Towards a Heuristic Research Model Linking Early Socioeconomic Adversity and Youth Cumulative Disease Risk: An Integrative Review

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Abstract This integrative review draws on previous empirical and theoretical work across multiple research traditions, including psychology, sociology, and neurology, to create a framework detailing pathways linking early socioeconomic adversity in childhood and adolescence to subsequent disease risk later in adolescence and young adulthood. A comprehensive search ultimately led to the socioeconomic adversity and disease risk model for youth, which is a heuristic research framework detailing four overarching pathways focused on early biological/genetic vulnerability, physiological responses (comprised of stress responses and hormonal activity), psychosocial resource trajectories, and behavioral and mental health trajectories as well as potential modifiers of these pathways. Other central constructs include brain development/neuroplasticity and adolescent life stressors. The heuristic research framework calls for an integrated perspective taking a long-view over early life course to explain cumulative physical disease risk by combining knowledge from developmental, social, neurocognitive, and biological sciences.

Keywords Socioeconomic adversity · SES · Disease risk · Stress · Physical health

Introduction

Previous research has documented the persistent association between early socioeconomic adversity (i.e., in childhood or early adolescence) and subsequent physical disease risk in later adolescence and young adulthood (Costello et al. 2003; Wickrama et al. 2015b), which leads to health inequalities in adulthood (e.g., Center on Social Determinants of Health 2008; Cohen et al. 2010; Nurius et al. 2015; Seeman et al. 2010). Here, adolescence is conceptualized as 13–19 years, young adulthood is defined as 25–40 years, and the transition from adolescence to young adulthood, or emerging adulthood, is conceptualized as 19–25 years of age. Previous empirical and theoretical work across several research traditions has identified different, but related, mediating and moderating mechanisms responsible for the connection between early socioeconomic adversity and physical health (e.g., physiological and behavioral processes as well as psychosocial resources and early biological vulnerability) (e.g., McEwen and Gianaros 2010; Miller and Cole 2012; Raposa et al. 2014). That is, the association between early socioeconomic adversity and physical health has been studied in various research domains: biological health, psychological health, and behavioral health and utilizing various lens from various perspectives: stress, resource, and resilience, to name a few. While these advanced studies have yielded a large body of research from which to draw, the lack of integration across disciplines is problematic for the development of a comprehensive understanding of how early socioeconomic adversity contributes to physical health risk.

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To this end, we believe that a review of these mechanisms resulting in testable models within an integrated framework is necessary to incorporate findings from multiple disciplines. This is particularly timely given that multi-dimensional datasets (e.g., data on genetics, biomarkers, and mental and physical health diagnoses) within multi-level, prospective longitudinal research designs are increasingly common. Consequently, the resulting socioeconomic adversity and disease risk (SADR) model serves as a heuristic organizing framework that can guide future research to advance multi-disciplinary research and interventions related to the early socioeconomic adversity-physical health connection. We label our SADR model as a heuristic organizing framework (where heuristic means to find or discover) because it was derived from our own exploration of existing research, both theoretical and empirical.

Our exploration and comprehension of the research was grounded in our understanding of the life course perspective and stress process theory (Elder et al. 2003; Pearlin et al. 2005). Particular life course concepts guiding the creation of our SADR model include a “long view” over the life course, the effects of cumulative influences, stress proliferation, path dependent and exposure dependent pathways, turning points in individuals’ lives, and a focus on the timing and sequence of life events (Elder and Giele 2009; Willson et al. 2007). Here, we build on existing work that has integrated ecological and biopsychosocial approaches to understand health disparities and inequalities. Such work has increasingly emphasized that the study of health and health behavior in isolation from the broader social and environmental context and life course experiences is incomplete (e.g., Ganzel et al. 2010; Solar and Irwin 2010). For instance, Glass and McAtee (2006) proposed a model with both temporal and multilevel structural dimensions for the investigation of health outcomes. The temporal dimension takes into account life course from early life to later life (i.e., upstream to downstream). The multilevel dimension identifies nested hierarchies of structure (i.e., under-water to uphill) each with opportunities and constraints. We expect to strengthen such existing frameworks with the addition of more specific testable mechanisms.

While we do not provide a thorough review of the life course perspective here, we briefly summarize how some of these concepts are relevant to the work at hand. Our heuristic research framework takes a “long view” of the early life course beginning with early stressful life experiences associated with early socioeconomic adversity (or affluence), which proliferate over adolescence and the transition to adulthood as the accrual of sequentially-contingent stressful circumstances (or resources) (i.e., stress trajectories). Also, consistent with the notion of risk (or resource) accumulation and cumulative disadvantage (or advantage) from the life course perspective (Dannefer 2003), we consider

disease risk development as a lifelong adaptive process (Braveman and Barclay 2009; Halfon and Hochstein 2002).

The Current Study

The aim of the current article is to utilize life course and stress process concepts to integrate previous research from multiple disciplines into an overarching and relatively comprehensive framework, which we termed the SADR model. While this broad view limits our ability to provide in-depth detailed discussion about responsible mechanisms, its strength is its presentation of a framework that can be utilized to derive testable models for future research exploring links from early socioeconomic adversity to subsequent physical disease risk. Figure 1 illustrates the SADR model with four pathways stemming from early socioeconomic adversity and leading to increased risk for various physical diseases in late adolescence and young adulthood. Table 1 provides examples of the various constructs contained within the larger heading titles shown in Fig. 1. The four pathways in Fig. 1, which are shown in colors other than white for emphasis, include early biological or genetic liabilities and vulnerabilities as well as three parallel pathways [i.e., physiological (stress response-hormonal), behavioral, and psychosocial resource pathways] or growth trajectories (e.g., Matthews and Gallo 2011; Wickrama et al. 2015b). The three parallel pathways represent not only the initial levels but also growth or development in these attributes over early life course. In Fig. 1, single-headed arrows depict uni-directional, additive influences whereas lines with arrows on each end depict bi-directional, often simultaneous, associations. Table 2 provides details of potential modifiers associated with these pathways.

