Macinko, S., & Starfield, B. (2001). The utility of social capital in research on health determinants. *The Milbank Quarterly, 79*(3), 387–427.

Navarro, V. (2004). Commentary: Is social capital the solution or the problem? *International Journal of Epidemiology, 33*, 672–674.

Putnam, R. D. (1993). *Making democracy work. Civic traditions in modern Italy*. Princeton: Princeton University Press.

Putnam, R. D. (2000). *Bowling alone. The collapse and revival of American community*. New York/London: Simon and Schuster.

Sundquist, J., Johansson, S. E., Yang, M., Sundquist, J., Johansson, S. E., Yang, M., et al. (2006). Low linking social capital as a predictor of coronary heart disease in Sweden: A cohort study of 2.6 million people. *Social Science and Medicine, 62*, 954–963.

Szreter, S., & Woolcock, M. (2004). Health by association? Social capital, social theory, and the political economy of public health. *International Epidemiology, 33*, 650–667.

Social Circumstance

- ▶ Sociocultural

Social Cohesion

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Definition

Social cohesion (SC) refers to the degree to which links between a society's members are strong and the degree to which people in a community share values and goals and are interdependent. Durkehiem conceptualized SC as a major protective variable against adversities including suicide. In his seminal work, Durkheim found that Catholics have lower suicide rates than Protestants, and he attributed this to greater social control and cohesion in the former than among the latter (Pickering and Walford 2000). Kelleher and Daly (1990) found that reduced SC (indexed by reduced marriage rates and increased separations) was among the variables possibly contributing to increased suicide rates in Ireland, between 1970 and 1985. The Dutch sociologist Geert Hofstede conducted the first empirically based quantification of international cultural dimensions in the 1970s in IBM plants around the world. Among

his four main cultural dimensions, individualism emerged, which can be seen as the opposite of SC. Individualism from Hofstede's scores does predict on a global level present suicide rates (Gidron and Ferreira 2012). Recent studies have also tested the relationship between SC and various health outcomes, pertinent to behavioral medicine. Chaix et al. (2008) found in Sweden that low SC predicted myocardial infarctions, independent of important demographic variables. More recently, Clark et al. (2011) found that each one point increase in SC predicted a reduction of 53% in deaths attributed to stroke, independent of confounders. SC was assessed individually by six items reflecting social interactions and contacts with neighbors. One mechanism could be that more cohesive societies (such as religious kibbutzim in Israel vs. secular ones) also promote a healthier psychosocial profile – higher sense of coherence and lower hostility levels (Kark et al. 1996). The concept of SC is thus one domain where concepts important in behavioral medicine and sociology may influence health at the more macro level of societies. One type of simple intervention which may increase SC is Walton and Cohen's social belonging method, where people are indirectly shown that others experience similar negative experiences as they do. This was found to raise academic performance in vulnerable or stigmatized people (Walton and Cohen 2007). This could have important implications for preventative medicine and public health. Programs for increasing the sense of belongingness and SC need to be developed and adapted to large-scale social contexts, and their effects on health outcomes can then be empirically examined.

Cross-References

- ▶ Social Support
- ▶ Suicide Risk, Suicide Risk Factors

References and Further Readings

Chaix, B., Lindström, M., Rosvall, M., & Merlo, J. (2008). Neighborhood social interactions and risk of acute myocardial infarction. *Journal of Epidemiology and Community Health, 62*, 62–68.

Clark, C. J., Guo, H., Lunos, S., Aggarwal, N. T., Beck, T., Evans, D. A., et al. (2011). Neighborhood cohesion is associated with reduced risk of stroke mortality. *Stroke, 42*, 1212–1217.

Gidron, Y., & Ferreira, O. (2012). *Culture & international suicide rates*. Paper presented at the sixth annual international conference on psychology, May, Athens.

Kark, J. D., Carmel, S., Sinnreich, R., Goldberger, N., & Friedlander, Y. (1996). Psychosocial factors among members of religious and secular kibbutzim. *Israel Journal of Medical Sciences, 32*, 185–194.

Kelleher, M. F., & Daly, M. (1990). Suicide in Cork and Ireland. *British Medical Journal, 157*, 533–538.

Pickering, W. S. F., & Walford, G. (Eds.). (2000). *Durkheim's suicide: A century of research and debate*. London: Psychology Press/Routledge.

Walton, G. M., & Cohen, G. L. (2007). A question of belonging: Race, social fit, and achievement. *Journal of Personality and Social Psychology, 92*, 82–96.

Social Conflict

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Synonyms

Interpersonal conflict; Negative social interaction; Relational distress; Relationship conflict; Social stress

Definition

Social conflict refers to the various types of negative social interactions that may occur within social relationships including, but not limited to, arguments, rejection, criticism, hostility, insensitivity, unwanted demands, and ridicule (Seeman 2001). Social conflict can also escalate to include physical violence. Conflict refers to overt behavior rather than subjective states. It typically results from purposeful interaction among two parties in a competitive setting in which the parties are aware of the incompatibility of positions. Social conflict can extend to different social relationships

including those with significant others such as spouses, family members, and friends, as well as less intimate relationships.

Description

Studies on social conflict and interpersonal stress suggest that negative aspects of social interactions including social conflict and social control are inversely related to emotional well-being and health (Miller et al. 2009). There is substantial evidence that interactions marked by acute conflict and negative emotions have direct physiological consequences. Epidemiological studies have linked social isolation, low social support, and high levels of social conflict with morbidity and mortality (Miller et al. 2009). Changes in immune function and elevated inflammation have been suggested as key pathways underlying the association between social conflict and health (Kiecolt-Glaser et al. 2010). Inflammation is a key pathogenic mechanism in many infections and cardiovascular and neoplastic diseases. Recent studies have also linked stressful interpersonal relationships to alterations in gene expression and intracellular signaling mechanisms (Miller et al. 2009).

Consistent evidence also points to the strong relationship between social conflict and psychological distress. Specifically, interpersonal stress and conflict with family and friends has been reliably associated with negative affect, depression, and emotional stress responses (Graham et al. 2007). Depression has been proposed as a particularly important psychological mechanism by which social conflict in close relationships affect immune function (Miller et al. 2009). The literature indicates that social conflict is associated with a range of physiological and psychological mechanisms that are in turn associated with concomitant alterations in the cardiovascular, endocrine, and immune systems. Family conflict and discord and domestic violence have also been linked to suicidal behavior (Van Orden et al. 2010). In drug abusers, interpersonal conflict has been also associated with an increased probability of a history of suicide attempt and ideation (Van Orden et al. 2010).

Social conflict has been assessed using self-report questionnaires about relationship quality and quantity as well as in experimental studies of laboratory-induced marital conflict (Kiecolt-Glaser et al. 2010). To date, there is not one standard measure to assess social conflict. Different types of study designs have been used to assess the impact of social conflict on physical and emotional well-being including correlational studies, experimental studies of couples, and animal studies (see Kiecolt-Glaser et al. 2010). Correlational studies suggest that social isolation, lack of social support, and interpersonal conflict are associated with biological markers of inflammation (i.e., C-reactive protein and interleukin-6). More recent studies have also suggested a plausible link between distressed pair-bond relationships and plasma levels of oxytocin in females and vasopressin in males. However, this research is still preliminary. In other correlation studies, men and women who had recently undergone a marital separation or divorce had poorer immune function than demographically matched married individuals (see Kiecolt-Glaser et al. 2010).

According to Kiecolt-Glaser et al. (1998), being married is not always protective, especially if there is frequent interpersonal conflict in the couple. In experimental studies with married couples, this group of researchers has shown that conflictive social interactions consistently result in heightened blood pressure and heart rate, especially for those with high trait hostility. Conflict discussion tasks are also widely used in marital research by this group and others. Discussion of marital problems has been associated with both immediate and longer term physiological changes related to the degree of negativity or hostility displayed during conflict (see Kiecolt-Glaser et al. 2010). Gender differences have been observed in these studies, with women evidencing greater sensitivity to negative marital interactions than men. Similarly, marital conflict shows a greater impact on health and physiological functioning in older adults compared to young couples. In general, this research suggests that relationships that are stable and long lasting but marked by social conflict have the potential to function as both an acute and chronic stressor that may impact health over an extensive period

of time (Kiecolt-Glaser et al. 2010). Relationship conflict and termination can also provoke detrimental health behaviors including disturbed sleep, unhealthy diets, less physical activity, smoking, and greater use of alcohol and other drugs. Thus, relationships characterized by hostility and conflict could have negative health consequences.

The most common social conflict models involving laboratory animals are variations of the resident-intruder model wherein one animal is placed in the home cage of another. Animal studies have shown that social conflict and disruption of social relationships have important immunological and endocrine consequences (Huhman 2006). In rodents, an aggressive social encounter is typically accompanied by elevated levels of stress hormones and changes in cellular and humoral immunity. Exposure to social conflict appears to have long-lasting behavioral and physiological effects not just in defeated/subordinate animals but also in dominant ones that appear to be moderated by developmental level (Huhman 2006). Data from experimental primate models also show that stressful social relationships can exacerbate viral infections by altering gene expression responses to infection. Social conflict models in animals may be useful for studying the physiological concomitants of a number of psychiatric disorders including major depression (see Huhman 2006).

Cross-References

- ▶ Family Stress
- ▶ Interpersonal Relationships
- ▶ Marriage and Health

References and Readings

Graham, J. E., Christian, L. M., & Kiecolt-Glaser, J. K. (2007). Close relationships and immunity. In R. Ader (Ed.), *Psychoneuroimmunology* (pp. 781–798). Burlington, MA: Elsevier Academic Press.

Huhman, K. L. (2006). Social conflict models: Can they inform us about human psychopathology? *Hormones and Behavior, 50*, 640–646.

Kiecolt-Glaser, J. K., Glaser, R., Cacioppo, J. T., & Malarkey, W. B. (1998). Marital stress: Immunologic, neuroendocrine and autonomic correlates. *Annuals of New York Academic Sciences, 840*, 656–663.

Kiecolt-Glaser, J. K., Gouin, J. P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews, 35*, 33–38.

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–524.

Seeman, T. (2001). How do others get under our skin: Social relationships and health. In C. D. Ryff & B. H. Singer (Eds.), *Emotion, social relationships and health* (pp. 189–209). New York: Oxford University Press.

Van Orden, K. A., Witte, T. K., Cukrowicz, K. C., Braithwaite, S. R., Selby, E. A., & Joiner, T. E. (2010). The interpersonal theory of suicide. *Psychological Review, 117*, 575–600.

Social Determinants of Health

▶ Social Factors

Social Ecological Framework

▶ Ecological Models: Application to Physical Activity

Social Ecological Model

▶ Ecological Models: Application to Physical Activity

Social Epidemiology

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Definition

While epidemiology is defined as the study of the distribution and determinants of disease and typically treats social determinants as background to

biomedical phenomena, social epidemiology is a sphere of enquiry in its own right. It is distinguished by explicitly investigating social determinants of population distributions of health, disease, and well-being. Social epidemiology was perhaps first coined as a discipline in the 1950s; postgraduate programs in social epidemiology are now offered.

Cross-References

- ▶ Epidemiology
- ▶ Socioeconomic Status (SES)

References and Further Reading

Berkman, L., & Kawachi, I. (Eds.). (2000). *Social epidemiology*. New York: Oxford University Press.
Krieger, N. (2001). A glossary for social epidemiology. *Journal of Epidemiology and Community Health, 55*(10), 693–700.

Social Factors

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Synonyms

Social determinants of health; Socioeconomic status (SES)

Definition

The conditions under which people are born, live, work, and age are collectively known as the social determinants of health. These social factors include both economic and social conditions and are may be responsible for the health inequities both within and between countries.

Description

Research into the social factors underpinning health arose from the dual observations of health disparities both within and between countries. These disparities can be easily (although crudely) demonstrated by way of differences in life expectancy for different groups.

Between-country differences in adult mortality are growing rapidly. Whereas mortality rose in Africa, Central and Eastern Europe between 1970 and 2002, global mortality fell overall during the same period (World Health Organization 2003). These differences in mortality rates are especially stark when comparing life expectancy by country. For example, while the average life expectancy at birth is 89.78 years in Monaco, it is just 29.3 years in Haiti (CIA 2011).

Large differences in morbidity and mortality are also apparent within the same country. For example, in Australia, the life expectancy for Aboriginal and Torres Strait Islanders is approximately 10 years below the national average (Australian Bureau of Statistics 2010).

Study of the social factors that underlie health provides possible mechanisms by which this large health disparity can be understood. The major social factors which influence health include both structural determinants of health, and the conditions of an individual's daily life. Research linking social factors to health outcome has identified a number of key social determinants of health (e.g., Wilkinson and Marmot 2003). These include:

- The social gradient
- Stress
- Early life
- Social exclusion
- Work and unemployment

These factors are thought to influence health both indirectly and directly. In many cases, social factors act in combination to have a cumulative impact on health. For example, while unemployment is known to have a direct influence on

health, it is also likely to have an indirect influence on health through its influence on other social factors (e.g., stress).

The Social Gradient

It is widely recognized that poverty is related to high levels of illness and disease. Data consistently shows that the health status of individuals in the poorest countries leads to significantly lower life expectancy and significantly higher risk of disease than that of individuals in richer countries.

The influence of socioeconomic status is also apparent within countries and appears to have an influence on health across the socioeconomic spectrum. This effect, known as the social gradient, shows that even within industrialized countries, both mortality and morbidity are linearly distributed across different levels of socioeconomic advantage and disadvantage. This research suggests that even moderate differences in wealth, and associated social factors, can have an important impact on health status.

For more information, see Social Class.

Early Life

Maternal deprivation and ill-health during pregnancy have both been linked to poor fetal development. Factors such as malnutrition, maternal stress, and inadequate prenatal care all increase the risk of childhood mortality and have profound impacts on health throughout later life.

Research has also linked circumstances during infancy to both physical and mental health in adult life. Social functioning, physical health, and the performance of health-protective behaviors have all been linked to experiences in early life.

For more information, see Maternal Stress; Birth Weight; Child Neglect; Child Development; Stress, Early Life.

Stress

Social and psychological circumstance can have a profound impact on the number of stressors an individual is exposed to and the level of stress that they experience. Increased levels of stress have been linked to increased risk of both morbidity and mortality.

Research suggests that exposure to stressors can have both direct and indirect influences on health. Chronic stress can directly increase risk of disease through its influence on physiological processes such as immune function. However, both acute and chronic stress can also lead to high rates of health-damaging behaviors such as alcohol consumption and smoking.

For more information, see Stress; Stressor.

Social Exclusion

Social exclusion relates to the isolation of certain member within a society and is associated with high rates of disease and mortality. It is most often related to relative poverty, low educational attainment, unemployment, and experiences of stigma and discrimination.

Individuals who experience social exclusion are subject to a pattern of multiple deprivations that prevent them from participating fully in society. This is often characterized by poor access to services, including health care, housing, education, and transport.

For more information, see Social Capital and Health; Stigma.

Work and Unemployment

Research suggests that work and unemployment have separate – but at times overlapping – influences on health.

In general, being unemployed places individuals at increased risk of a number of diseases (e.g., depression and heart disease). The effects of unemployment on health are linked to both

material deprivation that may occur due to a lack of income, and to the psychological consequences of being unemployed. The influence of unemployment on health appears to manifest itself even before individuals become unemployed, such that experiencing high levels of job insecurity (in many cases, a precursor of unemployment) can be as harmful to health as being unemployed.

In addition to the role of perceived job security, it appears that there are a range of factors that determine the extent to which employment is health protective. These include:

- Level of job control
- Level of job demand
- Adequacy of rewards for job performance

In particular it appears that professions that are characterized by low control but high demand place workers at increased risk of ill-health. Importantly social support and recognition of job performance, either through higher wages or increased social status, both appear to be health protective.

For more information, see Work-Related Stress.

Social Causation and Social Drift: Explanation of Possible Causal Relationships Between Social Factors and Health

When attempting to understand the influence of social factors on health, it is important to consider the possible causal relationships between these social factors and health. Broadly speaking, there are two possible ways of understanding the causal relationship between these factors. These explanations are described below using the example of the social gradient (Morrison and Bennett 2008).

The first explanation – called social causation – would suggest that low socioeconomic status is causally related to health problems (i.e., that there is something about low socioeconomic status that "causes" ill-health). For example, an individual who experiences being socioeconomically disadvantaged may be exposed to poor living conditions which cause later health problems.

The second explanation – called social drift – would suggest that ill-health is causally related to low socioeconomic status (i.e., that experiencing illness "causes" an individual to lose their socioeconomic position). For example, an individual who has to leave work due to illness may find that without a steady income, they are now socioeconomically disadvantaged.

Longitudinal studies provide evidence for both explanations. For example, studies have consistently shown that low socioeconomic status baseline is a predictor of heart disease at follow-up and that individuals who become unemployed are more likely to suffer from health complaints than individuals who stay employed. On the basis of this evidence, it would appear most likely that social factors and health have a bidirectional relationship (Morrison and Bennett 2008).

Cross-References

- ▶ Birth Weight
- ▶ Child Development
- ▶ Child Neglect
- ▶ Health Care Access
- ▶ Maternal Stress
- ▶ Social Capital and Health
- ▶ Stigma
- ▶ Stress
- ▶ Stress, Early Life
- ▶ Stressor
- ▶ Work-Related Stress

References and Readings

Australian Bureau of Statistics. (2010). *Experimental life tables for Aboriginal and Torres Strait Islander Australians, 2005–2007 (Cat. No. 3302.0.55.003)*. Canberra: Author.

CIA. (2011). *The world factbook: Life expectancy at birth*. Retrieved 18 Nov 2010 from https://www.cia.gov/library/publications/the-world-factbook/rankorder/2102rank.html#

Marmot, M. (2005). Social determinants of health inequalities. *The Lancet, 365*(9464), 1099–1104.

Morrison, V., & Bennett, P. (2008). *An introduction to health psychology*. New York: Prentice Hall.
Wilkinson, R. G., & Marmot, M. G. (2003). *Social determinants of health: The solid facts*. Copenhagen: World Health Organization.
World Health Organization. (2003). *World health report 2003: Shaping the future*. Geneva: Author.

Social Health

▶ Racial Inequality in Economic and Social Well-Being

Social Inhibition

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Synonyms

Behavioral inhibition

Definition

Social inhibition (SI) is a broad personality trait that refers to *the stable tendency to inhibit the expression of emotions and behaviors in social interaction* (Asendorpf 1993). Individuals who are high in SI are more likely to feel inhibited, tense, and insecure when with others. In children, the label *behavioral inhibition* is often used to describe this tendency (Gest 1997).

SI is more closely related to the *interpersonal dimension* of introversion/extraversion than to intrapsychic facets of extraversion such as positive affect or excitement seeking. Infants and children with an inhibited temperament tend to develop into adults who avoid people and situations that are novel or unfamiliar (Schwartz et al. 2003). Hence, individuals who are high in SI try to avoid potential "dangers" involved in social interaction, such as disapproval or criticism by others, through the deliberate inhibition of self-expression (Asendorpf 1993). They tend to experience discomfort in encounters with other people, may keep other people at a distance, and are less likely to actively seek social support.

Interestingly, recent imaging research demonstrates the effect of social inhibition on the *neural coding of threatening signals* in the human brain (Kret et al. 2011). Socially inhibited adults may show greater signal response in the brain to threatening stimuli than adults who are not socially inhibited (Schwartz et al. 2003). Other research also indicates that socially inhibited people tend to overactivate a broad cortical network in the brain when looking at fearful or angry facial and bodily expressions (Kret et al. 2011).

There are several reasons why it is important to account for individual differences in SI in clinical research and practice. First, SI has been related to *difficulties in coping with the challenges of everyday life*. For example, inhibited children may show a delay in establishing a first stable partnership and finding a first full-time job in early adulthood (Asendorpf et al. 2008). Second, socially inhibited individuals may seem quiet on the surface, while they may actually *avoid interpersonal conflict* through excessive control over their emotional and behavioral responses. Third, SI has been associated with an increased long-term *risk of developing internalizing problems* (Asendorpf et al. 2008), including anxiety disorders (Rapee 2002) and other forms of distress (Gest 1997) in adulthood.

SI may also increase the risk of physical health problems. Individuals who are high in SI display physiologic hyperreactivity to stress (Cole et al. 2003), and the active inhibition of emotions induces increased cardiovascular reactivity (Gross and Levenson 1997). In clinical research, SI has been associated with the progression of HIV (Cole et al. 2003) and with an increased risk of adverse cardiac events in patients with heart disease (Denollet et al. 2006).

SI can be reliably *assessed with the 7-item SI measure of the DS14* (Denollet 2005), a scale that was specifically designed to assess this broad and stable tendency to inhibit the expression of emotions and behaviors in social interaction.

Cross-References

▶ Type D Personality

References and Readings

Asendorpf, J. B. (1993). Social inhibition: A general-developmental perspective. In H. C. Traue & J. W. Pennebaker (Eds.), *Emotion, inhibition, and health* (pp. 80–99). Seattle: Hogrefe & Huber.

Asendorpf, J. B., Denissen, J. J., & van Aken, M. A. (2008). Inhibited and aggressive preschool children at 23 years of age: Personality and social transitions into adulthood. *Developmental Psychology, 44*, 997–1011.

Cole, S. W., Kemeny, M. E., Fahey, J. L., Zack, J. A., & Naliboff, B. D. (2003). Psychological risk factors for HIV pathogenesis: Mediation by the autonomic nervous system. *Biological Psychiatry, 54*, 1444–1456.

Denollet, J. (2005). DS14: Standard assessment of negative affectivity, social inhibition, and type D personality. *Psychosomatic Medicine, 67*, 89–97.

Denollet, J., Pedersen, S. S., Ong, A. T., Erdman, R. A., Serruys, P. W., & van Domburg, R. T. (2006). Social inhibition modulates the effect of negative emotions on cardiac prognosis following percutaneous coronary intervention in the drug-eluting stent era. *European Heart Journal, 27*, 171–177.

Gest, S. D. (1997). Behavioral inhibition: Stability and associations with adaptation from childhood to early adulthood. *Journal of Personality and Social Psychology, 72*, 467–475.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106*, 95–103.

Kret, M. E., Denollet, J., Grèzes, J., & de Gelder, B. (2011). The role of negative affectivity and social inhibition in perceiving social threat: An fMRI study. *Neuropsychologia, 49*, 1187–1193.

Rapee, R. M. (2002). The development and modification of temperamental risk for anxiety disorders: Prevention of a lifetime of anxiety? *Biological Psychiatry, 52*, 947–957.

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants "grown up": Adult amygdalar response to novelty. *Science, 300*, 1952–1953.

Social Integration

▶ Social Support

Social Isolation

▶ Loneliness
▶ Loneliness and Health

Social Marketing

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Synonyms

Cause marketing; Noncommercial advertising; Public interest advertising; Public service advertising

Definition

Social marketing is the application of marketing principles to non-tangible "products," including ideas, attitudes, and lifestyle changes. Unlike traditional marketing, the primary goal of social marketing is to improve public health, not to increase the marketer's profitability (Lefebvre and Flora 1988). Specifically, it is a planning and intervention model that targets audiences with marketing technologies to improve health and quality of life (Andreasen 1995). There are five principles addressed in the "marketing mix" of social marketing, known as the "5 Ps:" product, price, place, promotion, and positioning. "Product" is the target behavior (e.g., breastfeeding,

healthy eating). "Price" refers to the social, economic, and psychological costs involved in adopting the target behavior. "Place" is the setting, community context, or distribution channels for the product. "Promotion" includes the steps taken to make the audience aware of the ideas, behaviors, and their benefits and may also involve interpersonal communication, media messages, grassroots approaches, special events, and incentives. Lastly, "positioning" describes how the product is framed, namely, to maximize perceived benefits and minimize perceived costs.

Social marketing campaigns using mass media strategies have been effective in promoting positive health behavior outcomes. For example, the Stanford Five-City Project for Heart Disease Prevention, (Farquhar et al. 1985, 1990) which targeted specific audiences, developed a product (i.e., a 6-week quitting contest), and involved local television stations, resulted in a 30% increase in smoking cessation. Similarly, the VERB Campaign (Huhman et al. 2005; Wong et al. 2004) utilized mass media techniques such as advertising on television, on billboards, through a website, and through school- and community-based promotions to increase physical activity in ethnically diverse youth and their parents. While both of the aforementioned social marketing campaigns involved mass media strategies to promote their "products," social marketing campaigns that use interpersonal channels and target the social context may maximize success in promoting health behavior change.

Indeed, effective and sustainable social marketing campaigns are driven largely by social factors (e.g., social norms, social support) and involve the target population in the social marketing processes to increase the integration of the program into established community structures (Bryant et al. 2000). For example, Wilson et al. (2010) used a collaborative community process to develop a social marketing campaign which motivated citizens in a low-income, high-crime community to use a walking path. With the assistance of a communications firm, a community steering committee guided the development of the overall social marketing objectives and approach. One of five key objectives, including increasing perceptions of safety and social connectedness, was targeted each month using corresponding print materials (e.g., a 12-month calendar, matching door hangers). Through grassroots social networking, the program engaged residents to participate in walks with peers, allowing them to feel safe and connected to their neighbors. This study is an example of how involving constituents in the process of social marketing ensures that the approach is tailored and truly fits the needs of the target audience. Furthermore, social marketing campaigns that strategically provide opportunities for interactions between neighbors, friends, and families may influence social norms and support around a particular health behavior and foster a social climate of behavior change.

Cross-References

- Health Behaviors
- Health Communication
- Social Support

References and Readings

Andreasen, A. (1995). *Marketing social change: Changing behavior to promote health, social development, and the environment*. San Francisco: Jossey-Bass.

Bryant, C., Forthofer, M., Landis, D., & McDermott, R. (2000). Community-based prevention marketing: The next steps in disseminating behavior change. *American Journal of Health Behavior, 24*, 61–68.

Farquhar, J., Fortmann, S., MacCoby, N., Haskell, W., Williams, P., Flora, J., et al. (1985). The Stanford five-city project: Design and methods. *American Journal of Epidemiology, 122*(2), 323.

Farquhar, J., Fortmann, S., Flora, J., Taylor, C., Haskell, W., Williams, P., et al. (1990). Effects of communitywide education on cardiovascular disease risk factors: The Stanford five-city project. *Journal of the American Medical Association, 264*(3), 359.

Huhman, M., Potter, L., Wong, F., Banspach, S., Duke, J., & Heitzler, C. (2005). Effects of a mass media campaign to increase physical activity among children: Year-1 results of the VERB campaign. *Pediatrics, 116*(2), e277.

Lefebvre, C., & Flora, J. (1988). Social marketing and public health intervention. *Health Education & Behavior, 15*(3), 299.

Wilson, D. K., Trumpeter, N. N., St. George, S. M., Coulon, S. M., Griffin, S., Van Horn, M. L., et al. (2010). An overview of the "positive action for today's health" (PATH) trial for increasing walking in low income, ethnic minority communities. *Contemporary Clinical Trials, 31*, 624–633.

Wong, F., Huhman, M., Asbury, L., Bretthauer-Mueller, R., McCarthy, S., Londe, P., et al. (2004). VERB™-a social marketing campaign to increase physical activity among youth. *Preventing Chronic Disease, 1*(3), 1–7.

Social Media

▶ Social Networking Sites

Social Network

▶ Family Social Support
▶ Family, Relationships
▶ Social Support

Social Networking Sites

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Synonyms

Digital media; Social media; Virtual communities

Definition

Social networking sites, sometimes characterized under the umbrella term social media, are virtual communities or networks that allow individuals, communities, and organizations to create and disseminate user-generated content including but not limited to pictures, videos, text, memes, and profile pages for individuals or groups; social media content has greater virality than other web-based content. By connecting a user's profile (maintained by the social media organization) to other individuals or groups, social networks are formed. Well-known social media sites include Facebook, Myspace, Twitter, Instagram, and Snapchat. When social networking sites were first popularized, the average users were emerging adults, but now users represent a wide range of ages, races, and places of residence.

Associate Editor responsible for this entry: Dr. Ellen Beckjord

Description

Social media encompasses all online platforms on which media content is uploaded, and social networking sites are online, dynamic, interaction-based communities where online relationships can be created or maintained. These terms are often used interchangeably and are inherently related.

Social networking sites have undergone rapid and dramatic development since their creation. Emailing, with its ability to connect with others electronically and its inclusion of individualized "profiles" (i.e., email addresses), is considered to be one of the preliminary social network modalities. In the late 1990s, instant communication platforms were developed, such as instant messaging (e.g., AOL Instant Messenger), chat rooms, and online blog communities (e.g., Live Journal). Myspace, LinkedIn, and Facebook, all social networking sites, followed in the early 2000s; they allowed individual users and collective groups to create personalized page profiles from which they could "friend" one another, which adds the other user to the original user's social network. Users could publicly signify their identity and affiliations (e.g., schools, profession) and choose to connect with preexisting acquaintances or reach out to new ones. Although Facebook remains the most popular social networking site to date (Dagan and Beskin 2015), social networking sites that allow users to

comment on shared photographs and videos, such as Instagram and Snapchat, have grown in popularity. Online dating sites are popular, specialized, social networking sites. Most sites allow users to geo-tag posts to indicate their location; content can be aggregated by location.

Social networking sites are used by individuals across the life span, although the most prevalent users are young adults and adolescents (Pagoto et al. 2016), with 92% of teenagers reporting daily Internet access and 71% report using more than one social networking site (Pew Research Center 2015a). Widespread smartphone/tablet ownership has facilitated increased social networking site use from mobile phones: nearly two-thirds of American adults own smartphones, of which approximately 75% reported accessing social networking sites on their phones at least once in the last week (Pew Research Center 2015b).

Although many social networking sites were originally designed to connect with acquaintances, their size, speed, and accessibility have made them increasingly utilized platforms for consumers and corporations to disseminate information, communicate, and coordinate social action (Pagoto et al. 2016). Businesses and researchers have also turned to these sites to advertise products or memberships, collect data on users, and engage with a large sample of diverse consumers and patients. Additionally, Twitter users are more likely to elect sharing their information publicly, making Twitter an innovative and convenient data collection tool (Turner-McGrievy and Beets 2015). Users can also highlight important words or phrases by preceding them with a hashtag (#), a microblogging convention that allows users to see others' messages related to that topic; if many people tweet a common hashtag, such as related to a news sensation or popular artist, the hashtag is prominently featured as "trending" on the site (Gruzd and Haythornthwaite 2013). This potential for virality is a core defining characteristic that differentiates social media content from other Internet content.

Peer-to-peer health care, the use of social networking sites to engage with other patients and health care professionals, is a subtype of electronic health (eHealth) that has become an increasingly popular strategy for individuals to obtain and share information about health conditions and provide social support (Balatsoukas and Kennedy 2015; Pagoto et al. 2016). Beyond benefit to the individual, researchers and community health workers are harnessing the potential power of these sites to deliver prevention messages and intervention to improve health and outcomes through behavior change (Balatsoukas and Kennedy 2015). Social networking-based interventions can reduce barriers of traditional treatment (e.g., scheduling, transportation, social obligations; Pagoto et al. 2016). Messages exchanged between people in a social network may be more influential than messages delivered by an unknown outsider (Maher et al. 2014). These messages may also produce higher patient engagement and retention in interventions (Maher et al. 2014). Social networking sites have been effective in trials targeting smoking, sedentary behavior, weight loss, sedentary behavior, physical activity, and dietary awareness (Cheung et al. 2015; Maher et al. 2014).

Despite numerous advantages, there are many limitations for using social networking sites for research and intervention. While they are capable of quickly spreading useful, correct information, they are equally effective in facilitating the spread of misinformation. This is best illustrated in discussions of childhood vaccinations on social media sites, in which less than 25% of discussants were aware that the majority of evidence supports the safety and efficacy of vaccines (Seymour et al. 2015). Additionally, most health-related studies utilizing social networking sites have not isolated the effects of specific components, and extraneous variables may have influenced the results. Finally, numerous effective in-person interventions are challenging to modify for social networking-based delivery (Pagoto et al. 2016). Modifications may be needed to reach or surpass the success of in-person interventions. Controlled trials with foci on translation of delivery and patient engagement are necessary.

Social networking-based interventions may have massive impacts on health outcomes, but the current evidence is based primarily on qualitative data. Growing evidence with rigorous

methodology suggests that virtual communities represent one of the biggest opportunities for research and behavior change of this generation (Eysenbach et al. 2004). Smartphone ownership continues to rise among racial and ethnic minority groups (Pew Research Center 2015c), making social networking-based interventions an exciting opportunity to deliver high-quality behavioral interventions to individuals who are both typically underrepresented in research and often disproportionately affected by chronic health conditions. Research has indicated that the larger a group, the more contagious an idea becomes, highlighting social networking sites as a promising platform to impact healthy behavior changes (Luhmann and Rajaram 2015).

Cross-References

- ▶ eHealth
- ▶ Social Marketing

References and Further Readings

Balatsoukas, P., & Kennedy, C. M. (2015). The role of social network technologies in online health promotion: A narrative review of theoretical and empirical factors influencing intervention effectiveness. *Journal of Medical Internet Research, 17*(6), e141. https://doi.org/10.2196/jmir.3662.

Cheung, Y. T. D., Chan, C. H. H., Lai, C. K. J., Chan, W. F. V., Wang, M. P., Li, H. C. W., Chan, S. S. C., & Lam, T. H. (2015). Using whatsapp and facebook online social groups for smoking relapse prevention for recent quitters: A pilot pragmatic cluster randomized controlled trial. *Journal of Medical Internet Research, 17*(10), e238. https://doi.org/10.2196/jmir.4829.

Dagan, N., & Beskin, D. (2015). Effects of social network exposure on nutritional learning: Development of an online educational platform. *JMIR Serious Games, 3*(2), e7. https://doi.org/10.2196/games.4002.

Eysenbach, G., Powell, J., Englesakis, M., Rizo, C., & Stern, A. (2004). Health related virtual communities and electronic support groups: Systematic review of the effects of online peer to peer interactions. *British Medical Journal, 328*(7449), 1–6.

Gruzd, A., & Haythornthwaite, C. (2013). Enabling community through social media. *Journal of Medical Internet Research, 15*(10), e248. https://doi.org/10.2196/jmir.2796.

Luhmann, C. C., & Rajaram, S. (2015). Memory transmission in small groups and large networks: An agent-based model. *Psychological Science, 26*(12), 1909–1917. https://doi.org/10.1177/0956797615605798.

Maher, C. A., Lewis, L. K., Ferrar, K., Marshall, S., De Bourdeaudhuij, I., & Vandelanotte, C. (2014). Are health behavior change interventions that use online social networks effective? A systematic review. *Journal of Medical Internet Research, 16*(2), e40. https://doi.org/10.2196/jmir.2952.

Pagoto, S., Waring, M. E., May, C. N., Ding, E. Y., Kunz, W. H., Hayes, R., & Oleski, J. L. (2016). Adapting behavioral interventions for social media delivery. *Journal of Medical Internet Research, 18*(1), e24. https://doi.org/10.2196/jmir.5086.

Pew Research Center. (2015a). *Teens, social media and technology overview 2015*. Retrieved from http://www.pewinternet.org/2015/04/09/teens-social-media-.technology-2015/

Pew Research Center. (2015b). *The smartphone difference*. Retrieved from http://www.pewinternet.org/2015/04/01/us-smartphone-use-in-2015/

Pew Research Center. (2015c). *Technology device ownership: 2015*. Retrieved from http://www.pewinternet.org/2015/10/29/technology-device-ownership-2015/

Seymour, B., Getman, R., Saraf, A., Zhang, L. H., & Kalenderian, E. (2015). When advocacy obscures accuracy online: Digital pandemics of public health misinformation through an antifluoride case study. *American Journal of Public Health, 105*(3), 517–523. https://doi.org/10.2105/ajph.2014.302437.

Turner-McGrievy, G. M., & Beets, M. W. (2015). Tweet for health: Using an online social network to examine temporal trends in weight loss-related posts. *Translational Behavioral Medicine, 5*(2), 160–166. https://doi.org/10.1007/s13142-015-0308-1.

Social Networks

- ▶ Social Relationships

Social Norms

- ▶ Norms

Social Pain

- ▶ Loneliness

Social Problem-Solving Therapy (SPST)

▶ Problem Solving

Social Processes

▶ Interpersonal Processes

Social Relationships

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Synonyms

Interpersonal relationships; Social networks; Social ties

Definition

Broadly defined, social relationships refer to the connections that exist between people who have recurring interactions that are perceived by the participants to have personal meaning. This definition includes relationships between family members, friends, neighbors, coworkers, and other associates but excludes social contacts and interactions that are fleeting, incidental, or perceived to have limited significance (e.g., time-limited interactions with service providers or retail employees). Scientists interested in behavioral medicine often emphasize the informal social relationships that are important in a person's life, or the person's social network, rather than formal relationships, such as those with physicians, lawyers, or clergy. Relationship phenomena of interest to scientists encompass both the specific interactions that individuals experience with members of their social networks and the global perceptions of those interactions, which are shaped by past and current interactions with important social network members. The interactions that occur with social network members are often positive, and include the provision of emotional and material support, companionship, and encouragement of health-enhancing behaviors. Interactions with social network members also can be negative, however, and can include insensitive, unresponsive, hurtful, or intrusive actions by others.

Description

A large body of evidence suggests that social relationships are associated with health. Research has linked social relationships to mortality and morbidity (Berkman et al. 2000; Cohen 2004; House et al. 1988a). People with fewer social network ties have been found to have an elevated risk for a number of diseases, including cardiovascular disease and stroke, some forms of cancer, infectious disease, and possibly dementia (Cohen 2004; Uchino 2006). Social relationships also have been linked to the onset and progression of chronic illness, as well as illness adjustment, post-surgical recovery, disability, and survival (e.g., Seeman and Crimmins 2001). Increased confidence in the associations between social relationships and health stems from the fact that the associations emerge not only in large, well-controlled cross-sectional and longitudinal epidemiological studies, but also in experimental studies of humans and animals. Moreover, the strength of these associations is impressive, as evidenced by research suggesting that the effects of social relationships on health are comparable in size to the effects of conventional risk factors, such as smoking (House et al. 1988).

Structural Versus Functional Aspects of Social Relationships

Both structural and functional aspects of social relationships have been distinguished (Berkman et al. 2000). Structural aspects refer to the

existence and objective characteristics of social relationships, whereas functional aspects refer to the functions performed by and subjective qualities of social relationships (House et al. 1988b). Structural characteristics of interest to health researchers include the size of a person's social network, the frequency of contact with social network members, and the nature of the role relationships with network members (e.g., family member, friend, coworker). Research has demonstrated that some structural aspects of social relationships, such as social network size and frequency of contact, are related to health. For example, having more social ties and more frequent social interaction has been found to be associated with lower risks for mortality and poor mental and physical health outcomes. Similarly, the marital relationship has been found to be especially consequential for health, relative to other role relationships. Married individuals, compared to unmarried individuals, have a lower prevalence and incidence of both mental and physical health problems and a lower mortality risk (House et al. 1988). Furthermore, research suggests that men may derive more health benefits from marriage than do women. Poor quality marriages, however, and the disruption associated with divorce or widowhood appear to be particularly deleterious to mental and physical health (Burman and Margolin 1992).

Although numerous robust associations between structural aspects of social relationships and health have been documented, functional aspects also need to be examined in order to understand how and why social relationships impact health. The most commonly studied social network function that contributes to health is social support. A great deal of evidence suggests that social support can help to buffer people from the adverse effects of life stress (Cohen 2004; Uchino et al. 1996). Different types of social support have been distinguished (emotional, instrumental, informational), all of which are conceptualized as ways that social network members provide each other with care and aid in times of need. Different types of support are viewed as being important in the context of different stressors, although evidence suggests that emotional support is important across a very broad range of stressors (Cohen and Wills 1985).

Beyond social support, social relationships also serve as sources of companionship, which provides opportunities for enjoyable interaction and camaraderie. The positive affect and relief from stress afforded by companionship, in turn, help to sustain health and well-being (Rook 1987). Social network members also monitor each other's health behavior and intervene to discourage health-compromising behavior, leading researchers to be interested in the health effects of social regulation (or social control; Umberson 1987).

The beneficial functions of social relationships also have been posited to have negative counterparts (Rook 1998). Specifically, social relationships not only can provide support, companionship, and social regulation, but also can fail to provide support or can provide misguided support, can reject or neglect others, and can foster bad, rather than good, health practices. Even though such negative interactions with others are relatively rare, they can take a considerable toll on health and well-being (Rook 1998).

Pathways by Which Social Relationships Influence Health

Understanding the pathways by which social relationships influence health is a key goal for health researchers. Three main pathways have been identified (Berkman et al. 2000; Rook et al. 2010): The first pathway involves psychological processes and conditions associated with social relationships, such as positive and negative emotions, feelings of self-worth and self-efficacy, coping strategies, and depression. The second pathway involves physiological processes, or activation of bodily systems (endocrine, cardiovascular, and immune) in response to various kinds of social interactions. The third pathway involves health behaviors that are fostered by interactions with social network members, including health-enhancing behaviors (e.g., exercise) as well as health-compromising behaviors (e.g., smoking). All three of these pathways have the potential to have independent and joint effects on morbidity and, ultimately, mortality.

Pathways having beneficial health effects. Social support, companionship, and social regulation are believed to affect health through unique mechanisms. Social support is thought to dampen the emotional, physiological (neuroendocrine, cardiovascular, immune), and behavioral effects of stress by improving one's perceived ability to cope with stress (Uchino 2006; Uchino et al. 1996). Companionship, on the other hand, is thought to influence health and well-being by enhancing positive affect and providing a respite from stress (Rook 1987). Positive affect, in turn, has been linked to lower rates of morbidity, fewer symptoms and less pain from health conditions, and greater longevity (Pressman and Cohen 2005). Companionship also may activate physiological processes, such as the release of oxytocin, a neuropeptide that helps to counter harmful stress responses, including release of the stress hormone, cortisol. Social control is believed to affect health through two primary, but opposing, processes (Hughes and Gove 1981). Specifically, social control may discourage health-compromising behaviors and encourage health-enhancing behaviors, thereby contributing to better health and ultimately, a lower risk of mortality. Yet, at the same time, to the extent that social control involves constraints on others' behavior, it may provoke psychological distress, erode feelings of self-efficacy, and kindle relationship tensions. The psychological and relationship costs of social control may thus reduce or cancel the health benefits of social control, although the net effects of social control on health are not yet fully understood (Rook et al. 2010).

Pathways having detrimental health effects. Persistent conflict in social relationships, as well as the absence or loss of social relationships, also impact health through a number of mechanisms. Specifically, recurring strains and conflicts in social relationships lead to repeated activation of physiological systems (e.g., hypothalamic-pituitary-adrenal axis or sympathetic nervous system activity) and impaired immune functioning. These chronically activated and dysregulated physiological systems, in turn, may accelerate disease onset and progression. Additionally, social isolation and loneliness have been linked to negative emotions, chronic stress, cardiovascular activation, low physical activity, and impaired sleep (Cacioppo et al. 2002). Finally, it is important to recognize that social network members sometimes encourage undesirable, rather than desirable, health practices. For example, evidence suggests that adolescents sometimes recruit their peers to use illegal substances or to engage in other risky health behaviors. Thus, conflict and tensions in social relationships, social isolation and loneliness, and undesirable social influence all can increase the risk of disease onset and progression.

Conclusion

It is well established that social relationships are important for health and well-being, and research has identified key aspects of social relationships that warrant consideration in efforts to understand these links with health. The psychological, physiological, and behavioral pathways by which social relationships affect health also are beginning to be understood. As this literature evolves and expands to document patterns that exist across different sociodemographic and lifespan contexts, it may help to inform interventions designed to strengthen social relationships and, in turn, health.

Cross-References

- ▶ Family and Medical Leave Act
- ▶ Family Assistance
- ▶ Family Planning
- ▶ Family Stress
- ▶ Family Studies (Genetics)
- ▶ Family Systems Theory
- ▶ Family Violence
- ▶ Family, Caregiver
- ▶ Family, Income
- ▶ Family, Relationships
- ▶ Family, Structure
- ▶ Loneliness
- ▶ Psychosocial Characteristics
- ▶ Psychosocial Factors
- ▶ Social Capital and Health
- ▶ Social Cohesion

- ▶ Social Conflict
- ▶ Social Factors
- ▶ Social Stress
- ▶ Social Support

References and Readings

Berkman, L. F., Glass, T., Brissette, I., & Seeman, T. E. (2000). From social integration to health: Durkheim in the new millennium. *Social Science and Medicine, 51*, 843–857.

Burman, B., & Margolin, G. (1992). Analysis of the association between marital relationships and health problems: An interactional perspective. *Psychological Bulletin, 112*, 39–63.

Cacioppo, J. T., Hawkley, L. C., Crawford, L. E., Ernst, J. M., Burleson, M. H., Kowalewski, R. B., et al. (2002). Loneliness and health: Potential mechanisms. *Psychosomatic Medicine, 64*, 407–417.

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59*, 676–684.

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*, 310–357.

House, J. S., Landis, K. R., & Umberson, D. (1988a). Social relationships and health. *Science, 241*, 540–545.

House, J. S., Umberson, D., & Landis, K. R. (1988b). Structures and processes of social support. *Annual Review of Sociology, 14*, 293–318.

Hughes, M., & Gove, W. R. (1981). Living alone, social integration, and mental health. *The American Journal of Sociology, 87*, 48–74.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*, 925–971.

Rook, K. S. (1987). Social support versus companionship: Effects on life stress, loneliness, and evaluations by others. *Journal of Personality and Social Psychology, 52*, 1132–1147.

Rook, K. S. (1998). Investigating the positive and negative sides of personal relationships: Through a lens darkly? In B. H. Spitzberg & W. R. Cupach (Eds.), *The dark side of close relationships* (pp. 369–393). Mahwah: Lawrence Erlbaum.

Rook, K. S., August, K. J., & Sorkin, D. H. (2010). Social network functions and health. In R. J. Contrada & A. Baum (Eds.), *The handbook of stress science: Biology, psychology, and health* (pp. 123–136). New York: Springer.

Seeman, T. E., & Crimmins, E. (2001). Social environment effects on health and aging. Integrating epidemiologic and demographic approaches and perspectives. *Annals of the New York Academy of Sciences, 954*, 88–117.

Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine, 29*, 377–387.

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*, 488–531.

Umberson, D. (1987). Family status and health behaviors: Social control as a dimension of social integration. *Journal of Health and Social Behavior, 28*, 306–319.

Social Resources

- ▶ Family Social Support
- ▶ Social Support

Social Stress

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Synonyms

Interpersonal stress or conflict; Societal stress

Definition and Description

Social stress can be broadly defined as a perceived threat that is based on one's relation to or association with another person or group of people. There is great variability in the types of social stress that one may experience. It can arise from interpersonal interactions, such as those with family members, friends, professional colleagues, and strangers; from evaluated performances, including giving a speech in front of an audience; from sharing an experience with someone, such as patients and caregivers dealing with a chronic illness; or from group, community, or societal

dynamics, for instance, one's socioeconomic or professional status within a community or society.

Different types of social stress can lead to a range of observable and measurable responses. For instance, socially evaluated performances may lead to negative emotions and self-conscious thoughts. Severe interpersonal social stress, for example, verbal abuse between spouses, may lead to psychological distress (e.g., post-traumatic distress). Workplace social stress can have physiological impacts, including cardiovascular, endocrinological, immunological, and other system responses. Social discrimination, for instance, being treated unfairly or differently due to one's personal characteristics (e.g., gender, race, physical attributes, sexual orientation), can also be linked with negative physiological responses and with risky health behaviors (e.g., substance use, binge eating).

In order to overcome or deal with social stress, people engage in a variety of coping responses. These include active and passive coping strategies in which the individual either engages with or withdraws from the source of the social stress. Active coping strategies include problem-solving strategies (e.g., changing public policy steps to better provide healthcare services for underserved minority groups) and emotion-focused strategies (e.g., verbally communicating one's concerns with a friend about their drug use). Passive coping strategies include avoidant strategies (e.g., ignoring a rude co-worker) and distraction strategies (e.g., taking up exercise to overcome loss of a relationship).

Social stress and its associated responses, as well as the coping strategies used to manage them, can have serious health effects. For instance, acute (e.g., sudden job loss), frequent (e.g., repetitive marital strife), and chronic (e.g., bereavement) social stressors can compromise physiological functioning as well as self-reported physical and mental health conditions. Social stressors can also have long-lasting effects, such that social stress experienced early on in life (e.g., childhood) influences endocrine system responses (e.g., cortisol) to stress in adulthood. Furthermore, social status within a group, community, or society at large (e.g., socioeconomic status) can increase the risk for negative health outcomes and/or behaviors across the lifespan.

Cross-References

- ▶ Biobehavioral Mechanisms
- ▶ Psychosocial Factors
- ▶ Social Conflict
- ▶ Social Relationships
- ▶ Social Support
- ▶ Stress Reactivity

References and Further Reading

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355–391.

Juth, V., Silver, R. C., & Sender, L. (2015a). The shared experience of adolescent and young adult cancer patients and their caregivers. *Psycho-Oncology, 24*, 1746–1753.

Juth, V., Smyth, J. M., Carey, M., & Lepore, S. (2015b). Social constraints are associated with negative psychological and physical adjustment in bereavement. *Applied Psychology: Health and Well-Being, 7*, 129–148.

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–524.

Social Support

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Synonyms

Moderators/moderating factors; Social integration; Social network; Social resources

Definition

Social support refers to the belief that one is valued, cared for, and loved by others in a social network. Social support can generally be conceptualized as reflecting two broad factors (Cohen and Syme 1985; Cohen and Wills 1985). Structural support reflects properties of the social network and the degree to which a person participates in that network (i.e., social integration). Functional support refers to the ways by which network members aid the individual through tangible assistance or through psychological and emotional buffering. Social support is among the most widely studied and robust psychosocial moderators of health. This entry will expand upon this description, review evidence linking it to health, examine hypothesized mechanisms, and discuss future directions.

Description

Social support is a multicomponent construct reflecting the size, quality, and availability of one's social resources to moderate stress. Social support should be distinguished from conceptually related terms. *Social capital* is a sociological term referring to stock or stored social credit. *Social network* refers to the social collectives or groups to which a person may belong. *Social integration* refers to the degree to which a person is embedded in or a part of a social network. *Relationship* is a descriptor often used to characterize a specific type of social tie.

Social support is generally conceptualized as having structural (i.e., size of the social network, degree of social integration) and functional (i.e., support processes; support received versus support perceived) characteristics. In addition, the quality of support is increasingly recognized as an important moderator of the relationship between support and health. Importantly, relationships are not uniformly positive, with some acting as sources of stress (Uchino 2004).

Measures

Social support is generally measured by self-report. Data sources may include demographic data from hospital, census, or other records; interviews; psychometrically validated instruments; and ad hoc items and measures.

Structural Measures of Support

The simplest of the social integration measures is marital status which is consistently identified as a protective factor in large, prospective studies of mortality. Marital status is a particularly attractive measure as it is a common demographic characteristic and, thus, available for analysis with many kinds of health data. Many researchers are also interested in measuring the size of one's social networks and the degree to which a person is embedded in the networks. One well-validated multicomponent measure of social integration is the Social Network Index (SNI: Berkman and Syme 1979) which provides an aggregate score of social integration by sampling activity in multiple relationships. Lower SNI scores are predictive of a nearly two fold increase in mortality risk. Finally, the literature is replete with numerous ad hoc structural support measures including measures of number of networks, network size, as well as the number of specific kinds of relationships such as friendships, and others.

Functional Measures of Support

Functional measures can be classified into support received and support perceived. Interestingly, measures of received and perceived support tend not to be highly correlated. Measures of received supportive behavior allow assessment of the actual social resources available to an individual. For example, the Inventory of Socially Supportive Behaviors (ISSB: Barrera et al. 1981) is a 40-item structural support measure assessing the frequency with which one receives a variety of supportive actions. Measures of perceived social support assess an individual's beliefs about the social resources available to them. Perceived support measures generally consist of two factors:

beliefs about support available, and satisfaction with level of perceived support. A widely used example of a perceived support measure is Cohen and Wills (1985) Interpersonal Support Evaluation List (ISEL) which assesses perceptions of belonging, tangible support, support appraisals, and confidence in support perceptions.

Quality of Support

Relationship quality has generally been assessed along a single dimension ranging from positive to negative. However, emerging models hypothesize that relationships can have both positive and negative characteristics. The Quality of Relationship Inventory (QRI: Pierce, Baldwin, and Lydon, 1997) is among the most popular multidimensional measures. The 25-item, self-report measure yields discrete support and conflict scales as well as a total quality rating. Several authors have suggested that the relationship quality dimensions of positivity and negativity are orthogonal, yielding four basic relationship types: (1) high positive, low negative (i.e., supportive relationship), (2) low positive, high negative (i.e., conflictual relationship), (3) high positive, high negative (i.e., ambivalent relationship), and (4) low positive, low negative (i.e., benign/irrelevant relationship). The Social Relationships Index (SRI: Uchino 2004) is an example of this multidimensional relationship quality assessment approach. Although health outcome data is limited, this conceptualization appears to be gaining acceptance as a more theoretically sound approach.

Social Support and Health

Social support is among the most robust predictors of disease and all-cause mortality. For example, a recent meta-analysis of 148 studies estimated the effect of social relationships to improve survival rates by 50% (Holt-Lunstad et al. 2010). This effect varied by measurement, with multidimensional measures of social integration associated with more than 90% increase in survival rates. As noted by the authors, the magnitude of the effect of social support on mortality is likely diluted in these studies by unmeasured negative aspects of social relationships (e.g., a conflictual marriage). The results of this meta-analysis extend the work of previous systematic reviews and meta-analyses in identifying that social support is a substantial health and disease moderator with effects equivalent to more traditionally acknowledged risk factors such as cigarette smoking and obesity.

Cardiovascular Disease

The strongest evidence for a protective role of social support on health comes from studies of cardiovascular diseases such as coronary heart disease (CHD) morbidity and mortality (Smith and Ruiz 2002). Multiple prospective studies of initially healthy samples have demonstrated that lower social integration and social support are associated with greater CHD incidence, faster disease progression, risk of myocardial infarction (MI), and greater risk for all-cause mortality. Among persons with diagnosed CHD, lower social support is predictive of significantly greater risk of recurrent MI and death. For example, Welin et al. (2000) found an almost three-fold increased chance of cardiac mortality in post-MI patients with low perceived emotional support at 10-year follow-up. A similar effect size was found by Berkman et al. (1992). Some studies indicate that low social integration is only associated with survival in the most severely isolated of the population. In a review of the literature, Mookadam and Arthur (2004) estimated a two to three times increased risk of 1-year mortality in the socially isolated population, with little health benefits resulting from support systems above this threshold. Moreover, there is some evidence that the beneficial effects of social support on cardiac health are stronger among women and that functional measures of support are more strongly related to cardiac disease compared to structural measures.

Effect sizes for social integration in healthy samples for future development of CHD are

comparable to those of more traditional risk factors such as cigarette smoking (Orth-Gomer et al. 1993). Similarly, social integration is as strong a predictor of mortality among clinical populations as traditional risk factors such as cholesterol level, tobacco use, and hypertension in the patient population (Mookadam and Arthur 2004). An important conceptual issue is whether high social support connotes a cardiovascular benefit akin to physical activity or whether it is simply the absence of support that is relevant. In addition, the relative value of structural versus functional support remains an open question. Regardless, the cumulative evidence indicates social support is an important moderator of cardiovascular risk.

Cancer

The relationship between social support and cancer is quite mixed. A recent systematic review of the prospective longitudinal literature concluded that in the context of breast cancer, greater social support was associated with slower disease progression in five of seven well-designed studies. Structural indices were the more commonly used and significant measure of social support in these studies. In contrast, there were no associations between measures of social support and other types of cancer with the paradoxical exception of more social support related to faster cancer progression in a sample of colorectal patients (Villingshoj et al. 2006). A meta-analysis of 87 studies estimated the relative risk of perceived social support and measures of social network size on mortality among cancer patients to be .82 and .80, respectively, suggesting a beneficial effect (Pinquart and Duberstein 2010). Inconsistencies between studies may be partially explained by differences in support types measured and patient needs. The type of support associated with improved prognosis appears to vary by cancer site, with perceived social support representing a stronger predictor for leukemia and lymphoma, whereas breast cancer patients benefit more from have a large number of social ties (Pinquart and Duberstein 2010).

In contrast to the mixed findings regarding physical outcomes, a robust literature supports the beneficial effects of social support on emotional and psychological reactions to cancer and associated treatments. Moreover, cancer is often a shared interpersonal experience – affecting supports as well as patients. A meta-analysis of these *contagion effects* estimated the correlation between patient and caregiver distress is .35 (Hodges et al. 2005). Importantly, partner response to illness affects patient adjustment, particularly in terms of quality of life. The type of coping employed (e.g., positive versus negative), relationship maintenance behaviors, and the amount of communication about the relationship may also be important moderators of couples adjustment.

With respect to interventions, efforts have largely focused on increasing the patient's social network size and opportunity for emotional support. Perhaps the most well known of these interventions was conducted by Spiegel et al. (1989). Women with late-stage metastatic breast cancer were randomized to either supportive group therapy or wait-list control. Findings that women in the treatment condition survived approximately 18 months longer generated interest in the possible healing role of support. Several clinical and prospective trials have failed to replicate these results, fueling doubts about the potential physical benefits of the approach. Regardless, there is substantial effort to translate socially based interventions from bench to bedside where their emotional and quality of life benefits are well recognized.

HIV/AIDS

Conclusions regarding the benefits of social support on physical outcomes in the context of HIV/AIDS are limited by the small number of published studies. A prospective study of HIV-positive men with hemophilia demonstrated that lower levels of perceived support at baseline were predictive of faster decreases in CD4 T-lymphocyte levels, a key marker of AIDS status, over a 4-year follow-up (Theorell et al. 1995). Leserman and colleagues conducted a study of 82 asymptomatic gay men, in which greater satisfaction with social support was associated with slower progression to AIDS, regardless of

network size (Leserman et al. 1999). In contrast, other studies have found perceived support to be unrelated to progression to AIDS. More research is needed to adequately evaluate these relationships.

The social environment of the HIV/AIDS population may be uniquely important because of the social stigma associated with the diagnosis (Herek and Glunt 1988). Stigma is associated with both personal distress and hesitancy to self-disclose status to sexual partners, potentiating further transmission of the infection either by the individual or the individual's un-informed sexual partners. A meta-analysis of 21 studies showed that greater perceived social support was associated with increased likelihood of self-disclosing one's HIV-positive status (Smith et al. 2008). Disclosure of HIV status is associated with increased social support within those relationships, indicating that disclosure may be a positive method of eliciting support from others.

Mechanisms

Social support is hypothesized to affect health and well-being through two pathways. The *main effects* hypothesis suggests that having more social resources reduces the chances of exposure to stressful circumstances or the magnitude of threat associated with certain environments. For example, one is likely to be safer walking at night with a large group of friends than when walking alone. Numerous studies have shown that having more social resources (measured as the number of social ties, the degree of social integration, etc.) is predictive of lower disease incidence, better survival following illness, lower all-cause mortality, and greater longevity irrespective of the quality of those relationships. Interestingly, these social resources need not be human to have a beneficial effect. Several studies have demonstrated that having a loving pet is associated with less stress and better survival following disease incidents such as a heart attack.

Social support may also affect health by moderating or reducing the impact of stressful circumstances (i.e., *stress buffering hypothesis*). After a stressful romantic breakup, a friend may comfort you with a hug, by providing you with an opportunity to vent your emotions, or by introducing you to someone new. Substantial laboratory data demonstrates that provision of support reduces self-reported stress and acute physiological responses to lab stressors. For example, individuals who received a note communicating emotional support from a supportive friend experience less blood pressure increase during a subsequent speech task relative to those who receive a note from a less supportive person (Uno et al. 2002). Importantly, the perception of support appears to be more important than the actual provision of support. For example, imagining a supportive tie prior to a stressor results in less cardiovascular reactivity and less self-reported stress (i.e., buffering) compared to imagining an acquaintance (Smith et al. 2004). These findings support the idea that social support, received or perceived, can reduce stressful experiences.

Future Directions

Future research will continue to expand upon conceptual distinctions regarding sources of support and related actions. Longitudinal research is also needed to understand the biobehavioral mechanisms by which social support translates into disease risk. Further, more research is needed to determine whether it is better to have substantial support or simply to not be alone. Finally, emerging social phenomenon such as texting and online social networking through Facebook, Twitter, and other forums presents new challenges for researchers to conceptualize, measure, and gauge as moderators of health.

Cross-References

- ▶ Psychosocial Factors
- ▶ Psychosocial Predictors
- ▶ Psychosocial Variables
- ▶ Social Capital and Health
- ▶ Social Cohesion
- ▶ Social Conflict
- ▶ Social Factors
- ▶ Social Relationships

References and Further Readings

Barrera, M., Sandler, I. N., & Ramsay, T. B. (1981). Preliminary development of a scale of social support: Studies on college students. *American Journal of Community Psychology, 9*(4), 435–447.

Berkman, L. F., Leo-Summers, L., & Horwitz, R. I. (1992). Emotional support and survival after myocardial infarction. A prospective, population-based study of the elderly. *Annals of Internal Medicine, 117*(12), 1003–1009.

Berkman, L. F., & Syme, S. L. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of Alameda County residents. *American Journal of Epidemiology, 109*(2), 186–204.

Cohen, S., & Syme, S. L. (1985). Issues in the study and application of social support. In S. Cohen & S. L. Syme (Eds.), *Social Support and health* (pp. 3–22). San Francisco: Academic Press Inc..

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*(2), 310–357.

Herek, G. M., & Glunt, E. K. (1988). An epidemic of stigma: Public reactions to AIDS. *American Psychologist, 43*(11), 886–891.

Hodges, L. J., Humphris, G. M., & Macfarlane, G. (2005). A meta-analytic investigation of the relationship between the psychological distress of cancer patients and their carers. *Social Science & Medicine, 60*(1), 1–12. 1982.

Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine, 7*(7), e1000316–e1000316.

Leserman, J., Jackson, E. D., Petitto, J. M., Golden, R. N., Silva, S. G., Perkins, D. O., et al. (1999). Progression to AIDS: The effects of stress, depressive symptoms, and social support. *Psychosomatic Medicine, 61*(3), 397–406.

Mookadam, F., & Arthur, H. M. (2004). Social support and its relationship to morbidity and mortality after acute myocardial infarction: Systematic overview. *Archives of Internal Medicine, 164*(14), 1514–1518.

Orth-Gomer, K., Rosengren, A., & Wilhelmsen, L. (1993). Lack of social support and incidence of coronary heart disease in middle-aged Swedish men. *Psychosomatic Medicine, 55*, 37–43.

Pierce, T., Baldwin, M. W., & Lydon, J. E. (1997a). A relational schema approach to social support. In G. Pierce, S. Lakey, & B. R. Sarason (Eds.), *Sourcebook of social support and personality* (pp. 19–47). New York: Plenum Press.

Pierce, G. R., Sarason, I. G., Sarason, B. R., & Solky-Butzel, J. A. (1997b). Assessing the quality of personal relationships. *Journal of Social and Personal Relationships, 14*(3), 339–356.

Pinquart, M., & Duberstein, P. R. (2010). Associations of social networks with cancer mortality: A meta-analysis. *Critical Reviews in Oncology/Hematology, 75*(2), 122–137.

Smith, R., Rossetto, K., & Peterson, B. L. (2008). A meta-analysis of disclosure of one's HIV-positive status, stigma and social support. *AIDS Care, 20*(10), 1266–1275.

Smith, T. W., & Ruiz, J. M. (2002). Psychosocial influences on the development and course of coronary heart disease: Current status and implications for research and practice. *Journal of Consulting and Clinical Psychology, 70*(3), 548–568.

Smith, T. W., Ruiz, J. M., & Uchino, B. N. (2004). Mental activation of supportive ties, hostility, and cardiovascular reactivity to laboratory stress in young men and women. *Health Psychology, 23*(5), 476–485.

Spiegel, D., Bloom, J. R., Kraemer, H. C., & Gottheil, E. (1989). Effect of psychosocial treatment on survival of patients with metastatic breast cancer. *Lancet, 2*(8668), 888–891.

Theorell, T., Blomkvist, V., Jonsson, H., Schulman, S., Berntorp, E., & Stigendal, L. (1995). Social support and the development of immune function in human immunodeficiency virus infection. *Psychosomatic Medicine, 57*(1), 32–36.

Uchino, B. N. (2004). *Social support and physical health: Understanding the health consequences of relationships*. New Haven: Yale University Press.

Uno, D., Uchino, B. N., & Smith, T. W. (2002). Relationship quality moderates the effect of social support given by close friends on cardiovascular reactivity in women. *International Journal of Behavioral Medicine, 9*, 243–262.

Villingshoj, M., Ross, L., Thomsen, B. L., & Johansen, C. (2006). Does marital status and altered contact with the social network predict colorectal cancer survival? *European Journal of Cancer (Oxford, England: 1990), 42*(17), 3022–3027.

Welin, C., Lappas, G., & Wilhelmsen, L. (2000). Independent importance of psychosocial factors for prognosis after myocardial infarction. *Journal of Internal Medicine, 247*(6), 629–639.

Social Support at Work

► Job Satisfaction/Dissatisfaction

Social Ties

► Social Relationships

Societal Stress

► Social Stress

Society of Behavioral Medicine

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Synonyms

SBM

Definition

The Society of Behavioral Medicine (SBM) is a nonprofit organization founded in 1978. The organization strives to be multidisciplinary in nature, creating a dialogue between nursing, public health, psychological, and medical professionals to promote the study of interactions between behavior, biology, and the environment and to apply that knowledge to improve the health and well-being of individuals and communities. This is further illustrated through SBM's vision statement, which is "Better Health Through Behavior Change." In 2011, over 2000 behavioral and biomedical researchers and clinicians were members of SBM.

SBM hosts an annual meeting for its members and other behavioral medicine researchers and clinicians to share recent research findings and clinical strategies. SBM also sponsors two journals, *Annals of Behavioral Medicine* and *Translational Behavioral Medicine: Practice, Policy, and Research*.

References and Readings

Society of Behavioral Medicine. (2011). Society of Behavioral Medicine (SBM). Retrieved July 15, 2011, from http://www.sbm.org.

Sociocultural

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Synonyms

Social circumstance; Sociocultural context; Sociocultural factors; Socioeconomic position; Socioeconomic status (SES)

Definition

"Sociocultural" refers to a wide array of societal and cultural influences that impact thoughts, feelings, behaviors, and ultimately health outcomes. Sociocultural determinants of health and illness encompass socioeconomic status (SES) factors (traditionally assessed by income, education, occupation) and cultural factors. There are several dimensions encompassed by the term, which can include race, ethnicity, ethnic identity, sex, acculturation, language, beliefs and value systems, attitudes, and religion.

Description

Sociocultural factors are salient determinants of health and have been found to be associated with a

multitude of health outcomes including health behaviors (e.g., physical activity, diet, health screenings, and health-care utilization) and illness (e.g., cancer, diabetes, cardiovascular disease, and depression). Sociocultural factors are complex and may vary by sex, age, and racial/ethnic groups. In recent years, the term sociocultural has been extensively used in the literature in connection with physical and mental health outcomes.

Social and cultural factors play a central role in preventing illness, maintaining good health, and treating disease. Research has shown that an individual's social environment, family, neighborhood, school, and workplace have a significant impact on health. At the same time, cultural factors influence how physical and mental illness are viewed and diagnosed. A great advance in understanding the determinants of health and disease has been the identification of social and cultural factors influencing them. Social and cultural factors are pertinent not only to understanding individuals' health status but also recognizing the existing health disparities among different populations. In particular, a substantial body of research suggests that social factors stand at the root of health disparities (Marmot 2005).

Some of the most salient sociocultural factors studied in relation to health disparities, including morbidity and mortality, are SES and race/ethnicity. Levels of health within the USA vary dramatically among different social, economic, and racial/ethnic groups. Moreover, considerable research suggests that determinants of health often reflect economic disparities (Braveman et al. 2010). Higher income levels are linked with overall better health, including self-rated health, lower cardiovascular risk factors, and lower mortality (Braveman et al. 2011; Hajat et al. 2011). The incidence and prevalence of many diseases (e.g., cardiovascular disease, arthritis, diabetes, and cervical cancer) increases as SES decreases. In addition, SES differences in mortality have been observed for many causes of death including some cancers, diabetes, and cardiovascular disease. Similarly, individuals with higher SES have greater life expectancy rates than individuals with lower income levels (Braveman et al. 2011). In terms of health status, adults with lower incomes are more likely to report their health status as poor or fair compared with adults with higher incomes (Braveman et al. 2011).

Several explanations have been proposed to account for the association between socioeconomic standing and health. First, economic stability enables individuals to live in safer neighborhoods, access healthier food alternatives, have more leisure time for physical activity, and endure less stress. Second, income impacts access to high-quality health care such that lower SES individuals are less likely to be covered by health insurance and to receive high-quality health care (Braveman et al. 2011). Hence, the uninsured may have less access to preventive services (e.g., health screenings) and early diagnosis. Greater education is also linked with longer life expectancy. Individuals who have completed college have a greater life expectancy (at least 5 years longer) than individuals who have not completed high school (Braveman et al. 2011). Higher education levels are associated with greater knowledge regarding health and feelings of control (Braveman et al. 2011) over different domains of one's life. Therefore, education increases the likelihood that individuals will have the knowledge to prevent illness. As illustrated by these health patterns, SES disparities in health mirror a gradient pattern, with greater social and economic advantage being associated with better health.

Differences in health have also been observed based on race/ethnicity. For example, compared to non-Hispanic Whites, morbidity and mortality rates for cardiovascular disease (CVD) are higher among African-Americans (Payne et al. 2005). Compared to non-Hispanic Whites, African-Americans and Hispanics are more likely to have diabetes (Centers for Disease Control and Prevention 2011). Moreover, ethnic-minority and low-income groups have a disproportionate

burden of death and disability as a result of cardiovascular disease. In addition, although significant progress has been made in reducing cancer mortality rates in the USA, decreases in cancer mortality rates in ethnic minorities have been slower compared to non-Hispanic Whites (American Cancer Society 2011).

Culture refers to the shared values, beliefs, and norms held in common by a defined group of people. Within each culture, there is a set of behaviors and values related to health and illness which may vary between different groups, causing differing viewpoints toward illness. Each culture has a set of norms for behavior with related beliefs, knowledge, and customs. Acculturation, a related cultural construct, is often used to explain ethnic disparities in health outcomes. Acculturation as a predictive variable is based on the premise that culturally based knowledge, attitudes, and beliefs influence people to behave in particular ways and to select specific health choices. For limited English proficiency (LEP) individuals, language barriers can contribute to health disparities. For example, LEP individuals may encounter difficulties communicating with medical professionals, understanding printed health information or accessing health-related services due to lack of information about available services (American College of Physicians 2010). Moreover, some individuals may fear jeopardizing their immigration status by using health services. Research also suggests that cultural norms within the USA or Western society contribute to lifestyles and behaviors associated with risk factors for diseases (e.g., cancer, diabetes, cardiovascular disease) (Thomas et al. 2004). Therefore, health behavior interventions must address the target group's belief systems as well as cultural values.

Although the US population is diverse, health policies and interventions are often based on Western cultural assumptions. Often, minimal attention is given to aspects of culture from the perspective of individuals from diverse ethnic or SES membership groups. It is key to acknowledge that social and cultural factors may explain related health behaviors and, in part, elucidate disparities between ethnic/racial and SES groups. More specifically, research findings examined from the perspective of sociocultural differences may provide more meaningful information and help develop innovative intervention strategies for ameliorating some of the disparities in health outcomes and access to health care.

Cross-References

- ▶ Health Disparities
- ▶ Socioeconomic Status (SES)

References and Readings

American Cancer Society. (2011). *Cancer facts & figures*. Atlanta: American Cancer Society.

American College of Physicians. (2010). *Racial and ethnic disparities in health care*. Philadelphia: American College of Physicians.

Braveman, P. A., Cubbin, C., Egerter, S., Williams, D. R., & Pamuk, E. (2010). Socioeconomic disparities in the United States: What the patterns tells us. *American Journal of Public Health, 100*, S186–S196.

Braveman, P. A., Egerter, S. A., & Mockenhaupt, R. E. (2011). Broadening the focus: The need to address the social determinants of health. *American Journal of Preventive Medicine, 40*, S4–S18.

Centers for Disease Control and Prevention. (2011). *National diabetes fact sheet: National estimates and general information on diabetes and prediabetes in the United States, 2011*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

Hajat, A., Kaufman, J. S., Rose, K. M., Siddiqi, A., & Thomas, J. C. (2011). Long-term effects of wealth on mortality and self-rated health status. *American Journal of Epidemiology, 173*, 192–200.

Marmot, M. (2005). Social determinants of health inequalities. *The Lancet, 365*, 1099–1104.

Payne, T. J., Wyatt, S. B., Mosley, T. H., Dubbert, P. M., Guiterrez-Mohammed, M. L., Calvin, R. L., et al. (2005). Sociocultural methods in the Jackson Heart Study: Conceptual and descriptive overview. *Ethnicity & Disease, 15*, S6-38–S6-48.

Thomas, S. B., Fine, M. J., & Ibrahim, S. A. (2004). Health disparities: The importance of culture and health communication. *American Journal of Public Health, 94*, 2050.

Sociocultural Context

► Sociocultural

Sociocultural Differences

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Definition

Sociocultural approaches to understanding differences in health call attention to the roles of and potential interdependence between social and cultural factors for health outcomes.

Cultural attitudes, beliefs, values, history, and systems of knowledge are interdependent with the social environment that includes economic status, community and family systems, and interpersonal relationships. Together, sociocultural factors may impact health in numerous ways, such as influencing access/barriers to health care and service utilization preferences/patterns as well as affecting health behaviors such as diet and exercise.

Cross-References

► Aerobic Exercise
► Sociocultural

Sociocultural Factors

► Sociocultural

Socioeconomic Position

► Sociocultural
► Socio-economic Status

Socio-economic Status

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Synonyms

Socioeconomic position

Definition

Social class is often used by social scientists when the more generic socioeconomic status or socioeconomic position is preferable. Socioeconomic status refers to particular social strata in society and can be measured at a personal or geographical level. For an individual, indicators include occupational social class, occupational prestige, educational attainment, household income, housing tenure, household amenities, and car ownership. For a geographical area, composite measures are often derived from the characteristics of residents in a defined location (Galobardes et al. 2007).

In the context of epidemiology, socioeconomic status has been most commonly related to disease (particularly cardiovascular disease) and disease risk factors (particularly health behaviors such as smoking). Socioeconomic inequalities (variations) in health are essentially universal: with the exception of very few outcomes, poorer health is more common in poorer people. As such, reducing these differentials is a priority for many governments and health agencies.

References and Further Reading

Galobardes, B., Lynch, J., & Davey Smith, G. (2007). Measuring socioeconomic position in health research. *British Medical Bulletin, 81–82*, 21–37.

Socioeconomic Status (SES)

▶ Education, Lack of: As a Risk Factor
▶ Social Factors
▶ Sociocultural

Sodium

▶ Salt, Intake

Sodium Chloride

▶ Salt, Intake

Sodium, Sodium Sensitivity

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Definition

Sodium chloride (NaCl), commonly known as salt, is a molecule crucial for fluid balance and free-water homeostasis. However, overconsumption of sodium/salt plays an important role in the development of essential hypertension. Essential hypertension is seen almost exclusively in societies where average daily sodium consumption is greater than 2.3 g. In contrast, hypertension is rare in populations with low-sodium consumption (typically less than 1.2 g/day). These effects of sodium consumption appear independent of other potential causes of essential hypertension, such as obesity.

The blood pressure (BP) responsiveness to variations in sodium intake is known as salt sensitivity.

The change in BP to salt intake varies significantly between individuals and in the same individual at different times.

Salt sensitivity also increases with age and is more prominent in those with diabetes, obesity, and metabolic syndrome.

It may also be more common in African-Americans and other populations, in which excess salt intake may play an important role in the development of hypertension.

There is evidence to suggest that salt-sensitive individuals with normal blood pressure are at a greater risk of developing hypertension and at further risk of hypertension progression and poor blood pressure control. The mechanisms of salt sensitivity are incompletely understood but likely involve a combination of altered salt/water homeostasis, abnormal vascular signaling pathways, and other metabolic abnormalities such as type 2 diabetes and electrolyte abnormalities, such as hypokalemia.

References and Readings

Barba, G., Galletti, F., Cappuccio, F. P., Siani, A., Venezia, A., Versiero, M., Della Valle, E., Sorrentino, P., Tarantino, G., Farinaro, E., & Strazzullo, P. (2007). Incidence of hypertension in individuals with different blood pressure salt-sensitivity: Results of a 15-year follow-up study. *Journal of Hypertension, 25*(7), 1465–1471.

Obarzanek, E., Proschan, M. A., Vollmer, W. M., Moore, T. J., Sacks, F. M., Appel, L. J., Svetkey, L. P., Most-Windhauser, M. M., & Cutler, J. A. (2003). Individual blood pressure responses to changes in salt intake: Results from the DASH-Sodium trial. *Hypertension, 42*(4), 459–467.

Solid Fats

▶ Fat: Saturated, Unsaturated

Somatic Symptom Disorder

- ▶ Hypochondriasis
- ▶ Somatization
- ▶ Somatoform Disorders

Somatic Symptoms

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Definition

Mental health professionals commonly label bodily symptoms as "somatic" to distinguish them from cognitive, emotional, or other types of non-somatic symptoms (Kroenke 2007a). In contrast, bodily symptoms are more often referred to as "physical" symptoms by those practicing in general medical, surgical and other non-mental health care professions. Somatic symptoms are exceedingly prevalent, accounting for over half of all outpatient encounters. About half of these are pain complaints (e.g., headache, chest pain, abdominal pain, back pain, joint pains), a quarter are upper respiratory (e.g., cough, sore throat, ear or nasal symptoms), and the remainder are non-pain, non-upper-respiratory symptoms (e.g., fatigue, insomnia, dizziness, palpitations).

Description

Epidemiology

About 80% of individuals in the general population experience one or more symptoms each month, of who less than 1 in 4 seek care (Kroenke and Rosmalen 2006). This ubiquitous nature of somatic symptoms mandates that some thresholds be set to distinguish most "persons" who experience common symptoms from the smaller subset of individuals who qualify as "patients." Some thresholds might include severity of the symptom: its duration or persistence; the degree of occupational or social impairment; the level of patient distress, concerns or worries; the decision to seek treatment or use health care; and the direct and indirect financial costs.

An exact medical diagnosis that accounts for the symptom is often not established, with at least one-third of somatic symptoms lacking an adequate physical explanation and referred to by a variety of labels, including functional, idiopathic, atypical, somatoform, or unexplained.

About three-fourths of outpatients presenting with somatic complaints experience improvement within 2 weeks, while 20–25% of symptoms become chronic or recurrent.

Functional Somatic Syndromes

These conditions consist of a cluster of somatic symptoms for which the etiology is poorly understood and include disorders such as irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, temporomandibular disorder, interstitial cystitis, and others. Experts have questioned whether these are all separate disorders or instead part of a group of poorly explained somatic conditions sharing common features. Supporting the latter, literature syntheses have revealed that these disorders frequently overlap, both at the level of specific syndromes (half to two-thirds of patients with one syndrome also suffer from one or more additional syndromes) as well as in terms of individual symptoms (Kroenke and Rosmalen 2006). Second, they are similar in rates of psychiatric comorbidity, particularly depression and anxiety (Henningsen et al. 2003). Third, functional somatic syndromes respond similarly to certain therapies traditionally considered "psychological" treatments, such as antidepressants and cognitive-behavioral therapy.

Psychological Comorbidity

In patients presenting with poorly explained physical symptoms, a depressive disorder can be diagnosed 50–60% of the time, and an anxiety disorder 40–50% of the time, regardless of the type of symptom. While the specific type of symptom is not particularly important in terms of

predicting depression or anxiety, the number of symptoms is. In two primary care studies involving 1500 patients, those who endorsed 0–1, 2–3, 4–5, 6–8, or ≥ 9 physical symptoms on a 15-symptom scale, the proportion with a depressive or anxiety disorder was 6%, 20%, 33%, 58%, and 80% respectively, suggesting a "dose-response" effect between the number of physical symptoms and the likelihood of psychiatric comorbidity.

Two-thirds of primary care patients with major depression present exclusively with somatic complaints, and half report multiple, unexplained somatic symptoms. Also, depression is present in a quarter to a third of patients referred to medical specialty clinics and, if depressed, referred patients are only about a quarter as likely to have a physical diagnosis established as an explanation for their symptoms triggering the referral. Even disease-specific somatic symptoms (e.g., chest pain in patients with coronary artery disease, dyspnea in patients with pulmonary disease, joint pain in patients with arthritis) are at least as strongly associated with depression and anxiety as they are with objective physiologic measures of the medical disorder (Katon et al. 2007).

Overlap among somatic, anxiety and depressive symptoms (the *SAD triad)* is more common than the "pure" form of any of the three types of symptoms (Löwe et al. 2008). For example, very high levels of depressive, anxiety and somatic symptoms are present in 7%, 8%, and 10% of primary care patients, respectively. However, only 26% of depressed patients have depression alone (i.e., without high levels of anxiety and/or somatic symptoms), 43% of anxious patients have anxiety only, and 46% of patients with high somatic symptom levels have somatization alone. Predictors of psychological comorbidity in patients with somatic symptoms are summarized in Table 1.

Somatic Symptoms, Table 1 Predictors of psychological comorbidity in patients with somatic symptoms

Symptom remains medically unexplained after clinical assessment
Multiple symptoms
Three or more unexplained symptoms
Pain symptoms in two or more regions of the body
Multiple functional somatic syndromes
Chronic or recurrent symptom (s)
Excessive health care use
Medication history
Polypharmacy (especially for symptoms)
Poor response of symptoms to multiple medications
Nocebo response (nonspecific adverse effects to multiple medications)
Difficult clinician-patient relationship
Number of S4 predictors[a]
Stress recently
Symptom count is high
Self-rated health is low
Severity of symptom is high

[a]The four S4 predictors are (1) stress in past week (yes/no); (2) Patient reports being "bothered a lot" by five or more symptoms on the PHQ-15 scale of 15 somatic symptoms; (3) self-rated overall health of poor or fair on a 5-point scale (excellent, very good, good, fair, poor); (4) self-rated severity of presenting somatic symptom of $^{3}6$ on 0 (none) to 10 (unbearable) scale. The likelihood of a depressive or anxiety disorder with 0, 1, 2, 3, or 4 of these S4 predictors is 8%, 16%, 43%, 69%, and 94%, respectively

Somatoform Disorders

Somatoform disorders currently defined in the American Psychiatric Association's Diagnostic and Statistical Manual, 4th Edition (DSM-IV) include somatization disorder (chronic history of multiple medically unexplained symptoms), conversion disorder (unexplained neurological symptoms), hypochondriasis (preoccupation with having a serious medical illness that persists despite medical evaluations and reassurance), body dysmorphic disorder (distorted perceptions of specific bodily features), and chronic pain disorder. However, these are likely to be substantially revised in DSM-V (Kroenke et al. 2007). In particular, criteria for the most common type of somatoform disorder (full and abridged versions of somatization disorder) are likely to rely less on symptom counts or the degree to which symptoms are "medically explained" and more on positive psychological criteria characteristic of somatizing patients (e.g., excessive illness worry and health anxiety, inordinate health care use, catastrophizing).

Measuring Somatic Symptoms

The PHQ-15 is a brief, freely-available scale (www.phqscreeners.com) that measures 15 symptoms that account for more than 90% of non-upper-respiratory symptoms seen in primary

care (Kroenke et al. 2010). The PHQ-15 asks patients to rate how much they have been bothered by each symptom during the past month on a 0 ("not at all") to 2 ("bothered a lot") scale. Thus, the total score ranges from 0 to 30, with cutpoints of 5, 10, and 15 representing thresholds for mild, moderate, and severe somatic symptom severity, respectively.

Increasing scores on the PHQ-15 are strongly associated with functional impairment, disability, health care use, and somatoform disorder diagnoses. Also, items on the PHQ-15 overlap better with other validated somatization screeners than any other two screeners do with one another. There is emerging evidence that the PHQ-15 is responsive to treatment.

Treatment

Treatment of somatoform disorders as well as functional somatic syndromes has been recently reviewed (Abbass et al. 2009; Jackson et al. 2006; Kroenke 2007b). In addition to symptom-specific treatments (e.g., analgesics for pain, medications specific to the symptoms for irritable bowel syndrome, medications recently approved for fibromyalgia), the two most evidence-based treatments for both somatoform disorders and functional somatic syndromes are cognitive behavioral therapy and antidepressants, which have a beneficial effect on the somatic symptoms in these conditions independent of their effect on psychological symptoms such as depression and anxiety. Additionally, regular visits with a primary physician, avoidance of excessive testing, and evaluation of new symptoms (but not repeated evaluation of chronic symptoms) is beneficial. A clinical approach to the patient with unexplained somatic symptoms is outlined in Table 2.

Strategies for managing chronic somatization

- Schedule regular, brief appointments that are not related to symptom exacerbations
- Limit extensive diagnostic testing and multiple subspecialty referrals, especially for symptoms previously evaluated
- When new symptoms arise, conduct focused evaluations and testing rather than exhaustive work-ups

Somatic Symptoms, Table 2 Clinical approach to the patient with unexplained somatic symptoms

Initial visit	
Symptom-specific evaluation	Focus the interview and physical examination on the relevant symptom(s) Stratify symptoms into those that are at higher risk of a serious cause (e.g., angina-like chest pain; acute abdominal pain; syncope) and those that are seldom urgent (back pain, fatigue, insomnia). Identify "red flags" of a potentially serious cause (e.g., focal neurologic findings in patient with dizziness or headache; abnormal cardiac exam in patient with syncope)
Probe for symptom-specific concerns and expectations	"Is there anything else you were worried about?" (Serious cause? How long symptom might last?) "Is there anything else you wanted or thought might be helpful?" (Test? subspecialty referral? Specific treatment?)
Consider short-term use of symptom-specific medications (often available over-the-counter)	Simple analgesics for pain Gastrointestinal medications for dyspepsia or constipation Decongestants, antihistamines, cough suppressants for upper respiratory symptoms
Watchful waiting	Have patient follow-up in 2–6 weeks if symptom is not resolved
Follow-up visits	
Psychological screening	Especially for treatable depressive and anxiety disorders (see www.phqscreeners.com) Assess for somatization (PHQ-15 or other scale)
Diagnostic evaluation	Selective testing and/or specialty referral, especially if new unexplained symptom, if findings on interview or examination are worrisome for serious cause, or if patient insists

(continued)

Somatic Symptoms, Table 2 (continued)

Chronic symptom therapies	Pharmacological – analgesics; gastrointestinal medications; antidepressants, other Nonpharmacological – cognitive-behavioral therapy (CBT); other psychological or behavioral therapies; exercise; pain self-management programs; reattribution

- Empathize with the complaint. Do not dispute the reality of the symptom or associated impairment.
- Focus on symptom management/reduction rather than elimination (coping rather than cure).
- Strive for gradual rehabilitation (maximizing function despite the symptom) rather than chronic disability
- Emphasize that referral is for consultation or co-management rather than dismissal (i.e., you are not "dumping" the patient)

Cross-References

- ▶ Antidepressant Medications
- ▶ Anxiety Disorder
- ▶ Chronic Pain
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Depression: Treatment
- ▶ Medically Unexplained Symptoms
- ▶ Pain
- ▶ Somatization
- ▶ Somatoform Disorders
- ▶ Stress
- ▶ Symptoms

References and Readings

Abbass, A., Kisely, S., & Kroenke, K. (2009). Short-term psychodynamic psychotherapy for somatic disorders: Systematic review and meta-analysis of clinical trials. *Psychotherapy and Psychosomatics, 78*, 265–274.

Dimsdale, J. E., Xin, Y., Kleinman, A., Patel, V., Narrow, W. E., Sirvatka, P. J., & Regier, D. A. (Eds.). (2009). *Somatic presentations of mental disorders: Refining the research agenda for DSM-V.* Arlington: American Psychiatric Association.

Henningsen, P., Zimmermann, T., & Sattel, H. (2003). Medically unexplained physical symptoms, anxiety, and depression: A meta-analytic review. *Psychosomatic Medicine, 65*, 528–533.

Jackson, J. L., O'Malley, P. G., & Kroenke, K. (2006). Antidepressants and cognitive behavioral therapy for symptom syndromes. *CNS Spectrums, 11*, 212–222.

Katon, W., Lin, E., & Kroenke, K. (2007). The association of depression and anxiety with medical symptom burden in patients with chronic medical illness. *General Hospital Psychiatry, 29*, 147–155.

Kroenke, K. (2007a). Somatoform disorders and recent diagnostic controversies. *Psychiatric Clinics of North America, 30*, 593–619.

Kroenke, K. (2007b). Efficacy of treatment for somatoform disorders: A review of randomized clinical trials. *Psychosomatic Medicine, 69*, 881–888.

Kroenke, K., & Rosmalen, J. G. M. (2006). Symptoms, syndromes and the value of psychiatric diagnostics in patients with functional somatic disorders. *Medical Clinics of North America, 90*, 603–626.

Kroenke, K., Sharpe, M., & Sykes, R. (2007). Revising the classification of somatoform disorders: Key questions and preliminary recommendations. *Psychosomatics, 28*, 277–285.

Kroenke, K., Spitzer, R. L., Williams, J. B. W., & Löwe, B. (2010). The patient health questionnaire somatic, anxiety, and depressive symptom scales: A systematic review. *General Hospital Psychiatry, 32*, 345–359.

Löwe, B., Spitzer, R. L., Williams, J. B. W., Mussell, M., Schellberg, D., & Kroenke, K. (2008). Depression, anxiety, and somatization in primary care: Syndrome overlap and functional impairment. *General Hospital Psychiatry, 30*, 191–199.

S

Somatization

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Synonyms

Psychosomatics; Somatic symptom disorder

Definition

Somatization can be defined as "the tendency to experience a variety of somatic symptoms that are usually poorly described by biomedical disease processes"

Description

The term "somatization" goes back to psychodynamic theory and describes the transformation of unconscious conflicts and repressed emotions into somatic symptoms. Later on, Lipowski defined somatization as "a tendency to experience and communicate somatic distress in response to psychosocial stress and to seek medical help for it" (Lipowski 1986). Therefore, this definition postulates that psychosocial stress is the cause for somatization and that seeking medical help is a necessary feature of this syndrome. However, current concepts of somatization use this term more descriptively. According to these modern concepts, somatization could be defined as "the tendency to experience a variety of somatic symptoms that are usually poorly described by biomedical disease processes." In this definition, the presence of multiple somatic complaints is the core feature of somatization.

Somatization itself is not a diagnosis, but is frequently related to the former diagnostic group of "somatoform disorders," which has been reformulated to "Somatic Symptom Disorder" in DSM-5. If multiple somatic symptoms are part of a depressive syndrome or anxiety disorder, the term could be also used. It is not recommended to use the term "somatization" for a postulated association between psychological conflicts and serious medical conditions (e.g., cancer). However, medical conditions like cancer or diabetes can also be associated with multiple medical symptoms that are not explained by the biomedical disease itself. For this subgroup of patients with serious medical conditions, the additional use of the term "somatization" could be appropriate.

Current behavioral-medical concepts prefer the process of "somatosensory amplification" and other perceptual models to describe the development and maintenance of somatization. Somatosensory amplification summarizes the process of focusing attention to bodily perceptions; together with health worries and a catastrophizing style of interpreting bodily perceptions, selective attention leads to an amplified style of perceiving somatic symptoms. If symptoms are chronic, further sensitization processes might be involved. Further details on modern concepts of somatization can be found in Barsky (1992), Henningsen et al. (2018), and Rief and Martin (2014).

Cross-References

▶ Somatoform Disorders

References and Further Readings

Barsky, A. J. (1992). Amplification, somatization, and the somatoform disorders. *Psychosomatics, 33*, 28–34.

Henningsen, P., Gundel, H., Kop, W. J., Lowe, B., Martin, A., Rief, W., Rosmalen, J. G. M., Schroder, A., van der Feltz-Cornelis, C., Van den Bergh, O., & Grp, E.-S. (2018). Persistent physical symptoms as perceptual dysregulation: A Neuropsychobehavioral model and its clinical implications. *Psychosomatic Medicine, 80*(5), 422–431. https://doi.org/10.1097/psy.0000000000000588.

Lipowski, Z. J. (1986). Somatization: A borderland between medicine and psychiatry. *Canadian Medical Association Journal, 135*, 609–614.

Rief, W., & Martin, A. (2014). How to use the new DSM-5 diagnosis somatic symptom disorder in research and practice? A critical evaluation and a proposal for modifications. *Annual Review of Clinical Psychology, 10*, 339–367.

Somatoform Disorders

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Synonyms

Functional somatic symptoms; Medically unexplained physical symptoms; Somatic symptom disorder

Definition

The common feature of somatoform disorders is the presence of physical symptoms that could indicate a general medical condition, but the physical symptoms are not fully explained by a well-known biomedical disease, by the direct effects of a substance, or by another mental disorder. Somatoform disorders have been replaced by somatic symptom disorders in DSM-5. This group includes somatic symptom disorder, conversion disorder, illness anxiety disorder, and others.

Description

The term "somatoform disorder" has been introduced by DSM-III (*Diagnostic and Statistical Manual of Mental Disorders*, third version) as a category for a group of diagnoses. The common feature of somatoform disorders is the presence of physical symptoms that could indicate a general medical condition, but the physical symptoms are not fully explained by a well-known biomedical disease, by the direct effects of a substance, or by another mental disorder (e.g., panic disorder, depression). Symptoms must cause clinically significant distress or impairment. Somatoform disorders have to be distinguished from factitious disorders and malingering; the physical symptoms in somatoform disorders are not intentional or imagined, but patients perceive these symptoms similar to other physical symptoms caused by medical conditions.

After conceptual critique, the group of somatoform disorders has been replaced by the group of "somatic symptom disorder" (SSD). Again, somatic symptoms are in the core of classifying SSD, but the role of the etiology was discarded, while psychological features such as illness worries, ruminations, and others play a more crucial role. Beyond SSD, illness anxiety disorder, conversion disorder, psychological factors affecting other medical conditions, and factitious disorders are further diagnoses in this category. If pain is the predominant symptom, a diagnoses of somatic symptom disorder with predominant pain can be considered.

While most people suffer from somatic symptoms from time to time, a diagnosis of somatoform disorder or SSD should be only given if the symptoms cause clinically significant distress or impairment. A diagnosis of conversion disorder is justified if people suffer from motor or sensory symptoms or deficits that are not fully accounted by a medical condition. Illness anxiety disorder is considered if illness anxieties (such as in hypochondriasis) are predominant, but somatic symptoms are not significant (such as in SSD). In "Western" cultures, pain symptoms are the most frequent somatic symptoms. If pain duration is longer than 3 months, it should be considered to be chronic. In most cases, chronic pain conditions are not sufficiently understood with pure biomedical approaches, but psychological and social factors have to be also considered.

Somatoform disorders and somatic symptom disorders can develop as a result of the interaction of psychological factors (selective attention to bodily processes, overinterpretation of somatic sensations, illness fears, demoralization) with biomedical factors (e.g., traumatic injuries, car accidents, biological dysregulation of stress and immune responses) and with social factors (e.g., reinforcement of symptom expression by proxies, strong biomedical orientation of health-care systems, fears of doctors to overlook biomedical causes). While somatization typically refers to the experience of multiple medically unexplained symptoms, the concept of somatosensory amplification refers to the self-reinforcing circle of attention to somatic processes, overinterpretation of somatic sensations, intensified physical sensations, and behavioral consequences (Barsky 1992). Modern concepts emphasize perceptual processes (Henningsen et al. 2018). Somatoform disorders and SSD are more prevalent in women than in men. General somatic symptoms can be found in more than 10% of the population (Wittchen and Jacobi 2005).

The classification of "medically explained" versus "medically unexplained" symptoms was found to be unreliable, and medical doctors highly disagree in their ratings about causality of somatic symptoms. Therefore the former concept of somatoform disorders was criticized and accused to further continue a "mind-body separation"

instead of favoring a biopsychosocial model. The revision of this category in DSM-5 tries to overcome these shortcomings, but even the new classification bears some problems (Rief and Martin 2014).

To date, no well-founded pharmacological treatment is available for patients with somatoform syndromes. There is some evidence for the use of SSRIs (selective serotonin reuptake inhibitors) in patients with illness anxieties. Psychological interventions (especially behavioral-medical interventions) have been shown to be effective in all groups of somatoform disorders and somatic symptom disorders. However, effect sizes vary substantially, with highest effect sizes for the psychological treatment of illness anxiety, while effect sizes for the treatment of somatization disorder or chronic pain conditions are only in the low to medium range. The rate of overlooked medical conditions that can explain the somatic symptoms does not seem to be increased compared to other mental disorders: long-term studies reveal that less than 10% of patients with somatic symptom disorders must be considered to be misdiagnosed biomedical conditions.

Cross-References

- ▶ Functional Somatic Syndromes
- ▶ Somatization
- ▶ Somatic Symptom Disorder

References and Further Readings

Barsky, A. J. (1992). Amplification, somatization, and the somatoform disorders. *Psychosomatics, 33*, 28–34.

Henningsen, P., Gundel, H., Kop, W. J., Lowe, B., Martin, A., Rief, W., Rosmalen, J. G. M., Schroder, A., van der Feltz-Cornelis, C., Van den Bergh, O., & Grp, Euronet-Soma. (2018). Persistent physical symptoms as perceptual dysregulation: A Neuropsychobehavioral model and its clinical implications. *Psychosomatic Medicine, 80*(5), 422–431. https://doi.org/10.1097/psy.0000000000000588.

Rief, W., & Martin, A. (2014). How to use the new DSM-5 diagnosis somatic symptom disorder in research and practice? A critical evaluation and a proposal for modifications. *Annual Review of Clinical Psychology, 10*, 339–367.

Wittchen, H. U., & Jacobi, F. (2005). Size and burden of mental disorders in Europe – a critical review and appraisal of 27 studies. *European Neuropsychopharmacology, 15*, 357–376.

Spatial Analysis

▶ Geographic Information System (GIS) Technology

Spectral Analysis

▶ Quantitative EEG Including the Five Common Bandwidths (Delta, Theta, Alpha, Sigma, and Beta)

Speech and Language Pathology

▶ Speech and Language Therapy

Speech and Language Therapy

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Synonyms

Speech and language pathology; Speech therapy; Speech, language, and communication therapy

Definition

Speech and language therapy is an evidence-based discipline that anticipates and responds to the needs of individuals who experience speech, language, communication, or swallowing difficulties. Speech and language therapy works in partnership with individuals and their families and with other professions and agencies to reduce the impact of these often isolating difficulties on well-being and the ability to participate in daily life (Royal College of Speech and Language Therapists 2005).

Speech and language therapists (SLTs) are the lead experts regarding communication and swallowing disorders. This does not mean that others do not work within these areas or that others do not have many skills that may overlap with or complement those of SLTs. Rather, SLTs, through their preregistration education, and later experience, have greater depth and breadth of knowledge and understanding of these clinical areas and associated difficulties. This enables SLTs to lead on the assessment, differential diagnosis, intervention with, and management of individuals with communication and swallowing disorders.

Speech and language therapy assistants and bilingual co-practitioners are integral members of the speech and language therapy team, employed to act in a supporting role under the direction of a professionally qualified SLT.

Description

A wide range of individuals can potentially benefit from speech and language therapy, including:

- Babies with feeding and swallowing difficulties
- Children (from neonates to school age), adolescents, and adults with special needs in communication, communication disability, and/or swallowing disorders associated with diagnosed impairments, genetic and medical conditions, trauma, developmental delays, mental health problems, and learning disability
- Children (from neonates to school age), adolescents, and adults with special needs in the following areas: speech, voice, fluency, language, psychologically based communication disorders, social skills, problem solving, literacy, swallowing functions, and alternative and augmentative communication (AAC)
- Parents and families, caregivers, communication partners, friends, and colleagues of people with communication and swallowing disorders

Speech and language therapists work in and across a variety of settings.

Within education, these settings include:

- Local education authority nurseries and schools (mainstream and special)
- Language and communication units and colleges of further education
- Independent nurseries and schools
- Play groups
- Government-funded initiatives

Within health and social care, settings include:

- Hospitals inpatient and outpatient centers and hospices
- Specialist centers: child development centers, rehabilitation centers, specialist joint consultative clinics
- Primary care: community clinics, community day centers
- Supported living homes
- Mental health services
- Initiatives in areas of social deprivation (such as Sure Start)

Speech and language therapists have an increasing role within the legal system, including within the penal system/prisons, in court tribunals, and as part of adult and child protection services.

They also work in independent practice, as part of social enterprises and for the voluntary/charitable sector.

All speech and language therapy intervention is delivered on the basis of ongoing assessment and review of progress with the individual (and/or carer as appropriate) as measured against targeted outcomes. Various approaches or models of working have been developed to meet the needs of individuals and context.

The following are key principles guiding the provision of services:

- The rights, wishes, and dignities of each individual and their carers are respected at all times.
- Effective intervention is based on a holistic understanding of the individual, including their social, cultural, economic, political, and linguistic context.
- The safety of the individual is paramount.
- Speech and language therapy intervention aims to be efficient and effective, i.e., best results against targeted outcomes within given resources.

Speech and language therapy services may operate at the level of the person (working with individuals); the level of their environment (working with people, processes, or settings); and the level of the wider community (influencing attitude, culture, or practice). The form of intervention will vary according to the changing needs of the individual and contexts.