Method

In the paragraphs that follow, rather than reviewing previous research in detail or discussing disease-specific mechanisms in detail, we provide an overall summary of key findings from existing empirical studies to support the proposed pathways and associations leading to increased physical disease risk as depicted in Fig. 1 moving from left to right. Although, the pathways in Fig. 1 imply sequential processes, it should be noted that some associations depicted in the figure may be simultaneous and bi-directional. The search strategy included a review of APA PsycNet, PubMed, PsycINFO, and Google Scholar databases. Emphasis was placed on articles since 2000; however, seminal and well-cited research prior to 2000 were also incorporated. Primary search terms included: socioeconomic adversity, socioeconomic status, socioeconomic disadvantage, early

Fig. 1 The socioeconomic adversity and disease risk (SADR) model: an integrated research framework linking early socioeconomic adversity and adolescent/young adult cumulative disease risk. The four

proposed pathways are shown in colors other than white for emphasis. Proposed effect modifications for the paths presented here are detailed in Table 2

vulnerability, adolescent stress, puberty, adolescent physical health, young adult physical health, metabolic syndrome, inflammation, blood pressure, health behaviors, and allostatic load. We then sought to include literature (1) published in the English language, (2) incorporating neurobiological research findings into traditional life course stress process models, and (3) focusing on the early life course (infancy through adolescence) or—where appropriate—applying findings from adult populations to early life course development. This search yielded an outline of the organizing framework presented here. As a second step, supplementary searches were performed to flesh out more specific empirical details for each mechanism and ensure the existence of sufficient evidence for the proposed pathway and associations. These supplementary searches relied on key terms from specific articles found in the initial search as well as an examination of citations made by these articles and articles that cited these articles.

Early Socioeconomic Adversity: Socioeconomic Disadvantage and Stressful Life Contexts

Early socioeconomic disadvantage creates a stressful life context for children and adolescents. Early socioeconomic disadvantage is characterized by low

socioeconomic status (SES) and often marked by family characteristics, such as low family income, low parental education, and parents' disrupted marital status, as well as by adverse community characteristics and racial/ethnic minority status (Conger et al. 2010; Evans and Kim 2010). Thus, the influence of early adversity on youth's physical health should be evaluated in the context of the broader social and physical environments in which youth live (Schreier and Chen 2013). Family socioeconomic disadvantage generates stressful life contexts characterized by disrupted family processes, including parents' marital conflicts, ineffective parenting (Conger et al. 2010; Repetti et al. 2002), parental negligence, rejection and abuse (Anda et al. 2006), lack of cognitive stimulation (Stack et al. 2015), stressful family events (Ge et al. 2001), and family economic pressure (Conger et al. 2000). Community socioeconomic disadvantage is reflected by a lack of physical and/or social resources, high residential instability, a high poverty rate, high crime rates, and a high percentage of single parent households; like family disadvantage, these community disadvantages also generate stressful, dangerous, and often threatening, environments for adolescents (i.e., "community stress", Ross and Mirowsky 2009). Furthermore, minority youth (including racial/ethnic minorities as well as other minorities such as LGBT youth) frequently

Table 1 Important concepts comprising the SADR model illustrated in Fig. 1 (example constructs in parentheses)

Early socioeconomic adversity Parental education Family income Parent occupation (work complexity, prestige) Parental Marital Status (previous marital history) Community adversity (poverty rate, unemployment) Race/ethnic minority status (immigration status)	Psychosocial resource trajectories Personality (positive affect) Intelligence (GPA) Perceptions of self (self-esteem, self-efficacy) Psychological abilities (mastery, self control, resistance to peers) Altruism Spirituality Social support (from adults, teacher, peers) Acculturation	Emotional and behavioral stress responses Anxiety Anger Aggression Frustration Impatience Helplessness
Stressful socioeconomic contexts Ineffective parenting (negligence, rejection, and abuse) Lack of cognitive stimulation Family stressful events (parental divorce or conflict, deaths, illnesses) Family economic pressure Community stress (threats, violence) Minority stress Discrimination	Adolescent stressors Synchronization and timing of puberty Stressful life events (SLEs) Relationship difficulties (quality, satisfaction, conflict) Life transition patterns (timing and sequence of life events such as: graduation, marriage, pregnancy, parenthood) Parent–child differential acculturation	Physiological pathway (hormonal activity) <i>Neuroendocrine Systems</i> HPA Axis & SAM axis activation <i>Secondary Systems</i> Metabolic, cardiovascular, and immune system dysregulation
Brain development and neuroplasticity <i>Structure</i> Prefrontal cortex Amygdala Hippocampus <i>Functioning</i> Cognitive processing Emotional regulation	Health behavior and mental health trajectories Substance use (tobacco, alcohol, illegal drugs) Sexual behavior (onset, use of protection, number of partners) Physical inactivity (screen time) BMI Eating Behavior (fat and sugar content, fruits and vegetables) Behavioral withdrawal Depressive symptoms	Accumulating disease risk <i>Domains of health risk</i> Psychological health Cognitive health Behavioral health <i>Multisystem Dysregulation</i> Biomarker measures capturing allostatic load (BMI, BP, HR, A1c)
Early biological/genetic vulnerability Birthweight Pre-term birth disability Illness Impaired/delayed growth Biological programming Genetic expression		

encounter “minority stress” including prejudice and discrimination (Brody et al. 2006; Meyer 1995; Noh et al. 1999; Noh and Kaspar 2003).

Most previous developmental studies have examined these different dimensions of youth stressful life experiences stemming from early socioeconomic adversity one at a time (that is, in isolation from one another). For our purpose, however, we use the terms early socioeconomic adversity and early adversity when referring to both socioeconomic disadvantage and early stressful life contexts. Socioeconomic disadvantage components of early adversity include SES characteristics that are generally considered “structural,” such as lack of parental education. Early stressful life contexts components of early adversity include the stressful experiences related to the structural characteristics of SES. An example of an early stressful life context is parental rejection. This conceptualization of early adversity is in line with the multiple risk exposure notion (Evans

and Kim 2010), which posits that a cumulative measure of the risks associated with early socioeconomic adversity (both structural characteristics and stressful experiences) is a more powerful predictor of later outcomes compared to independent measures of single risk exposures because early risks co-vary. Furthermore, researchers have concluded that an additive formulation of these risk factors, rather than an interactive formulation, more adequately captures adolescents’ exposure to cumulative developmental risks (Bauman et al. 2006). For example, a study utilizing both structural characteristics (i.e., parental occupation) and stressful experiences (i.e., childhood maltreatment and childhood social isolation), found these early socioeconomic adversities independently predicted abnormalities in stress-sensitive biological systems, including nervous, immune, and endocrine/metabolic systems, in young adulthood (Danese et al. 2009). While acknowledging the unique influences of individual structural adversities and different