Benefits

Speech and language therapy can contribute to the following health, educational, and psychosocial benefits:

- Improvement in general health and well-being
- Increased independence
- Improved participation in family, social, occupational, and educational activities
- Improved social and family relationships
- Reduction in the negative effects of communication disability and the harm or distress this may cause to the individual and others
- Reduced risk of surgical intervention and poor nutrition in the case of individuals with swallowing disorders
- Reduced health risks and length of hospital stay through the prevention of respiratory problems associated with swallowing difficulties
- Reduced risk of surgical intervention by maintaining healthy voice mechanisms
- Reduced risk of educational failure
- Reduced risk of social isolation
- Prevention of certain speech, language, and communication disorders

Outcomes

The outcomes of speech and language therapy include:

- Diagnosis of communication and/or swallowing disorders
- Maintenance of optimal communication and/or swallowing abilities
- Improvement in the speech, language, and communication abilities of individuals
- Improved use of existing function
- Reduction of communication anxiety and avoidance
- Provision and use of AAC where oral communication is limited or precluded by a physical condition
- Improvement in interaction and effective social communication
- Increased awareness of others about communication and/or swallowing disorders, intervention, and management
- Improved communication environment
- Greater opportunities for communication
- Improvement in the individual's understanding of the nature and implications of a communication and/or swallowing disorder

In 2010, the Royal College of Speech and Language Therapists (RCSLT) commissioned analysts Matrix Evidence to review the existing evidence and undertake an economic evaluation

of the provision of speech and language therapy to four specific client groups. The aim of this was to pinpoint the benefits generated by speech and language therapy in relation to the costs of provision. The result was the UK-wide study "The economic case for speech and language therapy" (Marsh et al. 2010).

The Matrix research aimed to determine the costs and benefits for four common speech and language therapy client groups:

- Adults with dysphagia post stroke
- Adults with aphasia post stroke
- Children with speech and language impairment (SLI)
- Children with autism

Matrix Evidence undertook an evaluation of the costs and benefits of speech and language therapy intervention for each condition and compared either the effects of speech and language therapy with the effects of alternative forms of treatment, or the effects of intensive against less intensive therapy. Specifically, the analysis evaluated:

- Speech and language therapy for stroke survivors with dysphagia compared with "usual" care
- Enhanced NHS speech and language therapy for stroke survivors with aphasia compared with usual NHS therapy
- Enhanced speech and language therapy for children with SLI compared with existing therapy provision
- Enhanced speech and language therapy for children with autism compared with usual SLT treatment

The results of the Matrix report show that speech and language therapy for all four cohorts and conditions represents an efficient use of public resources. The net benefits of the interventions - including health and social care cost savings, quality of life, and productivity gains – are positive and exceed their costs. The report shows the total annual net benefit across aphasia, SLI, and autism is £765 million; it excludes dysphagia from the calculation since the two poststroke conditions are not mutually exclusive.

The RCSLT

Established as the College of Speech Therapists in 1945, the Royal College of Speech and Language Therapists (RCSLT) is the membership organization for UK SLTs, providing and promoting:

- Support and professional leadership for members, including the setting of standards
- Strategic direction for the profession
- Consistent, effective, and accurate professional representation to external bodies and the government
- Heightened public awareness of the medical, social, and emotional effects of communication, eating, drinking, and swallowing difficulties
- Heightened awareness of the contribution of speech and language therapy with the public, government, other professions, and the media

The RCSLT provides leadership so that issues concerning the profession are reflected in public policy and people with communication, eating, drinking, or swallowing difficulties receive optimum care.

Cross-References

▶ Alzheimer's Disease
▶ Brain Injury
▶ Brain Tumor
▶ Chronic Disease Management
▶ Cognitive Impairment
▶ Cost-Effectiveness
▶ Neurological
▶ Neuromuscular Diseases

- ▶ Nutrition
- ▶ Occupational Therapy
- ▶ Rehabilitation
- ▶ Stroke Burden

References and Further Reading

For more information, visit the RCSLT website: www.rcslt.org

Marsh, K., Bertranou, E., Suominen, H., Venkatachalam, M. (2010). An economic evaluation of speech and language therapy: Final report. December 2010. *Matrix Evidence*.

Royal College of Speech Therapists Language. (2005). *Communicating quality* (Vol. 3, pp. 2–28). London: RCSLT.

Speech Therapy

- ▶ Speech and Language Therapy

Speech, Language, and Communication Therapy

- ▶ Speech and Language Therapy

Sperm Donation

- ▶ In Vitro Fertilization, Assisted Reproductive Technology
- ▶ Surrogacy

Sperm Donor

- ▶ In Vitro Fertilization, Assisted Reproductive Technology
- ▶ Surrogacy

Spinal Muscular Atrophy

- ▶ Neuromuscular Diseases

Spiritual

- ▶ Religion/Spirituality

Spiritual Beliefs

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Synonyms

Religious beliefs; Spirituality

Definition

The concept of spiritual beliefs is a critical component of the broader terms *spirituality* or *religiousness* and is to some degree inseparable from them. The meanings of these terms, however, remain elusive. In fact, the paramount importance, and difficulty, with defining spiritual concepts has proven a prominent obstacle to establishing a cohesive body of literature. The empirical research is littered with varying, and sometimes incompatible, ways of understanding spiritual constructs (Hill and Pargament 2003; Kapuscinski and Masters 2010). Researchers disagree, for example, regarding the relationship of religiousness and spirituality. This issue takes on special significance when *beliefs* in particular are the subject of consideration because Western notions of religion often differentiate believers from unbelievers based on their convictions (beliefs), rather than behaviors. Historically, spirituality and religiousness were considered to be, if

not the same entity, intimately tied to one other. One's personal ideas about and experiences of the sacred were both informed by and occurred in the context of institutional religious beliefs and practices. In recent years, however, a growing minority of Americans have begun to identify themselves as "spiritual but not religious." The distinction often involves the term spiritual being used to signify personal beliefs or experiences of the sacred, apart from traditional religious doctrine and organizations. Interestingly, although most people recognize religion and spirituality as somewhat different concepts, they also see the terms as sharing much in common, including traditional religious concepts of God, Christ, and the church (Zinnbauer et al. 1999).

Some researchers have taken the connotation of spirituality as an individual's internal communion with the transcendent and sharply separated it from the idea of religiousness now defined narrowly as involvement with organized beliefs and practices (Hill and Pargament 2003; Zinnbauer et al. 1999). In some cases, the supernatural or sacred is completely removed from the notion of spirituality or spiritual beliefs, leaving a search for meaning or perspective that is unrelated to the transcendent. Some researchers (Kapuscinski and Masters 2010; Koenig 2010) do not support this separation on several grounds, including that the removal of the sacred creates a somewhat artificial notion of spirituality, devoid of any substance that separates it from mental health variables like optimism and purpose in life. Therefore, the conceptual state of affairs on these topics does not allow spiritual beliefs to be cleanly separated from spirituality or religiousness. All of these concepts overlap significantly.

Nevertheless, despite the overlap, thematic elements in the literature suggest that spiritual beliefs per se may be defined as convictions about self, others, and the world, which emerge from a search for the transcendent or sacred, and include the values regarding lifestyle and moral conduct derived from these convictions. Spiritual beliefs may be roughly synonymous with the notion of spiritual worldview, the basis of which is a belief in the transcendent. Beliefs may or may not include doctrine associated with religious institutions (e.g., that an omnipotent God created the universe, as in the Judeo-Christian tradition).

Description

Most of the questionnaires designed to assess spirituality emphasize beliefs as a critical element of what is measured, and a few, like the Beliefs and Values Scale (King et al., 2006) and the Royal Free Interview for Spiritual and Religious Beliefs (King et al. 2001), focus on beliefs specifically. Thus, consistent with the discussion above, the spiritual beliefs component of religiousness and spirituality (R/S) is strongly embedded in research findings on R/S, even when studies do not claim to focus specifically on beliefs. A wealth of evidence demonstrates that both R/S in general, and sometimes beliefs in particular, influence physical and mental health.

Physical Health

Available research generally indicates a relationship between R/S variables and better physical health and implicates the value of incorporating this aspect of culture into health-promoting interventions. Masters and Hooker (2011) provide an overview of the R/S and health literature. Religious service attendance stands out as a variable that consistently predicts mortality. Frequent attendees are at reduced risk of mortality compared to those who never attended services, even when standard controls are included, and the relationship is especially strong for African Americans. Importantly, this relationship appears to transcend culture, with research demonstrating that R/S serves as a protective factor against mortality in a variety of countries, including non-Western societies such as China and Israel. In this light, it is very important to note that service attendance does not measure spiritual beliefs per se. Nevertheless, the literature also indicates that spiritual beliefs are related to improved outcomes for seriously ill individuals. In cancer patients, for example, spiritual beliefs predict positive psychological adjustment and higher rates of perceived

cancer-related growth. Studies also indicate spiritual beliefs and practices may be helpful for cardiac patients, putting them at reduced risk of morbidity, complications, and depression following surgery. However, the relationship between R/S and health is complex, with some studies indicating a detrimental relationship between certain R/S variables and health outcomes. For instance, cardiac patients who viewed God as responsible for their *illnesses* had more difficult recoveries, whereas individuals who attributed their recoveries to God enjoyed better outcomes. Psychologists have theorized several pathways to explain the relationship between R/S and health variables, including the idea that R/S is associated with increased social support, positive coping skills, and the adoption of a healthy lifestyle (e.g., abstinence from smoking and alcohol use, regular exercise, and utilization of preventative health care).

Mental Health

The vast majority of research also indicates a positive association between mental health and spiritual beliefs (Koenig 2010). Individuals scoring higher on measures of spirituality are consistently less likely to have depressive symptoms or disorders, with an effect equivalent in size to that of gender and depression. Similarly, many studies indicate that anxiety is lower for individuals who are more spiritual and that spiritually based interventions result in reduced anxiety. However, the results are mixed – with some research indicating a positive correlation between certain spiritual variables (e.g., spiritual struggle) and anxiety. The relationship between spiritual beliefs and substance use is less ambiguous, with the preponderance of evidence indicating that more spiritual individuals are significantly less likely to engage in substance use, misuse, and abuse.

Despite marked disagreement regarding conceptualization of spiritual constructs, the interaction (whether beneficial or detrimental) of spiritual beliefs with both physical and mental health highlights the significance of recognizing this dimension of human experience in both the science and practice of psychology.

Cross-References

- ▶ Religion/Spirituality
- ▶ Spirituality and Health
- ▶ Spirituality, Measurement of

References and Readings

Hill, P. C., & Pargament, K. I. (2003). Advances in the conceptualization of religiousness and spirituality: Implications for physical and mental health research. *The American Psychologist, 58*, 64–74. https://doi.org/10.1037/0003-066X.58.1.64.

Kapuscinski, A. N., & Masters, K. S. (2010). The current status of measures of spirituality: A critical review of scale development. *Psychology of Religion and Spirituality, 2*, 191–205. https://doi.org/10.1037/a0020498.

King, M., Jones, L., Barnes, K., Low, J., Walker, C., Wilkinson, S., et al. (2006). Measuring spiritual belief: Development and standardization of a beliefs and values scale. *Psychological Medicine, 36*, 417–425. https://doi.org/10.1017/S003329170500629X.

King, M., Speck, P., & Thomas, A. (2001). The royal free interview for spiritual and religious beliefs: Development and validation of a self-report version. *Psychological Medicine, 31*, 1015–1023. https://doi.org/10.1017/S0033291701004160.

Koenig, H. G. (2010). Spirituality and mental health. *International Journal of Applied Psychoanalytic Studies, 7*, 116–122. https://doi.org/10.1002/aps.239.

Koenig, H. G., McCullough, M. E., & Larson, D. B. (Eds.). (2001). *Handbook of religion and health*. New York: Oxford University Press.

Masters, K. S., & Hooker, S. A. (2011). Impact of religion and spirituality on health. In J. Aten, K. O'Grady, & E. Worthington (Eds.), *The psychology of religion and spirituality for clinicians: Using research in your practice* (pp. 357–386). New York: Routledge.

Zinnbauer, B. J., Pargament, K. I., & Scott, A. B. (1999). The emerging meanings of religiousness and spirituality: Problems as prospects. *Journal of Personality, 67*(6), 889–919. https://doi.org/10.1111/1467-6494.00077.

Spiritual Coping

▶ Religious Coping

Spiritual Struggle

▶ Religious Coping

Spirituality

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Synonyms

Religion; Religiosity; Religiousness

Definition

Spirituality is a very unclear concept that has no concrete definition. By its very nature, the concept of spirituality is deeply rooted in religion, yet in contemporary spirituality, there is an incremental divide emerging between religion and spirituality. Therefore, in present-day society, the formation of a dichotomy with spirituality representing the personal, subjective, inner-directed, unsystematic, liberating expression and religion signifying a formal, authoritarian, institutionalized inhibiting expression is being witnessed. Spirituality has also been defined as a subjective and fluid approach to experiences which leads one to search for enlightenment, whereby behaviors are practiced in accordance with these sacred beliefs. Similarly, one can also consider spirituality to be something personal, which is defined by individuals themselves and is mostly likely devoid of the rules and regulations associated with religion.

Description

Pathways Linking Spirituality to Health

Rendering a congruent spiritual outlook on life has been associated with an enhanced quality of life, better mental and physical health, and improved recovery from various illnesses. However, the precise mechanisms behind these relationships remain unclear. This can be partly attributed to several reasons: first, the lack of a clear conceptual definition of what spirituality is; second, researchers using both concepts of religion and spirituality interchangeably; third, measuring both concepts with similar assessments (e.g., denomination and frequency of religious observance) which clearly are not adequate to capture a measure of one's spiritual beliefs; and finally, social acceptance that being spiritual entails performing soothing activities such as meditation, yoga or praying, etc., which may be tied to social interactions. Thus, it is necessary to distinguish social factors involved in these practices from spirituality itself. All of the above make it difficult to draw any firm conclusions; thus, caution must be warranted when interpreting the data from such studies. Nonetheless, a number of pathways linking spirituality and health have been proposed, from direct physiological mechanisms (e.g., immune functioning) to more indirect stress buffering (e.g., coping strategies) and lifestyle choices (e.g., dietary and exercise behaviors coupled to one's spiritual beliefs) (Miller and Thorensen 2003). For example, a positive correlation between spirituality and T-cell percentages in HIV-positive women has been reported, suggestive of a more direct physiological route, while other studies support a more indirect pathway; spiritual beliefs have been found to be linked to lower stress, better nutrition, and more exercise, all of which are associated with positive health indices (Koenig et al. 2001). However, what is more interesting is the strong evidence coming from intervention research; a number of studies have been conducted along this line, but one study found that those taught spiritual meditation had greater decreases in anxiety, negative affect, and frequency of migraine headaches compared to those who practiced internally focused secular meditation, externally focused secular meditation, or muscle relaxation (Wachholtz and Pargament 2009). Taken together, these studies indicate that spirituality influences health and that interventions targeting this concept can bring health benefits.

However, despite these positive benefits, a word of caution is warranted when investigating these relationships as there are also negative health consequences associated with spirituality. For example, a spiritual struggle denotes the

anxiety and pressure about spiritual concerns inside oneself, with others, and the godly. Indeed, this spiritual struggle is associated with poor mental and physical health among suffers in some traditional faith practices (Rosmarin et al. 2009); hence, it is sometimes difficult to determine whether spirituality is a resource or a liability and adds to the complexity that already exists in this spirituality-health relationship, which in some instances may not be linear (Gallagher et al. 2015). Further, there have been attempts to adopt the concept of spirituality into an overall definition of health, and some now argue that spiritual health and growth are equally important for quality of life (Sawatzky et al. 2005); thus, spirituality can now be viewed and measured as an endpoint itself.

Measuring Spirituality

Measuring spirituality is a very difficult task due to lack of agreed-upon definition and its close-knit ties with religion. However, there have been some admirable attempts to capture the concept using self-report methods, and some of the measures include the Daily Spiritual Experience Scale (Underwood and Teresi 2002), the Intrinsic Spirituality Scale (Hodge 2003) and the Spiritual Well-Being Scale (Paloutzian and Ellison 1991), and the Theistic Spiritual Outcome Survey (Richards et al. 2005), while the Spiritual Transcendence Scale (Piedmont 1999), the Beliefs and Values Scale (King et al. 2006), and Spiritual Connection Scale (Wheeler and Hyland 2008) offer a conceptualization of spirituality which is nonreligious; these may be useful when one is dealing with individuals who are more inclined to adopt a contemporary view of spirituality.

Cross-References

- ▶ Religion
- ▶ Religion/Spirituality
- ▶ Religiousness/Religiosity
- ▶ Spiritual Beliefs

References and Further Readings

Gallagher, S., Phillips, A. C., Lee, H. A. N., & Carroll, D. (2015). The association between spirituality and depression in parents caring for children with developmental disabilities: Social support and/or last resort. *Journal of Religion and Health, 54*, 358–370.

Hill, P. C., & Pargament, K. I. (2003). Advances in the conceptualization and measurement of religion and spirituality: Implications for physical and mental health research. *American Psychologist, 58*, 64–74.

Hodge, D. R. (2003). The intrinsic spirituality scale. *Journal of Social Service Research, 30*(1), 41–61.

King, M., Jones, L., Barnes, K., Low, J., Walker, C., Wilkinson, S., et al. (2006). Measuring spiritual belief: Development and standardization of a beliefs and values scale. *Psychological Medicine, 36*(3), 417–425.

Koenig, H. G. (2009). Research on religion, spirituality, and mental health: A review. *Canadian Journal of Psychiatry, 54*, 283–291.

Koenig, H. G., McCullough, M. E., & Larson, D. B. (Eds.). (2001). *Handbook of religion and health*. New York: Oxford University Press.

Miller, W. R., & Thorensen, C. E. (2003). Spirituality, religion, and health: An emerging research field. *American Psychologist, 58*, 24–35.

Paloutzian, R. E., & Ellison, C. W. (1991). *Manual for the spiritual well-being scale*. Nayack: Life Advance.

Peterman, A. H., Fitchett, G., Brady, M. J., Hernandez, L., & Cella, D. (2002). Measuring spiritual well-being in people with cancer: The functional assessment of chronic illness therapy-spiritual well-being scale (FACIT-Sp). *Annals of Behavioral Medicine, 24*, 49–58.

Piedmont, R. L. (1999). Does spirituality represent the sixth factor of personality? Spiritual transcendence and the five-factor model. *Journal of Personality, 67*, 985–1013.

Richards, P. S., Smith, T., Schowalter, M., Richard, M., Berrett, M. E., & Hardman, R. K. (2005). Development and validation of the theistic spiritual outcome survey. *Psychotherapy Research, 15*, 457–469.

Rosmarin, D. H., Pargament, K. I., & Flannelly, K. J. (2009). Do spiritual struggles predict poorer physical/mental health among Jews? *The International Journal for the Psychology of Religion, 19*, 244–258.

Sawatzky, R., Ratner, P. A., & Chiu, L. (2005). A meta-analysis of the relationship between spirituality and quality of life. *Social Indicators Research, 72*, 153–188.

Underwood, L. G., & Teresi, J. A. (2002). The daily spiritual experience scale: Development, theoretical description, reliability, exploratory factor analysis and preliminary construct validity using health-related data. *Annals of Behavioral Medicine, 24*, 22–33.

Wachholtz, M. A. B., & Pargament, K. I. (2009). Migraines and meditation: Does spirituality matter? *Journal of Behavioral Medicine, 3*, 351–366.
Wheeler, P., & Hyland, M. E. (2008). The development of a scale to measure the experience of spiritual connection and the correlation between this experience and values. *Spirituality Health, 9*, 193–217.

Spirituality and Health

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Synonyms

Faith and health; Religiousness and health

Definition

Spirituality is an elusive term for which definitional consensus has yet to be reached. Many definitions include concepts concerning that which is beyond the material world and may include features of life that are not commonly perceptible by the physical senses. This is sometimes said to include a search for the sacred or belief in the transcendent. Common themes in definitions of spirituality include connectedness or relationship, subjectivity, personal experience, behaviors reflecting sacred or secular beliefs, and belief in something transcendent. This entry regards spirituality as related to health, an area of significantly increased research activity over the last 15–20 years.

Description

Over the last 15–20 years, scholars have become significantly more interested in determining if there is a relationship between spirituality and health, what the strength of this relationship might be, and if the relationship varies depending on the specific dimensions of both spirituality and health under consideration. Clearly both spirituality and health are multidimensional constructs, and thus, any general statement on their relationship will be an oversimplification.

The first major problem that investigators have in this area is, indeed, defining both terms. In this entry, the definitional focus is on spirituality. An important first question pertains to the similarities and differences between religion and spirituality. Most scholars currently agree that they are related, but not synonymous constructs. This understanding diverges from the historic conceptualization that viewed spirituality and religion as indivisible entities but coincides temporally with the increasing secularization of Western culture (Zinnbauer et al. 1999). Compared to spirituality, religion is often viewed as including more organized social or group practices with well-defined rituals, doctrinal creeds, and statements of faith. Spirituality, on the other hand, is thought to be more subjective and personal, lacking in the organizational elements that characterize religion. Shahabi et al. (2002) reported that 10% of individuals in a US national stratified sample classified themselves as spiritual, but not religious. Nevertheless, most highly religious individuals note that their spirituality is pursued within the context of their religion. Further, a common theme for definitions of both religion and spirituality is reference to a higher power, and 70% of spirituality definitions referred to traditional concepts of God, Christ, and the church as constituting what is sacred (Zinnbauer et al. 1997). Based on a review of the nursing literature, Emblen (1992) defined spirituality as "a personal life principle which animates a transcendent quality of relationship with God" (p. 45). More recently, Saucier and Skrzypinska (2006) demonstrated that subjective spirituality and tradition-oriented religiousness are empirically independent and correlate quite distinctly with personality dimensions. It has also been observed that neither spirituality nor religiousness can be

simply reduced to a commonly measured personality variable (Piedmont 1999; Saroglou and Muñoz-García 2008; Saucier and Skrzypinska 2006).

Given the definitional problems with spirituality, it is not surprising that there are measurement concerns as well. Recently, Kapuscinski and Masters (2010) reviewed work in this area. They not only observed the lack of definitional consensus and related measurement confusion but also suggested that, given the dearth of lexical studies of spirituality, it is quite likely that the spiritual construct as defined by researchers differs from popular understanding and use of the term.

Clearly, these basic problems limit the extent that strong statements can be made regarding spirituality and health. At this point, most of the research has not focused on spirituality per se, as differentiated from religiousness, but rather has used measures such as religious service attendance as proxies for spirituality or has confounded religion and spirituality often using the R/S naming convention to represent both simultaneously and therefore demonstrate that no attempt at conceptual separation was made. There is significant research suggesting that some practices that are often associated with spirituality or considered behavioral indicators of spirituality can be effective. For example, meditation has a strong history of beneficial findings in the area of stress management and has been an important component in some major lifestyle interventions such as the Ornish Lifestyle Heart Trial (Ornish et al. 1983, 1990). Nevertheless, intervention studies that include a specifically spiritual intervention are almost nonexistent, and observational research, even if longitudinal, is severely limited in considering cause and effect relations. For example, it is clear that people tend to pray more when they are ill (negative relationship with health), but it is quite likely that it is the illness that is "causing" the increase in prayer rather than prayer having a negative impact on health. Investigations on frequency of prayer and health are, at this point, nonconclusive and largely characterized by cross-sectional research designs of self-report data. A few studies have investigated the content of prayer and found that, for example, self-esteem is higher among older adults when they pray, believing that only God knows when and how to best answer prayer (Krause 2004). Krause (2003) also found that prayer for material things did not alleviate the burden of financial strain on physical health. But these are very preliminary findings, and much work in this area remains.

Finally, there are many behavioral and cognitive practices that could be considered either spiritual or not spiritual depending on how they are conceptualized and contextualized. These include concepts such as gratitude, forgiveness, relaxation, compassion, hope, optimism, faith, and connectedness among others. The extent that these may be considered aspects of spirituality depends on many factors notably the extent to which they are conceptualized as aspects of relating to the transcendent or sacred.

Cross-References

- ▶ Spiritual Beliefs
- ▶ Spirituality, Measurement of

References and Readings

Emblen, J. D. (1992). Religion and spirituality defined according to current use in nursing literature. *Journal of Professional Nursing, 8*, 41–47. https://doi.org/10.1016/8755-7223(92)90116-G.

Kapuscinski, A. N., & Masters, K. S. (2010). The current status of measures of spirituality: A critical review of scale development. *Psychology of Religion and Spirituality, 2*, 191–205.

Krause, N. (2003). Praying for others, financial strain, and physical health status in late life. *Journal for the Scientific Study of Religion, 42*, 377–391.

Krause, N. (2004). Assessing the relationships among prayer expectancies, race, and self-esteem in late life. *Journal for the Scientific Study of Religion, 42*, 395–408.

Ornish, D., Brown, S. E., Scherwitz, L. W., Billings, J. H., Armstrong, W. T., Ports, T. A., et al. (1990). Can lifestyle changes reverse coronary heart disease? The lifestyle heart trial. *Lancet, 336*, 129–133.

Ornish, D., Scherwitz, L. W., Doody, R. S., Kesten, D., McLanahan, S. M., Brown, S. E., et al. (1983). Effects of stress management training and dietary changes in treating ischemic heart disease. *JAMA, 249*, 54–59.

Piedmont, R. L. (1999). Does spirituality represent the sixth factor of personality? Spiritual transcendence and the five-factor model. *Journal of Personality, 67*, 983–1013. https://doi.org/10.1111/1467-6494.0080.

Saroglou, V., & Muñoz-García, A. (2008). Individual differences in religion and spirituality: An issue of personality traits and/or values. *Journal for the Scientific Study of Religion, 47*, 83–101.

Saucier, G., & Skrzypinska, K. (2006). Spiritual but not religious? Evidence for two independent dispositions. *Journal of Personality, 74*, 1257–1292. https://doi.org/10.1111/j.1467-6494.2006.00409.x.

Shahabi, L., Powell, L. H., Musick, M. A., Pargament, K. I., Thoresen, C. E., Williams, D., et al. (2002). Correlates of self-perception of spirituality in American adults. *Annals of Behavioral Medicine, 24*, 59–68. https://doi.org/10.1207/S15324796ABM2401_07.

Zinnbauer, B. J., Pargament, K. I., Cole, B., Rye, M. S., Butter, E. M., Belavich, T. G., et al. (1997). Religion and spirituality: Unfuzzying the fuzzy. *Journal for the Scientific Study of Religion, 36*, 549–564. https://doi.org/10.2307/1387689.

Zinnbauer, B. J., Pargament, K. I., & Scott, A. B. (1999). The emerging meanings of religiousness and spirituality: Problems as prospects. *Journal of Personality, 67*, 889–919. https://doi.org/10.1111/14676494.

Spirituality, Measurement of

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Definition

Quantitative assessment of spirituality, typically in the form of self-report questionnaires.

Description

Measuring spirituality poses a serious challenge to research in the psychology of religion and spirituality. The principal source of difficulty is researchers' inability to reach a consensus on how to define spirituality, especially regarding its relationship to the concept of religiousness (Kapuscinski and Masters 2010). As language in the United States has shifted over the past few decades, such that religion and spirituality are no longer considered to be synonymous, researchers have created instruments intended to measure spirituality as a concept distinct from religiousness. However, the definitions used to generate measures of spirituality are highly diverse, resulting in some instruments that appear to share little overlap in content (Kapuscinski and Masters 2010). For instance, some measures define spirituality using language stemming from traditional religious institutions (e.g., "God"), whereas others include no reference to the transcendent or sacred in their conceptualizations. An additional concern is that some themes in the way researchers tend to understand spirituality stand in contrast to how the term is used in common language by the general public (Hill and Pargament 2003; Zinnbauer et al. 1999). Specifically, researchers tend to sharply divide spirituality from religion, such that religion is considered to be comprised of external behaviors (e.g., religious service attendance) and is associated with formal institutions, whereas spirituality is comprised of personal, inner experience that is separate from organized religion. Further, researchers are inclined to view spirituality as a health-promoting quality associated with positive psychological and societal benefits, and religiousness, in contrast, is perceived to be restrictive and unhealthy. The empirical literature, however, indicates that nonresearchers regard the concepts as overlapping considerably and view both religiousness and spirituality as positive qualities. Moreover, researchers' connotations are not consistent with research linking both religion and spirituality to positive mental and physical health outcomes (Masters and Hooker 2011).

Further, theoretical concerns arise regarding the feasibility of quantifying and operationally defining a concept that seems to be, by its nature, highly experiential and personalized. The literature indicates that like researchers, individuals differ in how they understand spirituality (Zinnbauer et al. 1999). Interestingly, the majority of researchers do not consult participants regarding conceptualization of spirituality when developing new measures (Kapuscinski and Masters 2010). Miller and Thoresen (2003) comment that

from the believer's perspective, scientists can at best explore mere reflections of spirituality, which approximate but never fully capture its essence.

The disagreement regarding how to conceptualize spirituality is implied by the sheer number of available spirituality instruments. Reviews of measures (Hill and Hood 1999; MacDonald et al. 1995; MacDonald et al. 1999) indicate that over 100 measures exist that are designed to measure a variety of spiritual constructs, including spirituality, intrinsic spirituality, spiritual experiences, spiritual meaning, spiritual development, spiritual transcendence, spiritual transformation, and spiritual well-being. Most measures appear to address interest in or search for the sacred and focus on assessing cognitive (e.g., meaning, beliefs, values) or emotional (e.g., peace, hope, connection) experiences associated with the sacred. Several high-quality measures should be considered as appropriate for use in health psychology research. The FACIT Spiritual Well-Being Scale (Peterman et al. 2002) was designed specifically to assess aspects of spirituality relevant to quality of life for chronically ill individuals. The Daily Spiritual Experiences Scale (Underwood and Teresi 2002) includes language that is relevant to individuals from various faith traditions and has demonstrated a relationship to important health variables such as alcohol consumption and depression. Additionally, the Spiritual Transcendence Scale (Piedmont 1999), which does not include language associated with institutional religion, is noteworthy for its high-quality scale development and validation practices and includes an observer report form. When selecting a measure for use, researchers should carefully consider whether or not the content of scale items is consistent with the conceptualization of spirituality relevant to their study and population of interest.

Cross-References

- ▶ Spiritual Beliefs
- ▶ Spirituality
- ▶ Spirituality and Health

References and Readings

Hill, P. C., & Hood, R. W. (Eds.). (1999). *Measures of religiosity*. Birmingham: Religious Education.

Hill, P. C., & Pargament, K. I. (2003). Advances in the conceptualization of religiousness and spirituality: Implications for physical and mental health research. *American Psychologist, 58*, 64–74. https://doi.org/10.1037/0003-066X.58.1.64.

Kapuscinski, A. N., & Masters, K. S. (2010). The current status of measures of spirituality: A critical review of scale development. *Psychology of Religion and Spirituality, 2*, 191–205. https://doi.org/10.1037/a0020498.

MacDonald, D. A., LeClair, L., Holland, C. J., Alter, A., & Friedman, H. L. (1995). A survey of measures of transpersonal constructs. *Journal of Transpersonal Psychology, 27*, 171–235.

MacDonald, D. A., Friedman, H. L., & Kuentzel, J. G. (1999). A survey of measures of spiritual and transpersonal constructs: Part one – research update. *Journal of Transpersonal Psychology, 31*, 137–154.

Masters, K. S., & Hooker, S. A. (2011). Impact of religion and spirituality on health. In J. Aten, K. O'Grady, & E. Worthington (Eds.), *The psychology of religion and spirituality for clinicians: Using research in your practice* (pp. 357–386). New York: Routledge.

Miller, W. R., & Thoresen, C. E. (2003). Spirituality, religion and health: An emerging research field. *American Psychologist, 58*, 24–35. https://doi.org/10.1037/0003-066X.58.1.24.

Peterman, A. H., Fitchett, G., Brady, M. J., Hernandez, L., & Cella, D. (2002). Measuring spiritual well-being in people with cancer: The functional assessment of chronic illness therapy – spiritual well-being scale (FACIT-Sp). *Annals of Behavioral Medicine, 24*, 49–58. https://doi.org/10.1207/S15324796ABM2401.

Piedmont, R. L. (1999). Does spirituality represent the sixth factor of personality? Spiritual transcendence and the five-factor model. *Journal of Personality, 67*, 986–1013. https://doi.org/10.1111/1467-6494.00080.

Underwood, L. G., & Teresi, J. A. (2002). The daily spiritual experience scale: Development, theoretical description, reliability, exploratory factor analysis and preliminary construct validity using health-related data. *Annals of Behavioral Medicine, 24*, 22–33. https://doi.org/10.1207/S15324796ABM2401_04.

Zinnbauer, B. J., Pargament, K. I., & Scott, A. B. (1999). The emerging meanings of religiousness and spirituality: Problems as prospects. *Journal of Personality, 67*(6), 889–919. https://doi.org/10.1111/1467-6494.00077.

Squamous Cell Carcinoma of the Cervix (SCCC)

- ▶ Cancer, Cervical

Stages of Change Model

► Transtheoretical Model of Behavior Change

Stages-of-Change Model

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Definition

The Stages-of-Change Model was developed by James Prochaska and Carlo DiClemente as a framework to describe the five phases through which one progresses during health-related behavior change (Prochaska und DiClemente 1983). It is part of their broader Transtheoretical Model, which not only assesses an individual's readiness to act to eliminate a problem behavior but also includes strategies and processes of change to guide the individual through the stages. The Stages-of-Change Model originated in research related to psychotherapy and the cessation of addictive behaviors, such as smoking, alcohol and substance abuse, and issues related to weight management (Buxton et al. 1996). Although Prochaska and DiClemente initially hypothesized that individuals progress linearly through a series of discrete stages of change, researchers now believe that a cyclical or "spiral" pattern more accurately represents how most people change unhealthy behavior over time. Since its development, the Stages-of-Change Model has been related to a variety of problem behaviors, associated with treatment outcomes, and integrated in stage-based interventions. Although most scientists and clinicians agree that the model has heuristic value, it has been criticized by some researchers.

Description

History of the Model

Stage theories have been integral to the field of psychology since its inception and include Freud's and Erikson's psychosexual stages, Kohlberg's stages of moral development, Piaget's stages of cognitive development, and Maslow's hierarchy of needs (Dolan 2005). DiClemente and Prochaska's Stages-of-Change Model uses language similar to Horn. Specifically, Horn hypothesized four stages of change associated with health-related behavior: (1) contemplating change, (2) deciding to change, (3) short-term change, and (4) long-term change (Horn 1976). DiClemente and Prochaska initially identified four stages of changes associated with smoking cessation and maintenance: (1) thinking about change (contemplation), (2) becoming determined to change (decision making), (3) actively modifying behavior and/or environment (action), and (4) maintaining new behaviors (maintenance). Precontemplation was later identified as a separate stage preceding contemplation.

Description of Stages

Precontemplation. The individual in the precontemplation stage has no intention to change his or her behavior in the foreseeable future (Prochaska und Norcross 2001). Although individuals are unaware of their problems, their families, friends, neighbors, and employees are often very aware of these problems. Individuals presenting for treatment in the precontemplation stage generally do so because of pressure from others.

Contemplation. During the contemplation stage, individuals are aware that a problem exists and seriously consider overcoming it. However, they have not yet made a commitment to do so. According to Prochaska and Norcross, individuals often remain stuck in this stage for long periods.

Preparation. The preparation stage combines intention and behavioral criteria. Individuals in the preparation stage intend to enact change in the next month and have unsuccessfully attempted

to do so in the past year. These individuals report small behavioral changes, but they have not yet reached a criterion for effective action, such as abstinence from smoking or sufficient weight loss. These individuals do intend to take action in the very near future.

Action. Individuals in the action stage modify their behavior, experiences, and environment in order to overcome their problems. This stage involves the most overt behavioral changes and requires considerable commitment of time and energy. Modifications of the problem behaviors made in this stage are most visible to others and tend to elicit others' recognition. Individuals in this stage must have successfully altered their problem behavior for a period of 1 day to 6 months.

Maintenance. Individuals in the maintenance stage concentrate on preventing relapse and consolidating the gains attended during the previous stage. These individuals must have remained free of their problem behavior and consistently engaged in a new incompatible behavior for more than 6 months.

Termination. Individuals who reach this stage have completed the change process and no longer have to work to prevent relapse. This stage involves total confidence or self-efficacy across all high-risk situations and no temptation to relapse.

Assessing Stages of Change

Multiple ways for measuring stage of change have been proposed and devised, and researchers/clinicians usually assign people to stages on the basis of their responses to questions concerning their prior behavior and current behavioral intentions (Weinstein, Rothman, & Sutton, 1998). A Stages-of-Change Questionnaire has been developed as a brief and reliable instrument for measuring stages of change in psychotherapy and has been adopted to evaluate stages of change for specific problem behaviors (McConnaughy, Prochaska, & Velicer, 1983). This continuous measure includes questions such as "As far as I'm concerned, I don't have problems that need changing" (precontemplation), "I have a problem and I really think I should work on it" (contemplation), "I am working really hard to change" (action), and "I may need a boost right now to help me maintain the changes I've already made" (maintenance). Given that attributes that define the stages of change are mainly internal to the individual (e.g., beliefs, plans, attributions), measurement is often imperfect (Weinstein, Rothman, & Sutton, 1998).

Stages of Change and Specific Health Behaviors

The Stages-of-Change Model has been used to understand a variety of problem behaviors including smoking cessation, cessation of cocaine use, weight control, high-fat food consumption, adolescent delinquent behaviors, risky sexual behaviors, sunscreen use, radon gas exposure, exercise acquisition, mammography screening, and physicians' preventive practices with smokers (Prochaska et al. 1994).

Stage-Based Treatments

The Stages-of-Change Model has been use to aid in treatment planning and to develop stage-based treatments. Prochaska (1991) has argued that a person's stage of change provides proscriptive and prescriptive information about appropriate treatments (Prochaska 1991). For example, those who are in the preparation or action stages presumably benefit from action-oriented therapies, whereas those in the precontemplation or contemplation stages likely may benefit more from insight-oriented, consciousness raising interventions.

Several interventions based on stage-based models of change have been developed to modify risk behaviors. These interventions are tailored to take into account the current stage an individual has reached in the change process in contrast to "one size fits all" interventions. A systematic review of these interventions identified 37 RCTs of such interventions aimed at smoking cessation, promotion of physical activity, dietary change, multiple lifestyle changes, mammography screening, and treatment adherence (Riemsma et al. 2002). The authors of this review concluded that there is little evidence to suggest that stage-based interventions are more effective compared to

non-stage-based interventions, no intervention, or usual care. Nonetheless, they recommend additional studies of tailored interventions which involve frequent reassessment of patients' readiness to change. Reviews of stage-based lifestyle interventions in primary care (Van Sluijs, Van Poppel, & Van Mechelen, 2004) and stage-based interventions for smoking cessation (Riemsma et al. 2003) have likewise resulted in limited scientific evidence in support of these interventions.

Revised Stages-of-Change Model

Freeman und Dolan (2001) offered a revised Stages-of-Change Model with five additional changes to more precisely determine a psychotherapy patient's position on the continuum of change (Freeman & Dolan). This revised model has been recommended as a more dynamic and flexible one that provides clinicians with a more experience-centered focus from which to make treatment decisions.

Non-contemplation is the stage during which an individual is not considering or even thinking about changing. These individuals do not actively avoid, resist, or oppose change; they are rather unaware of their need to change or of the effect their behavior has on others. Anti-contemplation involves the process of becoming reactive and violently opposed to the notion of needing change, a response often seen in individuals who are legally mandated to attend treatment or who come to treatment at the urging of their family, friends, or significant others. Freeman and Dolan's precontemplation and contemplation stages are identical to those of Prochaska and DiClemente. Their action planning stage occurs when the clinician and patient have collaboratively developed a treatment focus and treatment plan. Patients in this stage are actively willing to plan change, and next progress to the action stage of Prochaska and DiClemente's model. Prelapse, lapse, and relapse stages may then occur prior to the maintenance stage. During prelapse, an individual experiences overwhelming thoughts, desires, and cravings to engage in the problem behavior. During lapse, the skills needed to maintain the action stage decrease or are ignored. During relapse, the individual returns to the problem behavior. As in Prochaska and DiClemente's model, the maintenance stage occurs when the individual actively works toward maintaining the cessation of the problem behavior.

Evaluation of the Stages-of-Change Model

The Stages-of-Change Model is not without criticism. Those who have criticized the model argue that its inherent concept of discrete stages involves arbitrary distinctions; it falsely assumes that individuals make coherent and stable plans, and it neglects the role of reward, punishment, and associative learning that contribute to the maintenance of problem behaviors (West 2005). Others have found that minimal supportive evidence for the Stages-of-Change Model exists (Whitelaw, Baldwin, Bunton, & Flynn, 2000) and questioned the model's internal validity (Ahijevych und Wewers 1992; Bandura 1997; Farkas et al. 1996), external validity (Clarke und Eves 1997), and ethical difficulties associated with interventions derived from the Stages-of-Change Model (Piper und Brown 1998).

Notwithstanding the criticisms and absence of evidence discussed above, the Stages-of-Change Model provides a pragmatic framework for practitioners and clinical researchers, and it is intuitively appealing to many in the fields of clinical psychology, behavioral medicine, public health, and other fields. Future research promises to offer improved measurement of stages, qualitative case studies of practitioner utilization, and process-based implementation evaluation of the model in various settings (Whitelaw, Baldwin, Bunton, & Flynn, 2000).

Cross-References

- ▶ Health Beliefs/Health Belief Model
- ▶ Transtheoretical Model of Behavior Change

References and Readings

Ahijevych, K., & Wewers, M. (1992). Processes of change across five stages of smoking cessation. *Addictive Behaviors, 17*(1), 17–25.

Bandura, A. (1997). Editorial: The anatomy of stages of change. *American Journal of Health Promotion, 12*, 8–10.
Buxton, K., Wyse, J., & Mercer, T. (1996). How applicable is the stages of change model to exercise behaviour? A review. *Health Education Journal, 55*(2), 239–256.
Clarke, P., & Eves, F. (1997). Applying the transtheoretical model to the study of exercise on prescription. *Journal of Health Psychology, 2*(2), 195–207.
Dolan, M. (2005). Stages of change. In A. Freeman et al. (Eds.), *Encyclopedia of cognitive behavior therapy* (pp. 387–390). New York: Springer.
Farkas, A., Pierce, J., Zhu, S., Rosbrook, B., Gilpin, E., Berry, C., et al. (1996). Addiction versus stages of change models in predicting smoking cessation. *Addiction, 91*(9), 1271–1280.
Freeman, A., & Dolan, M. (2001). Revisiting Prochaska and DiClemente's stages of change theory: An expansion and specification to aid in treatment planning and outcome evaluation. *Cognitive and Behavioral Practice, 8*(3), 224–234.
Horn, D. (1976). A model for the study of personal choice health behavior. *International Journal of Health Education, 19*(1), 89–98.
McConnaughy, E., Prochaska, J., & Velicer, W. (1983). Stages of change in psychotherapy: Measurement and sample profiles. *Psychotherapy: Theory, Research & Practice, 20*(3), 368–375.
Piper, S., & Brown, P. (1998). Psychology as a theoretical foundation for health education in nursing: empowerment or social control? *Nurse Education Today, 18*(8), 637–641.
Prochaska, J. (1991). Prescribing to the stage and level of phobic patients. *Psychotherapy: Theory, Research, Practice, Training, 28*(3), 463.
Prochaska, J., & DiClemente, C. (1983). Stages and processes of self-change of smoking: toward an integrative model of change. *Journal of Consulting and Clinical Psychology, 51*(3), 390–395.
Prochaska, J., & Norcross, J. (2001). Stages of change. *Psychotherapy: Theory, Research, Practice, Training, 38*(4), 443.
Prochaska, J., Velicer, W., Rossi, J., Goldstein, M., Marcus, B., Rakowski, W., et al. (1994). Stages of change and decisional balance for 12 problem behaviors. *Health Psychology, 13*, 39–39.
Riemsma, R., Pattenden, J., Bridle, C., Sowden, A., Mather, L., Watt, I., et al. (2002). A systematic review of the effectiveness of interventions based on a stages-of-change approach to promote individual behaviour change. *Health Technology Assessment, 6*(24), 1–231.
Riemsma, R., Pattenden, J., Bridle, C., Sowden, A., Mather, L., Watt, I., et al. (2003). Systematic review of the effectiveness of stage based interventions to promote smoking cessation. *British Medical Journal, 326*(7400), 1175.
Van Sluijs, E., Van Poppel, M., & Van Mechelen, W. (2004). Stage-based lifestyle interventions in primary care: Are they effective? *American Journal of Preventive Medicine, 26*(4), 330–343.
Weinstein, N., Rothman, A., & Sutton, S. (1998). Stage theories of health behavior: Conceptual and methodological issues. *Health Psychology, 17*, 290–299.
West, R. (2005). Time for a change: Putting the transtheoretical (stages of change) model to rest. *Addiction, 100*(8), 1036–1039.
Whitelaw, S., Baldwin, S., Bunton, R., & Flynn, D. (2000). The status of evidence and outcomes in stages of change research. *Health Education Research, 15*(6), 707–718.

Standard Deviation

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Definition

A simple measure of dispersion is the range, the arithmetic difference between the greatest (maximum) and the least (minimum) value in a data set. While this characteristic is easily calculated and useful in initial inspections of data sets, by definition it only uses two of the values in a data set. In a large data set most pieces of numerical information are therefore not used in the calculation of the range, and it is not known whether many data points lie close to the minimum, maximum, or mean, or in any other distribution pattern.

Two more sophisticated measures of dispersion are variance and the standard deviation. These measures are intimately related to each other and take account of all values in a data set. The calculation of variance involves calculating the deviation of each data point from the mean of the data set, squaring these values, and summing them. The process of squaring the deviation is mathematically necessary: If the raw deviations were to be summed they would always sum to zero. However, the squaring process creates the problem that the units of measurement of variance are not the same as the units of measurement of the original data. In the vast majority of cases, the data

points in our studies are not simply numbers, but numerical representations of information measured in certain units. For example, a systolic blood pressure measurement of "125" is actually a measurement of 125 millimeters of mercury (mmHg). Since the calculation of variance involves squaring certain values, the variance of a set of blood pressure data points would actually be measured in squared millimeters of mercury, a nonsensical unit.

Fortunately, this problem can be solved by simply calculating the square root of the variance. The resulting value is called the standard deviation (SD), and the unit of measurement of the SD is the same as the unit of measurement of the original data points. The SD is a very commonly presented descriptor in research studies. It is usually presented in conjunction with the mean in the form "mean ± SD."

Cross-References

► Variance

Standard Normal (Z) Distribution

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Synonyms

Z distribution

Definition

The Standard Normal distribution, also known as the Z distribution, is one particular form of the Normal distribution in which the mean is zero (i.e., 0) and the variance is unity (i.e., 1). This can be written as ($\mu = 0$, $\sigma = 1$).

Before presenting the Z distribution, it is necessary to discuss the Normal distribution in general. Imagine that the heights of a large number of adult males (or females) are measured and the results plotted as a histogram. Height is plotted in inches on the x-axis and the number of people within each height category is plotted on the y-axis. There would be many more people close to the middle of the histogram than close to either end, since more individuals are close to the mean height, and very few are very tall or very short. Given a large sample and decreasingly thin bars in the histogram (that is, the width of the measurement intervals along the *x*-axis becomes infinitely small such that the height data become continuous), a curve can be superimposed on this histogram. One particular version of a density curve is called the Normal distribution. This distribution is of considerable interest since height and many physiological variables conform very closely (but not perfectly) to this distribution. Since the word normal is used in everyday language, and since its meaning in Statistics is different and important, the word is written in this entry with an upper case N when it is used in its statistical sense.

The Normal distribution has several notable properties:

- The highest point of the Normal curve occurs for the mean of the population. The properties of the Normal distribution ensure that this point is also the median value and the mode.
- The shape of the Normal curve (relatively narrow or relatively broad) is influenced by the standard deviation (SD) of the data. The sides of the curve descend more gently as the standard deviation increases and more steeply as it decreases.
- At a distance of approximately ±2 SDs from the mean, the slopes of the downward curves change from a relatively smooth downward slope to a curve that extends out to infinity and thus never quite reaches the *x*-axis. For practical purposes, the curve is often regarded as intercepting the *x*-axis at a distance of ±3 SDs from the mean, but this is an approximation.

Area Under the Normal Curve

The area under the Normal curve is of considerable interest in the discipline of Statistics. That is, it is of considerable interest to define and quantify the area bounded by the Normal curve at the top and the *x*-axis at the bottom. This area will be defined as 1.00, or as 100%. Given this interest, the final bullet point in the previous list raised an issue that appears problematic. That is, it appears that, if the two lower slopes of the Normal curve never quite reach the *x*-axis, the area under the curve is never actually fully defined and can therefore never be calculated precisely. Fortunately, this apparent paradox can be solved mathematically.

The solution is related to the observation that the sum of an infinite series can converge to a finite solution. An example that effectively demonstrates the solution here is the geometric series "1/2 + 1/4 + 1/8 + . . . *ad infinitum*." That is, the series starts with 1/2, and every subsequent term is one half of the previous term. Given this, the terms of the series never vanish to zero. However, the sum of them is precisely 1.00. The proof of this is as follows, where the series is represented as *S*:

$$S = 1/2 + 1/4 + 1/8 + \ldots \textit{ad infinitum} \quad (1)$$

Both sides of this equation are then multiplied by the same value, namely, 2 (multiplying both sides of an equation by a constant means that the sides are still of equal value):

$$2S = 1 + 1/2 + 1/4 + \ldots \textit{ad infinitum} \quad (2)$$

The value *S* is then subtracted from both sides (subtracting a constant from both sides of an equation means that the sides are still of equal value). First, consider the left-hand side (LHS) of Eq. 2:

LHS of Eq. 2: $2S - S$, which equals *S*.

Now consider the right-hand side (RHS) of Eq. 2. Subtracting *S* from this quantity can be represented as:

RHS of Eq. 2: (1 + 1/2 + 1/4 + . . . *ad infinitum*) − *S*, which equals what, exactly?

To determine the unknown value we can use Eq. 1, which shows that *S* is equal to (1/2 + 1/4 + 1/8 + . . .*ad infinitum*). Therefore, the right-hand side of Eq. 2 can be written as follows:

RHS of Eq. 2: (1 + *S*) − *S*, which equals 1.

Equation 2 can therefore be rewritten as:

$$S = 1$$

Therefore, despite the initial paradoxical nature of the statement, it can indeed be shown that the sum of an infinite series can converge to a finite solution.

Returning to the topic of immediate interest, i.e., the area under the Normal curve, the statement that the terms of the geometric series never vanish to zero can be reinterpreted in this context as saying that the curves of the Normal curve never intercept the *x*-axis. Despite this statement, however, an adaptation of the proof just provided shows that the area under the Normal curve is indeed precisely equal to 1.00, or 100%. The visual equivalent of this is that there is indeed a defined area under the Normal curve, bounded by the curve and the *x*-axis, and the value of this area can be represented as 1.00, or 100%. This can be demonstrated formally using integral calculus. It can also be thought of as analogous to the statement that the probability of all mutually exclusive events must sum to 1.00.

It is of particular interest in Statistics that the means of many large samples taken from a particular population are approximately distributed in this Normal fashion, i.e., they are said to be Normally distributed. This is true even when the population data themselves are not Normally distributed. The mathematical properties of a true Normal distribution allow quantitative statements of the area under the curve between any two points on the *x*-axis. It was just demonstrated that the total area under the Normal curve is 1, or 100%. It is also of interest to know the proportion of the total area under the curve that lies between two points that are equidistant from the mean. These points are typically represented by multiples of the standard deviation (SD). From the properties of the mathematical equation that

governs the shape of the Normal curve, it can be shown that:

- The central 90% of the area under the curve lies between the mean ±1.645 SDs
- The central 95% of the area under the curve lies between the mean ±1.960 SDs
- The central 99% of the area under the curve lies between the mean ±2.576 SDs

The area under the curve is representative of the number of data points falling within that range. That is, the percentage of the area under the curve translates directly into the percentage of data points falling between the two identified points. Of particular relevance for many research studies is that 95% of the area under the curve lies between the mean ±1.960 SDs. The value of 1.960 is often rounded up to 2, leading to the statement in many practical examples in textbooks that 95% of the data points fall within the mean ±2 SDs.

The Z Distribution

The Z distribution is such that the mean and the standard deviation in the immediately preceding statements can be removed, leading to the following statements:

- The central 90% of the area under the curve lies between ±1.645.
- The central 95% of the area under the curve lies between ±1.960.
- The central 99% of the area under the curve lies between ±2.576.

This distribution is used extensively in Statistics, and underpins many more complex statistical procedures (Durham and Turner 2008).

Cross-References

- ▶ Data
- ▶ Hypothesis Testing
- ▶ Median
- ▶ Mode
- ▶ Probability
- ▶ Standard Deviation

References and Further Reading

Durham, T. A., & Turner, J. R. (2008). *Introduction to statistics in pharmaceutical clinical trials*. London: Pharmaceutical Press.

Standardized Tobacco Packaging

- ▶ Plain Tobacco Packaging

State Anxiety

- ▶ Anxiety

Static Exercise

- ▶ Isometric/Isotonic Exercise

Statins

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Synonyms

HMG-CoA reductase inhibitors

Definition

Statins, also known as HMG-CoA reductase inhibitors, are a class of cholesterol lowering agents that are prescribed worldwide to hyperlipidemic patients who are at high risk for cardiovascular disease. Statins currently on the market include pravastatin, simvastatin, fluvastatin, atorvastatin, rosuvastatin, and pitavastatin. Statins exert their effect through inhibition of 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, the rate-limiting enzyme of cholesterol biosynthesis in the liver. In many clinical trials, statins have been beneficial both in the primary and secondary prevention of coronary heart disease. Recent clinical and experimental data suggest that the benefit of statins may extend beyond their lipid lowering effects. Those cholesterol-independent or "pleiotropic" effects of statins involve improving endothelial dysfunction, enhancing the stability of atherosclerotic plaques, decreasing oxidative stress and inflammation, and inhibiting the thrombogenesis.

Cross-References

▶ Hyperlipidemia

Statistical Inference

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Synonyms

Inferential statistics

Definition

Statistical inference is a process of using the precise data and results from a specific group of subjects in a research study to infer the likely responses to the same treatment in the general population of patients who would receive the treatment or intervention if it entered general behavioral medicine practice.

The ultimate purpose of the results from a single behavioral medicine study, such as a randomized clinical trial, is not to tell us precisely what happened in that trial, but to gain insight into likely responses to the behavioral treatment or intervention in patients with the disease or condition of clinical concern who would receive the treatment. Inferential statistics allows us to do this.

The treatment effect calculated from the data in the single study is regarded as the treatment effect point estimate. Confidence intervals are then placed around this point estimate. The range of values between the lower limit and the upper limit of the confidence interval represents a range that covers the true but unknown population treatment effect with a specified degree of confidence.

Cross-References

▶ Hypothesis Testing
▶ Randomized Clinical Trial

Statistical Inquiry

▶ Surveys

Statistics

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Definition

For present purposes, the discipline of Statistics (recognized here by the use of an upper case

"S") can be usefully defined as an integrated discipline that is critically and fundamentally important in all of the following activities associated with clinical research in behavioral medicine:

- Identifying a research question that needs to be answered.
- Deciding upon the design of the study, the methodology that will be employed, and the numerical information (data) that will be collected.
- Presenting the design, methodology, and data to be collected in a study protocol. This study protocol specifies the manner of data collection and addresses all methodological considerations necessary to ensure the collection of optimum quality data for subsequent statistical analysis.
- Identifying the statistical techniques that will be used to describe and analyze the data in a section within the protocol or in an associated statistical analysis plan, which should be written in conjunction with the study protocol.
- Describing and analyzing the data. This includes analyzing the variation in the data to see if there is compelling evidence that the treatment is safe and effective. This process includes evaluation of the statistical significance of the results obtained and, very importantly, their clinical significance.
- Presenting the results of a clinical study to the research and clinical communities in conference talks and posters, and in journal publications.

Cross-References

▶ Clinical Decision-Making
▶ Hypothesis Testing
▶ Statistical Inference

Stem Cells

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Definition

Stem cells are found in all multicellular organisms. They are defined by the ability to renew themselves through mitotic cell division (self-renewal) and to generate all the differentiated cell types of the tissue (multipotency). For this definition, one stem cell divides into one father cell that is identical to the original stem cell and another daughter cell that is differentiated.

The mammalian stem cells are divided into two broad types: embryonic stem cells and adult somatic stem cells. Embryonic stem cells are isolated from the inner cell mass of blastocysts, and adult somatic stem cells that are found in adult tissues. In a developing embryo, stem cells can differentiate into all of the specialized embryonic tissues. The stem cells can become any tissue in the body, excluding a placenta. Only the morula's cells are totipotent, able to become all tissues and a placenta.

In adult organisms, stem cells and progenitor cells act as a repair system for the body, replenishing specialized cells, but also maintain the normal turnover of regenerative organs, such as blood, skin, or intestinal tissues. In addition to the definition described above, adult stem cells are thought to be quiescent within the niche, dividing infrequently to generate one stem cell copy and a rapidly cycling cell (transient amplifying cell). Transient amplifying cells undergo a limited number of cell divisions and differentiate into the functional cells of the tissues.

Recently, the third stem cells, artificially established, induced pluripotent stem cells (iPSCs) are generated. Previously, nuclear transfer of embryo into the adult somatic cells is known to be able to reprogram the somatic cells. These are not adult stem cells but rather reprogrammed

cells (e.g., epithelial cells) given pluripotent capabilities. So reprogramming factors are thought to exist in the embryo or embryonic stem cells. Using genetic reprogramming with transcription factors, pluripotent stem cells equivalent to embryonic stem cells have been derived from human adult tissue. Shinya Yamanaka and his colleagues used the transcription factors Oct3/4, Sox2, c-Myc, and Klf4 in their experiments on cells from human faces. Another groups used a different set of factors, Oct4, Sox2, Nanog, and Lin28, and more limited combination of these factors (Oct3/4, Sox2, Klf4 (OSK), OS, only Oct3/4) can reprogram the adult tissue.

Stem cell therapy has the potential to dramatically change the treatment of human disease. A number of adult stem cell therapies already exist, particularly bone marrow transplantation that is used to treat hematological disease (leukemia, lymphoma, etc.). In the future, stem cell therapy will be broadened to treat a wider variety of diseases including cancer, neurological diseases, several inherited diseases, and so on.

Cross-References

► Genetics
► Hematopoietic Stem Cell Transplantation

Stepped Care Models

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Synonyms

Adaptive interventions; Minimally intensive interventions

Definition

Stepped care models are minimally intensive care models for treating conditions or changing behaviors. Patients are initially typically provided with an easy-to-disseminate, low-cost, minimally intensive intervention. If this does not produce remission of undesirable symptoms or sufficient behavior change, patients are provided with a slightly more intensive and more costly intervention. This continues until patients receive an intervention that produces the desired outcome. Ideally, each patient receives the least resource-intensive yet most effective treatment they need. Stepped care models require repeated assessments to determine if a treatment is effective (and can be stepped down), too burdensome (and should be stepped down), ineffective (and must be stepped up), when another treatment becomes available and more likely to produce better effects (requiring deimplementation of the current treatment and initiation of the new treatment), or inappropriate/unlikely to produce effects (and must be stepped down or discontinued altogether).

Description

Stepped care models involve the systematic process of care for an individual through stages of treatment meant to address the severity of their condition(s). Stepped care models can take many forms at various steps, such as bibliotherapy, computerized services, medications, and in-person treatment (e.g., cognitive behavioral therapy, a medical procedure).

There is a necessity for resource-efficient psychiatric, behavioral, and medical care. Frequently, individuals encounter insufficiently available care options (Jacobi et al. 2017; Carels et al. 2009). Many individuals do not have the financial means, transportation, or time to get the care they need, they do not have access to high-quality care, or the initial care they receive is insufficient to cause meaningful remission of symptoms; rather than pursue adequate care, they discontinue services or experience crisis requiring higher acuity treatment. Such patterns of care utilization are

frequently seen in smoking cessation, psychotherapy for depression or severe mental illness, and weight management.

Stepped care models seek to solve these problems by being a resource-efficient form of care that is accessible, cost-effective, and innovative. Stepped care models have shown clinical and medical benefit for numerous populations and treatment needs. For example, stepped care approaches for weight loss have produced better outcomes or similar weight losses (with lower costs) when compared to standard treatments (Waring et al. 2014). Stepped care approaches have also been found to be effective for the treatment of substance use disorders, including reduced overall drug use (Kidorf et al. 2007).

Stepped care programs may use weekly monitoring and initial assessment data to determine whether a patient needs to step up or down in the stepped care model. Patients may be evaluated for the severity and type of disorder they have, and the patient's preferences are also considered when deciding whether to go into the lowest level of care or higher (Waring et al. 2014; Carels et al. 2009). After treatment initiation, a follow-up assessment of regular symptom monitoring helps the clinician to determine if the original treatment should be transitioned to more intensive treatments, less intensive treatments, or if the treatment should be kept the same, according to how the patient is responding to treatment (Carels et al. 2009).

An example of the stepped model program developed for chronic pain includes pain self-management strategies and cognitive behavioral therapy (CBT). A clinical trial targeted musculoskeletal pain in veterans. In this case, the stepped care model kept participants engaged by including constant contact (biweekly) to check engagement in treatment. Participants were also taught self-management strategies on how to deal with their pain, such as goal setting, positive self-talk, and other relaxation techniques (Bair et al. 2015). Results found that a combination of analgesics, self-management strategies, and CBT effectively reduced the effects of pain on veterans' day-to-day lives (Bair et al. 2015).

The stepped care model has also been effective for weight management. In one program using motivation interviewing (MI), a therapeutic technique used to increase motivation for changes in behavior (Carels et al. 2007), participants met for 45–60 min weekly for MI until they reached their weight loss goal. Participants who were in the stepped care condition lost significantly more weight in comparison to non-stepped care participants. Participants in the stepped care condition also self-reported increased physical activity (Carels et al. 2007). In the context of weight management, the stepped care model may be useful in increasing participation and motivation in weight loss programs.

Stepped care models can be incredibly impactful if adopted. The affordability, accessibility, effectiveness, resource efficiency, and immediacy of care of this model could lead to higher rates of individuals in need receiving treatment without undue stress on the healthcare system.

Cross-References

- ▶ Agile Science
- ▶ Health Care System
- ▶ Multilevel Intervention
- ▶ Patient-Centered Care

References and Further Readings

Bair, M. J., Ang, D., Wu, J., Outcault, D. S., Sargent, C., Kempf, C., Froman, A., Schmid, A., Damush, T., Yu, Z., Davis, W. L., & Kroenke, K. (2015). Evaluation of stepped care for chronic pain (ESCAPE) in veterans of the Iraq and Afghanistan conflicts: A randomized clinical trial. *JAMA Internal Medicine, 175*(5), 682–689. https://doi.org/10.1001/jamainternmed.2015.97.

Carels, R. A., Darby, L., Cacciapaglia, H. M., Konrad, K., Coit, C., Harper, J., Kaplar, E. M., Young, K., Baylen, A. C., & Versland, A. (2007). Using motivational interviewing as a supplement to obesity treatment: A stepped-care approach. *Health Psychology, 26*(3), 369–374. https://doi.org/10.1037/0278-6133.26.3.369.

Carels, R. A., Wott, C. B., Young, K. M., Gumble, A., Darby, L. A., Oehlhof, M. W., Harper, J., & Koball, A. (2009). Successful weight loss with self-help: A stepped-care approach. *Journal of Behavioral Medicine, 32*(6), 503–509. https://doi.org/10.1007/s10865-009-9221-8.

Jacobi, C., Beintner, I., Fittig, E., Trockel, M., Braks, K., Schade-Brittinger, C., & Dempfle, A. (2017). Web-based

aftercare for women with bulimia nervosa following inpatient treatment: Randomized controlled efficacy trial. *Journal of Medical Internet Research, 19*(9). https://doi.org/10.2196/jmir.7668.

Kidorf, M., Neufeld, K., King, V. L., Clark, M., & Brooner, R. K. (2007). A stepped care approach for reducing cannabis use in opioid-dependent outpatients. *Journal of Substance Abuse Treatment, 32*(4), 341–347. https://doi.org/10.1016/j.jsat.2006.09.005.

Waring, M. E., Schneider, K. L., Appelhans, B. M., Busch, A. M., Whited, M. C., Rodrigues, S., Lemon, S. C., & Pagoto, S. L. (2014). Early-treatment weight loss predicts 6-month weight loss in women with obesity and depression: Implications for stepped care. *Journal of Psychosomatic Research, 76*(5), 394–399. https://doi.org/10.1016/j.jpsychores.2014.03.004.

Steptoe, Andrew (1951–)

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Biographical Information

Andrew Steptoe was born in London on April 24, 1951. He is British Heart Foundation Professor of Psychology at University College London, UK, where he is also the Director of the Division of Population Health, a grouping of academic departments including Epidemiology and Public Health, Primary Care and Population Health, Infection and Population Health, and the Medical Research Council Clinical Trials Unit. Steptoe graduated in Natural Sciences from Cambridge in 1972 and completed his Doctorate at Oxford University in 1976. He was appointed lecturer in psychology at St. George's Hospital Medical School in 1977, becoming professor and chair of the Department in 1988. He moved to his present research chair at University College London in 2000, where he is the Director of the Psychobiology Group. Steptoe is also the Director of the English Longitudinal Study of Ageing, a population cohort of older men and women in England.

Major Accomplishments

Steptoe was one of the small group of behavioral medicine specialists, who developed the International Society of Behavioral Medicine (ISBM) in the 1980s. He served as the third President of the ISBM from 1994 to 1996. Additionally, he was the former President of the Society for Psychosomatic Research in the UK. He was the cofounding editor of the *British Journal of Health Psychology* along with Jane Wardle and has served as an associate editor of *Psychophysiology*, the *Annals of Behavioral Medicine*, the *British Journal of Clinical Psychology*, and the *Journal of Psychosomatic Research* and is on the editorial boards of six other journals.

Steptoe is the author of more than 550 journal articles and papers, and author or editor of 17 books, most recently the *Handbook of Behavioral Medicine* (Steptoe 2010) and *Stress and Cardiovascular Disease* (Hjemdahl et al. 2012). His research has addressed many topics in behavioral medicine, including stress and health, socioeconomic status, the determinants of health behavior, health behavior change, cardiovascular disease, respiratory disorders, aging, and positive well-being. His collaborative research with Professor Marmot has focused on understanding the biological processes through which lower socioeconomic status and psychosocial risk factors influence cardiovascular disease risk. This work has involved laboratory studies of the influence of psychosocial factors on cardiovascular, neuroendocrine, and immune function, and naturalistic studies of blood pressure, cortisol, and other biological measures.

Steptoe has advanced our understanding of the psychobiology of health and diseases and the multiple associations between affect and biology in everyday life. He and his team found that people from lower socioeconomic groups

tend to suffer the biological effects of stress for longer than more affluent people. This is a potential pathway linking low socioeconomic status with increased risk for coronary heart disease. Further studies by Steptoe's group showed that in some patients, intense episodes of anger and stress occurred in the hours immediately before the onset of chest pain. In experiment settings, episodes of mental stress, similar to those encountered in everyday life, were found to cause transient (up to 4 h) endothelial dysfunction in healthy young individuals. These studies have been important in demonstrating the role of emotional factors in the triggering of coronary ischemia and acute coronary syndromes. Steptoe is also one of the leading scientists on the protective effects of positive affect in physical health.

Steptoe has received many honors for his work. He is a Fellow of the Society of Behavioral Medicine and the Academy of Behavioral Medicine Research in the USA, the Academy of Medical Sciences, the Academy of Learned Societies for the Social Sciences, and the British Psychological Society.

Cross-References

- ▶ Cardiovascular Disease
- ▶ Psychological Stress
- ▶ Socioeconomic Status (SES)
- ▶ Stress

References and Further Reading

Hjemdahl, P., Rosengren, A., & Steptoe, A. (Eds.). (2012). *Stress and cardiovascular disease*. London: Springer.

Kunz-Ebrecht, S. R., Kirschbaum, C., Marmot, M., & Steptoe, A. (2004). Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoneuroendocrinology, 29*, 516–528.

Steptoe, A. (Ed.). (2006). *Depression and physical illness*. Cambridge: Cambridge University Press.

Steptoe, A. (Ed.). (2010). *Handbook of behavioral medicine*. New York: Springer.

Steptoe, A., & Appels, A. (Eds.). (1990). *Stress, personal control, and health*. Chichester: Wiley.

Steptoe, A., & Wardle, J. (Eds.). (1994). *Psychosocial processes and health: A reader*. Cambridge: Cambridge University Press.

Steptoe, A., Pollard, T. M., & Wardle, J. (1995). Development of a measure of the motives underlying the selection of food – The food choice questionnaire. *Appetite, 25*, 267–284.

Steptoe, A., Doherty, S., Rink, E., Kerry, S., Kendrick, T., & Hilton, S. (1999). Behavioural counselling in general practice for the promotion of healthy behaviour among adults at increased risk of coronary heart disease: Randomised trial. *BMJ, 319*, 943–947.

Steptoe, A., Cropley, M., Griffith, J., & Kirschbaum, C. (2000). Job strain and anger expression predict early morning elevations in salivary cortisol. *Psychosomatic Medicine, 62*, 286–292.

Steptoe, A., Willemsen, G., Owen, N., Flower, L., & Mohamed-Ali, V. (2001). Acute mental stress elicits delayed increases in circulating inflammatory cytokine levels. *Clinical Science, 101*, 185–192.

Steptoe, A., Wardle, J., & Marmot, M. (2005). Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes. *PNAS, 102*, 6508–6512.

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*, 901–912.

Stereotypes

- ▶ Stigma

Steroid Hormones

- ▶ Estrogen
- ▶ Steroids

S

Steroids

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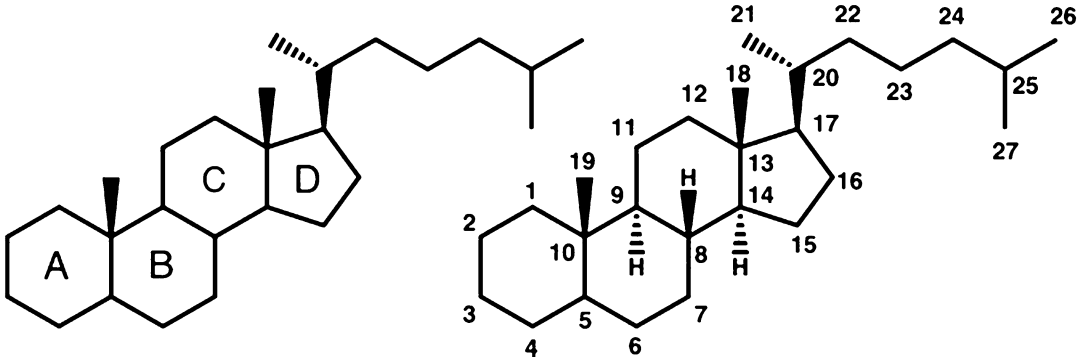
Synonyms

Steroid hormones



Steroids, Fig. 1 Structure and carbon numbering scheme for cholesterol and other steroids

Definition

A steroid or steroid hormone is a biomolecule derived from cholesterol, with a characteristic structure containing four fused rings (see Fig. 1).

Cholesterol is the precursor for five major classes of steroid hormones: progestagens, glucocorticoids, mineralcorticoids, androgens, and estrogens. These hormones are powerful signaling molecules that are released in order to elicit a specific response.

Androgens, such as testosterone, are responsible for the development of male sex characteristics, whereas estrogens are responsible for the development of female sex characteristics. Dehydroepiandrosterone (DHEA) is the most abundant circulating steroid in humans, and is a precursor for the sex hormones, testerosterone and estradiol. In addition, DHEA is a cortisol antagonist.

Glucocorticoids, such as cortisol, promote the formation of glycogen and inhibit the inflammatory response. They enable humans (and animals) to respond to stress.

Steroids act by interaction with cellular receptors that serve as transcription factors to regulate gene expression. Steroids are incredibly potent and elicit very specific responses due to their interaction with steroid receptors.

Cross-References

- ▶ Androgen
- ▶ Cortisol
- ▶ Estrogen
- ▶ Inflammation

References and Further Reading

Berg, J. M., Tymoczko, J. L., & Stryer, L. (2002). *Biochemistry* (5th ed.). New York: WH Freeman.

Nussey, S., & Whitehead, S. (2001). *Endocrinology*. Oxford: BIOS Scientific Publishers.

Sterol

- ▶ Cholesterol

Stigma

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Synonyms

Deviance; Discrimination; Prejudice; Stereotypes; Stigmatization

Definition

A stigma is a personal attribute, mark, or characteristic that is socially devalued and discredited (Goffman 1963). A wide variety of attributes are stigmas, including physical illnesses (e.g., HIV/AIDS, tuberculosis, epilepsy), mental illnesses (e.g., schizophrenia, mental disability), social norm violations (e.g., homosexuality, sex work, drug use, obesity), and certain demographic characteristics (e.g., racial/ethnic background, gender, socioeconomic status). People who possess a stigma are perceived and treated negatively by others and ultimately suffer worse physical, psychological, and behavioral outcomes than people who do not possess a stigma.

The following sections describe how certain attributes become socially devalued, how stigma impacts individuals who possess a stigma (i.e., stigmatized people) and who do not possess a stigma (i.e., nonstigmatized people) via a series of stigma mechanisms, and other considerations relevant to stigma. Because HIV/AIDS is one of the strongest stigmas throughout the world and has received a great deal of empirical attention related to stigma (e.g., Aggleton and Parker 2002), it used as the primary example of stigma throughout these sections.

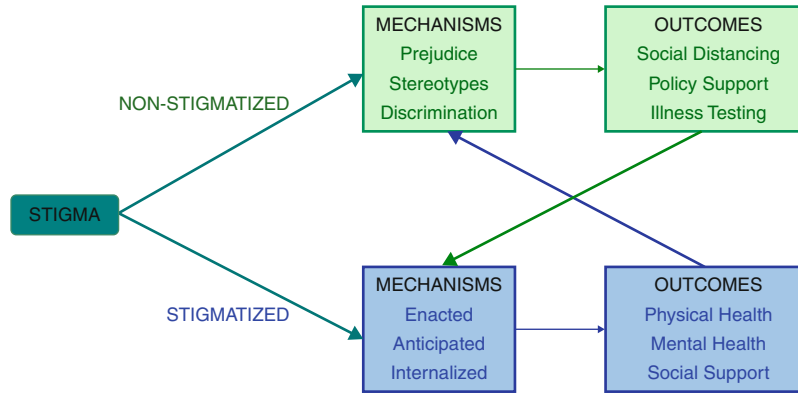
The Social Construction of Stigma

Stigmas are socially constructed (Crocker et al. 1998). In other words, certain attributes become devalued as the result of a social process (Link and Phelan 2001) rather than as the result of innate differences between people who possess the attribute and people who do not possess the attribute. This social process involves stereotyping by associating the attribute with negative characteristics. For example, people living with HIV/AIDS (PLWHA) may be stereotyped to be promiscuous. The social process also involves separating the people who have the attribute into out-group categories. HIV-negative people may view other HIV-negative as part of their social group (i.e., part of "us") but PLWHA as part of a different social group (i.e., part of "them"). Finally, the social process involves experiences of status loss and discrimination by people who have the attribute. PLWHA may not be given medical care because of their HIV-status. Importantly, the social process that results in stigma relies on power. Nonstigmatized people have more power than stigmatized people and use this power to produce and reproduce social inequities and inequalities. This happens in both subtle ways (e.g., stigmatized people being paid systematically less than nonstigmatized people) and blatant ways (e.g., stigmatized people being enslaved by nonstigmatized people).

Because stigma results from a social process, the extent to which an attribute is devalued varies across different social contexts. Social contexts include both individual relationships and cultural contexts. For example, individual people (e.g., friends, family members, employers, and healthcare providers) vary in the degree to which they view HIV/AIDS as a devalued attribute. Similarly, HIV/AIDS may be more devalued in some sociocultural contexts (e.g., Asian cities) than others (e.g., North American cities; Rao et al. 2008). The extent to which an attribute is devalued also varies across time. For example, devaluation associated with HIV/AIDS decreased during the 1990s within the United States (Herek et al. 2002). Taken together, the degree of devaluation associated with a stigma is not universal or fixed; instead, it changes relative to specific social contexts and time periods.

Stigma Mechanisms and Outcomes

Individuals are impacted by stigma via a series of stigma mechanisms. Stigma mechanisms refer to the ways in which people react to either possessing or not possessing a particular stigma (Earnshaw and Chaudoir 2009). Stigma mechanisms, in turn, result in physical, psychological, and behavioral outcomes for both stigmatized and nonstigmatized people. Figure 1 is adapted from the HIV Stigma Framework (Earnshaw and Chaudoir 2009) and shows how stigma leads to stigma mechanisms, which in turn lead to outcomes. Because they represent the link between the social process of stigma and outcomes associated with stigma, stigma mechanisms are often measured by researchers.



Stigma, Fig. 1 Stigma framework

Individuals who do not possess the stigma experience the stigma mechanisms of prejudice, stereotyping, and discrimination. Prejudice refers to negative emotions and feelings toward people who possess the stigma. For example, HIV-negative people may feel disgust toward PLWHA. Stereotypes refer to group-based beliefs about people who possess the stigma. HIV-negative people may believe that PLWHA are mostly gay men. Finally, discrimination refers to behavioral expressions of prejudice directed toward people who possess the stigma. HIV-negative people might refuse to hire, medically treat, or give housing to PLWHA. Although people who do not possess the stigma have more power than people who possess the stigma, people who do not possess the stigma may still suffer negative consequences due to stigma mechanisms. For example, an HIV-negative person who endorses the stereotype that PLWHA are mostly gay men may be less likely to engage in safe sex, be tested for HIV, or seek health care for HIV-related symptoms if they do not identify as a gay man. Consequently, belief in stereotypes can put HIV-negative people at risk for contracting HIV and not receiving treatment.

Individuals who possess the stigma experience the stigma mechanisms of anticipated stigma, enacted stigma, and internalized stigma. Enacted stigma, also called experienced stigma, refers to experiences of prejudice, stereotyping, and/or discrimination. For example, PLWHA may experience social rejection from friends and family members. Anticipated stigma refers to expectations of prejudice, stereotyping, and/or discrimination. PLWHA may expect that they will not be hired by a potential employer. Finally, internalized stigma refers to the endorsement of prejudice and stereotypes associated with one's stigma and applying them to the self. PLWHA may feel that they are dirty due to their HIV status. Further, stigma mechanisms experienced by individuals who do not possess the stigma may impact the outcomes of individuals who do possess the stigma. Discrimination perpetuated by HIV-negative people may be experienced as enacted stigma by PLWHA.

Stigma mechanisms profoundly impact the physical, psychological, and behavioral outcomes of stigmatized individuals. Meta-analytic evidence has shown that stigma mechanisms are associated with decreased physical health (e.g., increased physical illnesses and illness symptoms; Pascoe and Smart Richman 2009) and mental health (e.g., increased depression and decreased self-esteem; Mak et al. 2007). Additionally, stigma mechanisms are associated with maladaptive behaviors which may further undermine the health of stigmatized individuals. For example, stigma mechanisms are related to behaviors such as delayed HIV treatment initiation and non-adherence to medication regimens (Chesney and Smith 1999). Stigma mechanisms are further related to decreased likelihood of disclosure among individuals living with concealable

stigmatized identities including HIV/AIDS (Smith et al. 2008).

Other Considerations

Intersectional stigma and layered stigma refer to the possession of multiple stigmas. For example, a gay man living with HIV possesses two stigmas: homosexuality and HIV. Possessing multiple stigmas may exacerbate the impact of stigma on individuals.

Associative stigma, courtesy stigma, and affiliate stigma refer to being connected to someone who possesses a stigma. For example, an HIV-negative man who has a daughter living with HIV/AIDS may experience associative stigma due to his connection to his daughter. People who possess an associative stigma may experience negative outcomes via stigma mechanisms typically reserved for people who possess a stigma.

Concealable stigmas refer to devalued attributes that cannot be seen by others. Examples of concealable stigmas include many physical and mental illnesses and some social norm violations such as homosexuality and drug use. In contrast, visible stigmas refer to devalued attributes that can be seen by others. Examples of visible stigmas include many demographic characteristics such as racial/ethnic background and gender. Whereas people with concealable stigmas can hide their stigma from others in some social interactions, people with visible stigmas cannot.

Cross-References

- ▶ Discrimination and Health
- ▶ Health Disparities

References and Readings

Aggleton, P., & Parker, R. (2002). *A conceptual framework and basis for action: HIV/AIDS stigma and discrimination*. Geneva: Joint United Nations Programme on HIV/AIDS.

Allport, G. W. (1954). *The nature of prejudice*. Oxford, UK: Addison-Wesley.

Brewer, M. B., & Brown, R. J. (1998). Intergroup relations. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (4th ed., pp. 554–593). Boston: McGraw-Hill.

Chesney, M. A., & Smith, A. W. (1999). Critical delays in HIV testing and care: The potential role of stigma. *The American Behavioral Scientist, 47*, 1162–1174. https://doi.org/10.1177/00027649921954822.

Crocker, J., Major, B., & Steele, C. (1998). Social stigma. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (Vol. 2, 4th ed., pp. 504–553). Boston: McGraw-Hill/Distributed exclusively by Oxford University Press.

Earnshaw, V. A., & Chaudoir, S. R. (2009). From conceptualizing to measuring HIV stigma: A review of HIV stigma mechanism measures. *AIDS and Behavior, 13*, 1160–1177. https://doi.org/10.1007/s10461-009-9593-3.

Goffman, E. (1963). *Stigma: Notes on the management of spoiled identity*. New York: Simon & Schuster.

Herek, G. M., Capitanio, J. P., & Widaman, K. F. (2002). HIV-related stigma and knowledge in the United States: Prevalence and trends, 1991–1999. *American Journal of Public Health, 92*, 371–377. https://doi.org/10.2105/ajph.92.3.371.

Jones, E. E., Farina, A., Hastorf, A. H., Markus, H., Miller, D. T., & Scott, R. A. (1984). *Social stigma: The psychology of marked relationships*. New York: W. H. Freeman and Company.

Link, B. G., & Phelan, J. C. (2001). Conceptualizing stigma. *Annual Review of Sociology, 27*, 363–385. https://doi.org/10.1146/annurev.soc.27.1.363.

Major, B., & O'Brien, L. T. (2005). The social psychology of stigma. *Annual Review of Psychology, 56*, 393–421. https://doi.org/10.1146/annurev.psych.56.091103.070137.

Mak, W. W. S., Poon, C. Y. M., Pun, L. Y. K., & Cheung, S. F. (2007). Meta-analysis of stigma and mental health. *Social Science & Medicine, 65*(2), 245–261. https://doi.org/10.1016/j.socscimed.2007.03.015.

Pascoe, E. A., & Smart Richman, L. (2009). Perceived discrimination and health: A meta-analytic review. *Psychological Bulletin, 135*(4), 531–554. https://doi.org/10.1037/a0016059. (Supplemental).

Rao, D., Angell, B., Chow, L., & Corrigan, P. (2008). Stigma in the workplace: Employer attitudes about people with HIV in Beijing, Hong Kong, and Chicago. *Social Science & Medicine, 67*, 1541–1549. https://doi.org/10.1016/j.socscimed.2008.07.024.

Smith, R., Rossetto, K., & Peterson, B. L. (2008). A meta-analysis of disclosure of one's HIV-positive status, stigma and social support. *AIDS Care, 20*(10), 1266–1275. https://doi.org/10.1080/09540120801926977.

Stigmatization

▶ Stigma

Stop Smoking

▶ Smoking Cessation

Strain

▶ Psychological Stress

Stranger Anxiety

▶ Anxiety

Strength Model of Self-Control

▶ Self-Regulatory Capacity

Stress

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Synonyms

Anxiety; Distress; Mental stress; Pressure; Psychological stress

Definition

Stress is a transactional process occurring when an event is perceived as relevant to an individual's well-being, has the potential for harm or loss, and requires psychological, physiological, and/or behavioral efforts to manage the event and its outcomes (Lazarus and Folkman 1984). The stimuli or events that cause stress are referred to as stressors (Mason 1975). Stress often results in psychological distress and efforts to cope with the event. Physiological stress responses are often in support of efforts to manage the stressful event and protect the organism from harm (McEwen and Seeman 1999). Stress may not be a uniformly negative experience, as stressful events may also include the potential for benefit and growth (Lazarus & Folkman). Early views of stress focused on physical stress – events that perturb the resting homeostasis of the body, such as changes in temperature or physical injury (McEwen and Seeman 1999). Current views of stress focus heavily on social and psychological sources, with appraisals of the event and the perceived coping resources as key features. Stress may be categorized by its severity, its time course (acute, repeated, or chronic), and degree of control over the stressor. Distinctions also have been made between active coping stressors and passive coping stressors (Obrist 1981). Active coping stressors require overt behavioral action, such as giving a speech or performing a reaction time task, whereas passive coping stressors require that the individual endure without behavioral action, such as watching gruesome photos. Psychological and physiological stress responses have been shown to differ based upon these dimensions (Tomaka et al. 1993).

Cross-References

▶ Coping
▶ Family Stress
▶ Life Events
▶ Perceived Stress
▶ Perceptions of Stress
▶ Physiological Reactivity
▶ Psychological Stress
▶ Stress Responses
▶ Stress, Emotional
▶ Trier Social Stress Test

References and Readings

Cohen, S., Kessler, R. C., & Gordon, L. U. (1995). *Measuring stress: A guide for health and social scientists*. New York: Oxford University Press.
Hobfoll, S. E. (1989). Conservation of resources: A new attempt at conceptualizing stress. *American Psychologist, 44*, 513–524.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.
Mason, J. W. (1975). A historical view of the stress field (Parts I and II). *Journal of Human Stress, 1*, 6–12 & 22–36.
McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the NY Academy of Science, 896*, 30–47.
Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum Press.
Selye, H. (1956). *The stress of life*. New York: McGraw-Hill.
Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisals. *Journal of Personality and Social Psychology, 65*, 248–260.

Stress and Occupational Health

► Psychosocial Work Environment

Stress Appraisals

► Perceptions of Stress

Stress Cascade

► Neuroendocrine Activation

Stress Diathesis Models

► Stress Vulnerability Models

Stress Disorder

► Anxiety Disorder

Stress Management

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Synonyms

Coping with stress; Relaxation techniques; Stress reduction

Definition

Stress management techniques and interventions can be broadly categorized into skills that are provided via education, relaxation training, psychosocial interventions, and group formats, alone or in combination, that aim to reduce the stress response by targeting coping strategies and relaxation skills.

Description

The deleterious effects of stress on health and well-being are well documented and have led to the incorporation of stress management techniques into many psychosocial treatment protocols, alone or in combination with medical treatments. Chronic stress has been linked to physiologic changes such as neuroendocrine and immune dysregulation, and worsened disease profiles. One of the most prominent associations between stress and poor health has been in the area of cardiovascular risk. For example, evidence suggests that chronic stress is associated with increased sympathetic cardiovascular activity and damage to endothelial functioning,

which increases the risk of several cardiovascular conditions, such as arterial hypertension, coronary artery disease, and arrhythmias. Therefore, incorporation of stress management into clinical and research protocols designed to improve health and well-being has become increasingly popular.

The field of stress management is comprised of a variety of methods and techniques. Relaxation skills training methods with the most empirical support include progressive relaxation or progressive muscle relaxation, autogenic training, biofeedback, mental imagery, and other Eastern or Westernized meditation methods. Various cognitive techniques and in some cases, pharmacotherapy, are also part of stress management programs. Other methods that have been incorporated into stress management include listening to relaxing music, massage, aerobic exercise, diaphragmatic breathing, and postural relaxation methods. Some evidence suggests that "active" stress management techniques (e.g., breathing-guided relaxation training) may be more effective in reducing stress symptoms and inducing improved autonomic cardiovascular regulation than "passive" techniques (e.g., massage). Several standardized stress management interventions have been developed and shown to be efficacious in reducing symptoms of stress and improving various indicators of physical and mental health and quality of life. Empirical evidence supports the use of stress management techniques in the treatment of migraines, pain, and other somatic complaints, and have also been used to reduce stress symptoms, improve adjustment, well-being, and quality of life in a range of patient populations, including those diagnosed with type 2 diabetes, coronary heart disease, fibromyalgia, chronic fatigue syndrome (CFS), human immunodeficiency virus (HIV), and cancer.

Stress management intervention strategies range from single session psychosocial treatments to multifaceted treatments involving psychosocial and behavioral modification components. Likewise, intervention aims and target outcomes also range from behavior and lifestyle changes (e.g., diet, exercise, and utilization of stress management techniques) to changes in psychological and physical well-being (e.g., relief of depressive or anxious symptoms, cortisol regulation). It is hypothesized that if one can manage stress effectively, an individual's ability to adopt lifestyle changes that positively impact health outcomes will be maximized. Conversely, chronic stress has been associated with negative lifestyle factors including poor diet, sedentary lifestyle, alcohol and substance use, and poor adherence to medical regimens. Therefore, many stress management programs include medical endpoints such as disease risk factors, disease morbidity, and mortality. For example, stress management interventions in coronary heart disease may include known risk factors as relevant endpoints, such as being overweight or obese, smoking status, blood pressure, cholesterol, lipids, cardiac ischemia (e.g., angina), and number of cardiac events and/or procedures (e.g., myocardial infarction, angioplasty). Endpoints may be measured at proximal, intermediate, or distance time points, depending on the outcome of interest.

Cultural factors have also been shown to be associated with stress and stress management. Although all cultures experience stress and its sequelae, psychosocial sources of stress, the expression of stress symptoms, and the use and acceptability of stress management techniques varies across cultures. For example, evidence suggests that Hispanics are more likely to experience somatic symptoms in response to stress, compared to non-Hispanic Whites. Cultural differences in stress symptoms will likely lead to varying intervention and treatment approaches. Individuals who present with emotional symptoms of stress without a somatic component, for example, receive more treatment in the United States compared to South Korea, a collectivist culture in which individuals are less likely to express signs of emotional distress directly. However, there is no cross-cultural difference between the United States and South Korea in treatment of somatic symptoms of stress, suggesting that treatment methods may be culturally biased.

Stress Management Techniques and Interventions

Many different methods of stress management have been employed with empirical support across a range of populations. Progressive relaxation or progressive muscle relaxation consists of consecutively tensing and relaxing different sets of muscle groups throughout the body, generally starting with the feet and systematically progressing up to the head. Diaphragmatic breathing consists of taking deep breaths in which the diaphragm contracts and the abdomen, rather than the chest, is extended. This type of breathing involves a slow and deep inhalation through the nose, usually to a count of 10, followed by slow and complete exhalation for a similar count; the process is repeated for a preferred number of times to facilitate relaxation. Using mental imagery as a stress management tool involves imagining a scene, place, or event that is considered safe, peaceful, and restful; one that is associated with affective feelings of happiness, job, and contentment. Alternatively, images may involve mental pictures of stress flowing out of the body or being locked away in a padlocked chest. All of the senses are incorporated into the mental imagery exercise and it is encouraged to develop details and complex images that invoke sensual perceptions. The imagined place is used as a retreat from environmental stressors, with the goal of having the body react to imagined scenes of peace and tranquility as if they were real, counteracting the adrenergic effects of stress. These methods have been shown to be beneficial through self-report measures of stress and well-being and physical measures of the body's stress response through biofeedback methods.

Biofeedback is a method that keeps track of the body's physiological responses in real time, generally through machines that measure heart rate, muscle tension, or brain waves. Most often, biofeedback is used as a tool to facilitate control over the stress response sequelae. Individuals are taught to recognize the stress response when it is underway and employ relaxation techniques (e.g., deep breathing, mental imagery, or adaptive cognitive replacement) to calm physiological arousal. Theory and empirical evidence suggest that the real-time feedback of physiological changes in response to stress and the utilization of stress management techniques facilitate learning and adoption of effective relaxation methods into daily life.

Stress Management Programs

Many stress management interventions for clinical populations consist of a combination of methods that frequently incorporate a variety of cognitive and behavioral techniques. For example, cognitive-behavioral stress management (CBSM) interventions have been employed and been shown to have beneficial effects in stressed nonclinical subjects and a range of patient populations. These interventions typically aim to improve adaptive coping and reduce psychological distress through the use of emotion regulation strategies and relaxation training. Didactic training typically addresses stress appraisal and cognitive coping strategies to improve stress management of general- and disease-specific stressors. Often, a psychoeducational component about the nature and consequences of stress and disease processes is also incorporated into intervention protocols. The CBSM protocol consists of a 10-week manualized group intervention in which groups meet for 2 h per week; sessions consist of 90 min of didactic discussion and exercises and 30 min of relaxation training. During the didactic portion of each session, participants are provided information regarding stress awareness, physical responses to stress, and the appraisal process and are taught a variety of cognitive-behavioral techniques designed to manage general- and disease-specific stress. Cognitive techniques include learning to identify cognitive distortions and cognitive restructuring processes (e.g., rational thought replacement), effective coping strategies (e.g., emotional-focused vs. problem-focused coping), and anger management and assertiveness training (e.g., effective communication). Information related to disease physiology, diagnosis, and treatment are also provided and health maintenance strategies are reviewed.

During the relaxation portion, participants are taught a variety of techniques through group relaxation exercises, including progressive muscle relaxation, guided imagery, meditation, and diaphragmatic breathing. Participants are encouraged to practice the techniques at home on a daily basis. The CBSM intervention has been shown to be effective in increasing stress management skills, which has been related to improvements in a number of quality-of-life domains, in HIV, cancer, and CFS populations.

Similarly, some have demonstrated the efficacy of a group-based stress management program that included progressive muscle relaxation training, didactic training in the use of cognitive and behavioral skills to bring awareness to and reduce physiological symptoms of stress (e.g., guided imagery, deep breathing techniques, thought stopping, and recognition of life stressors), and education on the negative effects of stress on health in improving glycemic control in patients with type 2 diabetes. Participants learned stress management techniques in a group-based intervention format and were instructed to practice muscle relaxation at home twice daily with the aid of an audiotape. Specific instructions were given to encourage "mini-practices" (i.e., brief, 30-s versions of a progressive relaxation session) to facilitate the use of stress management and relaxation techniques into daily life. Although similar interventions have been conducted and shown positive effects on blood glucose, findings have been mixed as several others have failed to show a therapeutic effect of stress management on diabetes control.

Mindfulness-based stress reduction (MBSR) is a clinically standardized meditation program, originally developed as a group-based intervention for chronic pain but has since demonstrated efficacy for patients with a range of mental and physical disorders, as well as healthy subjects. The MBSR protocol consists of three different stress management techniques: body scan, which involves focusing on different parts of the body, bringing attention and awareness to sensations and feelings, breath awareness, and relaxation; sitting meditation, which involves focusing on body sensations and breathe, as well as nonjudgmental awareness of the cognitions and the stream of thoughts and distractions that pass through the mind; and Hatha yoga practice, which consists of breathing exercises, stretching, and postural exercises designed to strengthen and relax the musculoskeletal system. The MBSR program is an 8- to 10-week group intervention in which sessions typically last 2.5 h, with an additional single all-day session per course. Homework of at least 45 min a day, 6 days a week, is also encouraged, which may consist of meditation practice, mindful yoga, and/or incorporating mindfulness into daily life. Groups may be either homogenous or heterogeneous with regard to illnesses or presenting problems of participants. Empirical evidence provides consistent and relatively strong effect sizes regarding the efficacy of MBSR on a number of psychological (e.g., depressive and anxiety symptoms, coping style) and physical (e.g., medical symptoms, sensory pain, physical impairment, and functional quality of life) well-being in clinical and nonclinical populations. Clinical populations have included patients diagnosed with anxiety disorders, depression, chronic pain, fibromyalgia, cancer, and stress related to environmental contexts (e.g., medical school, prison life), as well as relatively healthy individuals interested in improving their ability to cope with normal stressors of daily living.

Stress management interventions for healthy and patient populations are promising. However, findings have been mixed and the literature is limited by measurement and research design problems, insufficient information regarding intervention components and protocol fidelity, clinical relevance of statistically significant effects, and feasibility and dissemination concerns regarding the translation of research protocols into clinical practice. Nevertheless, given the detrimental effects of stress on psychological and physical well-being, further research is needed using large-scale, randomized clinical trials, with sound methodological procedures that include objective markers of health and disease status, in addition to self-report measures of psychosocial well-being and functional indicators of distress.

Cross-References

► Anger Management

References and Readings

Antoni, M. H., Lechner, S. C., Kazi, A., Wimberly, S. R., Sifre, T., Urcuyo, K. R., Phillips, K., Glück, S., & Carver, C. S. (2006). How stress management improves quality of life after treatment for breast cancer. *Journal of Consulting and Clinical Psychology, 74*(6), 1143–1152.

Blumenthal, J. A., Sherwood, A., Babyak, M. A., Watkins, L. L., Waugh, R., Georgiades, A., Bacon, S. L., Hayano, J., Coleman, R. E., & Hinderliter, A. (2005). Effects of exercise and stress management training on markers of cardiovascular risk in patients with ischemic heart disease. *Journal of the American Medical Association, 293*(13), 1626–1634.

Brown, J. L., & Vanable, P. A. (2008). Cognitive-behavioral stress management interventions for personal living with HIV: A review and critique of the literature. *Annals of Behavioral Medicine, 35*, 26–40.

Chiesa, A., & Serretti, A. (2009). Mindfulness-based stress reduction for stress management in healthy people: A review and meta-analysis. *The Journal of Alternative and Complementary Medicine, 15*(5), 593–600.

Gaab, J., Blattler, N., Menzi, T., Pabst, B., Stoyer, S., & Ehlert, U. (2003). Randomized controlled evaluation of the effects of cognitive-behavioral stress management on cortisol responses to acute stress in healthy subjects. *Psychoneuroendocrinology, 28*, 767–779.

Lehrer, P. M., Carr, R., Sargunaraj, D., & Woolfolk, R. L. (1994). Stress management techniques: Are they all equivalent, or do they have specific effects? *Biofeedback and Self-Regulation, 19*(4), 353–401.

Lehrer, P. M., Woolfolk, R. L., & Sime, W. E. (Eds.). (2007). *Principles and practice of stress management*. New York: Guildford.

Lucini, D., Malacarne, M., Solaro, N., Busin, S., & Pagani, M. (2009). Complementary medicine for the management of chronic stress: Superiority of active versus passive techniques. *Journal of Hypertension, 27*, 2421–2428.

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews, 87*, 873–904.

Smith, J. E., Richardson, J., Hoffman, C., & Pilkington, K. (2005). Mindfulness-based stress reduction as supportive therapy in cancer care: Systematic review. *Journal of Advanced Nursing, 52*(3), 315–327.

Surwit, R. S., van Tilburg, M. A., Zucker, N., McCaskill, C. C., Parekh, P., Feinglos, M. N., Edwards, C. L., Williams, P., & Lane, J. D. (2002). Stress management improves long-term glycemic control in type 2 diabetes. *Diabetes Care, 25*(1), 30–34.

Stress Mindset

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Synonyms

Beliefs about stress; Implicit theories; Lay beliefs

Definition

Stress mindset refers to a set of beliefs individuals hold about the consequences of experiencing stress. This includes the belief that stress has enhancing consequences (i.e., a stress-is-enhancing mindset) and the contrasting belief that stress has debilitating consequences (i.e., a stress-is-debilitating mindset) for health and vitality, performance and productivity, and learning and growth (Crum et al. 2013). Stress mindset contrasts with transactional stress appraisal (Lazarus & Folkman 1984); in that the former concerns beliefs about the stress response in general which is theorized to apply across situations, whereas the latter is a single response to a stressor in a particular situation.

Description

Mindsets refer to the beliefs about the malleability of personal qualities that serve as a mental lens or framework through which people make predictions about and judge the meaning of life events (Dweck et al. 1995; Yeager & Dweck 2012). Mindsets are otherwise known as implicit theories in the literature. The term "implicit" is ascribed to these beliefs as they tend not to be explicitly articulated (Dweck et al. 1995; Yeager & Dweck 2012). However, it is not clear whether mindsets are truly implicit and operating outside of conscious awareness, consistent with dual-process theories of cognition and behavior.

Crum et al. (2013) applied the mindset concept to stress research, finding that stress mindset influences a range of stress-related outcomes and is distinct from other important variables in the stress process, such as stressor appraisal and amount of stress. Supporting the notion that stress mindset is a distinct variable in the stress process, Kilby and Sherman (2016) found stress mindset exhibited a moderate correlation with challenge appraisal and no significant correlation with threat appraisal following a mathematics stressor task. They also found no significant difference in stress mindset scores before and after the stressor task, suggesting that stress mindset may not be influenced by stressful events. The potential implicit operation of stress mindset was explored by Keech et al. (2018) using an implicit association test (IAT) based on the single-category procedure (Karpinski & Steinman 2006). Despite the reliability of the test being adequate, the test was not related to any of the outcome variables or the explicit measure of stress mindset. Given this was preliminary research, further studies are needed to understand the processes through which stress mindsets operate.

Mean stress mindset scores across a range of observational studies and experimental studies premanipulation indicate that on average people by default tend to endorse a stress-is-debilitating mindset (Crum et al. 2013, 2017; Keech et al. 2018; Kilby & Sherman 2016; Park et al. 2018). Crum et al. (2013) argue that this is due to public health messages being framed around the requirement of reducing stress to prevent negative consequences. Despite these defaults, several studies have demonstrated that stress mindsets are malleable, with mean mindset scores rising to a level that reflects endorsing a stress-is-enhancing mindset following an experimental manipulation (Crum et al. 2013, 2017).

Measuring Stress Mindset

To date, two self-report measures of stress mindset have been published: the Stress Mindset Measure (SMM; Crum et al. 2013) and the Stress Control Mindset Measure (SCMM; Keech et al. 2018). The SMM is an eight-item scale that examines the extent to which an individual holds a stress-is-enhancing mindset. There is a general and a stressor-specific version. No known studies have been published using the specific version following its initial publication. Crum et al. (2013) describe stress-is-enhancing and stress-is-debilitating mindsets as two ends of a spectrum, and items in the SMM are presented as fixed-enhancing (e.g., "Experiencing stress enhances my performance and productivity") versus fixed-debilitating (e.g., "Experiencing stress debilitates my performance and productivity"). However, many people have non-enhancing experiences of stress, and stress can be both enhancing and debilitating. Preliminary evidence from experimental research also suggests that presenting a balanced view of stress rather than a positive or negative view of stress alone yields lower heart rates and diastolic blood pressure following a laboratory-induced stressor (Liu et al. 2017). Arguing that the polarized approach to measurement of stress mindset diverges from how the construct was conceptualized by Crum et al. (2013) and from mindset theory more broadly (e.g., Dweck et al. 1995; Job et al. 2015), Keech et al. (2018) developed the 15-item SCMM. The SCMM is framed to assess the extent to which an individual holds the mindset that stress "can be" enhancing (e.g., "Stress can be used to enhance your performance and productivity").