Table 2 Examples for potential modifiers of the associations depicted in Fig. 1

Association			Example modifiers/resilience factors
Path #	Exogenous Construct	Endogenous Construct	
2a	Early socioeconomic disadvantages/context	Adolescent stressors	Psychological resources (e.g., personality, self-regulation, impulse control, “John Henryism”, “shift and persist”) Social resources (e.g., social support, high quality parenting) Genetic polymorphisms
3a	Early socioeconomic disadvantages/context	Psychosocial resources	Early biological damages, genetic predisposition (e.g., disability, impaired growth, genetic polymorphism)
4a	Early socioeconomic disadvantages/context	Health behavior and mental health	Psychological resources (e.g., personality, self-regulation, impulse control, “John Henryism”, “shift and persist”) Social resources (e.g., social support, effective parenting, positive peer relations, adult role models) Genetic polymorphisms
2b, 2c	Adolescent stressors	Physiological pathway	Psychological resources (e.g., personality, self-regulation, impulse control, “John Henryism”, “shift and persist”) Social resources (e.g., social support, effective parenting, positive peer relations, adult role models) Early biological damages Early stressful life experiences (“stress potentiation”) (e.g., parental rejection, negligence, community stress)
2b, 4b	Adolescent stressors	Health behavior and mental health	Psychological resources (e.g., self-regulation, acculturation, spirituality) Social resources (e.g., social support, effective parenting, positive peer relations, adult role models) Early stressful life experiences (“stress potentiation”)
2e, 4c	Physiological, and health behavior pathways	Cumulative disease risk	Early biological damages (e.g., impaired or delayed growth, physiological “programming”, compromised immune functions) Genetic polymorphisms

stressful experiences on adolescent biological, behavioral, and psychosocial resource pathways, in the present review we highlight the impact of multiple risk exposure on cumulative disease risk.

Early Socioeconomic Adversity and Cumulative Physical Disease Risk

We focus on cumulative physical disease risk in young adulthood (see the bolded boxes in Fig. 1) partly because the consequences of various early socioeconomic disadvantages and stressful contexts are typically non-specific and not limited to one particular disease. Factors that contribute to an increased risk of developing one disease often overlap considerably with the risk of developing a number of other physical diseases. Consequently, the impacts of early social disadvantage may be underestimated in disease-specific studies (Aneshensel 2005). Thus, in the present review, we focus broadly on physical disease risk in young adulthood,

paying particularly attention to a range of cardiovascular and metabolic disease risks, because these increase significantly during the transition from adolescence to young adulthood (Wickrama et al. 2015). Moreover, our focus on cumulative physical disease risk is consistent with research on multiple system dysregulation, which we highlight in the present review.

Early Adversity and Brain Development and Neuroplasticity

The brain is the primary mediator of the pathways in our heuristic research framework because, in part, childhood and early adolescence is a critical time of significant brain growth and cognitive skill development (Lupien et al. 2009). Furthermore, three of the four pathways proposed are linked directly to brain development and neuroplasticity, and these pathway associations are bi-directional, and often simultaneous, with brain functioning impacting and

being impacted by trajectories of psychosocial resources, physiological functioning, and health behavior/mental health. Childhood/adolescence is a sensitive period for many aspects of brain development (Knudsen 2004), which can be impeded by stressful life experiences. As depicted by path ABC, a number of previous studies have provided evidence that childhood socioeconomic adversity, particularly family poverty and parental neglect, detrimentally influence neurobiological development and neurocognitive functioning in the brain (Duncan and Brooks-Gunn 1997; Gunnar and Vasquez 2006; Kishiyama et al. 2009; McEwen 2012; Shonkoff et al. 2009). Also, studies have documented that children raised in family environments characterized by low SES and neglect exhibit growth abnormalities in various regions of brain (e.g., hippocampus and amygdala) (De Bellis et al. 1999; Grassi-Oliveira et al. 2008). Compared to other children, children raised in these environments also average less grey matter in these brain regions (Gianaros et al. 2007; Hanson et al. 2011) and a lower brain volume (Noble et al. 2012). These brain deficiencies are important because several studies suggest that disruptions in brain development and brain functioning during childhood may have long-term implications for stress sensitivity and for behavioral and psychosocial resource trajectories (paths 2c, 3b, and 4b).

Even in adolescence, the brain is not a “finished product” (Konrad et al. 2013). Brain maturation continues during adolescence, particularly the acquisition of higher order functions, such as decision making and impulse control (Gogtay et al. 2004; Lopez et al. 2008). Furthermore, “an imbalance in maturation” of the brain during adolescence has been noted because the subcortical area develops earlier than the prefrontal area (Konrad et al. 2013). This imbalance may result in heightened sensitivity to rewards and reduced sensitivity to goal-directed behavior, which may help explain why adolescence is often a time of increased risk-taking behavior, along with the more normative and adjustive nature of risk taking during this developmental period (Moffitt 1993). However, as in childhood, adolescent brain maturation is also influenced by the environment. For example, a recent longitudinal study found that years lived in poverty across ages 11–18 forecasted diminished hippocampal and amygdala volumes in young adulthood for a control group; however, these effects were ameliorated in a group receiving a family based intervention designed to enhance supportive parenting at age 11 (Brody et al. 2016).