Associations Between Stress Mindset and Health-Related Outcomes

Several experimental and correlational studies have observed effects of stress mindset on health and wellbeing, physiological, and affective outcomes in response to laboratory stressor tasks and ecological stress experiences over short periods of time. For example, Crum et al. (2017), in an experimental study examining the influence of stress mindset on a range of outcomes in response to a laboratory stressor, found those who were primed with a stress-is-enhancing mindset exhibited greater positive but not negative affect when anticipating and following the induced stressor. Other studies have examined the association between stress mindset and health and wellbeing outcomes in the context of ecological experiences of stress. Park et al. (2018) found

that stress mindset moderated the effect of stressful life events on perceived distress (mediator) and in turn self-control. Keech et al. (2018) found that stress mindset predicted proactive coping behavior under stress, perceived general somatic symptoms, perceived distress, perceived physical health, and, indirectly, psychological wellbeing. Crum et al. (2013) observed a small effect of a stress mindset manipulation on depression and anxiety symptoms from baseline to three days postintervention among company employees.

Two studies have examined the physiological effects of stress mindset when faced with laboratory-induced stressors. Crum et al. (2013) reported that stress mindset moderated the effect of a laboratory-induced stressor on cortisol, yet Crum et al. (2017) found no effect of stress mindset on cortisol in anticipation of, and following, a laboratory-induced stressor. These physiological effects should therefore be interpreted with caution and further research is required to replicate these findings. Crum et al. (2017) also found a quadratic effect of stress mindset on dehydroepiandrosterone sulfate (DHEAS) – a neurosteroid hormone which in reduced levels may increase vulnerability to neurotoxic effects of stress (Maninger et al. 2009) – with those in the enhancing condition experiencing sharper increases and sharper declines in DHEAS prior to and following the laboratory-induced stressor.

Two correlational studies support that stress mindset influences coping responses. Measuring workload anticipation and approach coping daily over five days, Casper et al. (2017) found that employees endorsing a stress-is-enhancing mindset made more approach-coping efforts in anticipation of a high workload. Keech et al. (2018) also found in a cross-sectional study that stress mindset predicted proactive coping behavior under stress.

Providing further evidence to support these notions that beliefs about stress can influence health outcomes, two large national longitudinal studies have found the perception that stress is affecting one's health to be associated with premature death in the United States (Keller et al. 2012) and adverse cardiovascular events in the United Kingdom (Nabi et al. 2013).

How Could Stress Mindset Improve Health?

Keech et al. (2018) proposed a stress beliefs model outlining two mechanisms through which stress mindsets may improve health and wellbeing outcomes. The first of which is that those endorsing more of an enhancing stress mindset would favor more proactive behaviors when coping with stress. The study demonstrated empirically that proactive behavior under stress mediated the influence of stress mindset on psychological wellbeing and perceived stress. Further research is required to evaluate causal links and to examine the role of more emotion-focused coping behaviors under stress in this process.

The second mechanism proposed by Keech et al. (2018) is that those endorsing more of an enhancing stress mindset may interpret the physiological arousal characteristic of the stress response as less adverse than those holding more of a debilitating stress mindset. Keech et al. (2018) provided preliminary empirical support for this process, finding that self-reported perceived general somatic symptoms mediated the effect of stress mindset on psychological wellbeing, perceived stress, and perceived physical health. Further investigation of this process is required. One line of future enquiry is examining whether stress mindsets influence the use of situational cognitive strategies such as arousal reappraisal when under stress (Jamieson et al. 2012).

Conclusion and Future Directions

While preliminary evidence points to positive outcomes being associated with eliciting a stress-is-enhancing mindset prior to the experience of laboratory-induced stressors and ecological stressors over a short period of time, observed effects have been small to medium in magnitude, and it remains unclear whether these mindsets would be activated during the experience of acute and chronic ecological stressors with established links to adverse health outcomes over longer periods of time. Future research should seek to investigate the influence of stress mindset in these contexts to better understand

whether these mindsets can be leveraged to meaningfully reduce the impact of stress on health. Further exploration of the mechanisms through which stress mindsets may influence outcomes is also important for maximizing intervention effects and for determining appropriate applications of these interventions.

Cross-References

- ▶ Lay Beliefs
- ▶ Perceived Stress
- ▶ Perceptions of Stress
- ▶ Stress Management
- ▶ Stress: Appraisal and Coping

References and Further Reading

Casper, A., Sonnentag, S., & Tremmel, S. (2017). Mindset matters: The role of employees' stress mindset for day-specific reactions to workload anticipation. *European Journal of Work and Organizational Psychology, 26*(6), 798–810. https://doi.org/10.1080/1359432X.2017.1374947.

Crum, A. J., Salovey, P., & Achor, S. (2013). Rethinking stress: The role of mindsets in determining the stress response. *Journal of Personality and Social Psychology, 104*(4), 716–733. https://doi.org/10.1037/a0031201.

Crum, A. J., Akinola, M., Martin, A., & Fath, S. (2017). The role of stress mindset in shaping cognitive, emotional, and physiological responses to challenging and threatening stress. *Anxiety, Stress, and Coping, 30*(4), 379–395. https://doi.org/10.1080/10615806.2016.1275585.

Dweck, C. S., Chiu, C., & Hong, Y. (1995). Implicit theories and their role in judgments and reactions: A word from two perspectives. *Psychological Inquiry, 6*(4), 267–285. https://doi.org/10.1207/s15327965pli0604_1.

Jamieson, J. P., Nock, M. K., & Mendes, W. B. (2012). Mind over matter: Reappraising arousal improves cardiovascular and cognitive responses to stress. *Journal of Experimental Psychology: General, 141*(3), 417–422. https://doi.org/10.1037/a0025719.

Job, V., Walton, G. M., Bernecker, K., & Dweck, C. S. (2015). Implicit theories about willpower predict self-regulation and grades in everyday life. *Journal of Personality and Social Psychology, 108*(4), 637–647. https://doi.org/10.1037/pspp0000014.

Karpinski, A., & Steinman, R. B. (2006). The single category implicit association test as a measure of implicit social cognition. *Journal of Personality and Social Psychology, 91*(1), 16–32. https://doi.org/10.1037/0022-3514.91.1.16.

Keech, J. J., Hagger, M. S., O'Callaghan, F. V., & Hamilton, K. (2018). The influence of university students' stress mindsets on health and performance outcomes. *Annals of Behavioral Medicine*, *52*(12), 1046–1059. https://doi.org/10.1093/abm/kay008.

Keller, A., Litzelman, K., Wisk, L. E., Maddox, T., Cheng, E. R., Creswell, P. D., & Witt, W. P. (2012). Does the perception that stress affects health matter? The association with health and mortality. *Health Psychology, 31*(5), 677. https://doi.org/10.1037/a0026743.

Kilby, C. J., & Sherman, K. A. (2016). Delineating the relationship between stress mindset and primary appraisals: Preliminary findings. *SpringerPlus, 5*, 336. https://doi.org/10.1186/s40064-016-1937-7.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer Publishing.

Liu J. J. W., Vickers K., Reed M., Hadad M. (2017). Re-conceptualizing stress: Shifting views on the consequences of stress and its effects on stress reactivity. *PLoS ONE*, *12*, e0173188

Maninger, N., Wolkowitz, O. M., Reus, V. I., Epel, E. S., & Mellon, S. H. (2009). Neurobiological and neuropsychiatric effects of dehydroepiandrosterone (DHEA) and DHEA sulfate (DHEAS). *Frontiers in Neuroendocrinology, 30*(1), 65–91. https://doi.org/10.1016/j.yfrne.2008.11.002.

Nabi, H., Kivimäki, M., Batty, G. D., Shipley, M. J., Britton, A., Brunner, E. J., . . ., & Singh-Manoux, A. (2013). Increased risk of coronary heart disease among individuals reporting adverse impact of stress on their health: The Whitehall II prospective cohort study. *European Heart Journal*, *34*(34), 2697–2705. https://doi.org/10.1093/eurheartj/eht216.

Park, D., Yu, A., Metz, S. E., Tsukayama, E., Crum, A. J., & Duckworth, A. L. (2018). Beliefs about stress attenuate the relation among adverse life events, perceived distress, and self-control. *Child Development*, 89(6), 2059–2069. https://doi.org/10.1111/cdev.12946.

Yeager, D. S., & Dweck, C. S. (2012). Mindsets that promote resilience: When students believe that personal characteristics can be developed. *Educational Psychologist*, *47*(4), 302–314. https://doi.org/10.1080/00461520.2012.722805

Stress Reactivity

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Synonyms

Stress responsivity

Definition

Stress reactivity is the capacity or tendency to respond to a stressor. It is a disposition that underlies individual differences in responses to stressors and is assumed to be a vulnerability factor for the development of diseases.

Description

People respond differently when exposed to the same stressor. Such differences can be observed in all four major stress response domains, namely, physiology, behavior, subjective experience, and cognitive function. Within the physiological domain, two response systems are of particular importance: cardiovascular responses (indicated by blood pressure and heart rate), driven by sympathetic nervous system (SNS) activity, and output of the glucocorticoid hormone cortisol from the adrenal cortex, driven by hypothalamic-pituitary-adrenal (HPA) axis activity. Stress reactivity is assumed to be stable over time, i.e., persons showing high responses at an initial assessment also show high responses when the assessment is repeated at a later time. Stress reactivity can be conceptualized as specific or general. Whereas specific stress reactivity reflects reactivity of a particular response system (e.g., cardiovascular stress reactivity; endocrine stress reactivity; affective stress reactivity), general stress reactivity is indicated by aggregation of responses across domains and/or stressors.

The relevance of stress reactivity for behavioral medicine rests primarily on the assumption that high stress reactivity is assumed to be a vulnerability factor for disease that predicts disease outcome variance independently of well-established risk factors. As early as in the first half of the twentieth century it was proposed that the size of blood pressure responses to placing the hand in cold water (cold pressor test) would indicate the risk of later development of hypertension. More recently, a growing body of evidence from longitudinal studies that used laboratory stress tests support the assumption that cardiovascular stress reactivity is indeed a risk factor for subclinical and clinical cardiovascular disease. This has been shown for both physiological stressors such as the cold pressor test and psychological stressors such as pressure to perform in a social situation (e.g., public speaking). On the basis of this evidence it has been concluded that cardiovascular stress reactivity is a risk factor for cardiovascular disease in addition to the classic risk factors of family history, obesity, smoking, diabetes mellitus, and hypercholesterolemia. As for endocrine stress reactivity, a few longitudinal studies found evidence for associations between endocrine stress reactivity and increased risk for disease. However, this research area is much less developed than that for cardiovascular stress reactivity, and the evidence is less robust. Finally, it has been suggested that endocrine and affective stress reactivity might be a risk factor for the development of mental disorders such as psychosis, depression, and anxiety disorders. Again, research in this area is still relatively scarce and the evidence mostly relies on cross-sectional studies.

In contrast to specific stress reactivity, the concept of general stress reactivity emphasizes generalizability of responses across response systems and stressors. It is based on the notion that stress responses have a common origin in brain areas that mediate activation of the HPA axis and the SNS, as well as behavioral and subjective-emotional responses. In particular, hippocampus, amygdala, and prefrontal cortex are higher central mediators of subjective-emotional responses. These areas are functionally connected to the hypothalamic and brainstem nuclei which are critical in activating the HPA axis and the SNS. If a stressor is processed in higher brain areas, stress responses are expected to show relatively high covariance. This is to be expected for psychological stressors, but less so for physiological stressors. Thus, dissociations between responses systems might reflect individual differences at both levels. In addition, observed physiological stress responses are influenced by peripheral factors such as receptor sensitivity in the periphery or vascular resistance. Although high covariance between response systems would be expected, a number of factors such as different dynamics of the response systems, habituation effects, measurement error, and limited variance due to limited

stressor intensity act at attenuating associations between responses.

Complementary to the notion of general stress reactivity, the concepts of individual response specificity (IRS) and stimulus response specificity (SRS) reflect observations of dissociation between response systems or stressors. The concept of IRS describes individual differences in patterns of responses, for example, one person might respond to stressors with a high blood pressure but low cortisol increase, whereas another person might also show a high cortisol increase. SRS describes such response patterns as related to stressors, for example, it has been proposed that the HPA axis in humans is activated by stressors that include social evaluative threat, but not by cognitive effort without that component. In research, it is often difficult to reliably detect general stress reactivity, IRS or SRS response patterns. Although there is now increasing evidence for the concept of general stress reactivity, associations between responses are usually moderate. Therefore, it is important to note that it is not possible to use the stress response in one domain or system as a general indicator of responses in other domains. Although it has been suggested that such dissociations might present useful information about psychobiological responses systems of an individual that could be valuable for behavioral medicine, to date little is known about the stability and implications of response dissociations.

Stress reactivity can be assessed both in the laboratory and in daily life. The major advantage of laboratory stress tests is the high degree of standardization over the conditions implemented. However, stress responses in the laboratory have limited ecological validity, i.e., do not necessarily reflect stress reactivity in daily life. For that reason, ambulatory assessment methods are increasingly used and present the opportunity to assess real-time stress reactivity in daily life for both research and clinical practice. A number of factors influence stress reactivity, with implications for clinical decisions. For example, it is known that stress reactivity is associated with sex, age, ethnicity, personality factors, preexisting disease, and the presence or absence of chronic stress. Due to such moderating factors there is a wide range of associations between stress reactivity and disease outcome. Therefore, the predictive value varies across individuals, making it difficult to use stress reactivity scores in clinical practice. Although the reliability of stress reactivity assessments could be increased by assessing aggregated stress responses in a repeated measure/multiple stressors design, few studies have implemented this design due to the high demands on resources. It would be expected that stronger and more consistent association with disease could be observed with such a design. Finally, an important conclusion from the variety of findings on associations between stress response systems is that, as mentioned above, a single assessment of one response system cannot be used as an indicator for stress responses in other systems. The notion of a consistent "gold-standard" indicator of stress reactivity is not supported by the research literature.

As the assessment of physiological stress responses is relatively expensive and fraught with practical problems, clinicians often rely on retrospective self-reports of individual stress experience to assess stress reactivity. However, such subjective measures of stress experience confound individual stress reactivity with frequency of exposure to daily life stress. Recently, a self-report instrument for the assessment of perceived stress reactivity has been developed and evaluated. Although retrospective self-report methods cannot replace real-time measures, they present an opportunity to assess patients' perceptions of their own stress reactivity in daily life when resources to assess real-time responses are lacking or when such self-observations are of primary interest.

Stress reactivity is assumed to be a consequence of the individual genetic makeup and early environmental factors. Quantitative genetic studies using mainly twin designs have concluded that approximately 50% of the variance in stress responses is due to genetic factors. However, there is some variability of estimates between studies, and the amount of heritability seems to change with repeated exposure to the same stressor. Molecular genetic studies have revealed a number of single gene variants that might influence stress responses in different systems, although many of these effects of candidate genes need replication

before valid conclusions can be drawn. In addition to genetic makeup, factors of the environment early in life have been shown to influence stress reactivity. A number of studies suggest that an adverse prenatal environment might exert long-term effects on stress reactivity in the offspring. Similarly, adverse early postnatal environmental factors such as maternal care or abuse have been shown to affect stress reactivity, with consequences for the risk of mental disorder later in life. Animal studies have suggested that such early environmental effects might be mediated by epigenetic changes in specific brain areas.

Despite good evidence for the prediction of cardiovascular disease by cardiovascular stress reactivity, potential pathways and causal chains of effects are unclear. In addition, not all individuals with high cardiovascular stress reactivity later develop cardiovascular disease. This points at another factor implicated in the pathway. It is likely that high stress reactivity leads to disease particularly if a highly stress reactive individual is exposed to chronic stress in daily life (diathesis-stress model). Other areas of discussion are the potential adaptive function of high stress reactivity in an evolutionary context, and the significance of low levels of stress reactivity for the development of diseases and disorders.

Cross-References

- ▶ General Adaptation Syndrome
- ▶ Hypothalamic-Pituitary-Adrenal Axis
- ▶ Individual Differences
- ▶ Physiological Reactivity
- ▶ Stress
- ▶ Stress Responses
- ▶ Stress Test
- ▶ Stress Vulnerability Models
- ▶ Stressor
- ▶ Sympathetic Nervous System (SNS)

References and Readings

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension, 55*(4), 1026–1032.

Contrada, R. J., & Baum, A. (Eds.). (2010). *The handbook of stress science: Biology, psychology, and health*. New York: Springer.

Heim, C., & Nemeroff, C. B. (2001). The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. *Biological Psychiatry, 49*(12), 1023–1039.

Kudielka, B. M., Hellhammer, D. H., & Wust, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology, 34*(1), 2–18.

Lovallo, W. R. (2005). *Stress and health: Biological and psychological interactions* (2nd ed.). Thousand Oaks: Sage.

Manuck, S. B., & McCaffery, J. M. (2010). Genetics of stress: Gene-stress correlation and interaction. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 455–478). New York: Springer.

Myin-Germeys, I., & van Os, J. (2007). Stress-reactivity in psychosis: Evidence for an affective pathway to psychosis. *Clinical Psychology Review, 27*(4), 409–424.

Phillips, D. I. (2007). Programming of the stress response: A fundamental mechanism underlying the long-term effects of the fetal environment? *Journal of Internal Medicine, 261*(5), 453–460.

Schlotz, W., Yim, I. S., Zoccola, P. M., Jansen, L., & Schulz, P. (2011). The perceived stress reactivity scale: Measurement invariance, stability and validity in three countries. *Psychological Assessment, 23*(1), 80–94.

Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65*(1), 46–62.

Ulrich-Lai, Y. M., & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews Neuroscience, 10*(6), 397–409.

S

Stress Reduction

- ▶ Stress Management

Stress Response

- ▶ Immune Responses to Stress
- ▶ Neuroendocrine Activation
- ▶ Psychophysiologic Reactivity

Stress Responses

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Synonyms

Stress; Stress reactivity

Definition

Stress responses are psychological, physiological, and behavioral responses to an event perceived as relevant to one's well being with some potential for harm or loss and requiring adaptation. Psychological stress responses often include negative emotions, such as anxiety, distress, or anger, although positive emotional states related to feeling challenged and driven may also occur. Cognitive efforts aimed at coping with the stressor, such as planning, distancing, and/or reinterpreting, also occur (Lazarus and Folkman 1984). Physiological stress responses are often those that are in support of coping with or fleeing from the stressor, and protecting the organism from potential harm. These responses include, but are not limited to, changes in heart rate, blood pressure, cortisol, and immune function (Sapolsky 1994). Behavioral stress responses involve actions also aimed at coping with or fleeing from the stressful event, such as actively performing a task or withdrawing effort from a situation perceived as impossible (Lazarus and Folkman 1984).

Cross-References

- ▶ Coping
- ▶ Physiological Reactivity

References and Readings

Cacioppo, J. T. (1994). Social neuroscience: Autonomic, neuroendocrine, and immune responses to stress. *Psychophysiology, 31*, 113–128.
Cohen, S., Kessler, R. C., & Gordon, L. U. (1995). *Measuring stress: A guide for health and social scientists*. New York: Oxford University Press.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.
Sapolsky, R. M. (1994). *Why zebras don't get ulcers*. New York: Holt.

Stress Responsivity

- ▶ Stress Reactivity

Stress Test

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Synonyms

Mental stress task; Psychological stress task; Psychological stressor

Definition

Laboratory mental stress tasks are commonly used in behavioral medicine to assess the physiological responses to a standardized stressor in a controlled setting (Turner 1994).

Description

Even though originally it was thought that particularly exaggerated physiological responses to mental stress can be predictive of cardiovascular disease (Obrist 1981), there is now growing evidence that blunted physiological responses can

also be associated with poor health (Carroll et al. 2009). Other evidence is available that not the responses to mental stress itself but the physiological recovery upon completion of the stress task can be related with the poor health outcomes (Larsen and Cristenfeld 2011). There is a large body of research that explores the associations between psychological traits (e.g., competitiveness and hostility) and mental disorders (e.g., depression and anxiety) with the individual differences in physiological responses to mental stress (Lovallo 1997). Participants that have been included in these stress studies comprise of young healthy participants, elderly people, and a wide variety of clinical populations. This section will describe the general setup of a laboratory stress task and the most commonly used mental stress tasks in behavioral medicine. Even though the focus will be on mental stress tasks, it is worth noting that physical tests, such as exercise, tilt test, and cold pressor, are also readily used in laboratory settings.

In order to assess the physiological responses to a mental stress task, it is important to assess the resting physiological state of the participant. To quantify the stress response, a reactivity score is calculated, which is the difference between the physiological activity during the stress task and the activity during the rest period (Turner 1994). A typical stress session starts with a resting period of 15–30 min, during which the participant is relaxed. Relaxation can be facilitated by listening to music, reading magazines, or watching a low-stimulating video. This is followed by an explanation of the stress task with, where appropriate, a brief practice session and the actual stress task. Upon completion of the stress task, a recovery period is started, during which the participant is asked again to relax, similar to the baseline rest period. The duration of the recovery period is depending on the variables that are under investigation. Whereas heart rate is known to return to baseline relatively soon after the end of the stress task, changes in other variables, in particular blood-based measures such as cytokines, will not be seen until 30 min or longer (Steptoe et al. 2007). In general, physiological data collection is conducted throughout each of these periods.

When exploring the effects of individual differences in physiological responses to mental stress, it is crucial that the testing procedures are identical between participants (Turner 1994). The conditions in the laboratory, such as temperature and number of experimenters present, but also time of day, should be kept consistent. Care should also be taken to standardize the instructions of the task, which can be done by having the instructions prerecorded. Finally, adherence of the participants to the presession instructions is important. These involve most commonly avoiding strenuous exercise, food, caffeine, and smoking, as well as instructions about the medication which could influence the physiological measurements. The presession instructions are dependent on the physiological measures that are under investigation.

Mental Stress Tasks

Public Speaking – The participant is asked to give a speech in front of an audience and/or a video camera, following a brief preparation period. The topic of speech is psychologically stressful such as "pretend that you are falsely accused of shoplifting and that you have to defend yourself to the shop owner" or "describe your personal strengths and weaknesses" or "describe a recent event that caused anger." The participant will be told that the audience will be critically evaluating the content and delivery of the speech (Van Eck et al. 1996).

Mental Arithmetic – Different varieties are available for mental arithmetic tasks, which include serial subtraction or addition of double-digit numbers or serial addition of single-digit numbers with an element of retention. These tasks, even though not complicated in nature, have been developed to be provocative by adding components of increased time pressure, competition, harassment when a wrong answer is given, and social evaluation (Veldhuijzen van Zanten et al. 2004).

Trier Social Stress Test – This is a combination of mental arithmetic task followed by a public speech, all under conditions of social evaluation.

In addition to the two varieties of mental stress, this task also has a postural component as the speech is conducted while upright (Kirschbaum and Hellhammer 1993).

Computer Games – A variety of computer games have been used to induce stress in participants, which has been mainly conducted in younger participants. These tasks often have a strong component of competition; participants are either directly competing against the experimenter (often in a modified situation to standardize the success rate between participants) or competing against the other participants in the study.

Stroop Color Word Task – The participants are presented with words which describe colors, but the color of the letters is incongruent with the color that the word is written in. For example, the word red is written with yellow ink, and the word yellow is written with red ink. The participant is asked to call out the color of the ink (Stroop 1935).

Mental stress tasks are subject to the effects task novelty and habituation (Turner 1994). For example, even though all of these tasks provoke an increase of heart rate throughout the task, typically, the peak heart rate response is seen at the start of the test. Particularly when a participant is asked to complete the task on different occasions, it is important to maintain the engagement of the participant in each session. It has been shown that the addition of stressful elements such as social evaluation and competition will help to facilitate this. To ensure that the desired levels of stress are obtained, it is common practice to add a measure of self-reported perceptions of the task to each session. These can vary from a simple Likert scale related to perceived stressfulness and difficulty or measures of state stress and anxiety levels both before and after the stress task.

The stress tasks vary in terms of generalizability to real-life settings. Interestingly, an overview of various stress task revealed that public speaking tasks were most consistently effective in inducing myocardial ischemia in patients with coronary heart disease (Strike and Steptoe 2003). It is possible that this is due to the more naturalistic nature of the task than, for example, mental arithmetic or Stroop task. However, care should be taken when interpreting the effectiveness of a certain task to induce physiological changes between studies, as it is hard to compare the stressfulness of tasks between studies. Ambulatory recording techniques are available for the assessment of physiological measurements in real-life setting. Even though these field studies cannot be standardized between participants, it is worth noting that there is evidence that the laboratory cardiovascular responses to mental stress were predictive of ambulatory physiological assessments (Strike and Steptoe 2003).

Cross-References

- ▶ Cardiovascular Recovery
- ▶ Immune Responses to Stress
- ▶ Mental Stress
- ▶ Psychological Stress
- ▶ Psychophysiologic Reactivity
- ▶ Stressor
- ▶ Trier Social Stress Test

References and Further Reading

Carroll, D., Lovallo, W. R., & Phillips, A. C. (2009). Are large physiological reactions to acute psychological stress always bad for health? *Social and Personality Psychology Compass, 3*, 725–743.

Kirschbaum, C., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test' – A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76–81.

Larsen, B. A., & Cristenfeld, N. J. (2011). Cognitive distancing, cognitive restructuring, and cardiovascular recovery from stress. *Biological Psychology, 86*, 143–148.

Lovallo, W. R. (1997). *Stress & health, biological and psychological interactions*. Thousand Oaks: Sage.

Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum Press.

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*, 901–912.

Strike, P. C., & Steptoe, A. (2003). Systematic review of mental stress-induced myocardial ischaemia. *European Heart Journal, 24*, 690–703.

Stroop, J. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643–662.

Turner, J. R. (1994). *Cardiovascular reactivity and stress*. New York: Plenum Press.
Van Eck, M. M., Nicolson, N. A., Berkhof, H., & Sulon, J. (1996). Individual differences in cortisol responses to a laboratory speech task and their relationship to responses to stressful daily events. *Biological Psychology, 43*, 69–84.
Veldhuijzen van Zanten, J. J. C. S., Ring, C., Burns, V. E., Edwards, K. M., Drayson, M., & Carroll, D. (2004). Mental stress-induced hemoconcentration: Sex differences and mechanisms. *Psychophysiology, 41*, 541–551.

Stress Testing

► Exercise Testing

Stress Vulnerability Models

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Synonyms

Stress diathesis models

Definition

Vulnerability models are used to identify factors that are causally related to symptom development. Stress vulnerability models describe the relation between stress and the development of (psycho) pathology. They propose an association between (1) latent endogenous *vulnerability factors* that interact with stress to increase the adverse impact of stressful conditions, (2) *environmental factors* that influence the onset and course of (psycho) pathology, and (3) *protective factors* that buffer against or mitigate the effects of stress on pathological responses.

Description

The prevalence of stress-related mental disorders encompassing mood and anxiety disorders in Europe is above 20%. This morbidity is associated with high health-care costs, disability, and potential mortality. It is widely acknowledged that there are individual differences in how stressful people judge a particular event to be as well as in their ability to cope with adverse stressful life events. While historically stress was said to play an initiating role in the development of pathology, only a minority of people who experience adverse stressful life events go on to develop pathology. To distinguish people who develop pathology from people who do not (i.e., are resilient), vulnerability processes are suggested that predispose individuals to psychopathology when confronted with severe stressors. In the late 1970s, Zubin and Spring were the first to introduce this idea in the field of behavioral medicine by postulating a vulnerability model for schizophrenia. They suggested that humans inherit a predisposition to mental illness but that an interaction between the genetic vulnerability and biological or psychosocial stressors is necessary to develop the disorder. The relationship between predispositional factors (or diathesis) and development of pathology has been described in four basic stress vulnerability models.

Stress Vulnerability Models

The first and most simple stress vulnerability model, the *dichotomous interactive* model, suggests that when predispositional factors are absent, even severe stress will not result in pathology. Instead, it is only when predispositional factors are present that stress may, depending on the severity of the stress, lead to the expression of pathology. Alternatively, the *quasi-continuous* model suggests varying degrees of predisposition with a continuous effect of predispositional factors on pathology once a threshold has been exceeded. The third, more extensive *threshold* model incorporates a specific individual threshold that is determined by the degree of vulnerability

and the level of experienced stress. Finally, perhaps the most comprehensive model is the *risk-resilience continuum* model that incorporates different levels of severity of pathology by postulating a continuum that ranges from vulnerability to resilience. This model explicitly emphasizes resiliency characteristics that can make people more resistant to the impact of stress. Note that according to this latter model, even highly resilient individuals might still be at risk for developing pathology when experiencing extreme stress, but their individual threshold will be higher and the symptomatology of post-trauma pathology is likely to be less severe. Collectively, these four models are used to describe the relation between predispositional factors and the development of various pathologies.

Vulnerability Factors

In general, stress vulnerability models postulate that a genetic vulnerability interacts with adverse life events or stressors to produce pathology. This gene-environment interaction with regard to stress and the development of pathology has been most extensively investigated in mood disorders such as depression. Gene-environment interaction studies use monozygotic twin, adoption, and family studies as tools to identify predispositional factors in shared and non-shared environments in order to differentiate genetic from environmental influences. In twin studies, a higher prevalence of pathology in monozygotic twins reared in different environments is used to confirm a genetic predisposition, whereas in adoption studies, the effect of the environment (adoptive parents) can be offset against the effect of genes (biological parents). Using these methods the heritability of major depression, for example, has been estimated at around 40%.

At the neurochemical level, the serotonin (5-HT) system has been implicated in depression. 5-HT regulates among other mood, activity, sleep, and appetite. Accumulating evidence indicates that individuals with a serotonergic vulnerability, manifested in a more sensitive brain serotonergic system, have an increased likelihood of developing mood-related disorders. Specifically, polymorphisms in the 5-HT transporter system (5-HTT) have been associated with stressful life events, a heightened risk for depression, and reactivity to negative emotional stimuli. Individuals carrying two copies of the short variant of the 5-HTT allele (i.e., 5-HTTLPR), a less active gene resulting in fewer 5-HTT transporters, for instance, display an increased sensitivity to the impact of mild stressful life events and an excessive amygdala activity to fearful faces and produce elevated and prolonged levels of cortisol in response to a laboratory stressor compared to individuals with the long variant of the 5-HTT allele. The heritability of the stress hormone response has also been investigated with family studies in relatives of patients with depression using neuroendocrine functioning tests. For example, studies with the dexamethasone suppression test, a drug test used to measure the effectiveness of the negative feedback mechanism of the hypothalamic-pituitary-adrenal (HPA) axis at the level of the pituitary, have found an amplified set point of the HPA axis in relatives of depressed patients compared to healthy controls.

Moreover, 5-HT is also involved in the modulation of the HPA axis and its associated regulatory actions in the secretion of cortisol, the major human glucocorticoid stress hormone. Cortisol binds to two corticosteroid receptors in the brain, namely, the mineralocorticoid receptor (MR) and the glucocorticoid receptor (GR). Two mechanisms of cortisol binding are known. First, cortisol can bind to the hormone response element on DNA to influence gene expression (intracellular MR and GR binding properties). Secondly, cortisol can bind to membrane versions of the corticosteroid receptors to influence glutamate transmission and gene expression in the brain. The MR controls the basal HPA activity through inhibition of the HPA axis, facilitating the selection of adaptive behavioral responses and preventing minor adverse stressful life events to disturb homeostasis. In contrast, the GR promotes recovery after stress as well as the storage of information for future events. The balance

between the MR and GR receptors determines the threshold and termination of the HPA axis response to stress. Increased MR expression or functionality has been associated with increased adaptability to cope with stressors. For example, animal studies have demonstrated that increased MR expression contributes to a more adaptive stress response via regulation of the tonic inhibition. Moreover, MRs play a role in memory formation and retrieval during stressful situations, and impaired memory formation alters the appraisal of stressful situations. Likewise, studies have demonstrated that individuals with polymorphisms in the GR gene display higher cortisol responses and inefficient recovery of the HPA axis following standardized laboratory stress tests, thus revealing predisposition factors for stress-related pathology.

Genes can have a direct effect on the development of various brain systems. To illustrate this point, altered gene expression can reduce plasticity in brain circuits regulating mood, anxiety, and aggression, thereby decreasing one's ability to cope with stressful life events. Moreover, genes can bias brain circuits to inefficient information processing which can result in the expression of pathology (e.g., intrusive memories in patients suffering from posttraumatic stress disorder). Genetic polymorphisms are then viewed as vulnerability factors given that they produce an increased sensitivity to the impact of stressful life events. However, it should be kept in mind that replication studies of candidate gene associations in pathology are relatively sparse and that most disorders are polygenetic. Additionally, the net outcome of a stressor is at least in part determined by the individual's personality traits that may be formed by genes, potentially indirectly influencing the selection of environments and thus the risk of exposure to adverse effects.

Lifespan models have examined the relation between early-life stressful events, later stressful life events, and pathology development. Undifferentiated neuronal systems are dependent on early experience during development. It is suggested that early-life stress results in inefficient information processing and sensitization of brain circuits involved in regulating stress reactivity, which may ultimately render people more vulnerable. Different brain structures have specific developmental trajectories resulting in a variety of pathological response after stress across the lifespan. For example, prenatal stress originating from maternal stress or postnatal environmental stress such as the quality of parental care influences the regulation of the HPA axis. Specifically, different early stress experiences result in either a HPA axis hyper- or hypofunction, due to a different regulation of glucocorticoid receptors in the hippocampus. In turn, altered HPA axis activity shapes the activity in prefrontal areas as well as the connectivity between prefrontal areas and the amygdala, which in turn influences the processing of emotions and stress-coping strategies. However, exposure to a manageable stressor during childhood can also desensitize the stress circuits, producing experience-based resilience in which brain systems tend to become less reactive to future stress. Early-life stress can hence be protective in that it can negate or diminish the negative outcomes or alternatively promote adaptive functioning in the context of adverse stressful life events. Additionally, other psychosocial factors during development like social support, parental care, and affective style have been identified as potentially protective factors that can enhance adaptive coping during or after stress. In a similar vein, brain frontal alpha asymmetry has been suggested to bias individuals' affective style and emotion regulation capacities. Specifically, left frontal activation has been linked to approach behavior and suggested to be an indicator of decreased vulnerability to depression, whereas right frontal activation is viewed as a predispositional factor, lowering the threshold for adverse impact of stressful conditions.

In sum, stress vulnerability models underscore that the nature and intensity of the stressor in combination with genetic vulnerability factors, phenotypic vulnerability factors (personality, neuroendocrine reactivity), and both genetic and phenotypic protective (resilience) factors determine the impact and sequela of adverse stressful life events.

Cross-References

- Corticosteroids
- Cortisol
- Family Stress
- Family Studies (Genetics)
- Gene-Environment Interaction
- Glucocorticoids
- Hypothalamic-Pituitary-Adrenal Axis
- Individual Differences
- Quantitative EEG Including the Five Common Bandwidths (Delta, Theta, Alpha, Sigma, and Beta)
- Resilience
- Stress
- Stress Reactivity
- Stress Responses
- Stress Test
- Stress, Caregiver
- Stress, Early Life
- Stress: Appraisal and Coping
- Stressor
- Trier Social Stress Test
- Twin Studies

References and Further Reading

Coan, J. A., & Allen, J. J. B. (2003). The state and trait nature of frontal EEG asymmetry in emotion. In K. Hugdahl & R. J. Davidson (Eds.), *The asymmetrical brain* (pp. 565–616). Cambridge, MA: The MIT Press.

Curtis, W. J., & Cicchetti, D. (2003). Moving research on resilience into the 21st century: Theoretical and methodological considerations in examining the biological contributors to resilience. *Development and Psychopathology, 15*, 773–810.

DeRijk, R. H., & de Kloet, E. R. (2008). Corticosteroid receptor polymorphisms: Determinants of vulnerability and resilience. *European Journal of Pharmacology, 583*, 303–311.

Gotlib, I. H., Joormann, J., Minor, K. L., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. *Biological Psychiatry, 63*, 847–851.

Huizink, A., & De Rooij, S. (2018). Prenatal stress and models explaining risk for psychopathology revisited: Generic vulnerability and divergent pathways. *Development and Psychopathology, 30*(3), 1041–1062. https://doi.org/10.1017/S0954579418000354.

Ingram, R. E., & Luxton, D. D. (2005). Vulnerability-stress models. In B. L. Hankin & J. R. Z. Abela (Eds.), *Development of psychopathology: A vulnerability-stress perspective* (pp. 32–46). Thousand Oaks: Sage.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*, 434–445.

Oitzl, M. S., Champagne, D. L., van der Veen, R., & de Kloet, E. R. (2010). Brain development under stress: Hypotheses of glucocorticoid actions revisited. *Neuroscience and Biobehavioral Reviews, 34*, 853–866.

Stahl, S. M. (2008). *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (3rd ed.). New York: Cambridge University Press.

ter Heegde, F., De Rijk, R. H., & Vinkers, C. H. (2015). The brain mineralocorticoid receptor and stress resilience. *Psychoneuroendocrinology, 52*, 92–110.

Van Praag, H. M., de Kloet, E. R., & van Os, J. (2004). *Stress, the brain and depression*. New York: Cambridge University Press.

Zubin, J., & Spring, B. (1977). Vulnerability-a new view of schizophrenia. *Journal of Abnormal Psychology, 86*, 103–126.

Stress, Caregiver

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Synonyms

Caregiver burden; Caregiver hassle; Caregiver strain

Definition

The stress of caregivers is defined as a feeling experienced when a person thinks that the demands of caregiving exceed the personal and social resources the individual is able to mobilize (Lazarus and Folkman 1984).

Description

An illness affects not only the quality of life of individuals with the disease but also that of their family members and close friends who care for the

patients. The stress of caregivers is defined as a feeling experienced when a person thinks that the demands of caregiving exceed the personal and social resources the individual is able to mobilize (Lazarus and Folkman 1984). The caregiver role of family members incorporates diverse aspects involved in dealing with an illness of the relative. This role includes providing the patient with cognitive/informational, emotional, financial/legal, daily activity, medical, and spiritual support, as well as facilitating communication with medical professionals and other family members and assisting in the maintenance of social relationships (Kim and Given 2008). All of these aspects can contribute to caregivers' stress when they perceive it difficult to mobilize their personal and social resources to carry out each of the caregiving-related tasks. Therefore, identifying the gaps between resources available for caregiving and the caregiving demands, unmet needs in caregiving, should be the initial step in the development of programs designed to reduce caregivers' stress and enhance their quality of life.

In addition to assessing the diverse aspects of caregivers' stress and unmet needs, understanding how caregivers' stress varies across the illness trajectory is an important concern (Kim et al. 2010). For example, in the early phase of caregivership, caregivers' stress is often associated with providing informational and medical support to the patients. During the remission phase, dealing with uncertainty about the future, fear that the disease may come back, the financial burden of extended treatment needs of the patients, and changes in social relationships are major sources of caregivers' stress. After the death of the patients, spiritual concerns and psychological and physical recovery efforts from caregiving strain are the challenges caregivers face.

Another important aspect of caregivers' stress is their own unmet needs – things that are not directly related to caring for the patient but represent important personal needs to the caregivers. That is, in addition to caring for the individual with an illness, family caregivers likely have responsibilities for self-care and care for other family members that may have to be set aside or ignored in order to carry out the caregiver role.

This complex construct of caregiver stress has been associated with caregivers' demographic characteristics (Kim et al. 2010; Pinquart and Sörensen 2003, 2005). For example, younger caregivers have reported greater stress in providing psychosocial, medical, financial, and daily activity support during the early phase of the illness trajectory. During the remission years after the illness onset, however, younger caregivers have reported greater stress only in daily activity. Gender has been also an important factor (Kim and Loscalzo 2018). Female caregivers have reported greater stress from dealing with psychosocial concerns of the patients, other family members, and themselves. Ethnic minorities tend to report lower levels of psychological stress but greater levels of physical stress from caregiving. Studies have found mixed associations of caregiver stress to other demographic characteristics, such as education, income, and employment status.

Caregivers' poorer mental health has also been related to higher levels of stress from meeting the medical needs of the patients during the early phase of illness, whereas during remission, poorer mental health has been related to financial stress from caregiving. Furthermore, cancer caregivers' increased risk of poor physical health has recently been recognized by a large epidemiological study using Swedish registries (Ji et al. 2012). This study showed that spouses of persons with cancer, compared with unaffected spouses, were more vulnerable to poor cardiovascular health, including risk of coronary heart disease (13% increase) and stroke (26% increase) up to 20 years after their spouse's cancer diagnosis. Perceived level of stress from providing care has been significantly related to the caregivers' quality of life years later (Kim et al. 2015). Caregivers' depressive symptoms have also shown to be the unique predictor of physical health decline over 6 years (Shaffer et al. 2017). Caregivers who reported higher levels of psychosocial stress from caregiving have shown poorer mental health consistently and strongly across different phases of the illness trajectory.

The physical burden of caregiving, documented in objective measures, has also been

considerable. For example, compared with matched non-caregivers, caregivers for a spouse with dementia report more infectious illness episodes, have poorer immune responses to influenza virus and pneumococcal pneumonia vaccines, show slower healing for small standardized wounds, have greater depressive symptoms, and are at greater risk for coronary heart disease (Vitaliano et al. 2003). A recent meta-analysis (2003) concluded that compared with demographically similar non-caregivers, caregivers of dementia patients had a 9% greater risk of health problems, a 23% higher level of stress hormones, and a 15% poorer antibody production. Moreover, caregivers' relative risk for all-cause mortality was 63% higher than non-caregiver controls.

Immune dysregulation has been identified as a key mechanism linking caregiving stress to physical health. Chronically stressed dementia caregivers have numerous immune deficits compared to demographically matched non-caregivers, including lower T-cell proliferation, higher production of immune regulatory cytokines (interleukin-2 [IL-2], C-reactive protein [CRP], tumor necrosis factor-alpha [TNF- α], IL-10, IL-6, D-dimer), decreased antibody and virus-specific T-cell responses to influenza virus vaccination, and a shift from a Th1 to Th2 cytokine response (i.e., an increase in the percentage and total number of IL10+/CD4+ and IL10+/CD8+ cells) (Segerstrom and Miller 2004; Vitaliano et al. 2003). A 6-year longitudinal community study (Kiecolt-Glaser et al. 2003) documented that caregivers' average rate of increase in IL-6 was about four times as large as that of non-caregivers. The mean annual change in IL-6 among former caregivers did not differ from that of current caregivers, even several years after the death of the spouse. There were no systematic group differences in chronic health problems, medications, or health-relevant behaviors that might otherwise account for changes in caregivers' IL-6 levels during the 6 years of the study period (2003).

Another mechanism linking caregiving stress to poor physical health is lifestyle behaviors. Family members with chronic strain from caring for dementia patients increase health-risk behaviors, such as smoking and alcohol consumption (Carter 2002). They also get inadequate rest and inadequate exercise and forget to take prescription drugs to manage their own health conditions, resulting in poorer physical health (Beach et al. 2000; Burton et al. 1997).

In summary, caregiver stress is a multidimensional construct that varies in nature across the illness trajectory. Certain caregivers by their demographic characteristics can be identified as a vulnerable subgroup to greater caregiving stress. Overall, however, caregiving stress takes a considerable toll on the caregivers' mental and physical health. Such effects deserve further systematic study to understand their psychological, biological, and behavioral pathways.

Cross-References

- Alzheimer's Disease
- Cancer Survivorship
- Caregiver/Caregiving and Stress
- Daily Stress
- Dementia
- Family Stress
- Family, Caregiver
- Family, Relationships
- Quality of Life

References and Readings

Beach, S. R., Schulz, R., Yee, J. L., & Jackson, S. (2000). Negative and positive health effects of caring for a disabled spouse: Longitudinal findings from the caregiver health effects study. *Psychology and Aging, 15*(2), 259–271.

Burton, L. C., Newsom, J. T., Schulz, R., Hirsch, C. H., & German, P. S. (1997). Preventive health behaviors among spousal caregivers. *Preventive Medicine, 26*(2), 162–169.

Carter, P. A. (2002). Caregivers' descriptions of sleep changes and depressive symptoms. *Oncology Nursing Forum, 29*(9), 1277–1283.

Ji, J., Zöller, B., Sundquist, K., Sundquist, J. (2012) Increased risks of coronary heart disease and stroke among spousal caregivers of cancer patients. *Circulation, 125*, 1742–1747.

Kiecolt-Glaser, J. K., Preacher, K. J., MacCallum, R. C., Atkinson, C., Malarkey, W. B., & Glaser, R. (2003).

Chronic stress and age-related increases in the pro-inflammatory cytokine IL-6. *Proceedings of the National Academy of Sciences of the United States of America, 100*, 9090–9095.
Kim, Y., & Given, B. A. (2008). Quality of life of family caregivers of cancer survivors across the trajectory of the illness. *Cancer, 112*(Suppl. 11), 2556–2568.
Kim, Y., & Loscalzo, M. (Eds.). (2018). *Gender in psycho-oncology.* New York, NY: Oxford University Press. ISBN 978-0-19-046225-3.
Kim, Y., Kashy, D. A., Spillers, R. L., & Evans, T. V. (2010). Needs assessment of family caregivers of cancer survivors: Three cohorts comparison. *Psycho-Oncology, 19*, 573–582.
Kim, Y., Carver, C. S., Shaffer, K. M., Gansler, T., & Cannady, R. S. (2015). Cancer caregiving predicts physical impairments: Roles of earlier caregiving stress and being a spousal caregiver. *Cancer, 121*, 302–310.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.
Pinquart, M., & Sörensen, S. (2003). Differences between caregivers and noncaregivers in psychological health and physical health: A meta-analysis. *Psychology and Aging, 18*(2), 250–267.
Pinquart, M., & Sörensen, S. (2005). Ethnic differences in stressors, resources, and psychological outcomes of family caregiving: A meta-analysis. *The Gerontologist, 45*(1), 90–106.
Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*, 601–630.
Shaffer, K. M., Kim, Y., Carver, C. S., & Cannady, R. S. (2017). Depressive symptoms predict cancer caregivers' physical health decline. *Cancer, 123*, 4277–4285.
Vitaliano, P. P., Zhang, J., & Scanlan, J. M. (2003). Is caregiving hazardous to one's physical health? A meta-analysis. *Psychological Bulletin, 129*, 946–972.

Stress, Early Life

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Synonyms

Adversity, early life; Trauma, early life

Definition

In order to define early life stress in humans, two main criteria must be considered: (a) the developmental age range that is subsumed under "early life" and (b) the characteristics of the events that would be considered as "stressful" during early life. There is no such generally agreed upon definition (Heim et al. 2003).

Many investigators use an upper age limit to define the early life criterion, usually between 12 and 18 years. An alternative approach is to define the early life period by developmental stage, using, for example, sexual maturation (such as menarche in girls) as a cutoff criterion.

As for the stress criterion, prevailing models suggest that stress is generally experienced when an individual is confronted with a situation, which is appraised as personally threatening and for which adequate coping resources are unavailable. In addition, threats to physiological homeostasis, such as injury or illness, elicit stress responses. Any such situation occurring within the defined developmental period may be classified as early life stress. The most salient forms of early life stress in humans are abuse (sexual, physical, emotional), neglect (emotional, physical), and parental loss (death, separation). Other forms of early life stress include accidents, physical illness, surgeries, natural disasters, and war or terrorism-related events. Less obvious experiences, which pose significant distress on a child, include unstable families, inadequate parental care, dysfunctional relationships between parent and child, and poverty.

Early life stress is often complex, inasmuch as various forms coexist or are associated among each other. While early life stress may be a single event, it more typically occurs as chronic or ongoing adversity in most cases. Taken together, there remains substantial ambiguity in the definition of early life stress in humans (Heim et al. 2003).

Description

It is well established that early life stress, such as childhood abuse, neglect, or loss, dramatically

increases the risk for developing a wide range of psychiatric disorders as well as certain medical diseases later in life. Among the major psychiatric disorders, depression and anxiety disorders have been most prominently linked to early life stress. Medical disorders, for which early-life stress induces risk, include ischemic heart disease, lung disease, cancer, gastrointestinal disorders, and chronic fatigue and pain syndromes among others. Early life stress has further been linked to a variety of risk behaviors, including smoking, alcohol, or drug abuse, impulsive behavior, promiscuity, teen pregnancy, and suicide (for further reading, see Anda et al. 2006). Many of the above disorders and risk behaviors are elicited or aggravated by acute stress, and individuals with early life stress experiences have decreased thresholds to exhibit symptoms and risk behaviors even upon mild challenge (Hammen et al. 2000).

The precise mechanisms that mediate the detrimental and persistent impact of early adversity on long-term adaptation and health have been the subject of intense inquiry over decades. Advances from neuroscience research have provided compelling insights into the enormous plasticity of the developing brain as a function of experience. For example, visual sensory input early in life is required for normal development of the visual cortex and perception, and disruptive experiences during such critical periods of plasticity can lead to lifelong and sometimes irreversible damage. The same principle may be applied to stress experiences during critical periods early in life that may permanently impact on the development of brain regions implicated in the regulation of emotion and stress responses (for further reading, see Weiss and Wagner 1998). Enduring effects of early life stress on the brain and its regulatory outflow systems, including the autonomic, endocrine, and immune systems, may then lead to the development of a vulnerable phenotype with increased sensitivity to stress and risk for a range of behavioral and somatic disorders (for further reading, see Heim et al. 2004).

Compelling support for this hypothesis comes from a burgeoning literature of studies in animal models that provide the direct and causal evidence that early adverse experience, such as prolonged maternal separation or naturally occurring low maternal care, leads to structural, functional, and epigenetic changes in a connected network of brain regions that is implicated in neuroendocrine control, autonomic regulation and vigilance, and emotional regulation or fear conditioning. These neural changes converge into lifelong increased physiological and behavioral responses to subsequent stress in animal models (see Heim et al. 2004; Lupien et al. 2009; Meaney 2001). These effects appear to be present across species and in different models of adversity, while the unifying element across studies is timing of the stressor in early life. Particularly intriguing are results from animal studies suggesting that epigenetic changes, stress sensitization, and maternal care behavior are transmitted into the next generation (Francis et al. 1999; Franklin et al. 2010).

Accumulating evidence suggests that these preclinical findings can be translated to humans. For example, adult women with histories of childhood sexual or physical abuse exhibit markedly increased neuroendocrine and autonomic responses to psychosocial laboratory stress, particularly those with depression (Heim et al. 2000). Other alterations in humans with early life stress experiences include glucocorticoid resistance, increased levels of inflammation, increased central corticotropin-releasing hormone activity and decreased activity of the prosocial neuropeptide, oxytocin (Carpenter et al. 2004; Danese et al. 2007; Heim et al. 2008; Heim et al. 2009). A small hippocampus has also been linked to early life stress in humans (Vythilingam et al. 2002). Early adversity has also been found to be associated with epigenetic changes of the glucocorticoid receptor gene in hippocampal tissue obtained by postmortem from suicide victims, leading to reduced glucocorticoid receptor expression and enhanced stress responses (McGowan et al. 2008).

Taken together, these neurobiological and epigenetic changes secondary to early life stress likely reflect risk to develop depression and a

host of other disorders in response to additional challenge. In several studies, these changes were not present in depressed persons without early life stress, suggesting the existence of biologically distinguishable subtypes of depression as a function of early life stress (Heim et al. 2004, 2008). These subtypes of depression were also found to be responsive to differential treatments (Nemeroff et al. 2003). Therefore, consideration of early life stress might be critical to guide treatment decisions.

Several genes moderate the link between childhood trauma and adult risk for depression and other disorders, including the serotonin transporter, corticotropin-releasing hormone receptor 1, FK506 binding protein 5, and oxytocin receptor genes. A more recent idea is that such genetic factors might reflect general sensitivity to the environment, inasmuch as persons who are susceptible to the detrimental effects of trauma might also be particularly amenable to the beneficial effects of a positive social environment or early psychological intervention (Binder et al. 2008; Bradley et al. 2008, 2011; for further reading, see Caspi et al. 2010).

References and Readings

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., et al. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience, 256*, 174–186.

Binder, E. B., Bradley, R. G., Liu, W., Epstein, M. P., Deveau, T. C., Mercer, K. B., et al. (2008). Association of FKBP5 polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults. *Journal of the American Medical Association, 299*, 1291–1305.

Bradley, R. G., Binder, E. B., Epstein, M. P., Tang, Y., Nair, H. P., Liu, W., et al. (2008). Influence of child abuse on adult depression: Moderation by the corticotropin-releasing hormone receptor gene. *Archives of General Psychiatry, 65*, 190–200.

Bradley, B., Westen, D., Binder, E. B., Jovanovic, T., & Heim, C. (2011). Association between childhood maltreatment and adult emotional dysregulation: Moderation by oxytocin receptor gene. *Development and Psychopathology, 23*(2), 439–452.

Carpenter, L., Tyrka, A., McDougle, C. J., Malison, R. T., Owens, M. J., Nemeroff, C. B., et al. (2004). CSF corticotropin-releasing factor and perceived early-life stress in depressed patients and healthy control subjects. *Neuropsychopharmacology, 29*, 777–784.

Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *American Journal of Psychiatry, 167*, 509–527.

Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. *Proceedings of the National Academy of Sciences United States of America, 104*, 1319–1324.

Francis, D., Diorio, J., Liu, D., & Meaney, M. J. (1999). Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science, 286*, 1155–1158.

Franklin, T. B., Russig, H., Weiss, I. C., Gräff, J., Linder, N., Michalon, A., et al. (2010). Epigenetic transmission of the impact of early stress across generations. *Biological Psychiatry, 68*, 408–415.

Hammen, C., Henry, R., & Daley, S. E. (2000). Depression and sensitization to stressors among young women as a function of childhood adversity. *Journal of Consulting and Clinical Psychology, 68*, 782–787.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., et al. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association, 284*, 592–597.

Heim, C., Meinlschmidt, G., & Nemeroff, C. B. (2003). Neurobiology of early-life stress and its relationship to PTSD. *Psychiatric Annals, 33*, 1–10.

Heim, C., Plotsky, P. M., & Nemeroff, C. B. (2004). Importance of studying the contributions of early adverse experience to neurobiological findings in depression. *Neuropsychopharmacology, 29*, 641–648.

Heim, C., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B. (2008). The link between childhood trauma and depression: Insights from HPA axis studies in humans. *Psychoneuroendocrinology, 33*, 693–710.

Heim, C., Young, L. J., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B. (2009). Lower cerebrospinal fluid oxytocin concentrations in women with a history of childhood abuse. *Molecular Psychiatry, 14*, 954–958.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*, 434–445.

McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonté, B., Szyf, M., et al. (2008). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience, 12*, 342–348.

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Reviews in Neuroscience, 24*, 1161–1192.

Nemeroff, C. B., Heim, C., Thase, M. E., Klein, D. N., Rush, A. J., Schatzberg, A. F., et al. (2003). Differential responses to psychotherapy versus pharmacotherapy in patients with chronic forms of major depression and childhood trauma. *Proceedings of the National Academy of Sciences United States of America, 100*, 14293–14396.

Vythilingam, M., Heim, C., Newport, J., Miller, A. H., Anderson, E., Bronen, R., et al. (2002). Childhood trauma associated with smaller hippocampal volume in women with major depression. *American Journal of Psychiatry, 159*, 2072–2080.

Weiss, M. J., & Wagner, S. H. (1998). What explains the negative consequences of adverse childhood experiences on adult health? Insights from cognitive and neuroscience research. *American Journal of Preventive Medicine, 14*, 356–360.

Stress, Emotional

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Synonyms

Emotional distress; Mental stress; Psychological stress; Stress

Definition

Emotional stress involves the experience of negative affect, such as anxiety, in the context of a physiological stress response that includes cardiovascular and hormonal changes. Emotional stress commonly occurs when an individual perceives that he or she does not have adequate personal resources to meet situational demands effectively (Lazarus 1966).

Description

Early conceptions of stress characterized its physical properties, with a focus on the disruption of homeostasis in an organism (Selye 1956). The stress concept subsequently evolved to include a greater emphasis on the influence of psychological factors on the stress process. The term "emotional stress" reflects the fact that the stress process in humans involves a substantial affective component.

Emotional stress includes both negative affect, such as anxiety and distress, as well as a cascade of physiological responses associated with the stress-response system. Physiological responses promote "fight or flight" and include activation of the hypothalamic pituitary adrenal (HPA) axis, which stimulates secretion of cortisol, and activation of the sympathetic nervous system, which increases heart rate (Sapolsky 1994). Behavioral responses may include attempts to flee or avoid the stressor or to actively address it.

Emotional stress can be triggered by various stress exposures, including major life events, chronic stressful situations, and daily hassles. Certain objective features of a stressor influence the likelihood that it will produce emotional stress. For instance, emotional stress is more likely to result from stressors that are not within an individual's control (e.g., a death) and affect central aspects of an individual's life (Dohrenwend 2000).

Individual differences are also critical components in predicting levels of emotional stress, particularly when the stressor is not extremely traumatic. Thus, the same stressor may produce emotional stress in one individual but not in another. Richard Lazarus and Susan Folkman's work has established the importance of appraisal processes in generating or buffering against stress (Lazarus and Folkman 1984). Emotional stress results from an appraisal that the situation is threatening and that efforts to address it effectively are not likely to be successful. In contrast, a sense of positive challenge may arise if the situation is not perceived as overly threatening or if the perceiver feels capable of an effective response. Similarly, a number of other factors

can increase risk for, or protect against, emotional stress. These factors include the ability to employ effective coping strategies and the presence of positive social supports (Kessler et al. 1985).

A life course perspective is important for understanding the etiology of vulnerability to emotional stress. Both vulnerability to stress and resilience are likely shaped over the life course by complex interactions of genetic factors, biological mechanisms, and environmental exposures. Emerging research suggests that exposure to stress and adversity during sensitive periods early in the life course (prenatal, early postnatal, childhood) may be especially critical in influencing genetic expression and impacting the developing stress-response system, with long-term effects on vulnerability to emotional stress (Andersen and Teicher 2009; Dudley et al. 2011; Shonkoff et al. 2009).

A key reason for continued interest in the study of emotional stress is its well-documented link with development of both mental and physical disorders. Major depressive disorder and posttraumatic stress disorder are two commonly studied psychiatric sequelae of emotional stress. Emotional stress has also been found to predict cardiovascular disease and other physical health problems (Brotman et al. 2007; Rozanski et al. 1999). Putative mechanisms linking emotional stress with psychiatric and physical disorders include stress-related neurobiological changes (e.g., dysregulation of the HPA axis) and increased cardiovascular reactivity to stress with slow recovery (Chida and Steptoe 2010; Hammen 2005); detailed understanding of these pathways requires more study.

A variety of psychosocial stress management interventions have been developed to reduce emotional stress and prevent its negative effects on health. Such interventions generally aim to enhance positive coping methods, including the use of relaxation techniques, exercise, and cognitive strategies for managing stress. Some stress management interventions have been shown to have positive effects on emotional and physical outcomes (e.g., Blumenthal et al. 2005).

Cross-References

- ▶ Cardiovascular Disease
- ▶ Coping
- ▶ Depression: Symptoms
- ▶ Relaxation
- ▶ Stress Reactivity
- ▶ Stress Responses
- ▶ Stress Vulnerability Models
- ▶ Stress, Early Life
- ▶ Stress: Appraisal and Coping
- ▶ Stressor
- ▶ Sympathetic Nervous System (SNS)

References and Readings

Andersen, S. L., & Teicher, M. H. (2009). Desperately driven and no brakes: Developmental stress exposure and subsequent risk for substance use. *Neuroscience and Behavioral Reviews, 33*, 516–524.

Blumenthal, J. A., Sherwood, A., Babyak, M. A., Watkins, L. L., Waugh, R., Georgiades, A., et al. (2005). Effects of exercise and stress management training on markers of cardiovascular risk in patients with ischemic heart disease: A randomized controlled trial. *Journal of the American Medical Association, 293*, 1626–1634.

Brotman, D. J., Golden, S. H., & Wittstein, I. S. (2007). The cardiovascular toll of stress. *Lancet, 370*, 1089–1100.

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension, 55*, 1026–1032.

Dohrenwend, B. P. (2000). The role of adversity and stress in psychopathology: Some evidence and its implications for theory ad research. *Journal of Health and Social Behavior, 41*, 1–19.

Dudley, K. J., Li, X., Kobor, M. S., Kippin, T. E., & Bredy, T. W. (2011). Epigenetic mechanisms mediating vulnerability and resilience to psychiatric disorders. *Neuroscience and Biobehavioral Reviews, 35*, 1544–1551.

Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology, 1*, 293–319.

Kessler, R. C., Price, R. H., & Wortman, C. B. (1985). Social factors in psychopathology: Stress, coping, and coping processes. *Annual Review of Psychology, 36*, 531–572.

Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw Hill.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis

of cardiovascular disease and implications for therapy. *Circulation, 99*, 2192–2217.
Sapolsky, R. M. (1994). *Why zebras don't get ulcers*. New York: Holt.
Selye, H. (1956). *The stress of life*. New York: Mc-Graw-Hill.
Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities. Building a new framework for health promotion and disease prevention. *Journal of the American Medical Association, 301*, 2252–2259.

Stress, Exercise

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Definition

Exercise is a form of physical activity that involves repeated body movements that are both structured and planned with the intention of maintaining or enhancing one's health or physical fitness. Typically, exercise is characterized as either aerobic or anaerobic with the former emphasizing the use of oxygen for sustained movements such as jogging or swimming, whereas the latter emphasizes the use of muscle glycogen supply and metabolism as sources of energy for higher-intensity activities such as strength and resistance training. Moreover, exercise has been defined in terms of being chronic/regular/habitual or as acute/single bout.

Stress represents a response among biopsychosocial systems in an effort to adapt to a challenge. The nature of the challenge has been delineated along several dimensions, including but not limited to its intensity, duration, frequency, quality, and familiarity. The stress response has been measured in naturally occurring situations as well as in laboratory settings and ranged from self-reported questionnaires to physiological indices of neuroendocrine, heart rate, and blood pressure reactivity.

Description

The health benefits of exercise and physical fitness have been well documented in the literature. In particular, the effects of exercise on reduced morbidity and mortality from cardiovascular disease have received a great deal of attention, in part, due to the association between stress and cardiovascular disease and the potential attenuation of physiological reactivity to stressors. Notably, an acute bout of exercise may also act as a stressor and elicit the same cardiovascular and neuroendocrine responses as psychosocial stressors without the detrimental effects health. As exercise becomes chronic and habitual, the physiological adaptations (e.g., reduced heart rate and blood pressure and increased parasympathetic activity) that occur are thought to yield similar responses and adaptations in the presence of psychosocial stressors. This beneficial adaptation and reduced sensitivity to stress is characterized in the literature as the cross-stressor-adaptation hypothesis. Thus, the effect of exercise on stress response is somewhat paradoxical as it is considered to be both a stressor and a potential modifier of stress. Most investigations of exercise-stress adaptations have relied upon laboratory stressors (e.g., mental arithmetic, public speaking and evaluative scenarios, cold pressor tests, reaction time, Stroop color-word test), cardiorespiratory responses, and cross-sectional designs. Although a number of studies have examined the association between exercise/fitness and psychophysiological stress responses, the findings have been rather mixed even among the reviews and quantitative analyses of the literature.

In an initial meta-analysis of the relationship between aerobic fitness and resistance to stress reactivity by Crews and Landers (1987), the overall effect size across multiple indices was $d = 0.48$ with effects ranging from 0.15 to 0.87. The findings from this review suggested that fitness/exercise training was beneficial in reducing reactivity

to stressors with regards to heart rate d = (0.39), diastolic blood pressure d = (0.40), systolic blood pressure d = (0.42), self-reported stress d = (0.57), skin response d = (0.67), and muscle tension d = (0.87). The review has since been criticized for a number of methodological limitations including confounding reactivity with recovery. Later qualitative reviews reported no beneficial effect for fitness on stress reactivity in terms of heart rate, blood pressure, or catecholamine responses, with the effect on stress recovery determined to be inconclusive (Claytor 1991; De Geus and Van Doornen 1993). More recently, Jackson and Dishman (2006) revealed in a meta-analytic review that fitness was associated with a slight increase in stress reactivity to laboratory psychosocial stressors. Notably, a small effect size was present between cardiorespiratory fitness and stress response recovery, indicating that physically fitter individuals appeared to have an enhanced and quicker recovery following their peak stress response. In contrast to these findings, Forcier et al.'s (2006) meta-analytic review examining the effects of aerobic fitness on stress reactivity and recovery revealed that, despite considerable heterogeneity present in the analyses and no significant moderation effects (e.g., gender, stressor intensity), significant effects for exercise/fitness were found for decreased heart rate and systolic blood pressure reactivity. Additionally, a significant effect was found for fitness and heart rate recovery, though no such effect was present for systolic blood pressure. Thus, the findings from this meta-analysis suggest a beneficial attenuated physiological reactivity and improved recovery from psychosocial stressors as a consequence of fitness/chronic exercise. Although the effects between fitness and reactivity/recovery appeared small in magnitude (e.g., 1.8 bpm heart rate reactivity, 3.7 mmHg systolic blood pressure reactivity), the differences are equivalent to 15–25% reductions in reactivity which may still be of clinical importance.

Evidence from a recent meta-analysis (Hamer et al. 2006) examining blood pressure response to a psychosocial stressor suggested that an acute bout of aerobic exercise may result in a significant reduction in reactivity. That is, medium effect sizes revealed that exercise resulted in beneficial attenuated stress reactivity for both diastolic and systolic blood pressure with reductions of a 3.0 mmHg and 3.7 mmHg in diastolic and systolic responses, respectively. The observed effects appeared most robust with psychosocial stressors administered up to 30 min postexercise with moderate to vigorous exercise intensity and durations lasting from 20 min to 2 h. The findings from this review offer the possibility that the effects of acute exercise may account for some of the mixed results from studies examining chronic exercise and blood pressure reactivity because habitually exercising may place an individual in the post-exercise "window" more frequently when encountering daily stressors and thereby result in attenuated stress responses.

Although evidence from meta-analytic reviews is lacking, some studies have reported relationships between physical fitness and reduced hypothalamic-pituitary-adrenal (HPA) axis psychosocial stress reactivity, such as cortisol and inflammatory cytokine production. However, the ability to draw solid conclusions in this area is currently limited. With regard to sympathetic-adrenal-medullary responses, the findings appear conflicting as a result of sampling methodology and exercise intensity and durations employed. Some studies have found no effect of fitness on norepinephrine and epinephrine levels, whereas other studies have reported higher norepinephrine levels in the early phases of a stress response or, conversely, an association between lower levels of fitness and an increased norepinephrine response. In animal analogue studies, levels of norepinephrine have been shown to increase in the frontal cortex following an acute bout of exercise, suggesting increased vigilance to threat and reactivity; however, reduced levels have been found in the hypothalamus and hippocampus which suggests diminished stress reactivity.

In summary, although it appears that acute and chronic exercise and fitness may favorably influence an individual's response to psychosocial stress, further research is needed to clarify this relationship. Reviews of the literature suggest

that there is a need for greater methodological rigor and reporting as well as examination of potential moderators of exercise-reactivity and exercise-recovery associations.

Cross-References

- ▶ Benefits of Exercise
- ▶ Physical Activity and Health
- ▶ Stress
- ▶ Stress Reactivity
- ▶ Stressor

References and Readings

Buckworth, J., & Dishman, R. K. (2002). *Exercise psychology*. Champaign: Human Kinetics.

Claytor, R. P. (1991). Stress reactivity: Hemodynamic adjustments in trained and untrained humans. *Medicine and Science in Sports and Exercise, 23*, 873–881.

Crews, D. J., & Landers, D. M. (1987). A meta-analytic review of aerobic fitness and reactivity to psychosocial stressors. *Medicine and Science in Sports and Exercise, 19*(Suppl), S114–S120.

De Geus, E. J. C., & Van Doornen, L. J. P. (1993). The effects of fitness training on the physiological stress response. *Work and Stress, 7*, 141–159.

Edenfield, T. M., & Blumenthal, J. A. (2011). Exercise and stress reduction. In A. Baum & R. Contrada (Eds.), *The handbook of stress science: Biology, psychology, and health* (pp. 301–319). New York: Springer.

Forcier, K., Stroud, L. R., Papandonatos, G. D., Hitsman, B., Reiches, M., Krishnamoorthy, J., et al. (2006). Links between physical fitness and cardiovascular reactivity and recovery to psychological stressors: A meta-analysis. *Health Psychology, 25*, 723–739.

Hamer, M., Taylor, A., & Steptoe, A. (2006). The effect of acute aerobic exercise on stress related blood pressure responses: A systematic review and meta-analysis. *Biological Psychology, 71*, 183–190.

Hand, G. A., Phillips, K. D., & Wilson, M. A. (2006). Central regulation of stress reactivity and physical activity. In E. Acevado & P. Ekkekakis (Eds.), *Psychobiology of physical activity* (pp. 189–201). Champaign: Human Kinetics.

Jackson, E. M., & Dishman, R. K. (2006). Cardiorespiratory fitness and laboratory stress: A meta-regression analysis. *Psychophysiology, 43*, 57–72.

Sothmann, M. S., Buckworth, J., Claytor, R. P., Cox, R. H., White-Welkley, J. E., & Dishman, R. K. (1996). Exercise training and the cross-stressor adaptation hypothesis. *Exercise and Sport Sciences Reviews, 24*, 267–287.

Stress, Posttraumatic

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Synonyms

PTS

Definition

Posttraumatic stress is a stress reaction characterized by a multitude of symptoms following a traumatic event. Symptoms can be affective, behavioral, cognitive, and/or physiological in nature. These symptoms appear only after exposure to an event that involved a threat to one's physical integrity such as actual or threatened death or serious injury to oneself, witnessing such a threat on another person, or learning of such a threat experienced by a family member or other close person. A traumatic event can come in many forms including but not limited to a natural disaster (e.g., earthquake, tornado), violent personal attack, (e.g., rape, robbery), military combat, terrorist attack, severe automobile accident, or diagnosis of life-threatening illness (American Psychiatric Association [APA] 1994). Although posttraumatic stress is similar to posttraumatic stress disorder (PTSD), a diagnosable psychological condition, it is distinctly different in that the symptoms of posttraumatic stress may not be as severe or as numerous as those of PTSD. Thus, not all of the criteria necessary for diagnosis of PTSD are met, and the symptoms typically do not cause clinically significant distress or impairment in important areas of functioning.

Description

Rates of trauma exposure are unclear, but it is estimated that a majority of people, over 60%, living within the United States have experienced

at least one traumatic event (Resick et al. 2008). Some of those affected will develop no signs of distress, whereas some will develop clinically significant, diagnosable signs of distress, and yet others, people experiencing posttraumatic stress, can be categorized as developing subclinical levels of distress. Like many other psychological difficulties, whether or not someone develops significant signs of distress after a traumatic event depends on many variables such as the individual's coping abilities, trauma history, support system, type of traumatic event, intensity of the event, proximity to the event, and so on. Individuals with diminished coping abilities, extensive trauma histories, weak support systems, and who experience more intense and physical traumas closer to their bodies will be at greater risk for suffering from more severe posttraumatic distress (APA 1994; Resick et al. 2008). Since the subclinical population does not warrant a diagnosis and most likely does not seek treatment due to the lack of or low level of impairment, it is difficult to estimate how many are afflicted by posttraumatic stress. However, individuals living with posttraumatic stress experience various difficulties, which can be categorized into groups of affective, behavioral, cognitive, and physiological symptoms.

Affective Symptoms

After a traumatic event, one can feel a variety of stressful emotions as a consequence for days, weeks, or even months after the experience. Intense fear and helplessness are characteristic markers of posttraumatic stress (APA 1994; Beidel and Stipelman 2007; Resick et al. 2008). A person can feel scared that the event might reoccur at any moment and horror that he or she will be unable to stop it from happening again. One might fear for their life and worry that death or severe injury is imminent, creating constant anxiety, and perhaps a sense of a foreshortened future (APA 1994; Beidel and Stipelman, 2007). Some people may react to a traumatic incident with guilt and/or shame, unwilling to talk to others about the incident believing that they are at fault for the event. Sadness and even depression can also arise as a result from experiencing a trauma. This can lead to no longer finding pleasure in activities that the person once enjoyed or in diminished social involvement (Beidel and Stipelman 2007). Lastly, an individual may experience a feeling of anger, manifested through irritability or outbursts (APA 1994). This anger may be focused on the event itself; it may also be geared toward others, perhaps blaming others for the occurrence of the event or fueled by mistrust of others. Overall, the affective symptoms that result from posttraumatic stress are typically negative and can become harmful if they linger for too long.

Behavioral Symptoms

Certain behaviors that were not present before a traumatic event can also surface as a result. Hypervigilance, a constant watchful eye on one's surroundings, is a hallmark behavior used to protect oneself from unexpected threats. This hypervigilance includes heightened sensory sensitivity with an intense, somewhat irrational reaction or an exaggerated startle response (APA 1994; Beidel and Stipelman 2007). For example, an individual who has personally experienced an earthquake may become more aware of slight tremors, flickering lights, rattling windows, or rumbling sounds that others do not notice. These stimuli might arouse fear or anxiety in the individual who might feel as if they are reexperiencing the traumatic event. A person may also purposely work toward not reexperiencing or remembering the event. He or she might avoid certain places, people, smells, sounds, topics of conversation, or anything else that might trigger memories of the incident and may even be unable to recall certain aspects of the event altogether (Beidel and Stipelman 2007; Resick et al. 2008). One might also learn to numb their feelings so that they no longer experience fear, anxiety, or anger as a protective measure from the various affective symptoms that may have developed after the trauma. However, this typically also leads to numbing positive affect such as joy and excitement as well, restricting one's range of affect (APA 1994; Beidel and Stipelman 2007). Avoidance and numbing can further cause a person to become somewhat reclusive and decrease involvement in social situations, resulting in a feeling of detachment or estrangement from friends and family. In

general, the behavioral symptoms that appear after a traumatic event are used as coping mechanisms to either protect oneself to keep such an event from reoccurring or to distance oneself from the event that has occurred.

Cognitive Symptoms

There are key cognitions that are representative of those individuals who have experienced a trauma. Most often, there is an overgeneralization of the event and the harm that it caused (Resick et al. 2008). In other words, an individual might believe that harmful or deadly events happen more often and in more places than previously thought. He or she might feel that he or she is always at risk and lacks safety, misappraising minor events or stimuli as much more dangerous than they really are. A victim might lose trust in others and think that others are out to harm him or her, particularly if another human being caused the trauma. Other distorted cognitions about power and control may surface as well (Resick et al.). Such beliefs might be that one has no control or power over events in their daily life, perhaps lowering self-esteem. Another distortion might be self-blame where the victim believes he or she had complete control and power in the situation, bringing the traumatic event upon him or herself. This can result in a feeling of shame or guilt.

Although cognitive symptoms usually only include cognitions or beliefs that a person holds, in the case of posttraumatic stress, they also include ideas, images, and impulses that might materialize in one's mind after a traumatic event. Individuals might have sudden, intrusive memories of images, smells, sounds, etc., of the event flash in his or her mind (APA 1994). The victim might also have distressing dreams about the incident or feel and act as if he or she is reliving the terrible moment (Resick et al. 2008). Due to the many stressors experienced after a traumatic event, concentration may be difficult for individuals with posttraumatic stress.

Physiological Symptoms

Posttraumatic stress causes physiological changes as well. Most notably, there is an increase in heart rate and sweat gland activity. Levels of cortisol may also rise. These symptoms may lead to exhaustion or various serious illnesses such as heart disease (Resick et al. 2008; Sarafino 2008).

Summary

Posttraumatic stress is a psychological stress reaction following a traumatic event. It is characterized by a multitude of affective, behavioral, cognitive, and physiological symptoms that negatively impact an individual. However, the symptoms are not severe enough to cause impairment in social, occupational, or other areas of functioning and therefore do not warrant a diagnosis. Nevertheless, if symptoms become more severe and do not diminish within a month's time, they may be a sign of a diagnosable psychological condition, posttraumatic stress disorder. In such a case, the individual should seek treatment from a professional psychologist or counselor.

Cross-References

- ▶ Post Traumatic Stress Disorder
- ▶ Posttraumatic Growth
- ▶ Stress

References and Readings

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Beidel, D. C., & Stipelman, B. (2007). Anxiety disorders, chapter 11. In M. Hersen, S. M. Turner, & D. C. Beidel (Eds.), *Adult psychopathology and diagnosis* (5th ed., pp. 349–409). Hoboken: Wiley.

Resick, P. A., Monson, C. M., & Rizvi, S. L. (2008). Posstraumatic stress disorder, chapter 2. In D. H. Barlow (Ed.), *Clinical handbook of psychological disorders: A step-by-step treatment manual* (4th ed., pp. 65–122). New York: Guilford Press.

Sarafino, E. P. (2008). Stress – Its meaning, impact, and sources, chapter 3. In E. P. Sarafino (Ed.), *Healh psychology: Biopsychosocial interactions* (6th ed., pp. 61–86). Hoboken: Wiley.

Stress: Appraisal and Coping

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Definition

Stress has been defined traditionally either as a *stimulus*, often referred to as a *stressor*, that happens to the person such as a laboratory shock or loss of a job, or as a *response* characterized by physiological arousal and negative affect, especially anxiety. In his 1966 book, *Psychological Stress and the Coping Process* (Lazarus 1966), Richard Lazarus defined stress as a relationship between the person and the environment that is appraised as personally significant and as taxing or exceeding resources for coping. This definition is the foundation of stress and coping theory (Lazarus and Folkman 1984).

Description

Stress and coping theory provides a framework that is useful for formulating and testing hypotheses about the stress process and its relation to physical and mental health. The framework emphasizes the importance of two processes, appraisal and coping, as mediators of the ongoing relationship between the person and the environment. Stress and coping theory is relevant to the stress process as it is experienced in the ordinary events of daily life, major life events, and chronic stressful conditions that stretch out over years.

Appraisal refers to the individual's continuous evaluation of how things are going in relation to his or her personal goals, values, and beliefs. Primary appraisal asks "Am I okay?" Secondary appraisal asks "What can I do?" Situations that signal harm or potential harm that is personally significant and in which there are few options for controlling what happens are appraised as stressful. Stress appraisals include harm or loss, which refer to damage already done; appraisals of threat, which refer to the judgment that something bad might happen; and appraisals of challenge, which refer to something that may happen that offers the opportunity for mastery or gain as well as some risk of an unwelcome outcome. Situations that are appraised as high in personal significance and low in controllability, for example, are usually appraised as threats, and situations that are high in personal significance and high in controllability are more likely to be appraised as challenges.

The concept of appraisal addresses the issue of variability of responses among people experiencing a similar stressor and why a given situation may be more stressful for one person than another. The situation may involve goals, values, or beliefs that are more personally significant for one person than for another, or one person may be better equipped than another to control the situation's outcome. Appraisal-based approaches now dominate the field (Pearlin et al. 1981).

Appraisals generate emotions that vary in quality and intensity according to the person's evaluation of personal significance (primary appraisal) and options for coping (secondary appraisal). Threat appraisals, for example, are often accompanied by fear, anxiety, and worry; harm/loss appraisals are often accompanied by anger, sadness, or guilt; and challenge appraisals are often accompanied by eagerness and excitement as well as a touch of threat.

People experience a complex array of emotions during real-life stressful events, including positive as well as negative emotions (Folkman 1997, 2008). Emotions indicate that something is happening that matters to the individual. Emotions also often signal what the person intends to do. Negative emotions have long been associated with the individual's preparation to approach or avoid, fight or flee (Lazarus 1991). Positive emotions have more recently been examined for their roles in the stress process. Positive emotions, for example, are associated with widened focus of attention, motivating meaning-focused coping, and eliciting social support (Folkman 2008; Fredrickson 1998).

Coping refers to the thoughts and actions people use to manage distress (emotion-focused

coping), manage the problem causing the distress (problem-focused coping), and sustain positive well-being (meaning-focused coping). Emotion-focused coping includes strategies such as distancing, humor, and seeking social support that are generally considered adaptive, and strategies such as escape-avoidance, day dreaming, and blaming others that are generally considered maladaptive. Problem-focused coping includes strategies such as information gathering, seeking advice, drawing on previous experience, negotiating, and problem solving. Meaning-focused coping includes strategies such as focusing on deeply held values, beliefs, and goals; reframing or reappraising situations in positive ways; and amplifying positive moments over the course of a day (Folkman and Moskowitz 2000).

Coping is influenced by the person's coping resources including psychological, spiritual, social, environmental, and material resources, and by the nature of the situation, especially whether its outcome is controllable or has to be accepted. Problem-focused coping is used more in situations that are controllable, and emotion-focused coping is used more in situations that have to be accepted. Meaning-focused coping appears to be used more in situations that are chronic and not resolvable, such as in caregiving or serious illness. It is hypothesized that meaning-focused coping becomes more active when initial coping efforts fail to make the situation better (Folkman 2011). Meaning-focused coping sustains other coping efforts and restores coping resources.

People use an array of coping strategies in real-life situations. Most situations involve more than one coping task or goal, each of which requires a coping strategy tailored to that task or goal. And people switch coping strategies when the ones they are using do not have the desired effect. Coping also changes as an encounter unfolds in response to changes in the environment, the situation, or to changes within the person.

Coping effectiveness is determined contextually because effective coping in one situation may be ineffective in another. For instance, distancing may be ineffective when a person should be problem solving or preparing for an upcoming challenge, whereas it may be effective when there is nothing to be done, as when waiting for a test result. Researchers often identify on an a priori basis the outcome that is desired, such as improved mood. In such cases, effective coping is the coping that is associated with the desired outcome.

Another approach to evaluating coping is to examine the goodness of the fit between the appraised options for coping and the choice of coping strategy. Problem-focused coping that is used when the situation is appraised as controllable, for example, would be a good fit, whereas the same form of coping in situation where nothing can be done would be a poor fit. Conversely, distancing that is used when there is nothing that can be done would be a good fit, whereas the same form of coping in a controllable situation that called for attention would be a poor fit.

Like appraisal, coping is key to understanding why the outcomes of given stressful situations can vary from person to person. Two people may cope quite differently with the same stressful situation because of differences in their resources, experiences, motivation, preferences, and skills for coping.

The dynamic quality of the stress process is evident in changes in the appraisal and reappraisal process, the fluidity of emotions, and changes in coping thoughts and actions as an encounter unfolds. The processes are also in reciprocal relationships. An outcome of appraisal and coping at Time 1, such as mood, for example, can become a predictor of appraisal and coping at Time 2.

References and Readings

Folkman, S. (1997). Positive psychological states and coping with severe stress. *Social Science and Medicine, 45*, 1207–1221.

Folkman, S. (2008). The case for positive emotions in the stress process. *Anxiety, Stress, and Coping, 21*, 3–14.

Folkman, S. (Ed.). (2011). *The Oxford handbook of stress, health, and coping*. New York: Oxford University Press.

Folkman, S., & Moskowitz, J. T. (2000). Positive affect and the other side of coping. *American Psychologist, 55*, 647–654.

Fredrickson, B. L. (1998). What good are positive emotions? *Review of General Psychology Special Issue: New Directions in Research on Emotion, 2*, 300–319.
Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw Hill.
Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
Pearlin, L. I., Lieberman, M. A., Menaghan, E. G., & Mullan, J. T. (1981). The stress process. *Journal of Health and Social Behavior, 22*, 337–356.

Stress-Associated Disorders

► Stress-Related Disorders

Stressful Life Event

► Psychosocial Factors and Traumatic Events

Stressful Life Events

► Life Events

Stress-Induced Asthma

► Asthma and Stress

Stress-Induced Disorders

► Stress-Related Disorders

Stressor

► Psychological Stress

Stress-Related Disorders

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Synonyms

Stress-associated disorders; Stress-induced disorders

Definition

The term "stress-related disorders" is used in various ways in the scientific literature. A narrow definition includes conditions that are clearly *caused* by stress. Consequentially, merely a handful of (mental) disorders (e.g., posttraumatic stress disorder) are deemed eligible for this label. By contrast, a broad definition includes all illnesses that are somehow adversely *impacted* by stress. This can mean that stress is involved in the predisposition, precipitation, and/or perpetuation of a particular condition. Given that stress exerts negative effects on numerous bodily systems (e.g., the autonomic nervous system, the endocrine system, and the immune system) and on health behaviors (e.g., eating, physical activity, substance use), virtually all mental disorders and a large proportion of somatic diseases can be classified as stress-related disorders according to this definition.

Description

The continuum between conditions in which stress is a causal versus a contributing factor is reflected by the various diagnostic codes for stress-related disorders. In the 11th edition of the International Classification of Diseases (ICD-11; WHO 2018b), stress is most prominently mentioned in "disorders specifically associated with stress" within the

mental disorders chapter. This category includes adjustment disorder and posttraumatic stress disorder (PTSD), among other disorders that are triggered by trauma, critical life events, or chronic stress, and is analogous to the category of "trauma- and stressor-related disorders" in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; APA 2013). In addition, stress is featured as a general "factor influencing health status" at the very end of the ICD-11 and as a "psychological factor affecting disorders or diseases classified elsewhere" within the chapter on mental disorders. These codes are usually used in somatic diseases in which stress is a risk or exacerbating factor (e.g., in cardiovascular or inflammatory diseases).

Stress-related symptoms are highly diverse. When stress is the main factor involved in the development of a particular illness (e.g., in PTSD or adjustment disorder), typical symptoms include depressed mood, anhedonia, problems in concentration, anxiety, irritability, and sleep disturbance. When stress is one among several factors contributing to the development and/or maintenance of a condition (e.g., in somatic symptom disorder or hypertension), any number of these symptoms may be found, in addition to the unique conglomerate of symptoms pertaining to the disease/disorder in question. However, it is often difficult to establish a clear distinction between stress-related symptoms and symptoms of other origins (e.g., a genetic abnormality), especially as stress may exacerbate almost any symptom via its negative effects on stress-responsive bodily systems (e.g., impaired immune functioning) and lifestyle behaviors (e.g., physical inactivity, smoking).

The contribution of stress-related disorders to the global burden of disease is high. Posttraumatic stress disorder alone has a lifetime prevalence of 3.9% (Koenen et al. 2017), and diseases that are adversely affected by stress are among the most debilitating conditions worldwide. According to the WHO (2018a), the top five current causes of disability-adjusted life years (DALYs) and death are ischemic heart disease, stroke, lower respiratory infection, chronic obstructive pulmonary disease (COPD), and road injury. Of these, all three noncommunicable diseases, that is, ischemic heart disease, stroke, and COPD, have direct and indirect links to stress. For instance, a meta-analysis of over 500,000 individuals showed that long working hours (i.e., more than 55 h per week) were directly associated with a significantly increased risk of both ischemic heart disease and stroke (Kivimaki et al., 2015). This can be attributed to physical inactivity and repeated activation of the stress response. Similarly, an extensive meta-analysis confirmed that smoking, which is often used as a means to cope with stress, is a major risk factor for COPD, causing airway obstruction, mucus hypersecretion, and emphysema (Forey et al. 2011).

Depending on the role of stress in the development and/or maintenance of a particular disorder, and depending on the type of stressor involved, different interventions can be applied to alleviate symptom burden. When stress is causal and traumatic (e.g., a rape victim developing PTSD), trauma-focused cognitive behavioral therapy (CBT) should be applied. This treatment comprises prolonged exposure to traumatic cues or memories as well as cognitive restructuring of dysfunctional trauma-related beliefs. In cases where stress is causal, but non-traumatic (e.g., a critical life event or chronic stress triggering an adjustment disorder), the choice of psychological intervention will depend on whether a patient shows symptoms of depression, anxiety, or disturbance of conduct. In the case of depression, behavioral activation or sleep hygiene may prove to be effective, whereas anxiety may best be managed by relaxation exercises or cognitive restructuring, and patients with disturbance of conduct may benefit from social skills training. Finally, when (chronic) stress is a contributing factor, stress management techniques may be a worthy addition to pharmacological or other somatic therapies (e.g., antidepressants for major depressive disorder, surgery after myocardial infarction). Such programs either target the stressor itself (e.g., effort-reward imbalance at work); the individual appraisal of potentially stressful situations, including personality traits that foster stress (e.g., perfectionism); or the immediate consequences of stress (e.g., elevated sympathetic activity, smoking as a coping strategy).

Cross-References

- ▶ Autonomic Nervous System (ANS)
- ▶ Cardiovascular Disease
- ▶ Chronic Obstructive Pulmonary Disease
- ▶ Cognitive Behavioral Therapy (CBT)
- ▶ Cognitive Restructuring
- ▶ Coping
- ▶ Health Behaviors
- ▶ Hypertension
- ▶ Ischemic Heart Disease
- ▶ Physical Activity
- ▶ Physical Inactivity
- ▶ Post Traumatic Stress Disorder
- ▶ Relaxation Techniques
- ▶ Smoking
- ▶ Stress
- ▶ Stress Disorder
- ▶ Stress Management
- ▶ Stress, Posttraumatic
- ▶ Stress Response

References and Further Readings

APA. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington D.C.: APA.

Forey, B. A., Thornton, A. J., & Lee, P. N. (2011). Systematic review with meta-analysis of the epidemiological evidence relating smoking to COPD, chronic bronchitis and emphysema. *BMC Pulmonary Medicine, 11*, 36. https://doi.org/10.1186/1471-2466-11-36.

Kivimaki, M., Jokela, M., Nyberg, S. T., Singh-Manoux, A., Fransson, E. I., Alfredsson, L., et al. (2015). Long working hours and risk of coronary heart disease and stroke: A systematic review and meta-analysis of published and unpublished data for 603,838 individuals. *Lancet, 386*(10005), 1739–1746. https://doi.org/10.1016/S0140-6736(15)60295-1.

Koenen, K. C., Ratanatharathorn, A., Ng, L., McLaughlin, K. A., Bromet, E. J., Stein, D. J., et al. (2017). Posttraumatic stress disorder in the world mental health surveys. *Psychological Medicine, 47*(13), 2260–2274. https://doi.org/10.1017/S0033291717000708.

WHO. (2018a). *Global Health estimates 2016: Deaths by cause, age, sex, by country and by region* (pp. 2000–2016). Geneva: WHO.

WHO. (2018b). International statistical classification of diseases and related health problems (11th revision). Retrieved from https://icd.who.int/browse11/l-m/en.

Stress-Related Growth

- ▶ Benefit Finding
- ▶ Perceived Benefits
- ▶ Posttraumatic Growth

Stroke Burden

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Definition

Description

Strokes are one of the leading causes of death and disability: each year, nearly 800,000 Americans experience a new or recurrent stroke. Approximately 600,000 of these are first events and roughly 180,000 are recurrent attacks. In the United States, strokes accounted for 1 out of every 16 deaths in 2004, and more than 50% of the deaths attributable to strokes occurred outside of the hospital. When separated from other cardiovascular diseases, stroke is the third leading cause of death, behind heart disease and cancer. Importantly, strides have been made in reduction of stroke mortality: the stroke death rate has fallen more than 20% from 1994 to 2004. However, important disparities remain. While Hispanic, Latino, American Indian, and Pacific Islander populations have somewhat lower stroke death rates than whites, black men and women continue to have significantly higher stroke death rates than all other populations, and the prevalence of stroke remains higher in minority populations than among whites. Lastly, because women live longer than men, in 2004, women accounted for greater than 60% of stroke deaths in the United States.

In addition to the significant mortality, the morbidity associated with stroke is considerable: more than 25% of stroke survivors older than age 65 are disabled 6 months later. The length of time to recover from a stroke depends on its initial severity. From 50% to 70% of stroke survivors

regain functional independence, but 15–30% are permanently disabled, and 20% require institutional care at 3 months after onset. Although 70% of strokes are a first cardiovascular event, 15% of survivors will have a recurrent event within 1 year, and 30% will have a recurrent event within 5 years. The period soon after an acute stroke is associated with the highest rate of stroke recurrence, and the risk of stroke following a transient ischemic attack (TIA) is over 10% for the following 90 days.

The medical costs of stroke are high: the estimated direct and indirect costs of stroke for 2008 are $65.5 billion USD. In comparison, the 2008 costs for coronary heart disease were estimated at $156.4 billion USD, making stroke one of the leading US health-care expenditures. The mean lifetime cost of ischemic stroke is over $140,000 USD, and 70% of the first-year costs following an acute stroke are due to inpatient hospital costs.

Finally, the burden associated with stroke contains an important association with other cardiovascular disease processes and risk. Many risk factors for stroke overlap with those of cardiac and peripheral vascular disease. This overlap has led to the concept of "global risk" for cardiovascular disease in general, of which stroke is one component. High blood pressure is the greatest risk factor for stroke, but factors like smoking, diabetes, and low high-density lipoprotein levels are important risk factors. Depression has been suggested to be an independent risk factor for stroke. The occurrence of a stroke is therefore likely to be the initial manifestation of a global cardiovascular disease process, with high morbidity, mortality, and cost, and risk factors that overlap with traditional cardiovascular risk factor categories.

References and Readings

Asplund, K., Stegmayr, B., & Peltonen, M. (1998). From the twentieth to the twenty-first century: A public health perspective on stroke. In M. D. Ginsberg & J. Bogousslavsky (Eds.), *Cerebrovascular disease pathophysiology, diagnosis, and management* (Vol. 2). Malden: Blackwell. Chap 64.

Rosamond, W. D., Folsom, A. R., Chambless, L. E., Wang, C. H., McGovern, P. G., Howard, G., Copper, L. S., & Shahar, E. (1999). Stroke incidence and survival among middle-aged adults: 9-year follow-up of the Atherosclerotic Risk in Communities (ARIC) cohort. *Stroke, 30*, 736–743.

Rosamond, W., Flegal, K., Furie, K., et al. (2008). Heart disease and stroke statistics-2008 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation, 117*, e25–e146.

Stroop Color-Word Test

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Synonyms

Mental stressor; Problem solving

Definition

The Stroop test is commonly used in psychophysiological studies as a problem-solving task to elicit mental stress. The test is an incongruent task that requires participants to identify the name of a color (e.g., "blue," "green," or "red") that is printed in a conflicting color not denoted by the name (e.g., the word "red" printed in blue ink instead of red ink). The task primarily evokes beta-adrenergically driven responses, resulting in increased heart rate and cardiac output. Functional neuroimaging studies of the Stroop effect have consistently revealed activation in the frontal lobe and more specifically in the anterior cingulate cortex and dorsolateral prefrontal cortex, two structures hypothesized to be responsible for conflict monitoring and resolution.

Cross-References

► Blood Pressure Reactivity or Responses
► Heart Rate

Structural Equation Modeling (SEM)

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Synonyms

SEM

Definition

Structural equation modeling (SEM) is a multivariate statistical methodology for the estimation of a system of simultaneous linear equations that may include both observed and latent variables. With origins in the path analysis work of the biometrician Sewell Wright and the factor analysis tradition of Charles Spearman, over the past 40 years, SEM has transitioned from a novel methodology for linear models to a mainstream statistical framework for the analysis of latent variable models. SEM-related techniques may be used to examine a wide variety of structures, including causal models, measurement models, growth models, latent classes or mixtures, and combinations of these. The generality and flexibility of SEM, the development of efficient estimation methods, and the availability of computer programs contribute to the utility of this methodology for addressing important research questions in behavioral medicine.

Description

The first step in an SEM analysis is the specification of the model. SEM models are specified via either a system of structural equations or, more commonly, a path diagram. Included in the path diagram are the observed variables to be analyzed, the constructs or latent variables to be inferred, the unobserved but ever-present errors or disturbances, and the ways in which these observed and unobserved variables are related to one another. The path diagram is a valuable tool in itself, helping investigators better understand their research questions and their data. A path diagram forces the investigator to think critically about every variable relevant to the phenomenon under study.

Observed variables (also called indicators) are the variables we measure directly. These could be exogenous (variables whose causes are not included in the model) or endogenous (variables whose causes are posited in the model). *Path analysis* is a special case of SEM in which all variables specified in a model are observed (except for the errors) and assumed to be perfectly reliable. However, when reliability is not perfect and indicators contain measurement error, as is often the case, parameter estimates may be biased. One of the key features of SEM is the possibility of combining multiple observed variables into *latent variables* (the constructs of interest). A latent variable, like a construct, is not observed directly, but is rather inferred from the covariances shared among its corresponding indicators. For example, we could combine multiple measures of depression into a latent variable to improve reliability. Thus, a general SEM model may be viewed as having two components: a structural model (which allows for the specification and testing of relationships among variables) and a measurement model (which offers the advantage of using latent variables to represent constructs of interest, modeling sources of measurement error and bias associated with directly observed variables).

Generally speaking, the purpose of analyzing SEM models is twofold. First, we wish to test whether the specified model fits the data. Second and simultaneously, we want to estimate the parameters of interest and test them for significance. The most common *method of estimation* used by available computer programs is maximum likelihood (ML), performed iteratively to arrive at an admissible solution. ML parameter estimates are unbiased, consistent, efficient, and normally distributed in large samples. Given a particular model specification, this method is used to generate and compare a model-implied

variance-covariance matrix to the data-based variance-covariance matrix. ML estimates are those that minimize the discrepancy between those two matrices, and as such, yield parameter values that have the greatest likelihood of having given rise to the sample values obtained, assuming a multivariate normal distribution. The typical output from a computer analysis will have indices of model fit, as well as the parameter estimates, their standard errors, and *z*-values used to test them for significance.

The primary index used to test model fit is a χ^2 statistic. However, given this statistic's direct dependence on sample size, with large sample sizes, even small differences between the two matrices may yield a significant χ^2 indicative of poor model fit. Several other indices have been developed and proposed as either alternatives or companions to the χ^2 in such cases, including the comparative fit index (CFI), the root mean squared error of approximation (RMSEA), and the standardized root mean residual (SRMR). Beyond overall measures of fit, it is important to make sure that parameter estimates make sense in relation to the problem being investigated.

In terms of the structural aspect of SEM, the *parameters of primary interest* are the path coefficients (or the direct effects among the variables). In SEM, the variances and covariances among the exogenous variables are also estimated, as well as the variances and covariances of the disturbances. In terms of the measurement aspect of SEM, the parameters of primary interest are the factor (or latent variable) loadings, the measurement error variances, and the variances and covariances of the latent variables.

When working within the SEM framework using ML estimation, it is possible to take advantage of its full information capabilities to include all of the available data. Often referred to as full information maximum likelihood (FIML), this approach to *missing data* has been shown to yield unbiased parameter estimates when missingness is related to variables that are accessible for analysis (Little and Rubin 2002; Schafer and Graham 2002). Comparable to multiple imputation, this method is superior to other missing data techniques such as listwise or pairwise deletion, or mean, regression, or hotdeck imputation, particularly when data are not missing completely at random (Enders 2006).

SEM is a large sample methodology and the appropriate sample size must be considered keeping in mind several issues including model complexity, estimation method, and statistical power. With samples less than 100 participants, models must be simple and the variables normally distributed, otherwise problems with model convergence are likely to arise. As a general rule, more complex models or non-normal data will require more participants.

Of note, testing of *alternative models* is necessary to strengthen the causal inferences often associated with SEM. Sometimes researchers improperly assume that models that fit the data represent reality, without recognizing there are always multiple alternative models that fit just as well. Models can be rejected but not proven. It is important to consider design features such as randomization, experimentation, longitudinal designs, instrumental variables, or inclusion of other variables to strengthen model interpretation.

In addition to testing relationships among variables, means and mean structures may be analyzed. For example, multiple groups may be compared with respect to means of latent variables, or mean changes over time may be modeled using *latent growth modeling*. Many other extensions are possible but their description is beyond the scope of this entry. However, this is an active area of research and readers are encouraged to learn more by consulting available textbooks, Web sites, and papers. A very readable conceptual introduction to SEM is provided by Kline (2010). A more detailed presentation of its use in behavioral medicine may be found in Llabre (2010). The Web site for Mplus (Muthen and Muthen 2011) – one of the more popular computer programs – contains a lot of useful information on the basics of SEM, as well as extensions to more complex models.

Cross-References

- ▶ Hierarchical Linear Modeling (HLM)
- ▶ Latent Variable
- ▶ Missing Data
- ▶ Randomization

References and Readings

Enders, C. K. (2006). Analyzing structural equation models with missing data. In G. R. Hancock & R. O. Mueller (Eds.), *Structural equation modeling: A second course* (pp. 313–344). Greenwich: Information Age Publishing.

Kline, R. (2010). *Principles and practice of structural equation modeling* (2nd ed.). New York: Guilford Press.

Little, R. J., & Rubin, D. B. (2002). *Statistical analysis with missing data* (2nd ed.). Hoboken: Wiley.

Llabre, M. M. (2010). Structural equation modeling in behavioral medicine research. In A. Steptoe (Ed.), *Handbook of behavioral medicine: Methods and applications* (pp. 895–908). New York: Springer.

Muthen, L., & Muthen, B. (1998–2011). *Mplus user's guide*. Los Angeles: Muthen & Muthen.

Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of art. *Psychological Methods, 7*, 147–177.

Structural Variant

- ▶ Copy Number Variant (CNV)

Structured Clinical Interview for DSM-IV (SCID)

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Definition

The structured clinical interview for DSM-4 (SCID) is a semistructured interview created to make reliable psychiatric diagnoses in adults according to the *Diagnostic and Statistical Manual*, fourth edition (DSM-IV). The SCID has two parts: one for DSM-IV Axis I disorders (SCID-I) and another for DSM-IV Axis II personality disorders (SCID-II).

In order to meet different needs, the SCID-I is available in two versions: the research version (SCID-I-RV; First et al. 2002) and the clinician version (SCID-CV; First et al. 1996). In contrast to the SCID-CV, the SCID-I-RV comprises more disorders, subtypes, severity, and course specifiers and is easier to modify. The SCID-I-RV itself is also available in different versions. The broadest SCID-I-RV version comprises ten self-contained diagnostic modules: mood episodes, psychotic and associated symptoms, psychotic disorders, mood disorders, substance use disorders, anxiety disorders, somatoform disorders, eating disorders, adjustment disorders, and an optional module which allows psychiatric diagnoses that may be of the interviewer's interest, such as the module on acute stress disorder and on minor depressive disorder.