The Early Biological/Genetic Vulnerability Pathway (Pathway 1 in Fig. 1)

Previous studies have documented the mediating and moderating roles of early biological vulnerabilities, such

as low birth weight, pre-term birth, physiological damages, and genetic vulnerability, in relation to the early adversity-health connection (Goodman et al. 2005; Miller et al. 2011; Packard et al. 2011; Repetti et al. 2011). With regard to these mediating influences, early stressful experiences biologically program developing physiological systems, beginning even in utero, and can cause physiological damages (Smith et al. 1997) as depicted by path 1a. Furthermore, because these physiological changes get “under the skin” during this sensitive developmental period (i.e., become irreversible and incubate), they can also compound and accumulate creating adverse trajectories of physiological changes resulting in disease risk years later as depicted by path 1d (Hertzman and Boyce 2010; Miller et al. 2011; Packard et al. 2011; Shonkoff et al. 2009). For example, escalating trajectories of blood pressure from childhood to young adulthood are influenced by adverse childhood experiences (Su et al. 2015). Also, coronary heart disease, hypertension, stroke, and diabetes in adulthood can be traced back to low birth weight and low growth rate in early infancy regardless of adult social status (Barker 1998; Barker et al. 1989; Forsén et al. 1997). Manifestations of this health impact are sometimes obvious in adolescence as well, as evidenced by early elevated levels of regulatory biomarkers, such as blood glucose level and BMI (Goodman et al. 2005). It has also been demonstrated that harsh family environments trigger a proinflammatory phenotype in children, which contributes to the development of chronic diseases as they age (Miller and Chen 2010).

Early biological vulnerabilities also influence the development and consolidation of individual psychosocial resources (or vulnerabilities) (path 1c). For example, low birth weight is associated with low self-esteem, reduced mastery, inhibited psychological and behavioral control, poor educational performance, and poor social relations (Lund et al. 2012; Ribeiro et al. 2011). In turn, these resources are negatively associated with young adult disease risk (path 3d), which we will discuss in detail in a later section.

With regard to moderating influences, early damages and impairments in physiological regulatory systems, as presented in Table 2, may amplify the health impact of experiencing adolescent stressors (Loman and Gunnar 2010; Repetti et al. 2011), as well as of other pathways (e.g., physiology and health risk behavior) leading to subsequent disease risk because early physiological damages (e.g., physiological “programming” including compromised immune functions) increase youth’s health vulnerability (Godfrey and Barker 2001). For example, children’s heightened heart-rate reactivity amplifies the health influence of daily stressors (Gannon et al. 1989).

Gene Expression, Genetic Vulnerability

In this review, genetic vulnerability is conceptualized as a part of early vulnerability or biological imbedding (Hertzman and Boyce 2010) because genetic disposition can be turned off or on depending on early life experiences (Non et al. 2016; Meaney 2010) (path 1a). That is, epigenetic research has shown that experiences, such as adversity, are related to how genes are “expressed” (i.e., phenotypes), and this genetic expression is a determining factor of emotional and behavioral problems (path 1b), psychosocial resources (path 1c), and disease risk (path 1d).

Genetic polymorphisms are variations in the structure of genes, and each variant of a gene is called an “allele”. Most molecular genetic research on youth behavior has focused on genetic variations that regulate neurotransmitter systems, such as serotonergic and dopaminergic neurotransmitter systems. We limit our review to a handful of these alleles or “candidate genes” that have been frequently studied (e.g., 5-HTT, DRD2, DRD4). 5-HTT is a key regulator of serotonergic neurotransmission (implicated in reward sensitivity). Individuals with one or two copies of the 5-HTT “short” allele display more behavioral and psychological difficulties (path 1b) as well as health problems (path 1d) (Gloster et al. 2015; Wargelius et al. 2012). The DRD2 candidate gene is linked to regulating the dopaminergic system (implicated in sensitivity to pleasure and punishment) (DeYoung et al. 2005), and youth with the A1 allele of DRD2 are more likely to encounter difficulties in close relationships (path 1c) (Waldman 2007; Walum et al. 2008). Furthermore, studies have shown that genetic influence increases over the early life course making genetic vulnerabilities more relevant during adolescence than childhood (gene×age interaction) (Shanahan and Boardman 2009).

Genetic Moderation

More importantly, as presented in Table 2, genetic polymorphisms may interact with stressful (or positive) life contexts to impact youth behavioral and mental health trajectories (moderation of path 4a), psychosocial resource outcomes (moderation of path 3a), and physiological outcomes (moderation of paths 2b and 2c). These gene-environment interactions are known as G×E effects. In addition, genetic differences may modify the effect of health risk behavior (e.g., alcohol use, eating behavior) on disease risk (e.g., risk of heart disease and obesity) (representing moderation of path 4c) (Corella et al. 2001; Loktionov et al. 2000).

The 5HTTLPR polymorphism of the 5-HTT gene is one of the most extensively studied genetic variants in G×E developmental studies and has been shown to moderate the influence of adolescents’ stressful life events on their

depressive symptoms (Caspi et al. 2003). The 5HTTLPR allele may also interact with early socioeconomic adversity to influence youth educational attainment, the occurrence of “off-time” life events, and cardiovascular and metabolic disease risk (Wickrama et al. 2016). Other research has shown that the influence of community adversity on adolescent BMI trajectories is greater for those with more sensitivity alleles (e.g., 5HTTLPR, DRD4) than those with fewer sensitivity alleles (Wickrama et al. 2013). Similarly, emerging adults manifest steeper escalating trajectories of drug use when they report a high level of life stress and carry an allele of DRD4 (Brody et al. 2012).

Although research has shown that this polymorphism produces adverse outcomes under stressful contexts (i.e., a diathesis hypothesis), the same gene may also act more generally to enhance environmental sensitivity; thereby producing positive youth outcomes in positive contexts (i.e., genetic plasticity) (Simons and Lei 2013; Wickrama and O’Neal 2015). Furthermore, multiple genes may cumulatively influence youth health and behavioral outcomes (i.e., shared genetic influence) (Belsky and Beaver 2011). Accordingly, individual differences in youth genetic disposition may partially shape developmental pathways beyond socioeconomic adversity (Belsky and Beaver 2011; Simons and Lei 2013; Wickrama and O’Neal 2015).