The SCID-I starts with an open-ended overview that includes questions about demographic information, work history, chief complaint, past and present periods of psychopathology, treatment history, and current functioning. This is followed by the diagnostic modules, which are presented in a three-column format: the left-hand column contains the interview questions, the middle column contains the corresponding DSM-IV criteria, and in the right-hand column ratings for the criteria are indicated. Besides rating the presence of the DSM-IV criteria for Axis I disorders, the SCID-I also enables rating of Axis III, IV, and IV of the DSM (see DSM-IV for more details).

The SCID-II (First et al. 1997) is only offered in a single version. It covers the ten standard DSM-IV Axis II personality disorders (avoidant, dependent, obsessive-compulsive, paranoid, schizotypal, schizoid, histrionic, narcissistic, borderline, antisocial personality disorder), as well as personality disorder not otherwise specified, and the appendix categories

depressive personality disorder and passive-aggressive personality disorder. The item format and the conventions of the SCID-II are very similar to those of the SCID-I. The SCID-II consists of several questions organized in sections in accordance with the DSM-IV diagnoses for personality disorders. In most cases, the questions correspond accurately with the criteria. To shorten overall administration time, the SCID-II is also provided with a self-report screening questionnaire that is intended to be administered at first. After this questionnaire has been filled out, only those items indicating personality abnormalities need to be inquired in more detail during the interview.

The SCID-II is often used in conjunction with the SCID-I. While administration of SCID-I typically takes between 45 and 90 min, the complete administration time of the SCID-II usually lasts about 1 h. Ideally, the SCID is administered by a trained interviewer familiar with the diagnostic criteria used in the DSM-IV. The SCID can be used in both healthy individuals and psychiatric patients. In individuals with either severe psychotic symptoms or severe cognitive impairments, the administration of the SCID is not recommended.

Overall, the SCID is a widely used assessment tool in both research and clinical settings in many countries. Various versions of the SCID have been translated into multiple languages, including Mandarin, Spanish, French, and German. The psychometric properties of the SCID-I and the SCID-II have been evaluated in several adult populations in numerous investigations, with encouraging results for most Axis I and Axis II disorders (e.g., Lobbestael et al. 2010). Computer-assisted versions of the SCID are also available.

Notably, since October 2015 the Structured Clinical Interview for DSM-5 diagnoses (SCID-5) has been available in English. The SCID-5 can be ordered in two versions: SCID-5-CV (clinician version; comparable with SCID-CV) and SCID-5-PD (personality disorders; comparable with SKID-II). For more information on the SCID-5, the reader is referred to the American Psychiatric Association Publishing website.

References and Further Reading

https://www.appi.org/scid5

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text revision.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured clinical interview for DSM-IV axis I disorders, clinician version (SCID-CV)*. Washington, DC: American Psychiatric Press.

First, M. B., Gibbon, M., Spitzer, R. L., Williams, J. B. W., & Benjamin, L. S. (1997). *Structured clinical interview for DSM-IV axis II personality disorders, (SCID-II)*. Washington, DC: American Psychiatric Press.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV-TR axis I disorders, research version, patient edition. (SCID-I/P)*. New York: Biometrics Research, New York State Psychiatric Institute.

Lobbestael, J., Leurgans, M., & Arntz, A. (2010). Inter-rater reliability of the structured clinical interview for DSM-IV axis I disorders (SCID I) and axis II disorders (SCID II). *Clinical Psychology & Psychotherapy, 18*(1), 75–79. https://doi.org/10.1002/cpp.693.

Study Methodology

► Research Methodology

Study Protocol

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Definition

The study protocol is “the most important document in clinical trials, since it ensures the quality and integrity of the clinical investigation in terms of its planning, execution, conduct, and the analysis of the data” (Chow and Chang 2007). The study protocol is a comprehensive plan of action that contains information concerning the goals of the study, details of subject recruitment, details of safety monitoring, and all aspects of design,

methodology, and analysis. (In some cases, a Statistical Analysis Plan, associated with and written at the same time as the study protocol, will contain the detailed description of the analyses to be conducted.)

Description

The creation of study protocol requires input from many individuals. Consider a study protocol for a pharmaceutical clinical trial, since this is likely to be more extensive (and complex) than some smaller trials for behavioral medicine interventions and treatments. By considering this more extensive version, one will be able to judge which parts are and are not needed on a case by case basis.

In the case of a pharmaceutical trial, input will be needed from clinical scientists, medical safety officers, study managers, data managers, and statisticians. Consequently, while one clinical scientist or medical writer may take primary responsibility for the protocol's preparation, many members of the study team make important contributions.

The requirements of a study protocol include:

- Objectives (usually primary and secondary objectives). These goals of the study are stated as precisely as possible.
- Measurements related to the drug's safety, and procedures to ensure the safety of all subjects while participating in the trial.
- Inclusion and exclusion criteria. These provide detailed criteria for subject eligibility for participation in the trial.
- Details of the procedures for physical examinations.
- Laboratory procedures. Full details of the nature and timing of all procedures and tests are provided.
- Electrocardiogram (ECG) measurement and any other measurements such as imaging.
- Drug treatment schedule. Route of administration, dosage, and dosing regimen are detailed. This information is also provided for the control treatment.
- In the case of later-phase trials, measurements of efficacy. The criteria to be used to determine efficacy are provided.
- In the case of later-phase trials, details of the method of diagnosis of the disease or condition of clinical concern for which the drug is intended.
- Statistical analysis. The precise analytical strategy needs to be detailed, here and/or in an associated statistical analysis plan.

Inclusion and exclusion criteria are central components of clinical trials. A study's inclusion and exclusion criteria govern which individuals interested in participating in the trial are admitted to the study as subjects. Criteria for inclusion in the study include items such as the following:

- Reliable evidence of a diagnosis of the disease or condition of clinical concern.
- Being within a specified age range.
- Willingness to take measures to prevent pregnancy during the course of treatment. This includes a female in the trial not becoming pregnant (she may be receiving the drug being tested), and a male participating in the trial not causing a female to become pregnant (he may be receiving the drug being tested).

Criteria for exclusion from the study may include:

- Taking certain medications for other medical conditions and which therefore cannot safely be stopped during the trial.
- Participation in another clinical trial within so many months prior to the commencement of this study.
- Liver or kidney disease.

While inclusion and exclusion criteria are typically provided in two separate lists in regulatory documentation, exclusion criteria can be regarded as further refinements of the inclusion criteria. Meeting all the inclusion criteria allows a person to be considered as a study participant, while not meeting any exclusion criteria is also necessary to allow the person to become a participant.

Cross-References

▶ Informed Consent

References and Further Reading

Chow, S.-C., & Chang, M. (2007). *Adaptive design methods in clinical trials: Concepts and methodologies*. Boca Raton: CRC/Taylor Francis.

Study Size

▶ Sample Size Estimation

Subethnic Groups

▶ Ethnicity
▶ Minority Subgroups

Subgroup Heterogeneity

▶ Minority Subgroups

Subject Characteristics

▶ Demographics

Subjective Well-Being

▶ Happiness and Health

Submissiveness

▶ Interpersonal Circumplex

Substance Abuse

▶ Alcohol Abuse and Dependence
▶ Dependence, Drug
▶ Lifestyle Changes

Substance Abuse: Treatment

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Synonyms

Addiction rehabilitation; Chemical dependency treatment; Drug abuse: treatment; Drug and alcohol treatment; Drug dependence treatment; Drug rehabilitation; Inpatient treatment; Outpatient treatment; Residential treatment

Definition

Treatment refers to a defined, empirically evaluated, data-based intervention intended to manage, remediate, or cure a diagnosed condition, here, impairing or problematic drug use. Historically, there have been two frameworks underpinning substance use treatment, the "disease" and "learning/conditioning" models (Higgins 1997). While some assumptions are divergent and perhaps irreconcilable, a broad integrative view dictates that genetic and other biological factors interact with behavior and environmental factors as composite determinants of substance use disorders. The diagnostic criteria applied to determining need for treatment are found in the International Statistical Classification of Diseases and Related Health Problems (ICD) 10 (*"Mental and behavioral disorders due to psychoactive substance use"–"a wide variety of disorders that differ in severity and clinical form but that are all attributable to the use of one or more psychoactive substances, which may or may not have been*

medically prescribed") and The American Psychiatric Association Diagnostic and Statistical Manual (DSM) IV, (*"Substance Related Disorders"–"The Substance-Related Disorders include disorders related to the taking of a drug of abuse (including alcohol), to the side effects of a medication, and to toxin exposure"*). The intervention may include behavioral, psychological, social, and pharmacological components delivered by skilled practitioners. There is a wide range of self-help and other efforts with little or no documented efficacy that are beyond the purview of this entry as are a number of criminal justice–based interventions.

Description

Underlying Disciplines, Principles, Goals, and Focus

In the public and lay domain, putative treatments for substance use disorders (i.e., "drug abuse" or "addiction") are legend and varied in their underpinnings. However, the core principles for systematic treatment of substance use disorders reside in empirically based interventions of psychology, psychiatry, pharmacology, behavioral science, and neuroscience. Despite diverse theoretical, conceptual, and terminological differences, there are many commonalities in both practice and behaviors and thoughts that are the focus of treatment. Emphasis is typically placed on avoiding drug use–related circumstances (physical or social) and replacing drug-seeking, drug use, and drug-related thoughts with other behaviors (e.g., coping skills, problem-solving skills). The interventions focus on altering biology, behavior, and social interaction through behavioral/psychological and pharmacological techniques. Longer term pharmacotherapy focuses on substituting a therapeutic medication (e.g., methadone) at controlled doses for the drug used (opiate) or a medication blocking (e.g., naltrexone) the effects of the drug used (opiate) with untoward consequences. Each strategy produces blunting or elimination subjective drug effects. Short-term alleviation of withdrawal symptoms or disturbed behavior is achieved with specific symptomatic treatment (e.g., anxiolytics, sedatives, antidepressants, antipsychotics).

Two important considerations in discussion of treatment are the determinants and correlates of the disorders and the not uncommon existence of co-occurring psychiatric/behavioral or medical problems. These may predate or be a consequence of the substance use disorder(s). The most common comorbid condition for treatment is multiple drug use (e.g., cocaine, heroin, alcohol). Next, the psychological/psychiatric diagnoses of problematic drug use or dependence are not uncommon among individuals also diagnosed for schizophrenia, depression, posttraumatic stress disorder, etc. Indeed, observation of common symptoms associated with extreme drug use, for example, disorganization, paranoid ideation, hallucinations, may reveal existence of the other psychiatric condition. Increasing awareness of co-occurrence of other psychiatric conditions with substance use disorders has resulted in extensive research, discussion, and review of conceptualization of treatment. For example, should specialized single interventions be applied to each presenting condition or should integrated treatments be devised? Questions arise as to correlation, association, and causation. Did the substance use cause psychiatric illness or precipitate its onset and reveal its existence or a predisposition? Did the psychiatric illness predispose to drug use, perhaps as a means to self-medicate the underlying condition? Finally, in instances of severe drug use, medical consequences (e.g., respiratory depression, cardiovascular event, accident-induced trauma) rather than psychiatric symptoms may lead to identification of problematic drug use and need for treatment.

Critical in establishing treatment strategies and regimens is understanding that the substance use disorder is the consequence of multiple determinants, some amenable to manipulation or intervention, others not. As with forms of some other common conditions, for example, hypertension, diabetes, obesity, a complex interplay of genetic, biological, behavioral, social, and other environmental factors contributes to the observed disease. However, limits must exist on diagnosis and treatment. Thus, while some patients present with

reasonably stable life styles, educational backgrounds, and work histories, others using the same drugs may have limited education and skills. In both cases, systematic focus on diagnosed conditions is essential. However, the myriad collateral circumstances are the purview of other domains (e.g., social service networks, job counselors, educational systems), which can be addressed more effectively, for example, by adept case managers and other experts, rather than health care providers. The two historical models continue to influence the perspective and goals of treatment. Underpinning an integrative behavioral learning perspective is the concept that just as one learns other behaviors (reading, using the internet, sports), one learns to use drugs. The behavior is maintained by biological, behavioral, and social rewards or reinforcers. The treatment strategy is establishment or learning of other behaviors sustained by non-drug reinforcers, while drug seeking and taking are diminished or eliminated. The disease model assumes a "chronic relapsing disease" evidenced when a predisposed individual is exposed to and begins using psychoactive drugs; complete and perpetual abstinence is the goal and relapse is always imminent (Higgins 1997). To the extent that biology underlies behavior, the integrated learning model accounts for genetic and biological differences but focuses on new behaviors and reinforcers rather than the chronicity of disease.

Treatment Settings, Dose, and Costs

Distinct from the intervention is the setting or environment in which treatment is provided. Settings include outpatient (e.g., office, clinics, emergency rooms), inpatient (e.g., hospital facilities), and residential (e.g., community-like living arrangements). The opportunity for access to patients is increased in controlled residential settings and some supplement the professionally driven therapy with a variety of "self-help" activities such as group discussions. Still others may have options for attending work or school. However, many of the same fundamental interventions can be applied in any of these settings. For example, a course of cognitive behavior therapy could be applied weekly in a mental health care practitioner's office, an inpatient facility, a residential care setting, or for that matter a prison. Another dimension of "setting" is whether care is provided to a single patient by a therapist, or with a group of patients, having the therapist interacting with individual members and also facilitating discussion among members. Attempts to match individuals to particular settings for treatment have been flawed and generally ineffective. For example, no data point to advantages of inpatient compared to outpatient care, regardless of severity for treatment of the behavior of substance use itself.

Similarly distinct is the actual amount or "dose" of intervention. Inpatient settings in which the patient resides continuously for days or weeks do not necessarily deliver more, or more efficacious therapeutic care than treatment delivered to a patient residing at home and functioning in her or his natural environment while receiving office-based care a few hours a week. For example, while inpatient or residential care may shield a patient from access to drugs, including alcohol and nicotine, it also prevents them from having exposure to the very environments in which it will be necessary to engage in life without problematic drug use; this exposure is both essential and therapeutic. As an aside, inpatient and residential care are extremely costly strategies that are well beyond the resources of most individuals and, with increasing health cost burdens for society, will be of diminishing importance except in unusual circumstances.

Evaluation and Monitoring

Drug use severity, as determined by substance used, frequency and amount of use, may be critical to determining treatment required. Thorough diagnostics and history determination permit tailoring treatment. An interview method termed Timeline Follow Back (Sobell et al. 1986) permits careful structuring of a history of use. The accepted diagnostic interviews conforming to elements of the DSM or ICD criteria are necessary to determine severity, existence of comorbid conditions, and plausible treatment plans. A collateral finding in many studies of therapeutic interventions for substance use disorders is that

individuals presenting with less severe conditions are more successful in treatment. For example, individuals who on entry for evaluation have negative biological screening tests for the drug used (see below) are more likely to continue successfully for a full course of treatment. Comorbid conditions and individual differences in a range of social and environmental circumstances must always be considered.

Both at intake and during treatment, objective measures of determining current use are essential. For alcohol use, this may be a breath test or urine screen. Tobacco smoking can be readily determined using CO monitoring while other tobacco use can be monitored with saliva or urine screens for cotinine, primary metabolite of nicotine. Virtually all other drug use can be monitored with simple and widely available urine screen procedures and this is essential, much as regular blood pressure evaluation is critical in treatment of hypertension or glucose level monitoring is critical to diabetes treatment.

Interventions: Behavioral, Pharmacological, and Combined

Behavioral therapy/psychotherapy and pharmacotherapy are the two broad classes of intervention applied to psychiatric disorders including substance use disorders. Current science points to remarkably effective interventions for specific types of substance use disorders, limited efficacy for some forms of substance use disorders, and for others, little efficacy or even exacerbation of drug use (e.g., olanzapine and cocaine use).

Some therapies are designed primarily to promote understanding of an existing problem or motivate entry into more extended or intensive treatment (e.g., motivational enhancement therapy). Other treatments are intended to provide a systematic course of intervention addressing the gamut of problems linked to substance use disorder and cessation of use (e.g., cognitive behavior therapy, contingency management). Still other interventions are composites with elements incorporated from a number of conceptual and pragmatic frameworks (e.g., community reinforcement approach, matrix model).

There is a clear need to distinguish between treatment of acute drug-related symptoms, notably withdrawal syndromes, and the complex behaviors of drug seeking and drug taking. A patient can achieve a "drug free" state within days by enforced abstinence. Sleep patterns and other biological functions and a variety of disrupted behaviors may stabilize within days or weeks. This normalization does not directly address the problems of drug seeking and drug use; however, for a small minority of individuals, a period of abstinence/cessation, however achieved, may be sufficient. Even this observation may be confounded since these individuals often undergo repeated cessation attempts before achieving behavioral change. For most, some supplemental intervention, ranging from brief, structured therapies to multiple structured sessions, will be required at some point in the drug use career to attain meaningful beneficial outcomes. Indeed, some scientists conceptualize substance use disorders as chronic for many individuals, requiring ongoing treatment and support, similar to obesity (McLellan 2002).

There are several behavioral, or psychotherapy, interventions for which strong supporting data of efficacy exist. One is cognitive behavior therapy; another is contingency management sometimes applied within the broader community-based reinforcement approach; and a third encompassing a range of theoretical frameworks entails variants of "talk therapy." Components or variants exist with labels such as "the matrix model" (Shoptaw et al. 1994), "relapse prevention," and "dialectical behavior therapy." Diverse techniques (e.g., hypnotism, acupuncture) have been incorporated into treatment models with little evidence of added efficacy compared to well-constructed behavioral therapies. All effective non-pharmacological therapies ultimately focus on behavior and thought directed at minimizing or eliminating drug use while increasing the frequency of a range of socially appropriate constructive behaviors. Among adults, these goals are more readily achieved when the individual arrives at therapy with an age and ability appropriate set of social skills, training, job history, and experience. Still, there are times when

additional focused therapeutic elements may be included, for example, parenting or marital counseling.

In the realm of pharmacotherapy, opiate replacement therapy (methadone, buprenorphine) provides a clear example of robust and effective treatment applicable to use and dependence ranging from iv heroin use to oral oxycodone use. Some promising data suggest potential efficacy of a similar replacement, or agonist-like strategy for stimulant dependence though there are currently no FDA-approved medications. Possible substitute medications for stimulant dependence include amphetamine analogs. Similarly, while there are currently no approved medications for treatment of marijuana use and dependence, a promising agent that represents substitution or replacement, tetrahydrocannabinol/cannabidiol, is currently being evaluated. A variety of strategies addressing different behaviors and pharmacological mechanisms have been applied to alcohol use (disulfiram, benzodiazepines, naltrexone, acamprosate) with greater or lesser efficacy. Similarly, several medications (nicotine preparations, bupropion, varenicline) have been shown to have varying degrees, but nevertheless modest effectiveness in treatment of tobacco use and nicotine dependence. There are several nicotine preparations (gum or polacrilex, lozenges, nasal spray, patches, inhalers) that alone produce relatively modest rates of cessation across broad populations but have meaningful public health consequences due to the prevalence of tobacco use and the costs of associated diseases. Disulfiram for alcohol dependence has a unique profile: through metabolic interference, it results in high levels of acetaldehyde and induced severe discomfort; integrated into a well-controlled regimen, it can be very effective but is not widely used. Naltrexone and acamprosate appear to have modest effects in reducing alcohol intake or sustaining abstinence. For individuals whose alcohol use is found to be highly correlated with diagnoses of comorbid depression or anxiety, effective treatment may stem from administration of anxiolytics or antidepressants. In some instances, use of two or more medications may be a useful therapeutic approach. A dilemma across most medical and psychiatric is nonadherence or noncompliance with medication regimens. This is particularly true for antagonist medications such as disulfiram and naltrexone.

Ultimately, for many instances of substance use disorders, benefit of joint action of concurrently applied behavioral and pharmacological interventions may be important in all but the least severe cases. This is analogous to treatment of other disorders with clear biological and behavioral determinants. For example, hypertension may be treated with combined behavioral (e.g., exercise, diet) and pharmacological (e.g., ACE inhibitors) interventions. Similarly, depression, while amenable to both behavioral and pharmacological treatments, may be most effectively treated with a combination (e.g., CBT and SSRIs). The need for continuing intervention in treatment of substance use disorders may vary. For example, pharmacotherapy with an effective course of behavior therapy establishing (or reestablishing) alternative behaviors and eliminating drug use may still entail long-term maintenance pharmacotherapy. The latter may be necessitated by biological perturbations that predated or were a consequence of drug use. Here continued medication sustains biological and behavioral stability that would otherwise not be achieved or would only continue with unnecessary behavioral, emotional, or biological burden on the patient. Exemplifying this is treatment of heroin dependence. On completing initial behavioral treatment combined with methadone or buprenorphine maintenance, some patients may successfully undergo gradual reduction in medication dose. In other instances, perhaps more severe and involving many years of heroin use, maintenance pharmacotherapy for years or decades is desirable and warranted. The need for maintenance may reside in engrained perturbations in biology and behavior, resulting from prolonged heroin use. Generally, the costs and inconvenience of maintenance replacement are outweighed by nearly inevitable return to drug dependence and associated dire psychosocial and medical consequences when abstinence without further treatment is undertaken.

Often discussed is the concept of “relapse,” “return to drug use,” or in some instances, “development of new drug use.” Early in treatment

(often within days but up to 3–6 months), there may be graded but rapid return to previous baseline levels of use. Some individuals may use a drug a few times, for example, smoke a cigarette, or consume alcohol. They may even engage in periodic use, heavy use, or a binge, but through sustained therapy eventually refrain from further use. The likelihood of return to use greatly decreases after a year. Confounding these general observations are individuals who enter treatment and never use the drug again as well as those who are abstinent for years and then resume use. In some cases, individuals treated for heavy use, for example, heavy alcohol drinking, may resume nominal social use without return to heavy drinking. Here the legal status of a drug is germane. Treatment with a goal of abstinence from cocaine or heroin use has positive implications beyond those related to reducing impairment from drug effects, for example, no risk of incarceration. For those engaged in heavy alcohol use, reduced use may be a realistic, achievable, beneficial goal. However, as with cigarette smoking, the entrenched habitual behavior combined with reinforcing effects of the drug may preclude a goal of moderation. Finally, a small subset of individuals who have well-established behaviors of problematic use may be successfully treated for use of one drug, but at some later date develop similarly problematic use of a different drug. This would reflect reestablishment of drug seeking and taking, but not "relapse" as the word is commonly used. As with any complex disorder with multiple determinants, the core constellation of symptoms, here drug seeking and drug taking, can be addressed directly with supplemental specialty intervention when appropriate.

References and Readings

Higgins, S. T. (1997). Applying learning and conditioning theory to the treatment of alcohol and cocaine abuse. In B. Johnson & J. Roache (Eds.), *Drug addiction and its treatment* (pp. 367–386). Philadelphia: Lippencott-Raven.

McLellan, A. T. (2002). Have we evaluated addiction treatment correctly? Implications from a chronic care perspective. *Addiction, 97*(3), 249–252.

Shoptaw, S., Rawson, R. A., McCann, M. J., & Obert, J. L. (1994). The matrix model of outpatient stimulant abuse treatment: Evidence of efficacy. *Journal of Addictive Diseases, 13*(4), 129–141.

Sobell, M. B., Sobell, L. C., Klajner, F., Pavan, D., & Basian, E. (1986). The reliability of a timeline method for assessing normal drinker college students' recent drinking history: Utility for alcohol research. *Addictive Behaviors, 11*(2), 149–161.

Substance Dependence

▶ Alcohol Abuse and Dependence

Substance H

▶ Histamine

Substance Use Disorders

▶ Dependence, Drug

Success

▶ Attribution Theory

Successful Aging

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Synonyms

Optimal aging

Definition

Successful aging has been addressed and discussed repeatedly with at least 29 different

definitions articulated from both cross-sectional and longitudinal research studies. Common themes across all of this work describe successful aging as the absence of physical and mental disability. Generally, successful aging is noted to occur when the individual has perceived "good health."

Description

Successful Aging

The concept of successful aging emerged in the late 1980s and early 1990s as a departure from the loss-focused geriatric and gerontological research that preceded the concept. In their groundbreaking 1987 article, Human Aging: Usual and Successful, Rowe and Kahn argued that the cognitive and physiological losses documented in the literature as age-related changes were mischaracterizations of the natural aging process. "We believe that the role of aging per se in these losses has often been overstated and that a major component of many age-associated declines can be explained in terms of lifestyle, habits, diet, and an array of psychosocial factors extrinsic to the aging process."

Definition

Successful aging has been addressed and there are at least 29 different definitions articulated from both cross-sectional and longitudinal research studies. Common themes across all of this work consider successful aging as the absence of physical and mental disability and perceived "good health." In addition, successful aging has increasingly been associated with resilience and maintenance of an active lifestyle and having good supportive relationships. Everyone has the opportunity to age successfully. From the outside, objectively, this may look very different across people. For some, aging successfully is still working at age 98. For others, it is sitting quietly in a room in assisted living and reliving past memories reviewing very rich, fulfilling lives.

More important than the absolute state of health of the individual is the notion of one's conceptualization and acceptance of his or her health status. Acceptance of changes and optimization of physical and mental health are the most critical aspects of aging successfully. Changes that occur as part of normal aging must be accepted, addressed, and adjusted to. Vision changes that occur starting around age 40 provide the first experience adults have to cope with and age successfully. Successful adaptation includes buying reading glasses over the counter as needed or getting eye examinations and vision testing done and getting new glasses! Increasingly baby boomers carry small lights with magnification to read restaurant menus, telephone books, and other pertinent information. Other changes, such as painful degenerative joint disease, are not so easily or commonly recognized, accepted, and adjusted to. It is being able to accept and adjust to changes that are at the core of successful aging.

As noted, resilience is central to successful aging. The word "resilience" comes from the Latin world "salire," which means to spring up and the word "resilire" means to spring back. Resilience, therefore, refers to the capacity to spring back from a physical, emotional, financial, or social challenge. Being resilient indicates that the individual has the human ability to adapt in the face of tragedy, trauma, adversity, hardship, and ongoing significant life stressors. Resilient individuals are able to adapt to all types of situations, especially with regard to social functioning, morale, and somatic health, and are less likely to succumb to illness. That is, a resilient individual may still get ill but will respond in a way in which he or she optimizes recovery, maintenance, or even death in a way that defines resilience. Resilience, as a component of the individual's personality, develops and changes over time through ongoing experiences with the physical and social environment. Resilience can, therefore, be perceived as a dynamic process that is influenced by life events and challenges. Increasingly, there is evidence that resilience is related to motivation, specifically the motivation to age successfully and to recover from physical or psychological traumatic events.

Resilience research has helped to uncover the many factors or qualities within individuals that are associated with resilience. These include such things as positive interpersonal relationships, incorporating social connectedness with a willingness to extend oneself to others, strong internal

resources, having an optimistic or positive affect, keeping things in perspective, setting goals and taking steps to achieve those goals, high self-esteem, high self-efficacy, determination, and spirituality which includes purpose of life, religiousness or a belief in a higher power, creativity, humor, and a sense of curiosity. Strengthening any of these factors will help individuals to become more resilient when faced with a challenge.

Older women who have successfully recovered from orthopedic or other stressful events describe themselves as resilient and determined and tend to have better function, mood, and quality of life than those who are less resilient. These are the women who at 97 engage in rehabilitation services post–hip fracture to their fullest ability and then following discharge home are insistent on getting back on their exercise equipment at the gym. Resilience has also been associated with adjustments following the diagnosis of dementia, widowhood, management of chronic pain, and overall adjustment to the stressors associated with aging. Thus, it is through resilience he or she adjusts, adapts, and addresses the physical, emotional, and mental challenges that are to be anticipated as one ages.

Older adults accrue a lifetime of experiences through which resilience develops. Resilient individuals generally age successfully. A resilient older adult is exemplified by such things as accepting the loss of a spouse; symptoms associated with an acute medical event; or a fracture and responding with determination to recover and grow through the experience. He or she does not waste energy on complaining about what has happened but rather on pooling the resources needed to overcome the challenging event.

Resilience alone is not sufficient to assure successful aging. It is believed that all individuals have the innate ability to be resilient and return to homeostasis successfully and to transform, change, and grow, regardless of age. He or she must, however, summon motivation in the face of adversity to be resilient. Thus, motivation may be present independent of resilience, but resilience depends on being motivated to successfully reintegrate. Resilient reintegration requires increased energy, or motivation, for resilience to successfully occur.

Motivation comes from within and is based on an inner urge that moves or prompts a person to action. This is in contrast to resilience which is stimulated in response to adversity or challenge. Motivation refers to the need, drive, or desire to act in a certain way to achieve a certain end. We do not need to be challenged to be motivated. We do have to be motivated to respond, with resilience, to a challenging event. There are some factors that are associated with both resilience and motivation such as determination, self-efficacy, being open and willing to experience new things, and social supports. The capacity to be resilient and/or motivated is present in everyone and choices are made in the face of routine and challenging situations to be motivated and/or resilient. Motivation related to engaging in physical activities may be high for some individuals while others are motivated to sit in a chair or lie in a bed. Conversely, some older adults are motivated to take classes in a senior center while others refuse to even consider this and are motivated to sit daily and watch television alone. Some individuals are resilient with regard to physical challenges but cannot cope with challenges associated with finances or cognitive changes. Thus, there are traits and characteristics of individuals associated with resilience and motivation as well as external factors that can impact resilience and motivation as individuals respond to challenges or activities within their lives.

Resilience, unlike motivation, relies on the individual experiencing a life challenge or some type of adversity. These challenges may be developmental challenges such as those associated with normal aging (e.g., vision changes), or they maybe social and/or economic challenges such as those experienced by the loss of employment, the loss of a spouse, or a move into an assisted living facility. Conversely, motivation is not dependent on an adverse event or challenge; rather motivation is a necessary component for all activity. Routine personal care activities such as bathing and dressing require motivation, as do making plans to have dinner with a friend or play cards. Resilience would be required, however, when he or she is faced with bathing and dressing challenges following a wrist fracture.

Keys to Aging Well

An individual has the ability to be resilient as long as he or she is motivated to do so. The first step in the process is to engage in appropriate health promotion activities, particularly exercise. Due to the changes that can occur with aging, underlying physical capability will vary among older individuals. For some individuals, maintaining a regular exercise program will involve running for 40 min on the treadmill. For others, it may be walking within their apartment building or long-term care facility, doing a sitting exercise program, or swimming or walking in the water. There is, however, an exercise program that matches each individual's needs. This is critically important to appreciate and recognize. The benefit of physical activity for older adults should not focus on preventing cardiovascular disease and managing blood pressure to prevent a stroke. Rather, it should be geared toward the mental health benefits associated with physical activity as well as the sense of achievement and physical benefit of maintaining function and improving balance and strength.

Successful Aging Guidelines

Meta-analytic reviews provide strong evidence to support the many benefits of exercise including decreased risk of coronary heart disease and stroke; decreased progression of degenerative joint disease; prevention of osteoporosis of the lumbar spine; decreased incidences of falls; increased gait speed if the activity is of sufficient intensity and dosage; improved cognitive function in sedentary older adults and in those with dementia; a modest benefit in quality of life for frail older adults; and a positive association with successful aging. Current guidelines from the American College of Sports Medicine and the American Heart Association recommend that all older adults engage in moderately intense aerobic exercise for 30 min daily at least 5 days a week or vigorous exercise 20 min a day, 3 days a week. In addition, each should do eight to ten strength training exercises, 10–15 repetitions of each exercise two to three times per week. These exercises are well described in a book published by the National Institute of Aging, *Exercise: A Guide from the National Institute of Aging*. For older adults at risk for falling, balance exercise is also recommended. It is not known, however, what dose of exercise is needed for each individual to age successfully. For some, a walk to get the mail is sufficient psychologically to constitute an exercise program. For others, one activity a day (e.g., playing bridge, going to dinner) makes them feel successful and engaged. The focus should be on helping the individual understand what successful aging means to them and helping them develop a plan to achieve that.

Unfortunately, all too many older adults assume that they cannot exercise or engage actively in routine life activities and social activities because of underlying disease, pain, shortness of breath, or other limiting symptoms. Health outcomes can be achieved at even relatively low levels of exercise intensity, particularly for those who have previously been sedentary. Thus, initiation of physical activity even at 1-min intervals is an important step to successful aging. Adjustments can be made to engage them in social activities via chair positioning that will help them maintain independence in public or remain comfortable to sit for a game of bridge or an evening eating with friends. Solving these challenges is part of the necessary resilience needed to age successfully. Health-care providers can serve as sources to help with overcoming such challenges and barriers.

In addition to physical activity, mental stimulation may also be important for successful aging. In a recent meta-analysis, there was not statistical support to indicate that cognitive stimulation through structured programs prevents or slows the progression of Alzheimer's disease. The current research, however, is limited by a lack of consensus on what constitutes the most effective type of cognitive training, insufficient follow-up times, a lack of matched active controls, and few outcome measures showing changes in daily functioning, global cognitive skills, or a decrease in disease progression. Keeping actively engaged mentally through volunteer work or activities within one's own living space certainly will build resilience and likewise assure successful aging. Opportunities abound for such activities,

however, as with physical activity the older individual must be motivated to initiate and engage in these activities.

Older adults should be encouraged to move beyond their level of comfort and engage in new and different activities to stimulate their minds and their bodies. If playing bridge or doing a crossword puzzle is lifelong acquired skill, newer activities such as learning a new language or playing an instrument will provide important mind stimulation and encourage plasticity and growth.

The Health-Care Provider's Role in Successful Aging

Informing young and older adults about the ways in which to age successfully is the first step toward facilitating successful aging for any individual. There are ways in which to measure resilience and motivation, although at a clinical level they will not necessarily direct interventions. Thus, the best approach is to motivate individuals toward resilient behaviors.

Cross-References

- ▶ Aging
- ▶ Elderly
- ▶ Exercise
- ▶ Geriatric Medicine
- ▶ Gerontology
- ▶ Physical Activity

References and Readings

Boardman, J., Blalock, C., & Button, T. (2008). Sex differences in the heritability of resilience. *Twin Research and Human Genetics, 11*(1), 12–27.

Bonanno, G., Galea, S., Bucciarelli, A., & Vlahov, D. (2007). What predicts psychological resilience after disaster? The role of demographics, resources, and life stress. *Death Studies, 31*(10), 863–883.

Chow, S., Hamagani, F., & Nesselroade, J. (2007). Age differences in dynamical emotion-cognition linkages. *Psychology and Aging, 22*(4), 765–780.

Hardy, S., Concato, J., & Gill, T. (2002). Stressful life events among community-living older persons. *The Journal of General Internal Medicine, 17*(11), 832–838.

Hardy, S., Concato, J., & Gill, T. (2004). Resilience of community-dwelling older persons. *Journal of the American Geriatrics Society, 52*(2), 257–262.

Harris, P. (2008). Another wrinkle in the debate about successful aging: The undervalued concept of resilience and the lived experience of dementia. *International Journal of Aging and Human Development, 67*(1), 43–61.

Hegney, D., Buikstra, E., Baker, P., Rogers-Clark, C., Pearce, S., Ross, H., et al. (2007). Individual resilience in rural people: A Queensland study, Australia. *Rural and Remote Health, 7*(4), 620–625.

Hicks, G., Simonsick, E. M., Harris, T. B., Newman, A. B., Weiner, D. K., Nevitt, M. A., & Tylavsky, F. A. (2005). Trunk muscle composition as a predictor of reduced functional capacity in the health, aging and body composition study: The moderating role of back pain. *The Journal of Gerontology Series A, Biological Sciences and Medical Sciences, 60*(11), 1420–1424.

Karoly, P., & Ruehlman, L. (2006). Psychological "resilience" and its correlates in chronic pain: Findings from a national community sample. *Pain, 123*(1–2), 90–97.

Lee, H., Brown, S., Mitchell, M., & Schiraldi, G. (2008). Correlates of resilience in the face of adversity for Korean women immigrating to the US. *Journal of Immigrant and Minority Health, 10*(5), 415–422.

O'Connell, R., & Mayo, J. (1998). The role of social factors in affective disorders: A review. *Hospital & Community Psychiatry, 39*, 842–851.

Ong, A., Bergeman, C., Bisconti, T., & Wallace, K. (2006). Psychological resilience, positive emotions, and successful adaptation to stress in later life. *Journal of Personality and Social Psychology, 91*(4), 730–749.

Resnick, B., Orwig, D., Zimmerman, S., Simpson, M., & Magaziner, J. (2005). The exercise plus program for older women post hip fracture: Participant perspectives. *The Gerontologist, 45*(4), 539–544.

Rossi, N., Bisconti, T., & Bergeman, C. (2007). The role of dispositional resilience in regaining life satisfaction after the loss of a spouse. *Death Studies, 31*(10), 863–883.

Sanders, A., Lim, S., & Sohn, W. (2008). Resilience to urban poverty: Theoretical and empirical considerations for population health. *American Journal of Public Health, 98*(6), 1101–1106.

Wagnild, G., & Young, H. (1993). Development and psychometric evaluation of the resilience scale. *Journal of Nursing Measurement, 1*(2), 165–177.

Werner, E., & Smith, R. (1992). *Overcoming the odds: High risk children from birth to adulthood*. Ithaca: Cornell University Press.

Sudden Cardiac Death

- ▶ Cardiac Death
- ▶ Death, Sudden

Suicidal Ideation, Thoughts

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Synonyms

Suicidal impulses; Suicidal thoughts

Definition

Suicidal ideation refers to thoughts or impulses of engaging in behavior intended to end one's life (Nock et al. 2008). Suicidal ideation should be distinguished from suicidal plan, which refers to the formulation of a specific method through which one intends to die, and from suicide attempt, which refers to an actual engagement in potentially self-injurious behavior in which there is at least some intent to die (Nock et al. 2008).

Description

Ongoing suicidal ideation is a chronic risk factor and has been considered predictive of suicidal behavior especially if accompanied with severe hopelessness, prior suicide attempts, not having a child under 18 years old living at home, and a history of alcohol and drug abuse (Tishler and Reiss 2009). Suicidal ideation represents an important phase in the suicidal process and often precedes suicide attempts or completed suicide. However, not all patients experiencing suicidal ideation attempt suicide (Weissman et al. 1999). While approximately 10–20% of the population across diverse countries report suicidal ideation and 3–5% have made a suicide attempt at some time in their life, only 0.01% will complete suicide (Kessler et al. 2005). Acute risk factors including severe anxiety, agitation, and severe anhedonia are most predictive of whether someone will commit suicide in inpatients (Tishler and Reiss 2009; Bostwick and Rackley 2007).

Physical as well as mental health problems have been associated with suicidal ideation. Patients with chronic medical illnesses, such as HIV and cancer, and especially those experiencing physical pain are more likely to report suicidal thoughts (Tang and Crane 2006). Certain medical illnesses, such as neurological disorders and some cancers, appear to have higher rates of suicidal ideation and suicide compared to other medical disorders (Hugues and Kleespies 2001). A higher prevalence of suicidal ideation has been observed in patients experiencing pain associated with a variety of medical conditions including migraines, musculoskeletal pain, fibromyalgia, and arthritis (Tang and Crane 2006). Research suggests that the location and type of pain as well as the intensity and duration of the pain may have implications for the risk of suicidal ideation (Tang and Crane 2006). In addition, sleep-onset insomnia associated with pain is also a significant discriminator of the presence or absence of suicidal ideation (Tang and Crane 2006).

Research in the elderly suggests that physical illness plays an important role in suicidal ideation and behavior (Szanto et al. 2002). In the elderly with physical illness, untreated or undertreated physical pain, anticipatory anxiety regarding the progression of the physical illness, fear of dependence, and fear of burdening the family have been reported as the major contributing factors for suicidal ideation (Szanto et al. 2002). For all age groups, social isolation is considered another important risk factor that has been shown to be associated with suicidal ideation.

Sociodemographic factors including age and income level have also been associated with suicidal ideation (Kessler et al. 2005). In general, younger age, lack of education, and unemployment have been associated with higher rates of suicidal ideation and may represent an increased risk associated with social disadvantage (Nock et al. 2008). For all age groups, social isolation is considered another important risk factor that has

been shown to be associated with suicidal ideation (Van Orden et al. 2010).

Prior suicide attempts and the presence of a psychiatric disorder are the most consistently reported risk factors associated with suicidal ideation and behavior (Nock et al. 2008). Mood disorders such as anxiety and particularly depression significantly increase the risk of suicidal ideation in the general population and in medical patients in particular. Suicidal ideation has also been associated with other mental illnesses including severe anxiety, psychotic and personality disorders, and alcohol and substance abuse (Van Orden et al. 2010). A number of psychological processes have been found to exacerbate suicidal ideation. Specifically, findings from cross-sectional as well as longitudinal studies have attested to the role of hopelessness and helplessness, feelings of defeat and entrapment, deficits in problem solving abilities, and avoidant coping in the development of suicidal ideation (Van Orden et al. 2010; Nock et al. 2008; Szanto et al. 2002).

The presence of suicidal ideation in a clinical or medical setting typically requires a thorough risk assessment including chronic and acute risk factors as well as the frequency, intensity, and duration of suicidal thoughts (see Tishler and Reiss 2009 for prevention in medical settings).

References and Readings

Kessler, R. C., Berglund, P., Borges, G., Nocke, M., & Wang, P. S. (2005). Trends in suicide ideation, plans, gestures, and attempts in the United States, 1990–1992 to 2001–2003. *JAMA: The Journal of the American Medical Association, 293*, 2487–2495.

Nock, M. K., Borges, G., Bromet, E. J., Cha, C. B., Kessler, R. C., & Lee, S. (2008). Suicide and suicidal behavior. *Epidemiologic Reviews, 30*, 133–154.

Szanto, K., Gildengers, A., Mulsant, B. H., Brown, G., Alexopoulus, G. S., & Reynoldsm, C. F. (2002). Identification of suicidal ideation and prevention of suicidal behavior in the elderly. *Drugs & Aging, 19*, 11–24.

Tang, N. K., & Crane, C. (2006). Suicidality in chronic pain: A review of the prevalence, risk factors, and psychological links. *Psychological Medicine, 36*, 575–586.

Tishler, C. L., & Reiss, N. S. (2009). Inpatient suicide: Preventing a common sentinel event. *General Hospital Psychiatry, 31*, 103–109.

Van Orden, K. A., Witte, T. K., Cukrowicz, K. C., Braithwaite, S. R., Selby, E. A., & Joiner, T. E. (2010). The interpersonal theory of suicide. *Psychological Review, 117*, 575–600.

Weissman, M. M., Bland, R. C., Canino, G. J., Greenwald, S., Hwu, H. G., Joyce, P. R., et al. (1999). Prevalence of suicide ideation and suicide attempts in nine countries. *Psychological Medicine, 29*, 9–17.

Suicidal Impulses

► Suicidal Ideation, Thoughts

Suicidal Thoughts

► Suicidal Ideation, Thoughts

Suicide

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Synonyms

Deliberate self-harm; Self-directed violence; Self-Inflicted injurious behavior; Self-murder

Definition

Suicide is the act of intentionally ending one's own life. The definition of suicide reflects three important components (Rudd et al. 2001): (a) that the person died, (b) that the person's behavior caused death, and (c) that the person intended to cause his or her own death. While intentionality is the most precise characteristic that distinguishes those who have died by suicide and those who had died by other causes, its assessment remains controversial.

Description

Suicide is a major public health problem, and reports from the World Health Organization (WHO) indicate that suicide is projected to become an increasingly important contributor to the global burden of disease over the coming decades. Suicide is the 11th leading cause of death among all ages in the United States and the 13th leading cause of death worldwide. Suicide rates vary significantly cross-nationally (Center for Disease Control and Prevention 2007). In general, rates are highest in Eastern Europe and lowest in Central and South America, with the United States, Western Europe, and Asia falling in the middle. Epidemiological surveys showed that 2.7% of the US population has made a suicide attempt (Nock et al. 2008). Most individuals that attempt suicide die during their first suicide attempt (Nock et al. 2008). Within medical settings, according to Joint Commission on Accreditation of Healthcare Organizations (JCAHO 2010), suicide ranks among the five top most frequent sentinel events in hospitals. Seeking help from mental health professionals can assist in the reduction of distressing psychological symptoms. However, according to a recent review, while 45% of people who successfully committed suicide contacted a primary care provider within 1 month of their death, only 20% contacted mental health services within the same time period (Lauma et al. 2002).

Rudd et al. (2001) provide in their book a good description on a number of psychiatric, psychological, demographic, and biological variables have been recognized as suicide risk factors. The presence of one or more psychiatric problem is a central variable explaining suicide acts. At least 90% of individuals who have died from suicide have had at least one psychiatric disturbance. In addition, past suicide attempts, history of childhood abuse, and family history of suicide are associated with increased risk of suicide. Other variables that contribute to suicide risk are psychological variables, such as hopelessness, impulsivity, problem-solving deficits, and perfectionism. In terms of demographic variables, men are more likely to die by suicide than are women. Death by suicide is more common in older, lower socioeconomic status, and veterans. Similarly, non-Hispanic White individuals have a higher rate of suicide than individuals from other ethnical background. In addition, individuals who are unemployed, single, divorced, or widowed are also at a higher risk. Biological variables have also been found to be associated with suicide behaviors. Family, twin, and adoption studies have found evidence for heritable risk of suicidality. Biological factors related to lower levels of serotonin metabolites in the cerebrospinal fluid, higher serotonin receptor binding in platelets, and fewer presynaptic serotonin transporter sites were found in individuals who died by suicide. In addition, biological factors that inhibit impulsive behaviors, such as greater postsynaptic serotonin receptors in specific brain areas, such as the prefrontal cortex, have been associated with suicide behaviors.

The high rate of medical illness among individuals who committed suicide shows that both mental disorders and physical illness are important risk factors. Research shows variability in the risk of suicide according to the type of medical diagnosis. According to Hughes and Kleespies (2001), medical illnesses such as AIDS, cancer, chronic pain, end-stage renal diseases, severe neurological disorders, and chronic obstructive pulmonary disease have been correlated with increased risk of suicide. In contrast, they reported that other medical conditions including sclerosis, heart transplant, hypertension, rheumatoid arthritis, neoplasms, and cervix and prostate cancer have not been associated with increased risk. Studies have shown that at least one quarter of inpatients that have committed suicide in medical/surgical units did not report any previous psychiatric history (JCAHO 2010). Furthermore, suicide among these patients tends to happen within the first 2 weeks of their initial diagnosis (JCAHO 2010). This highlights the need of assessing suicidality in individuals who have chronic medical problems or who have been recently diagnosed with a life-threatening illness. Additionally, significant differences have been found between individuals who have completed suicide in inpatient psychiatric facilities and inpatient medical units. According to these studies, individuals

who committed suicide in medical units tend to be older, their death did not include careful planning, and their means of committing suicide were significantly more violent (Nock et al. 2008).

In contrast to the vast literature on variables associated with suicide, there are some studies that have identified protective factors. Protective factors are those that decrease the probability of suicide in the presence of elevated risk. Rudd et al. (2001) summarized some of the most consistent findings in the literature are supportive social network or family, reasons for living, and religious beliefs. Being pregnant and having young children in the home are also protective against suicide.

Clinical providers face the difficulty of recognizing individuals at risk of committing suicide as no single factor is sufficient to trigger or protect an individual from a suicidal act. Suicide warning signs are the earliest detectable signs or symptoms that indicate high risk for suicide in the near term (i.e., within minutes, hours, or days before the suicidal act; Rudd et al. 2001). They provide immediate cues to loved ones or clinical providers of the imminent risk of attempting to end one's life. Some suicide warning signs include hopelessness, anger, dramatic changes in mood, acting recklessly or engaging in risky activities, reports of feeling trapped, increased alcohol or drug use, withdrawal from loved ones, agitation or anxiety, and drastic sleep changes (Rudd et al. 2001).

Due to the difficulty of preventing and recognizing suicide, the assessment of suicide risk should be completed in a standardized and systematic way. In addition to assessing risk and protective factors, as well as warning signs to understand the risk level of an individual, suicide ideation, suicide plan, intent to act, previous suicide attempts, and the medical lethality of means need to be considered. When assessing suicide risk, it is important to differentiate between chronic risk and acute risk (Rudd et al. 2001). Chronic risk involves the presence of risk factors. Acute risk is determined by suicide ideation, intention, and a suicide plan in combination with warning signs. Acute risk can be further classified into low, moderate, and high. Low acute risk involves the presence of suicide thoughts with no specific plan, intent, or behavior. Moderate acute risk involves the presence of suicide ideation and a plan without intent or behavior. High acute risk refers to the presence of persistent suicide ideation and a specific plan with the intent to die.

References and Readings

Centers for Disease Control and Prevention. (2007). Web-based injury statistics query and reporting system (WISQARS). Retrieved June 20, 2011, from Centers for Disease Control and Prevention, National Center for Injury and Prevention Control Website: http://www.cdc.gov/ncipc/WISQARS

Hughes, D., & Kleespies, P. (2001). Suicide in the medically ill. *Suicide and Life Threatening Behaviors, 31*, 48–59.

Joint Commission on Accreditation of Healthcare Organizations. (2010). *Sentinel event statistics data*. Retrieved June 20, 2011, from the Joint Commission Website: http://www.jointcommission.org/sentinel_event_statistics_quarterly/

Lauma, J. B., Martin, C. E., & Pearson, J. L. (2002). Contact with mental health and primary care providers before suicide: A review of the evidence. *The American Journal of Psychiatry, 159*(6), 909–916.

Nock, M. K., Borges, G., Bromet, E. J., Cha, C. B., Kessler, R. C., & Lee, S. (2008). Suicide and Suicidal Behavior. *Epidemiologic Reviews, 30*, 133–154.

Rudd, M. D., Joiner, T., & Rajab, M. H. (2001). *Treating suicide behavior: An affective, time-limited approach*. New York: Guilford Press.

Wenzel, A., Brown, G. K., & Beck, A. T. (2009). *Cognitive therapy for suicide patients: Scientific and clinical applications*. Washington, DC: American Psychological Association.

Suicide Risk, Suicide Risk Factors

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Definition

Death from suicide in 1998 was ranked by the World Health Organization (WHO) as the 12th

leading cause of mortality worldwide. Suicide is the cause of death of one million people a year worldwide (Lineberry 2009). Suicide has been committed via several methods, which vary across geographic regions (Ajdacic-Gross et al. 2008). It is a main cause of death in later adolescence (ages 15–24 years; Shields et al. 2006).

The main difficulty in suicide prevention is its prediction because it constitutes a rare event and because of the multiple risk factors of suicide. These include situational factors such as social stressors and life events (e.g., unemployment, poverty), psychological factors such as hopelessness and hostility, biological factors (e.g., reduced brain-derived neurotrophic factor, protein kinase A; Dwivedi and Pandey 2011), and mere access to means of suicide (e.g., arms). One main difficulty in predicting suicidal behavior is the reliance on self-reported instruments, where people either conceal their real replies or are unaware of them. Furthermore, most studies in this domain have been retrospective and thus carry multiple sources of bias including survivors' and family members' attempt to find an explanation rather than the real cause.