The Physiological (Stress-Neuroendocrine) Pathway (Pathway 2 in Fig. 1)

As depicted by paths 2b and 2c, the physiological pathway involves the mediating role of the brain in relation to the association between adolescent stressors and stress responses that lead to neuroendocrine hormonal activation (Ganzel et al. 2010; McEwen and Gianaros 2010; Seeman et al. 2010). Over time, repeated and/or continuous stress responses lead to physiological dysregulation through neuroendocrine responses including activity in the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic–adrenal–medullary (SAM) axis because these systems fail to adapt to future stressors. That is, although neuroendocrine hormonal activation may be protective insofar as it provides short-term adaption (i.e., allostasis), if the activation is excessive, it may contribute to physiological dysregulation in multiple biological systems (i.e., allostatic load), eventually leading to diseases with protracted etiologies (Matthews and Gallo 2011; McEwen and Gianaros 2010; Seeman et al. 2010) (path 2e).

Early stressful life contexts increase the likelihood that youth will encounter stressors later in adolescence and young adulthood in a successively contingent manner over adolescence, creating a stress trajectory or pathway (path 2a) (Attar et al. 1994; O’Rand and Hamil-Luker

2005). For instance, stressful life contexts encountered in childhood and adolescence, such as family economic hardship, parents' marital conflict, or community adversity often increase the likelihood that these youth will face additional hardship (e.g., Conger et al. 2002; El-Sheikh et al. 2013; Wickrama et al. 2005). The resulting stress trajectories may include the early onset of puberty (Ge et al. 2001), adolescent stressful life events and circumstances (Wickrama et al. 2015b), romantic relationship problems (Barr et al. 2016), and an off-time (early or late) transition to adulthood, including the acquisition of adult roles such as becoming a parent, cohabiting, and dropping out of school (Lee 2015; Wickrama et al. 2015a, 2005). Furthermore, off-time events and the related adverse transition patterns are chronically stressful for

youth because they often create a “rush to adulthood” for which the adolescent is often not emotionally, financially, and socially/relationally prepared (Foster et al. 2008; Lee 2015; Wickrama et al. 2010).

As depicted by paths 2b and 2c and also detailed in Fig. 2, adolescent stressors invoke physiological responses that produce various neuroendocrine hormones (Packard et al. 2011). That is, the neurocognitive stress process is mediated by the brain as it translates environmental stimuli (e.g., an adolescent stressor) into stress responses (Ganzel et al. 2010; McEwen 2012; McEwen and Gianaros 2010). First, the brain processes information about the stressor, including an appraisal of the threat. Second, the brain regulates the stress response by invoking neuroendocrine activation.

Fig. 2 The stress-physiological pathway: from adolescent stressor to physiological dysregulation in multiple systems

Neuroendocrine Systems

As illustrated in Fig. 2, the primary regulatory systems include the sympathathetic-adrenal-medullary (SAM) axis and the hypothalamic–pituitary–adrenal (HPA) axis (Seeman et al. 2010). When these systems are activated, they produce various neuroendocrine hormones, such as cortisol, epinephrine, and neuroepinephrine, which aid in productive short-term adaptations to stress.

However, exorbitant physiological stress responses (e.g., the excessive production of hormones through the HPA axis and SAM reactivity) are contemporaneously and longitudinally associated with adolescent emotional and behavioral problems (path 2d in Fig. 1) (Hastings et al. 2011; Seeman et al. 2010). In particular, changes in hormone levels associated with puberty have negative behavioral and mental health outcomes for adolescents (Blakemore et al. 2010), and variations in cortisol levels are linked to adolescent alcohol use (Ruttle et al. 2015).

Although these stress responses are thought to be adaptive as the body attempts to maintain stability through change (i.e., allostasis), chronic arousal of the brain and excessive activation of physiological systems can lead to cumulative physiological dysregulation in multiple systems (path 2e). This is because, as shown in Fig. 2, primary regulatory systems are associated with additional peripheral systems, including metabolic and cardiovascular systems as well as the immune system (inflammation); all of which are inter-related (Seeman et al. 2010). Particularly, hormonal activation, such as impaired endothelial functions, may mediate the stress and inflammation (impaired immune function; e.g., elevated levels of C-reactive protein) association (Black 2002) as well as the connection between stress and cardiovascular dysregulation (Ghiadoni et al. 1998). Physiological dysregulation establishes new baseline parameters of these regulatory systems (e.g., elevated blood pressure or blood glucose) (Boulos and Rosenwasser 2004; McEwen 1998; Seeman et al. 2010). For instance, low socioeconomic trajectories from adolescence to adulthood (where SES is defined as parental occupation and then their socioeconomic attainment) are related to long-term cortisol dysregulation (Gustafsson et al. 2010). Taken together, prior literature has provided strong evidence of the brain operating as a mediator between adolescent stressors and stress responses that lead to chronic levels of neuroendocrine hormonal activation with detrimental effects on a host of biological systems.

The Brain as a Target of the Stress Process

Recent stress response models emphasize that dysregulation in multiple systems can also create wear and tear on the brain as depicted by the left-directed arrow of path 2c

in Fig. 1. That is, the association between brain and physiological systems via neural and endocrine mechanisms work in both directions (Ganzel et al. 2010; McEwen and Gianaros 2010). For example, excessive production of stress hormones (e.g., cortisol) can affect the structure of brain regions and their functions resulting in changes in memory formation and memory deficits (Gould et al. 1997; Sapolsky 2000). Additionally, stress hormones associated with early socioeconomic adversity may deleteriously alter the functional connectivity among regions of the adolescent brain. Specifically, it has been demonstrated that, for females, higher socioeconomic adversity in the first year of life predicted higher subsequent cortisol levels, which were a determinant of decreased resting state functional connectivity between the amygdala and components of the prefrontal cortex at age 18 (Burghy et al. 2012). Thus, the brain is not only the primary mediator of the stress process but also a central target of the same stress process.

The Moderating Effect of Early Stressful Life Contexts: Stress Potentiation

Previous developmental research has suggested childhood/adolescent stressful contexts moderate several of the pathways illustrated in Fig. 1. Early stressful life experiences (e.g., stressful parent–child relationships, parental disconnection, lack of nurturing), which can also continue into adolescence, may result in heightened stress sensitivity, impaired self-regulation, and impaired coping mechanisms; all of which are implicated in increased disease risks (Dich et al. 2015; Loman and Gunnar 2010; Lovallo 2013; Repetti et al. 2011; Shonkoff et al. 2012; Bae and Wickrama 2017). That is, early stressful contexts not only influence the occurrence of later, adolescent stressors (path 2a), but also amplify the health effect of these later stressors through paths 2b, 2c, and 2e. This stress amplification is also known as stress potentiation (Loman and Gunnar 2010; Lovallo 2013; Shonkoff et al. 2012).

Moreover, neurobiological studies have shown that neurological processing of stressful life contexts and current stressors occur in different parts of the brain's emotional circuitry (Armony and LeDoux 1997) suggesting stressful life contexts (e.g., parental rejection) and stressful life events are two psychologically distinct constructs. Thus, they may have unique impacts on health outcomes through differential processes (Dich et al. 2015; Lovallo 2013; McCutcheon et al. 2010) and these processes may interact with each other to further influence health outcomes.

The Psychosocial Resource (or Vulnerability) Pathway (Pathway 3 in Fig. 1)

Previous studies have documented that intra- and inter-personal resource (or vulnerability) trajectories mediate the association between early socioeconomic adversity and young adult disease risk (paths 3a, 3b, and 3d in Fig. 1) (Zilioli et al. 2016). More importantly, as acknowledged in Table 2, there are moderating effects of intra- and inter-individual resources on behavioral and physiological pathways.

Developmental studies have documented that early socioeconomic conditions adversely influence the development of psychosocial resources (Donnellan et al. 2009; Whitbeck et al. 1991). Early adversity increases adaptive challenges faced by adolescents who are already dealing with the normative biological, cognitive, and social changes that occur during this life stage (Cicchetti and Rogosch 2002; Conger et al. 1994; Harter 2012; Hildyard and Wolfe 2002; Steinberg 2008). In particular, lower-SES parents are more likely to lack necessary financial and material resources and parental knowledge, utilize a harsher, authoritarian, and rejecting parenting style, and engage in parental neglect and abuse. All of these adverse circumstances have been shown to impair the developmental trajectories of cognitive processing capacity and emotional regulation capacity as well as trajectories of educational attainment, mastery, self-esteem, and future orientation (e.g., Farley and Kim-Spoon 2015; Hampson et al. 2007; Hardy 2006; Melby et al. 2008; Nam and Huang 2009; Oshri et al. 2016; Steinberg 2001; Zhang 2003).

Path 3a also depicts the influence of early socioeconomic adversity on the development of personality traits in adolescents. Socioeconomically disadvantaged youth may lack the environmental exposure and interactions necessary to develop positive personality characteristics implicated in socially desirable beliefs and behaviors. Thus, compared to other youth, socioeconomically disadvantaged youth are more likely to be less agreeable, less conscientious, and have high negative emotionality (Conger et al. 2010; Jonassaint et al. 2011). Furthermore, neuroticism and extraversion are associated with inflammation biomarkers while openness is negatively associated with inflammation biomarkers (Armon et al. 2013).

Health Impacts of Psychosocial Resources

Psychosocial resources are relevant for young adults' disease risk (path 3d). For example, research has shown that psychosocial resources, such as education and self-esteem, have health-protective biological correlates, including a lower cortisol level, better ambulatory heart rate, reduced cardiovascular stress reactivity, and lower rate of disease

(Steptoe et al. 2008, 2005). This may be attributed to increased access to health resources and services (Ross and Mirowsky 2010). This direct path also suggests a neurophysiological mechanism (Zilioli et al. 2016); in that, the association between psychosocial resources and lower disease risk may operate through enhanced brain functioning or increased resiliency to stress (e.g., Center on Social Determinants of Health 2008) (as depicted by the downward arrow of path 3b).

Additionally, maladaptive psychosocial developmental outcomes, such as impaired self-regulation, delayed discounting, increased impulsivity, and low self-esteem, are associated with various health risk behaviors in adolescence, particularly substance use, risky sexual behavior, and physical inactivity (e.g., Farley and Kim-Spoon 2015; Kogan et al. 2015; Oshri et al. 2016) (not shown in Fig. 1). Further still, there may be even more cascading consequences as previous literature has provided evidence that adolescents with impaired self-regulation are more likely to be caught in “snares”, which result in a greater likelihood of being trapped in harmful lifestyles during adulthood (see the downward pointing arrow of path 3c) (Moffitt et al. 2011).

Moderating Effect of Psychosocial Resources

Consistent with the traditional stress process model (Pearlin et al. 2005), and as depicted in Table 2, psychosocial resources can moderate the influence of early socioeconomic adversity on adolescent stress trajectories (path 2a) and adolescent health behavior and mental health trajectories (path 4a).

The moderation of path 2a may be attributed to the fact that individual psychological resources, such as sense of control and self-regulation, aid in avoiding situations that increase the likelihood of stressful events and circumstances. Even when stressful events occur, psychosocial resources may moderate the stress response (depicted by paths 2b and 2c) because individuals with psychosocial resources are generally more proactive in seeking solutions to problems and less traumatized by the occurrence of negative events (Pearlin et al. 1981). Furthermore, social resources appear to be moderators of the genetic influence on health behaviors (path 1b). For example, involved parenting attenuates the link between the 5-HTT gene and substance use (Brody et al. 2009). Conversely, psychological vulnerability, as manifested by a lack of psychosocial resources, amplifies the adverse health influence of stressful life circumstances (King et al. 2015).

However, individual psychosocial characteristics are not fully shaped by socioeconomic adversity (as suggested by path 3a). Instead, a substantial number of adolescents possess healthy levels of psychosocial resources regardless of their

socioeconomic background; these adolescents are capable of leveraging resources to successfully negotiate life challenges in a manner that allows them to altogether avoid, or at least minimize the detrimental impact of, many of the stressors encountered by youth who lack these psychosocial resources stressors (Bonanno 2005; Freitas and Downey 1998; Luthar et al. 2000; Rutter 1999). This represents the resilience of a number of youth who are able to develop psychosocial resources in the face of early adversity. This point is important because this resilience epitomizes why all children born into socioeconomic disadvantage are not destined to experience disease or other negative outcomes.