Conversely, some recent studies have shown that assessing implicit associations between the "self" and "life" versus "death," with the Implicit Association Test, predicted suicidal behavior beyond what was detected by traditional risk factors (e.g., depression, past attempts, clinicians' ratings; Nock et al. 2010). Similarly, attention biases to suicide-related words relative to neutral words, using the "emotional Stroop," predicted suicidal behavior better than traditional risk factors (Cha et al. 2010). More research needs to utilize such instruments to identify additional suicide risk factors assessed in such indirect manners. Often, an accumulation of risk factors occurs, culminating in the tragic event. To conclude, explicit and implicit psychosocial factors and biological and situational factors serve as risk factors for suicide and need to be considered in the important attempt to prevent this severe health outcome. Given that recent studies mentioned here found that attention bias to suicide-related concepts predicted risk of suicide, future studies may also wish to test whether targeting such biases via attention-modification training, which was found to reduce mental problems (e.g., Heeren et al. 2015), may also prevent suicide in people with known suicide risk factors.

Cross-References

▶ Hopelessness
▶ Suicide

References and Further Readings

Ajdacic-Gross, V., Weiss, M. G., Ring, M., Hepp, U., Bopp, M., Gutzwiller, F., et al. (2008). Methods of suicide: International suicide patterns derived from the WHO mortality database. *Bulletin of the World Health Organization, 86*, 726–732.

Cha, C. B., Najmi, S., Park, J. M., Finn, C. T., & Nock, M. K. (2010). Attentional bias toward suicide-related stimuli predicts suicidal behavior. *Journal of Abnormal Psychology, 119*, 616–622.

Dwivedi, Y., & Pandey, G. N. (2011). Elucidating biological risk factors in suicide: Role of protein kinase A. *Progress in Neuropsychopharmacology and Biological Psychiatry, 35*, 831–841.

Heeren, A., Mogoaşe, C., Philippot, P., & McNally, R. J. (2015). Attention bias modification for social anxiety: A systematic review and meta-analysis. *Clinical Psychology Review, 40*, 76–90.

Lineberry, T. (2009). Suicide rates in 2009. Do the economy and wars have an effect? *Minnesota Medicine, 92*, 49–52.

Nock, M. K., Park, J. M., Finn, C. T., Deliberto, T. L., Dour, H. J., & Banaji, M. R. (2010). Measuring the suicidal mind: Implicit cognition predicts suicidal behavior. *Psychological Science, 21*, 511–517.

Shields, L. B., Hunsaker, D. M., & Hunsaker, J. C., 3rd. (2006). Adolescent and young adult suicide: A 10-year retrospective review of Kentucky medical examiner cases. *Journal of Forensic Sciences, 51*, 874–879.

Sulcus

▶ Brain, Cortex

Summary Data

► Aggregate Data

Sun Exposure

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Definition

Sun exposure refers to the amount and manner in which people expose themselves to sunlight. This is a highly complex parameter for quantification, as it relies on self-report and its effects depend on multiple environmental and personal factors, as well as on frequency and location of exposure on the body. Most skin cancers are related to sun exposure, and a great majority of exposure to sun takes place before adulthood, making children and adolescents central target groups for assessment and prevention of sun exposure and skin cancers. Multiple studies have linked sun exposure to various cancers, particularly to skin cancer. In a review of 57 studies, intermittent sun exposure and history of burns were related to risk of melanoma. In contrast, high occupational sun exposure was inversely related to melanoma. Furthermore, factors such as country of study seem to moderate effects of sun exposure on melanoma, suggesting that effects of sun exposure depend on geographical factors as well. Indeed, geographic latitude synergistically interacts with history of sunburn in relation to occurrence of melanoma (Gandini et al. 2005). A major cause of melanoma due to sun exposure is ultraviolet light (Armstrong and Kricker 1993). Various questionnaires exist for assessing sun exposure, which consider the manner and duration of sun exposure, context (working vs. nonworking days), and cumulating measures in relation to various time frames (years or one's life time; Kricker et al. 2005). Importantly, to achieve greater test-retest reliability, it may be beneficial to assess sun exposure in relation to activities (e.g., with family, at work) rather than in relation to specific time periods (Yu et al. 2009). In children, important social factors related to usage of sun protective agents include parental reminders (Donavan and Singh 1999). Furthermore, in children, sun protective behavior decreases with age though sun exposure increases with age, possibly reflecting greater peer pressure and reduced parental control in older children (Pichora and Marrett 2010). Thus, sun exposure reflects a major and complex cause of various skin cancers and must be properly assessed and better controlled in attempt to prevent skin cancers. Future studies may wish to identify the social pressures people face concerning sun exposure and the cognitive barriers people have about protection from sun exposure, which could be then targeted by "psychological inoculation," an evidence-based cognitive method with far stronger effects than health education only (Duryea et al. 1990).

Cross-References

► Cancer Screening/Detection/Surveillance

References and Further Readings

Armstrong, B. K., & Kricker, A. (1993). How much melanoma is caused by sun exposure? *Melanoma Research, 3*, 395–401.

Donavan, D. T., & Singh, S. N. (1999). Sun-safety behavior among elementary school children: The role of knowledge, social norms, and parental involvement. *Psychological Reports, 84*, 831–836.

Duryea, E. J., Ransom, M. V., & English, G. (1990). Psychological immunization: Theory, research, and current health behavior applications. *Health Education & Behavior, 17*(2), 169–178.

Gandini, S., Sera, F., Cattaruzza, M. S., Pasquini, P., Picconi, O., Boyle, P., et al. (2005). Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure. *European Journal of Cancer, 41*, 45–60.

Kricker, A., Vajdic, C. M., & Armstrong, B. K. (2005). Reliability and validity of a telephone questionnaire for estimating lifetime personal sun exposure in epidemiologic studies. *Cancer Epidemiology, Biomarkers & Prevention, 14*, 2427–2432.

Pichora, E. C., & Marrett, L. D. (2010). Sun behaviour in Canadian children: Results of the 2006 national sun survey. *Canadian Journal of Public Health, 101*, 14–18.

Yu, C. L., Li, Y., Freedman, D. M., Fears, T. R., Kwok, R., Chodick, G., et al. (2009). Assessment of lifetime cumulative sun exposure using a self-administered questionnaire: Reliability of two approaches. *Cancer Epidemiology, Biomarkers & Prevention, 18*, 464–471.

Supervisory Attentional System

▶ Executive Function

Supplication

▶ Prayer

Supportive Care

▶ Palliative Care

Suprachiasmatic Nucleus

▶ Hypothalamus

Supraoptic Nucleus

▶ Hypothalamus

Surgery

▶ Cancer Treatment and Management

Surgical Resection

▶ Cancer Treatment and Management

Surrogacy

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Synonyms

ART, Assisted reproductive technology; Commissioning parent; Cross-border reproductive care; Egg donation; Egg donor; Embryo donation; Gestational carrier; ICSI; Infertility; Intended parent; IVF; Sperm donation; Sperm donor; Surrogate mother

Definition

A surrogacy arrangement is one in which, before the child is conceived, the commissioning parent/s (also known as intended parent/s) and the surrogate mother (also known as gestational carrier, or birth mother) and her partner (if she has one) agree that the surrogate will become pregnant with the intention that the child will, at birth, be given into the care of the commissioning parent/s to raise as their own.

Description

Common reasons for surrogacy include absence of the uterus (such as hysterectomy for women or men without a female partner), congenital malformation of the uterus, or a medical condition that compromises pregnancy making it unsafe for the commissioning woman (intended mother) or her prospective baby. Surrogate conception may occur in a number of ways including where the genetic material is provided by both commissioning parents or by one only of them, by both the surrogate mother and her partner or by one only of them, or by third-party donors (egg, sperm, or

embryo) who are not involved in the actual surrogacy arrangement. A surrogacy arrangement can take the form of a natural process, through the surrogate's self-insemination, or occur at an in vitro fertilization (IVF) clinic through assisted reproductive technology. Surrogacy, as practiced worldwide, is primarily IVF or gestational surrogacy that does not involve any genetic material of the surrogate or her partner but may involve third-party donor gametes. Insemination surrogacy (also known as genetic, traditional, or partial surrogacy) is less common.

The availability and conditions of surrogacy arrangements vary according to the jurisdiction in which the arrangement takes place (Jadva 2016). For example, in the USA, gestational surrogacy is primarily undertaken through IVF clinics with the gestational carrier being financially compensated for her contractual participation in the surrogacy arrangement. In comparison, in countries such as Australia and New Zealand, only altruistic surrogacy is legally permitted, with mostly gestational surrogacy (Hammarberg et al. 2011). In the UK surrogacy is altruistic, with both gestational and genetic insemination surrogacy (Van den Akker 2007). Commercial surrogacy has also been available in a number of developing countries including India (Lamba et al. 2018), Thailand, Mexico, Cambodia, and more recently, Greece and Ukraine. In many of these countries, the commissioning parents mostly originate from the developed world, with the surrogates having significantly less agency over decisions in the surrogacy arrangement, with subsequent legal and ethical implications. One of the greatest challenges arises from this type of cross-border surrogacy where there is uncertainty regarding the status of the parent and the child, particularly when considering the legal aspects of citizenship (Crockin 2013).

Despite the conception process being relatively straightforward medically, the psychological and social implications of surrogacy are complex. Social relationships between the surrogate and commissioning parents vary from close family or friends, to those where there is no prior or anticipated subsequent, relationship between the surrogate and the child born of the surrogacy arrangement. For many intended parents, the point of surrogacy may reflect years of physical, emotional, and financial stressors in the unsuccessful quest to conceive through IVF-type techniques. This, is turn, may lead the intended parents to be over vigilant and anxious about the surrogate's well-being throughout the pregnancy (Greenfeld 2014). It is critical that the capacity of the commissioning woman to manage the emotional challenges is considered prior to engaging in the surrogacy arrangement, including the challenges of having another woman carrying her baby. Potential conflicts may also arise between the intended parents and the surrogate from differences in values and preferences regarding diet, medications, delivery options, and even termination options, if required. Variations in the amount of contact that intended parents expect to have with the surrogate during the pregnancy and whether or not the surrogate will maintain contact antenatally can be sources of conflict, particularly as the expectations of all involved parties may fluctuate with changing circumstances surrounding the surrogacy. Trust is clearly a key factor underlying successful surrogacy arrangements, pointing to the need for adequate pre-surrogacy counselling and education for all involved parties (Fuchs and Berenson 2016). In best medical and psychological practice, counselling should address four key principles relating to the best interests of the child born of the surrogacy arrangement; the surrogate's ability to make free and informed decisions; the requirement that the surrogate be free of exploitation; and a requirement for legal clarity about the resulting parent-child relationship, including documented information of genetic and birth history (Hammarberg et al. 2011).

There is no universally agreed standard for implications counselling in surrogacy. Professional psychological counselling may be provided through a treating IVF clinic, or it may be provided by a mental health professional independent of the treating clinic. There may be a requirement for in-depth psychological assessment and screening such as is required in the USA for gestational carriers and egg (oocyte) donors (Fuchs and Berenson 2016). In some countries, such as Australia, there may be pre-surrogacy

psychosocial assessment of all parties to the surrogacy arrangement including the intended parents. In the UK pre-surrogacy counselling of all parties is recommended, but not mandated (Norton et al. 2015). Overall, there is more intensive and comprehensive attention to the psychosocial issues in countries such as the USA, UK, Australia, Canada, and New Zealand, with less being provided in countries such as Ukraine, Greece, Mexico, India, Thailand, and Cambodia.

With increasing uptake of surrogacy arrangements worldwide, there is a need to consider the short- and long-term outcomes for all parties concerned. In general, surrogates appear to score within the normal range on personality tests and to have medically and psychologically healthy pregnancies (Klock and Covington 2015). The greatest psychological challenge for surrogates appears to be at the time for the surrogate to hand over the child following the birth (Jadva 2016). Regarding the adjustment of children born of surrogates, one systematic review of 55 studies reported that by 10 years of age, these children had similar levels of psychological functioning as children naturally conceived and those conceived through assisted reproductive technology (ART) (Söderström-Anttila et al. 2016). The findings from one study of gay men parenting a child conceived through surrogacy further support the view that this practice has no adverse effects on child health outcomes (Carone et al. 2018). Moreover, it appears that disclosure to the child about the surrogacy arrangement is common in such families, reflecting a greater level of openness within the family than has been found in children conceived from other assisted third-party reproduction (Ilioiv et al. 2017). Despite a growing literature in this area, unfortunately there is limited high-quality research, with few studies providing adequate control groups and sample sizes and most studies having low-response rates questioning the generalizability of these findings (Söderström-Anttila et al. 2016). Clearly, there is an urgent need for more methodologically rigorous investigations regarding the psychosocial correlates and consequences of individuals involved in surrogacy arrangements.

Cross-References

- ▶ Assisted Reproductive Technology
- ▶ Infertility
- ▶ Infertility and Assisted Reproduction: Psychosocial Aspects
- ▶ Infertility-Related Stress
- ▶ In Vitro Fertilization
- ▶ In Vitro Fertilization, Assisted Reproductive Technology

References and Further Reading

Carone, N., Lingiardi, V., Chirumbolo, A., & Baiocco, R. (2018). Italian gay father families formed by surrogacy: Parenting, stigmatization, and children's psychological adjustment. *Developmental Psychology, 54*(10), 1904–1916.

Crockin, S. L. (2013). Growing families in a shrinking world: Legal and ethical challenges in cross-border surrogacy. *Reproductive Biomedicine Online, 27*(6), 733–741.

Fuchs, E. L., & Berenson, A. B. (2016). Screening of gestational carriers in the United States. *Fertility and Sterility, 106*(6), 1496–1502.

Greenfeld, D. A. (2014). Use of gestational carriers: Psychological aspects. In J. M. Goldfarb (Ed.), *Third party reproduction: A comprehensive guide* (pp. 79–83). New York: Springer.

Hammarberg, K., Johnson, L., & Petrillo, T. (2011). Gamete and embryo donation and surrogacy in Australia: The social context and regulatory framework. *International Journal of Fertility and Sterility, 4*(4), 176–183.

Ilioiv, E., Blake, L., Jadva, V., Roman, G., & Golombok, S. (2017). The role of age of disclosure of biological origins in the psychological wellbeing of adolescents conceived by reproductive donation: A longitudinal study from age 1 to age 14. *Journal of Child Psychology and Psychiatry, 58*(3), 315–324.

Jadva, V. (2016). Surrogacy: Issues, concerns and complexities. In S. Golombok, R. Scott, J. B. Appleby, M. Richards, & S. Wilkinson (Eds.), *Regulating reproductive donation* (pp. 126–139). Cambridge: Cambridge University Press.

Klock, S. C., & Covington, S. N. (2015). Results of the Minnesota multiphasic personality inventory-2 among gestational surrogacy candidates. *International Journal of Gynecology & Obstetrics, 130*, 257–260.

Lamba, N., Jadva, V., Kadam, K., & Golombok, S. (2018). The psychological well-being and prenatal bonding of gestational surrogates. *Human Reproduction, 33*(4), 646–653.

Norton, W., Crawshaw, M., Hudson, N., Culley, L., & Law, C. (2015). A survey of UK fertility clinics' approach to surrogacy arrangements. *Reproductive Biomedicine Online, 31*, 327–338.

Söderström-Anttila, V., Wennerholm, U.-B., Loft, A., Pinborg, A., Aittomäki, K., Bente Romundstad, L., & Bergh, C. (2016). Surrogacy: Outcomes for surrogate mothers, children and the resulting families – A systematic review. *Human Reproduction Update, 22*(2), 260–276.

Van den Akker, O. B. (2007). Psychosocial aspects of surrogate motherhood. *Human Reproduction Update, 13*(1), 53–62.

Surrogate

► In Vitro Fertilization, Assisted Reproductive Technology

Surrogate Decision Making

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Synonyms

Proxy

Definition

Surrogate Decision Making

If an individual is unable to make decisions about personal health care, some other individual can be authorized to provide direction. Such a person is called the surrogate decision maker, a proxy, or some other term specific to the type of authorization. For example, under an advance directive, the decision maker is typically an "agent"; under a durable power of attorney for health care, the decision maker is the "attorney in fact" (although in slang, some health-care providers refers to such persons as "the POA"); an individual may be judicially appointed as a "guardian," or state law may identify certain next of kin or other close persons as eligible to serve as a surrogate. State law will determine the role of domestic partners in the absence of an advance directive appointing someone as an agent.

Each type of surrogate or proxy has a scope of authority to make health-care decisions for an individual depending on the source of the surrogate or proxy's authority and state law. For example, the surrogate may have broad authority to consent to health care and to refuse or direct the withdrawal of health care but with limitations if the care is life-sustaining. An agent under an advance directive from an individual may have immediate, broader authority to direct the withholding or withdrawal of life-sustaining treatment than a surrogate acting only under state law. Guardians may nor may not need court approval for certain actions involving life-sustaining treatment.

Surrogates or proxies appointed by the individual patient have decision making priority. Where there is no such person, state law determines the process for consulting with next of kin or others, which can include close friends, potentially in an order of hierarchy as to who priority, and how to resolve disagreements among those with the same level of relationship. Where there are no such persons or there is an irreconcilable disagreement, a judicial guardianship may be needed. Ethics committees or Patient Care Advisory Committees are examples of bodies within a health-care facility that can be very helpful in gathering an evaluating information about an individual's clinical condition, treatment options and prognosis, previously expressed wishes, interpretations of available documents, varying points of view, and related considerations.

In a recent study, it was noted that surrogate decision making is often required for older Americans at the end of life. Among a sample of 4246 deaths of respondents in the Health and Retirement Study, proxies reported that 42.5% of these individuals needed decision making about medical treatment before death; 70.3% of subjects lacked the capacity to make those decisions themselves and, overall, 29.8% required decision making at the end of life but lacked decision-making capacity. These findings suggest that more than a quarter of elderly adults may need surrogate decision making before death.

Cross-References

▶ End-of-Life Care

References and Readings

Silveira, M. J., Kim, S. Y. H., & Langa, K. M. (2010). Advance directives and outcomes of surrogate decision making before death. *The New England Journal of Medicine, 362*, 1211–1218.

Surrogate Mother

▶ Surrogacy

Surveys

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Synonyms

Micro data collection and analysis system; Opinion poll; Statistical inquiry

Definition

Survey is a methodology and a practical tool used to collect, handle, and analyze in a systematic way information from individuals. These individuals or micro units can be of various types, such as people, households, hospitals, schools, businesses, or other corporations. The units can be simultaneously available from two or more levels such from households and their members. Information in surveys may be concerned various topics such as people's personal characteristics, their behavior, health, salary, attitudes and opinions, incomes, poverty and housing environments, or characteristics and performance of businesses. Survey research is unavoidably interdisciplinary, although the role of statistics is most influential since the data for surveys is constructed in a quantitative form. Correspondingly, many survey methods are special statistical applications. However, surveys exploit substantially many other sciences such as informatics, mathematics, cognitive psychology, and theory of submatter sciences of each survey topic.

Basic Survey Concepts

A key concept in surveys is *target population* the universe of which should be exactly determined and realistic. It is possible that there are more than one target population (e.g., hospitals of certain types and their clients during a specific period). Before determining a strict target population, a researcher can have in mind *population of interest*, but this is often too difficult to reach, and hence, a realistic population is chosen. For example, more or less heavy alcohol drinkers may be interest for a researcher, but such people cannot be found from any data source. Respectively, such a target population is not realistic, but fortunately, one can try with a larger target population where there are also nondrinkers or light drinkers. The study itself can, among others, concentrate on those heavy drinkers, and results are correct if there are in data enough such people in order to get appropriate results. This requires also that we have a good frame and *frame population* from which reasonable data are downloaded. A drawback is that although the frame seems to be ideal, it includes such people who do not belong to our realistic target population, nevertheless, and secondly, we cannot get responses from all selected people due to *nonresponse*. Thus, when designing data collection, it is necessary to predict nonresponse and other gaps as well as possible in order to get enough respondents for the study.

Sampling

Survey data can cover the whole target population, but if it is large, it is rational to use sampling. This leads to plan an optimal sampling design. The design may be more or less complex. The simplest one is to use completely random selection in which case every frame unit has an equal *inclusion probability* to be selected in the sample. Such a design is rarely rational since the data collection may be too expensive or such a frame is not available. For these reasons, the most common strategy in people surveys is in the first stage,

to select so-called *clusters* (small areas, service houses, households), and in the second stage, the desired sample units (target people, clients, household members) are to be interviewed and studied. This strategy is called *two-stage sampling*, but *three-stage sampling* is also applied. These both strategies lead to *probability sampling* that is definitely a valid method. Naturally, next steps must have been done successfully too.

Two- and three-stage samplings are generally called multistage sampling, but if the study units are selected directly from the frame, it is one-stage sampling, but this term is not much used. In contrast, the term *element sampling* is common. Multistage sampling is *hierarchical* in its nature, that is, we approach to study units by stage by stage. This strategy is different from *multiphase sampling* in which case after one sample selection, a new sample from the first sample has been selected. This is much used in *panels* in which case the second or the consecutive samples have been selected even with 100% from those who have responded in the first phase. This panel approach gives opportunity to follow respondents over time. It is common in health, poverty, and living condition research, for instance. Panel designs can be very complex too. *Rotating design* is much used when needed to analyze data both *cross-sectionally* (for a specific time point or time period) and *longitudinally* (for following individuals over time). Long panels might be hard to do well, and there can be met too much nonresponse that leads a worsening data quality.

Two-phase sampling is also useful when analyzing how respondents differ from nonrespondents. This information is necessary for survey quality documentation, but it can be used also for improving the quality. The second phase could in this case lead to draw a subsample of the nonrespondents, and then attempts to get some information from them. Naturally, this is not easy since they are reluctant, but most nonrespondents still are willing to answer to some simple but key questions of the survey.

Moreover, it is often advantageous to exploit *stratification* in surveys. This leads to create a number of strata that are like subpopulations. Sampling designs for each stratum may be similar or different. The main varying point is maybe that the sample size has been allocated for each stratum so that the research targets are satisfied ideally. Typically, the relative sample size is larger for a smaller stratum population and smaller for a larger population, respectively. This is due to an ordinary target to get about as accurate estimates (results) for each stratum.

Data Collection Tools and Methods

Data collection is not ready after selection a sample (or the full data). Three other major tasks are needed: (1) design the questionnaire, (2) decide the data collection mode (mail or phone or face-to-face interviewing or web or mixed mode such as web plus phone), (3) collect supported data (auxiliary) both for sampling and further data handling. Such data are required both from respondents and nonrespondents. *Auxiliary data* are very advantageous for adjusting *sampling weights* from the initial ones. Consequently, the bias in estimates will be reduced.

Data Cleaning and Analysis

Survey data should be cleaned before starting the analysis. In addition to computing the sampling weights, the following tasks are needed: data editing, imputation of missing data, data documentation (called metadata), data collection process documentation (called paradata), and selection of a good IT format (e.g., SPSS or SAS). The sampling design strategy is necessary to correctly take into account in the analysis.

Cross-References

- ▶ Clusters
- ▶ Cohort Study
- ▶ Data
- ▶ Internet-Based Studies
- ▶ Interview
- ▶ Multivariate Analysis
- ▶ Odds Ratio
- ▶ Participation Bias
- ▶ Population Stratification
- ▶ Probability
- ▶ Randomization
- ▶ Regression Analysis
- ▶ Retrospective Study

- ▶ Sample Size Estimation
- ▶ Selection Bias
- ▶ Standard Deviation
- ▶ Statistics
- ▶ Study Methodology

References and Readings

Bethlehem, J. G. (2009). *Applied survey methods: a statistical perspective* (375 pp). Hoboken: Wiley.

de Leeuw, E. D., Hox, J. J., & Dillman, D. A. (Eds.). (2009). *International handbook of survey methodology*. New York\London: Lawrence Erlbaum Associates \Taylor and Francis Group. 549 pp.

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Biographical Information

Richard S. Surwit received his B.A. from Earlham College and Ph.D. in clinical psychology from McGill University. He completed a postdoctoral fellowship in psychophysiology at Harvard University and joined the Duke faculty in 1977. He is currently professor of medical psychology and vice chair for research in the Department of Psychiatry and Behavioral Science at Duke University Medical Center, Durham, NC.

Major Accomplishments

Surwit's early work focused on the utility of biofeedback in the treatment of medical disorders, including Raynaud's disease and hypertension, and he was instrumental in establishing one of the first clinical biofeedback facilities in the country. Over the course of a long and productive career, his scientific contributions range from basic research on the use of mouse models to examine genetic and behavioral interactions in the development of obesity to clinical investigations of the effects of stress and hostility on glucose metabolism and diabetes. He also pioneered the use of computers in the medical management of such chronic diseases as diabetes and congestive heart failure. He has received multiple grants from the National Institutes of Health, private foundations, and industry to support his research program. He has played a key administrative role in the Department of Psychiatry and Behavioral Sciences at Duke University as vice chair for research and is a former president of the Society of Behavioral Medicine. He also has served on the editorial boards of such journals as *Health Psychology*, the *Journal of Consulting and Clinical Psychology*, *Obesity*, and *Metabolism*. He is the recipient of numerous awards and honors including the Research Career Award in Health Psychology from the American Psychological Association.

Cross-References

- ▶ Diabetes

Dr. Surwit recently received the Distinguished Scientist Award from the Society of Behavioral Medicine.

Sustainability

- ▶ Ecosystems, Stable and Sustainable

Sympathetic

▶ Autonomic Balance
▶ Heart Rate Variability

Sympathetic Nervous System (SNS)

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Definition

The sympathetic nervous system (SNS) is one of two main branches or subsystems of the autonomic nervous system (ANS). It originates in the thoracic and upper lumbar segments of the spinal cord and commonly – but not always – yields peripheral adjustments that are complementary to those produced by its counterpart, the parasympathetic nervous system (PNS).

Description

The sympathetic nervous system is one of two main branches or subsystems of the autonomic nervous system, the physical system responsible for unconsciously maintaining bodily homeostasis and coordinating bodily responses. Working with the second main branch, the parasympathetic nervous system, the sympathetic nervous system regulates a wide range of functions such as blood circulation, body temperature, respiration, and digestion. Sympathetic activation commonly leads to adjustments on organs and glands that are complementary to those produced by parasympathetic activation and suitable for high activity ("fight and flight" as opposed to "rest and digest"). Examples of high-activity adjustments are constriction of blood vessels in the skin, dilation of blood vessels in the skeletal muscles and lungs, and increased heart rate and contraction force. Although sympathetic adjustments tend to complement parasympathetic adjustments, they do not always. For example, both sympathetic nervous system arousal and parasympathetic nervous system arousal increase salivary flow, although to different degrees and yielding different compositions of saliva.

Basic functional units of the sympathetic nervous system are preganglionic and postganglionic neurons. Preganglionic neurons have cell bodies in the thoracic and upper lumbar segments of the spinal cord and axons that extend to cell bodies of postganglionic neurons. Postganglionic neurons have cell bodies that are clustered in the so-called ganglia and relatively long axons that innervate target organs and glands. The major neurotransmitters of the sympathetic nervous system are acetylcholine and norepinephrine. Acetylcholine is the neurotransmitter of all preganglionic neurons. Stimulation of cholinergic receptors of the nicotinergic subtype located on the cell bodies of the postganglionic neurons by acetylcholine leads to an opening of nonspecific ion channels. This opening permits transfer of potassium and sodium ions, which depolarizes the postganglionic cell and initiates an action potential. Norepinephrine is the neurotransmitter of most sympathetic postganglionic neurons and stimulates adrenergic receptors lying on targeted visceral structures. All adrenergic receptors are coupled with G-proteins, but transmission pathways depend on the receptor subtype. Activation of alpha-1 receptors changes the calcium concentration in the cell, which in turn triggers the specific effect on the targeted visceral structure. Alpha-2 and beta receptors trigger visceral responses by affecting cAMP production in the cell. Specific effects depend on the receptor subtype and on the innervated visceral structure. For instance, stimulation of alpha-1 receptors on blood vessels of skeletal muscles leads to vasoconstriction and reduced blood flow, whereas stimulation of alpha-1 receptors on the radial pupil muscle leads to muscle contraction and increased pupil size. Stimulation of beta-2

receptors on the heart leads to increased heart rate and contraction force, whereas stimulation of beta-2 receptors on skeletal muscle blood vessels leads to vasodilation and increased blood flow.

In working jointly with the parasympathetic nervous system, the sympathetic nervous system does not function in an all-or-none fashion, but rather activates to different degrees. Depending on the affected visceral structure and situation, it may be more or less active than the parasympathetic nervous system. Shifts in the magnitude of sympathetic and parasympathetic influence can occur locally within a single visceral structure (e.g., the eye) or across visceral structures, with local shifts occurring to meet highly specialized demands (e.g., a change in ambient light) and global shifts adapting the body to large-scale environmental changes (e.g., the appearance of a substantial physical threat). Autonomic control is maintained by structures in the central nervous system that receives visceral information from an afferent (incoming) nervous system. A key central nervous system structure is the hypothalamus, which integrates autonomic, somatic, and endocrine responses that accompany different organism states.

Cross-References

- ▶ Acetylcholine
- ▶ Autonomic Activation
- ▶ Autonomic Balance
- ▶ Autonomic Nervous System (ANS)
- ▶ Epinephrine
- ▶ Parasympathetic Nervous System (PNS)

References and Further Readings

Berne, R. M., Levy, M. N., Koeppen, B. M., & Stanton, B. A. (2004). *Physiology* (5th ed.). St. Louis: Mosby.

Cacioppo, J. T., & Tassinary, L. G. (1990). *Principles of psychophysiology: Physical, social, and inferential elements*. New York: Cambridge University Press.

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (2000). *Handbook of psychophysiology* (2nd ed.). New York: Cambridge University Press.

Ganong, W. F. (2005). *Review of medical physiology* (22nd ed.). New York: McGraw-Hill.

Levick, J. R. (2009). *An introduction to cardiovascular physiology* (5th ed.). London: Hodder.

Sympathetic Nervous System (SNS) Activation

▶ Sympatho-adrenergic Stimulation

Sympatho-adrenergic Stimulation

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Synonyms

Adrenergic activation; Sympathetic nervous system (SNS) activation

Definition

Activation of one of the three branches (the sympathetic nervous system) of the autonomic nervous system via disruption of physiological homeostasis which results in the release of epinephrine/adrenaline and norepinephrine/noradrenaline from the adrenal medulla.

Description

The sympatho-adrenomedullary (SAM) system is one of two major components of the stress system. Stress activates the sympathetic nervous system and the goal of this response is to return the individual to physiological homeostasis. Epinephrine release from the adrenal medulla causes physiological alterations in cardiovascular tone, respiration rate, and blood flow to the muscles. Epinephrine does not cross the blood–brain barrier, but acts indirectly on the brain via the vagus nerve, which projects to the nucleus of the solitary tract (NTS), resulting in noradrenergic projections to the amygdala, as well as other brain regions. The release of epinephrine and norepinephrine from the adrenal

medulla increases blood glucose levels and enhances alertness, learning, and memory.

Cross-References

► Norepinephrine/Noradrenaline

References and Readings

Cannon, W. B. (1914). The interrelations of emotions as suggested by recent physiological research. *The American Journal of Psychology, 25*(2), 256–282.

Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *Journal of the American Medical Association, 267*(9), 1244–1252.

Hollenstein, T., McNeely, A., Eastabrook, J., Mackey, A., & Flynn, J. (2011). Sympathetic and parasympathetic responses to social stress across adolescence. *Developmental Psychobiology.* https://doi.org/10.1002/dev.20582.

Sherwood, L. (2008). *Human physiology: From cells to systems* (7th ed., p. 240). Stamford: Cengage Learning.

Tilders, F. J. H., & Berkenbosch, F. (1986). CRF and catecholamines; their place in the central and peripheral regulation of the stress response. *Acta Endocrinology, 113*, S63–S75.

Symptom Magnification Syndrome

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Synonyms

Learned symptom behavior; Maladaptation of symptom behaviors to chronic illness; Malingering; Martyr behavior; Secondary gain

Definition

Symptom magnification is a self-destructive, socially reinforced behavioral response pattern consisting of reports or displays of symptoms which function to control the life of circumstances of the sufferer.

Description

Symptom magnification syndrome (SMS) may be described as a conscious or unconscious self-destructive learned pattern of behavior which is maintained through social reinforcement and typically controls the individual's life activities. SMS may be labeled "malingering" or exaggerated psychological complaints which can be associated with individuals who are seeking financial compensation or a secondary gain, e.g., increased attention from a family member, from symptom reporting. The validity scales of the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) are widely used for the detection of exaggerated psychological complaints.

Cross-References

► Psychosocial Adjustment
► Psychosocial Predictors
► Psychosocial Variables
► Psychosocial Work Environment
► Psychosomatic
► Psychosomatic Disorder
► Somatic Symptoms
► Somatization
► Somatoform Disorders

References and Readings

Kopel, S., Walders-Abramson, N., MsQuaid, E., Seifer, R., Koinis-Mitchell, D., Klein, R., et al. (2010). Asthma symptom perception and obseity in children. *Biological Psychology, 84*, 135–141.

Matheson, L. N. (1986). *Work capacity evaluation: Systematic approach to industrial rehabilitation*. Anaheim: Employment and Rehabilitation Institute of California.

Matheson, L. N. (1987). *Symptom magnification casebook*. Matheson: Employment and Rehabilitation Institute of California.

Theodore, B., Kishino, N., & Gatchel, R. (2008). Biopsychosocial factors that perpetuate chronic pain, impairment, and disability. *Psychological Injury and Law, 1*, 182–190.

Tsushima, W., & Tsushima, V. (2009). Comparison of MMPI-2 validity scales among compensation-seekin Caucasian and Asian American medical patients. *Assessment, 16*, 159–164.

Symptom Scale

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Synonyms

Symptoms Inventory

Definition

Symptom scales are psychometric instruments aimed at assessing the frequency or severity of any type of symptom associated with a mental or physical health condition. Development of symptom scales requires the same type of rigor as any other self-report instrument requires, including tests of internal reliability and test-retest reliability and face, concurrent, construct, and predictive validity. Questions on such scales can be asked in relation to a specific time frame (e.g., the present moment, the past week) and in relation to certain severity levels. For example, in the assessment of pain symptoms, patients may be asked to rate their level of average and worse pain in a given time frame. Finally, some scales also ask the extent to which certain symptoms interfere with one's daily functioning, as is often done in the domain of quality of life or pain. Another issue is whether the scale includes items concerning general symptoms or disease-specific symptoms. One of the earliest developed psychological symptom scales is the Symptom Checklist 90 (SCL-90) which was designed to assess psychological symptoms for evaluating the outcomes of mental health interventions and for research purposes. The symptoms are assessed in relation to the past 7 days and are categorized into nine dimensions (e.g., psychoticism, depression). Its internal reliability is adequate (e.g., Cronbach's alpha of 0.77–0.90 on its dimensions). Its concurrent, construct, and predictive validities have been shown as well. A physical symptom scale is the Patient Health Questionnaire 15 (PHQ-15; Kroenke et al. 2002). This scale assesses 15 common physical symptoms including stomach, back, head, and chest pains, dizziness, shortness of breath, etc. Scores on the PHQ-15 correlate with functional status and with health-care utilization. Numerous other instruments exist for assessment of various psychiatric symptoms including depression, anxiety, and post-traumatic stress disorder and for disease-specific symptoms. The latter include symptom scales of upper respiratory infections (Orts et al. 1995), the Rose chest pain questionnaire (Rose et al. 1977), and many others. More recently, Baxter et al. (2011) developed a brief pictorial scale for assessing nausea in children which can be used for pediatric oncological treatments. Symptom scales are a basic element in diagnosis and monitoring of treatment effects and in research in many health disciplines including medicine and behavior medicine. However, it is noteworthy to consider the limitations of symptom scales, as in most self-report scales. These include reporting biases, memory, and lack of self-awareness. One important factor known to underlie reporting biases is neuroticism or negative affectivity, which, when elevated, often leads to inflated symptom reporting and needs to be considered when patients complete symptom scales (Watson and Pennebaker 1989).

Cross-References

► Somatic Symptoms

References and Further Readings

Baxter, A. L., Watcha, M. F., Baxter, W. V., Leong, T., & Wyatt, M. M. (2011). Development and validation of a pictorial nausea rating scale for children. *Pediatrics, 127*, e1542–e1549.

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2002). The PHQ-15: Validity of a new measure for evaluating the severity of somatic symptoms. *Psychosomatic Medicine, 64*, 258–266.

Orts, K., Sheridan, J. F., Robinson-Whelen, S., Glaser, R., Malarkey, W. B., & Kiecolt-Glaser, J. K. (1995). The reliability and validity of a structured interview for the assessment of infectious illness symptoms. *Journal of Behavioral Medicine, 18*, 517–529.

Rose, G., McCartney, P., & Reid, D. D. (1977). Self-administration of a questionnaire on chest pain and intermittent claudication. *British Journal of Preventive & Social Medicine, 31*, 42–48.

Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review, 96*, 234–254.

Symptom-Limited Exercise Test

▶ Maximal Exercise Stress Test

Symptoms

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Synonyms

Indicators

Definition

Symptoms are physical sensations or changes in internal state that a person recognizes, interprets, and reports (Pennebaker 1982). Although symptoms are a key indicator of disease, it has been shown that perceived symptoms do not always correspond with objective, physiological pathology. Thus, researchers have sought to examine the various factors that can influence the perceptual processing and attributing of physical symptoms.

Because perception involves attention to certain cues while ignoring others, a person is more likely to recognize symptoms if their external world is not supplying them with information or distractions (Pennebaker 1982). For example, a person with a boring job is more likely to report symptoms than one with a fast-paced job. The assumption is that the person with the boring job can focus more attention on his internal, physical state rather than managing the external.

There is much evidence that people selectively search for information when interpreting their symptoms. People tend to focus on information which either confirms their expectations or shows the potential symptoms to be benign (Leventhal et al. 1998; Pennebaker 1982). Situational cues may also influence a person's search for information. For example, a recent outbreak of influenza in one's town may make a person more sensitive to his physiological changes and more likely to interpret his symptoms as signs of the flu. Previous experience with similar symptoms may also prime a person to attribute symptoms as being indicators of a particular disease (Jemmott III et al. 1988).

Finally, researchers have found that individual differences like gender, age, and personality can affect the amount that people report symptoms (Pennebaker 1982). For example, women tend to report more physical symptoms than men, older adults report more than young adults, and those high in the personality trait of negative affectivity report more than those high in positive affectivity. One reason suggested for these differences are differing tendencies to focus on one's internal state, resulting in more attention and recognition of symptoms.

Cross-References

▶ Cognitive Appraisal
▶ Common-Sense Model of Self-regulation
▶ Illness Cognitions and Perceptions

References and Readings

Jemmott, J. B., III, Croyle, R. T., & Ditto, P. H. (1988). Commonsense epidemiology: Self-based judgments from laypersons and physicians. *Health Psychology, 7*, 55–73.

Leventhal, H., Leventhal, E. A., & Contrada, R. J. (1998). Self-regulation, health, and behavior:

A perceptual-cognitive approach. *Psychology & Health, 13*, 717–733.
Pennebaker, J. W. (1982). *The psychology of physical symptoms*. New York: Springer.

Symptoms Inventory

▶ Symptom Scale

Syndrome X

▶ Metabolic Syndrome

Syntocinon (Synthetic Forms)

▶ Oxytocin
▶ Oxytocin, Social Effects in Humans

Syringe Exchange Programs

▶ Needle Exchange Programs

Systematic Bias

▶ Bias

Systematic Desensitization

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Synonyms

Graded exposure counterconditioning

Definition

Systematic desensitization or graded exposure is a behavioral intervention commonly used in the treatment of phobias and other anxiety-related disorders. Individuals with phobias tend to possess irrational fears of stimuli such as heights, close spaces, dogs, and snakes. In order to cope, the individual avoids such stimuli. Since escaping from the phobic object reduces anxiety temporarily, the individual's behavior to reduce the perceived fear is negatively reinforced. The aim of systematic desensitization is to overcome this avoidance by gradually exposing individuals to the phobic stimulus until their anxiety to the fear is extinguished (Sturmey 2008).

Description

Joseph Wolpe (1915–1997)

Joseph Wolpe was a South African-born American doctor. During his work as a medical officer, Wolpe's task was to treat soldiers who were diagnosed with "war neurosis" which is now referred to as post-traumatic stress disorder. It was argued at the time that by talking about their war experiences would lead to a resolution of their symptoms. However, this was not found to be the case, and Wolpe became increasingly disillusioned by Freud's psychoanalytic therapy. It was this which served as a catalyst for Wolpe to discover other more effective treatment strategies (Wolpe 1973).

Wolpe began to investigate behavioral strategies through laboratory experiments. One of his concepts was reciprocal inhibition by which anxiety is inhibited by a feeling which is incompatible such as relaxation (Wolpe 1961). He pioneered the intervention assertiveness training and deciphered that this approach was useful for people who were anxious about social situations. As they learned assertiveness skills, this assisted to minimize the anxiety associated with such situations and in turn relaxed. As this proved to be highly fruitful, it further led to the development of systematic desensitization (Wolpe 1973).

Systematic Desensitization

It was discovered that fears could be learned through the behavioral model of classical conditioning (see ▶ "Classical Conditioning"). Therefore, Wolpe (1973) sought to eliminate the fear response generated by the stimulus and replace it with a competing response of relaxation. The notion of systematic desensitization was based upon two principles of conditioning. The first was that an individual could not produce two different responses to stimuli such as fear and relaxation. Secondly, classical conditioning often involves stimulus generalization which refers to stimuli which are similar and lead to the learned response of fear (Sturmey 2008).

During the process of systematic desensitization, the therapist works in conjunction with the person who has a phobia to ascertain the exact stimulus which triggers the phobia. Next, the individual is taught relaxation-inducing techniques often related to a cue for relaxing. After this stage, the therapist and individual develop a list of fear-evoking stimuli, ranging from very mild to very intense anxiety. This list is referred to as a hierarchy of fears as the stimulus items are listed in order of the intensity of fear they evoke. Then, working gradually the therapist attempts to recondition the person so that the stimuli in the hierarchy become associated with a relaxed response rather than fear. Once the individual can eventually confront the stimulus which originally evoked the greatest anxiety and remains relaxed, then the phobia has been effectively extinguished (Sturmey 2008).

Systematic desensitization has been found to be highly effective in the treatment of phobias (Clark 1963), sexual disorders (Obler 1973), and traumatic nightmares (Schindler 1980).

Cross-References

- ▶ Anxiety Disorder
- ▶ Behavior Change
- ▶ Behavior Modification
- ▶ Behavioral Intervention
- ▶ Behavioral Therapy
- ▶ Classical Conditioning
- ▶ Cognitive Behavioral Therapy (CBT)

References and Further Reading

Clark, D. F. (1963). The treatment of monosymptomatic phobia by systematic desensitization. *Behaviour Research and Therapy, 1*(1), 63–68.

Obler, M. (1973). Systematic desensitization in sexual disorders. *Journal of Behavior Therapy and Experimental Psychiatry, 4*(2), 93–101.

Schindler, F. E. (1980). Treatment by systematic desensitization of a recurring nightmare of a real life trauma. *Journal of Behavior Therapy and Experimental Psychiatry, 11*(1), 53–54.

Sturmey, P. (2008). *Behavioral case formulation and intervention: A functional analytic approach*. Chichester: Wiley.

Wolpe, J. (1961). The systematic desensitization treatment of neuroses. *The Journal of Nervous and Mental Disease, 132*(3), 189–203.

Wolpe, J. (1973). *The practice of behavior therapy* (2nd ed.). New York: Pergamon Press.

Systematic Review

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Definition

Systematic reviews present a descriptive assessment of a collection of original research articles related to a specific research question. These reviews "collate, compare, discuss, and summarize the current results in that field" (Matthews 2006). Campbell et al. (2007) noted that "It has now been recognised that to obtain the best current evidence with respect to a particular therapy all pertinent clinical trial information needs to be obtained." This "overview process" (Campbell et al. 2007) has led to many changes in the way clinical trial programs are developed. They have become an integral part of evidence-based medicine, impacting decisions that affect patient care.

A considerable problem in writing such reviews is the retrieval of all relevant publications in the behavioral medicine literature, although the advent of computerized searchable databases has made this task much less arduous.

While such a narrative review can be very useful in its own right, it can also be the first step in a

two-step process that also includes conducting a meta-analysis. This provides a statistical (quantitative) answer, whereas the authors' conclusions in a systematic review will largely be qualitative.

Cross-References

► Meta-analysis

References and Further Reading

Campbell, M. J., Machin, D., & Walters, S. J. (2007). *Medical statistics: A textbook for the health sciences* (4th ed.). Chichester: Wiley.

Matthews, J. N. S. (2006). *Introduction to randomized controlled clinical trials* (2nd ed.). Boca Raton: Chapman & Hall/CRC Press.

Systems Theory

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Definition

An approach to science that emphasizes unity and wholeness (von Bertalanffy 1968) and views factors that influence phenomena as mutually affecting each other at various levels of complexity.

Description

Systems theory, which became popular in the mid-twentieth century, developed in opposition to some of the philosophical assumptions that permeated the sciences across a variety of disciplines (von Bertalanffy 1968). Specifically, systems theory opposed the idea that knowledge about the universe is best obtained through a perspective rooted in notions of reductionism, mechanism, and objectivism (Midgley 2000). Mechanism assumes that the universe and all phenomena contained within it can be likened to machines, composed of parts that operate in predictable, logical ways. Therefore, traditional scientific theory and methodology seeks to reduce complex phenomena to the functions of the smallest possible parts (reductionism), assuming that such analysis will yield complete understanding and ultimately control, in the area under study (Midgley 2000). For example, a reductionistic approach to psychology may pose that all behavior can ultimately be traced to genetic endowment or chemical reactions occurring at the level of individual brain cells.

Systems theorists, however, argue that the search for simple, linear, cause and effect relationships between variables may lead to the neglect of developing more holistic, comprehensive conceptual models (Midgley 2000), leaving the field of psychology impoverished in a couple of ways. First, mechanistic and reductionist approaches may struggle to explain emergent properties of systems, that is, those qualities of a system that cannot be explained merely as the sum of its individual parts (Bertalanffy 1968), such as the human capacity for agency, creativity, or love. Even the concept of life itself cannot be explained by the activity of individual cells (Bertalanffy), but emerges from a complex system of interacting cells. Second, reductionistic scientific disciplines tend to exist in relative isolation from each other, without attempts at integration that may benefit the individual field, as well as facilitate broader societal improvement. Since system thinking views the boundaries between disciplines as somewhat unnecessary and artificial, a behavioral medicine investigator working from a systems perspective may draw from many sciences to obtain a more holistic understanding of a given topic. Therefore, in contrast to the "zooming in" approach of reductionism, systems thinking values panning outward to examine different levels of contextual factors that may be influencing and be influenced by a particular aspect of a system – including concepts across disciplines.

Ecological models of health behavior (see Sallis et al. 2008) are a good example of research grounded in systems thinking. These models assume multiple influences on health behaviors at various levels, with influences interacting across the levels (Sallis et al. 2008). For example, consider the problem of cardiovascular disease.

A systemic approach to reducing the incidence of cardiovascular disease would aim to target a variety of factors that are interacting to determine one's degree of risk for developing the condition, including biological (e.g., hypertension, high cholesterol), psychological (e.g., hostility), social (interpersonal relationships and support), and behavioral (e.g., diet, exercise, medication compliance) contributors. Such conceptualization in behavioral medicine is often referred to as taking a biopsychosocial approach, a form of systems thinking. A systemic model would also examine contextual factors that affect the individual, such as health care utilization or level of education, which may influence the individual's exposure to preventative medicine and information about healthy eating habits. Further, the approach would likely be mindful of broader social and economic concerns (e.g., unemployment rate, racial inequality) that may, in turn, influence one's access to these resources and thus would also be considered relevant to intervention efforts. These types of ecological models have been developed to further understanding of various health behaviors such as physical activity, tobacco control, and diabetes management (Sallis et al. 2008).

Just as systems theory questions the validity of boundaries between disciplines, it also disputes the boundary between scientists and their objects of study (Midgley 2000). Systems theorists reject the position that one can passively observe reality, standing separate from the object of study, but holds that scientists interact with these objects to actively create reality and interpret observations based on the lens through which they view them (von Bertalanffy 1968). If objectivism is not possible and scientists can never capture "reality," then theories and methodologies become "ways of seeing" that are full of value (Midgley 2000). A critical implication of this shift in thinking is the possibility for theoretical pluralism, wherein investigators value contributions from various perspectives and types of research.

Influence on Psychotherapy

The principles inherent in the systems approach have influenced important shifts in the way psychotherapists understand the process of facilitating client change. First, there has been a shift away from the traditional psychoanalytic approach, which viewed the therapist as a relatively objective figure who could successfully remove his or her self from the process, providing the patient with a "blank screen" on which to project unconscious material for interpretation by the therapist. Modern psychoanalytic approaches, such as the object relations or two-person paradigms, assert that the therapist cannot avoid revealing the self and influencing the therapeutic encounter so as to create it along with the patient (Levenson 1995). When viewing the therapeutic relationship as a system, the interaction between the therapist and patient, including the therapists' behaviors and feelings, becomes critically important and should be considered to aid case formulation and planning interventions. Since a change in any part of a system affects the whole of the system (von Bertalanffy 1968), the therapist's attempts to foster a healthy relationship can encourage improved interpersonal functioning in the patient (Levenson 1995). Second, systems theory gave rise to another popular paradigm for understanding individual and family dysfunction, known as family systems theory. Family systems theory holds that an individual's or family's distress cannot be understood fully by looking at any one person in isolation, but must be viewed as the symptom of problematic patterns of interactions within the larger family structure (Smith-Acuña 2010). Systems therapists therefore seek to improve the functioning of the family as a unit.

Cross-References

- ▶ Ecological Models: Application to Physical Activity
- ▶ Family Systems Theory

References and Readings

Breunlin, D. C., Schwartz, R. C., & Mac Kune-Karrer, B. (2001). *Metaframeworks: Transcending the models of family therapy*. San Francisco: Wiley.

Levenson, H. (1995). *Time-limited dynamic psychotherapy: A guide to clinical practice*. New York: Basic Books.

Midgley, G. (2000). *Systemic intervention: Philosophy, methodology and practice*. New York: Plenum.
Sallis, J. F., Owen, N., & Fisher, E. B. (2008). Ecological models of health behavior. In K. Glanz, B. K. Rimer, & K. Viswanath (Eds.), *Health behavior and health education: Theory, research and practice* (pp. 465–485). San Francisco: Wiley.
Smith-Acuña, S. (2010). *Systems theory in action: Applications to individual, couple, and family therapy*. Hoboken, NJ: Wiley.
von Bertalanffy, L. (1968). *General systems theory*. London: Penguin.

Systolic Blood Pressure (SBP)

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Synonyms

Blood pressure

Definition

Systolic blood pressure is the force exerted by blood on arterial walls during ventricular contraction measured in millimeters of mercury (see Tortora and Grabowski 1996). It is the highest pressure measured; normal range for systolic blood pressure is <120 mmHg.

Cross-References

- ▶ Blood Pressure
- ▶ Blood Pressure Classification
- ▶ Blood Pressure, Measurement of
- ▶ Diastolic Blood Pressure (DBP)

References and Further Reading

Tortora, G. J., & Grabowski, S. R. (1996). *Principles of anatomy and physiology* (8th ed.). New York: Harper Collins College.