Furthermore, psychosocial resources also serve as a source of resilience, as these resources allow youth to thrive, or at least avoid more detrimental effects experienced by others, when faced with adversity. For instance, as illustrated in Table 2, adolescent stressors, such as relationship difficulties, are likely less detrimental for the mental health and behavioral health of youth with more psychosocial resources (e.g., self-esteem, mastery, etc). As previously noted, this stress resiliency may also be attributed to the association between psychosocial resources and enhanced brain functioning. Researchers contend that low SES youth who are exposed to positive role models may develop appropriate emotion regulation behaviors in the face of stress while fostering a future orientation because positive role models provide the stable attachment relationships that are necessary for promoting this positive development (Chen and Miller 2012). This allows youth to “shift” themselves in the face of immediate stressors while “persisting” in the broader perspective of life (i.e., a “shift and persist” strategy). Thus, these low-SES youth are less likely to display heightened physiological responses to stressors.

However, recent studies have shown that striving for psychosocial achievement (e.g., educational attainment) in the face of adverse socioeconomic conditions during adolescence may exert a physiological toll on youth years later as young adults (Brody et al. 2013; Wickrama et al. 2016b). This problematic moderating effect of social achievement is consistent with the John Henryism hypothesis, which suggests that positive developmental achievement in the presence of adversity often requires intense physical, mental, and/or social effort (see James 1994 for a detailed explanation of the hypothesis). Such intensity is often stressful and, consequently, increases the risk of disease.

The Behavioral and Mental Health Pathway (Pathway 4 in Fig. 1)

As shown by paths 4a, 4b, and 4c in Fig. 1, health risk behaviors, including substance use, poor eating habits, a

sedentary lifestyle, and risky sexual behavior, as well as poor mental health trajectories mediate the association between early socioeconomic adversity and youth disease risk. In addition, the physiological pathway connects early socioeconomic adversity to emotional and behavioral problems through various paths (e.g., the downward pointing arrow of path 2d). That is, research suggests that these associations operate through both neurophysiological and structural mechanisms.

Regarding structural mechanisms, numerous studies have found that family and community socioeconomic characteristics influence an individual’s health behaviors (path 4a) (Shin and Miller 2012). Adolescents from poor families often lack health resources, including access to healthy food, recreation facilities, and health services (Fitzgibbon et al. 1998). Furthermore, disadvantaged parents often transmit their unhealthy behaviors and risky lifestyles to their offspring (Wickrama et al. 1999). Poor families are often located within poor communities, which further hinders healthy behaviors because these communities are often unable to meet their residents’ dietary and health-related needs. For example, not only do unaffordable prices limit access to healthy foods in poor communities, but poor communities also have a greater number of unhealthy fast food restaurants compared to higher income communities (Kipke et al. 2007). Disadvantaged communities also often lack recreational activities, safe spaces for engaging in physical activities, and nearby health services (Wen and Maloney 2011). Studies have also documented the lack of social resources in disadvantaged communities. The lack of community support, positive role models, and close relationships, act as deterrents for a healthy lifestyle while simultaneously encouraging risky healthy behaviors (Umberson et al. 2008; Wickrama and O’Neal 2015).

Family and community adverse conditions and ethnic minority status also increase youth’s risk for emotional problems (path 4a). A lack of physical and/or social resources and opportunities associated with socioeconomic adversity combined with a threatening community environment, poor housing, and related physical hazards as well as racial discrimination often lead adolescents to feel trapped, angry, hopeless, and frustrated (Brody et al. 2006; Prawitz et al. 2013; Ross et al. 2000; Wickrama and Bryant 2003). These negative feelings make youth more susceptible to developing emotional and behavioral problems, such as depressive symptoms, substance use, and risky sexual activities (Kerrigan et al. 2006; Kogan et al. 2010; Wheaton and Clarke 2003). Using growth mixture modeling, previous studies have shown that youth depressive symptoms trajectories are socially stratified, and adverse depressive symptoms trajectories are associated with various health risk behaviors including risky sexual activity, crime, and substance use (Wickrama et al. 2016a, 2010). Furthermore,

these negative feelings play a role in the adoption of health risk behaviors (Browning and Cagney 2003; Kogan et al. 2010). Research has shown that emotional eating (eating more foods that are high in fat and less fruits and vegetables) is often utilized by distressed individuals as a coping mechanism for experiencing negative emotions (Macht 2008; Macht et al. 2005).

Behavioral trajectories are also influenced by brain development and functioning in childhood (path 4b). Also, heightened reward sensitivity during adolescence may contribute to risk taking in the form of risky health behaviors, particularly substance use (Chambers et al. 2003; Chein et al. 2011; Steinberg 2004). However, recent studies have shown that heightened reward sensitivity in adolescence may decrease risk taking behaviors if adolescents are engaged in prosocial activities (Telzer et al. 2013). Conversely, psychosocial resources, such as positive affect, self-regulation, self-esteem, social integration, and education reduce the likelihood of youth engaging in health risk behaviors. For example, extant literature has demonstrated that higher reward sensitivity is associated with earlier onset of substance use for adolescents with low inhibitory control (measured via both behavioral and neural indicators), but not among adolescents with high inhibitory control (Kim-Spoon et al. 2016). This association may be attributed to a neurological mechanism as enhanced brain functioning is associated with psychosocial resources (the downward pointing arrow of path 3b).

In addition, as depicted by the downward pointing arrow of path 2d, physiological stress responses, such as elevated levels of stress hormones, also have behavioral and emotional consequences, including the consumption of an overall less healthy diet, a sedentary lifestyle, and alcohol use (Konttinen et al. 2010; Ruttle et al. 2015). Conversely, as depicted by the upward pointing arrow of path 2d, there are physiological consequences of elevated levels of depressive and anxiety symptoms (Culpepper 2009; Fagundes et al. 2013). Also, as depicted by the left-directed arrow of path 4b, health risk behaviors (e.g., ongoing adolescent substance use) impair brain functioning (for a review see Squeglia et al. 2009).

As depicted by path 4c, it is well documented that health risk behaviors and poor mental health increase youth disease risk (Culpepper 2009; Raposa et al. 2015). For example, studies suggest that the consumption of a less healthy diet, physical inactivity, and sedentary behavior contribute to an increased risk for metabolic diseases, including obesity and diabetes (Biro and Wien 2010; Steyn et al. 2004). More importantly, these unhealthy behaviors have been shown to have proinflammatory effects related to disease risk (Stringhini et al. 2013). Furthermore, psychobiological research has shown that elevated depressive, anxiety, and hostility symptoms also contribute to increased disease risk

through dysregulation of physiological regulatory systems (the upward arrow of paths 2d and 2e) (Culpepper 2009; Fagundes et al. 2013).

Discussion

The SADR model draws heavily from recent studies on the stress process in adulthood and identifies the brain as a mediator, as well as a primary target, of the stress process. Consequently, our review led to the conclusion that the physiological pathway involving the brain plays the most prominent mediating role linking early and adolescent stressors to young adult disease risk. The prominence of this mediating pathway may be specific to adolescence as it is a time of rapid physical and cognitive development. To further explore this possibility, there is a need for more neurodevelopmental research focused specifically on adolescence while investigating these reciprocal influences in the context of brain development and maturation, especially given the cascading influence of these processes over the rest of the lifespan.

The current review emphasizes several important aspects for future research on youth's health outcomes. Across all of these avenues for future research is the need for more advanced cutting-edge analytical techniques involving multi-level data and longitudinal repeated measurements. First, future research investigating multiple dimensions of early socioeconomic adversity is needed. Such research should include both additive and multiplicative health impacts of multilevel factors (e.g., family, race, community) and should also capture both negative (e.g., ineffective parenting, community disorganization) and positive (e.g., effective parenting, collective socialization, and community affluence) dimensions of adversity. Similarly, there is also a need for future research incorporating both stress and resource pathways leading to young adult health outcomes. Second, future research should properly integrate the pathways proposed here, which include capturing psychosocial, biological (biomarkers), genetic (genetic polymorphism), and neurological changes simultaneously, within a "long view" perspective utilizing prospective data. Third, in addition to conducting research assessing additive and linear influences of these pathways, future research should focus on the multiplicative health influences of different pathways (e.g., health impact of conjoint mental health and physiological trajectories). Fourth, there is a need to identify additional modifiers of these pathways that are specific to adolescence. While the framework identifies the moderating effects of socioeconomic contexts and psychosocial resources on the physiological and behavioral pathways to accumulated disease risk (see Table 2), studies of these

moderating effects are somewhat underdeveloped, particularly as they relate to adolescence.

The material and biopsychosocial pathways that we discussed in the current review stem from early socioeconomic adversity and lead to young adult health inequalities. That is, in many instances, an early stressful socioeconomic context appears to be a primary impetus for subsequent health inequalities in young adults. Such a model, then, suggests the need for macro-level policy formulations and early prevention strategies addressing early socioeconomic adversity itself (Center on Social Determinants of Health 2008). These strategies are in addition to the previously discussed resilient, modifiable pathways that serve as potential local and grass-root level intervention points. Thus, federal, state, and county level policies and programs should be designed to reduce socioeconomic disadvantage and inequalities as this offers a strong preventative method for inoculating against the consequences of an early stressful context. More specific policies and programs are likely to develop from research testing aspects of the current heuristic organizing framework.

Relatedly, there is a need for additional interventions aimed at reducing the impact of early adversity on the initial levels and shapes of physiological, behavioral, and resource trajectories. It is imperative that the efficacy of such interventions be assessed so successful interventions can be adopted more broadly. For example, family-based interventions, such as the Family Check-Up (Fosco et al. 2013) and Strong African American Families program (Brody et al. 2016), have demonstrated efficacy in reducing exposure to early life adversities while positively altering said trajectories. Implementing interventions to reduce experiences of early adversity may also be able to ward off early biological damages with long-term health impacts.

This review study also brings to light several important practical implications for interventions targeting early socioeconomic adversity and subsequent health disparity outcomes. First, interventions should aim to target physiological mechanisms as well as the associated behavioral mechanisms (e.g., promoting proper eating habits and physical activity) earlier in the life course because infancy and childhood have been identified as life stages that are more malleable to positive influences in (e.g., parental attachment, cognitive stimulation). Targeting these mechanisms at earlier life stages can effectively reduce the incidence and severity of health risks in later years. Interventions focused on early brain development (Tomprowski et al. 2011), which is influenced by early socioeconomic context, are likely to have long-term positive effects. Such programs that work with parents to foster brain development in their young children can prevent and minimize early physiological damages and aid healthy physical and brain development. Also, the current review highlights the intervention

and prevention opportunities stemming from the psychosocial mechanism, which includes self-control, mastery, and self-esteem, and related opportunities for intervening with family processes, including affective parenting and close relationships. In addition to operating as a linking mechanism, interventions targeting the psychosocial mechanism may prove particularly efficacious because psychosocial resources appear to be a primary moderator of multiple pathways responsible for the adversity-health association. Because these psychosocial resources can bolster resilience to adversity, psychosocial resources represent a protective factor that can be leveraged to buffer youth who do encounter socioeconomic adversity from experiencing its negative consequences. More applied research is necessary to identify promising procedures for impeding in the adversity-health association. In many instances, such procedures may already be active in prevention/intervention projects in various settings (churches, schools, community centers).

Conclusion

The SADR model illustrated in Figs. 1 and 2 and Tables 1 and 2 serves as a heuristic organizing framework combining past empirical and theoretical evidence and calls for an integrated perspective taking a long-view over the early life course to explain disease risk by combining knowledge from developmental, social, neurocognitive, and biological sciences. The present review does not discuss specific mechanisms in an in-depth manner; instead, it identifies a broad comprehensive framework comprising four overarching pathways: early biological damages/epigenetic regulation as well as physiological, psychosocial, and health behavior/mental health mechanisms linking early socioeconomic adversity to subsequent physical disease risk. Our objective in creating the SADR model, as a comprehensive framework, is to provide a foundation for research conceptualizing physical health risk and its determinants, as well as moderators of these associations, more broadly in order to more accurately assess the various pathways implicated in physical health risk. Such comprehensive research accounts for multiple pathways simultaneously and considers adolescence as a life stage with deep connections to both preceding and following stages rather than an isolated period of development. Such research can have long-lasting implications for advancing the field's understanding of adolescent development.

Author Contributions KASW conducted the comprehensive literature review, conceived the model, and drafted the manuscript; CWO participated in reviewing the literature, constructing and organizing the model, and manuscript preparation; CH provided neurobiological and psychological expertise. All authors read and approved the final manuscript.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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