



Thinking Slow More Quickly: Development of Integrated Illness Scripts to Support Cognitively Integrated Learning and Improve Clinical Decision-Making

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Abstract

Illness scripts describe the mental model used by experienced clinicians to store and recall condition-specific knowledge when making clinical decisions. Studies demonstrate that novice clinicians struggle to develop and apply strong illness scripts. We developed the Integrated Illness Script and Mechanism of Disease (IIS-MOD) map framework to address this challenge.

Keywords Illness script · Cognitive integration · Curriculum development · Teaching and learning · Basic science education

The term “illness script” describes the mental model by which a clinician organizes, stores, and retrieves from long-term memory key concepts and their relationships to clinical problems (i.e., diseases, conditions, or syndromes) [1]. Experienced clinicians draw from a repertoire of scripts to drive purposeful clinical data gathering, compare and contrast diagnostic hypotheses, and direct initial management decisions [2]. Scripted knowledge of each condition is organized into three main components: Enabling Conditions (predisposing epidemiologic and structural factors that

influence a patient’s probability of the disease), Fault (the underlying pathophysiological insult), and Clinical Consequences (the patient’s chief concern, signs, and symptoms to which the Fault gives rise) [1, 2]. Importantly, research demonstrates that how a clinician “encapsulates” relevant information within these components facilitates or hinders accurate and efficient information retrieval during clinical decision-making [1].

The development of a holistic, organized knowledge base and a strong foundation of core illness scripts is a principal goal of health professions training. Initially, clinical learners must rely heavily on basic science understanding of core concepts and underlying pathophysiological mechanisms (e.g., the Fault) to slowly reason through clinical hypotheses and competing diagnoses. Not surprisingly, effective cognitive integration of basic and clinical science knowledge has been shown to enhance diagnostic accuracy of novice clinicians [3, 4]. However, left to their own devices’ learners will make connections, but seldom do they make the correct connections between biomedical knowledge and clinical features [3, 4]. The problem is further compounded by the difficulty basic science and clinical faculty face in “unpacking” their own deeply interconnected and encapsulated knowledge while teaching and collaborating [1].

We developed the model of an Integrated Illness Script (IIS) and related Mechanism of Disease (MOD) map to address these challenges and accelerate the development and effective use of scripts by faculty and learners (Fig. 1). The IIS provides

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ASTHMA: INTEGRATED ILLNESS SCRIPT (IIS)

OVERVIEW

Asthma is defined as a chronic reversible obstructive lung disease interspersed with periods of exacerbation that can be broadly classified into two forms: atopic (allergic) or non-atopic (non-allergic) asthma. Triggers for atopc asthma include common allergens such as pet dander, pollen, and dust mites. Triggers for non-atopic asthma include exercise, cold air, smoke, and physical exertion. Environmental factors such as weather changes, air pollution or occupational exposures, pharmacologic agents, and psychological or physiological stress.

The underlying pathophysiological insult in asthma is an airway inflammatory response that results in persistent airway epithelial inflammation, airway hyperreactivity, bronchoconstriction, airway remodeling, and hypersecretion upon repeated exposure to the triggers. In atopc asthma, an individual develops allergen specific IgE antibodies that bind to a high affinity receptor (Fc epsilon RI) on mast cells, a process known as sensitization. In a sensitized individual, the subsequent exposure and binding of the antigen to IgE on mast cells triggers activation of mast cells. Both IgE and IgG antibodies, as well as non-atopic triggers activate mast cells to degranulate and release inflammatory mediators such as histamine, prostaglandins, and leukotrienes, which causes prolonged tissue damage after mast cell activation to cause an acute phase of exacerbation that includes airway smooth muscle contraction and increased vascular permeability. Cytokines such as IL-4, IL-5, and chemokines are released later to recruit inflammatory cells rich in eosinophils, which causes prolonged tissue damage as a late phase of exacerbation, developing 2 to 4 days after the exposure. These mechanisms contribute to airway remodeling.

Genetically, asthma has been recognized as a problem of chronic inflammation of the airways, leading to bronchoconstriction and mucus hypersecretion that impact air flow, leading to tissues of oxygen and carbon dioxide regulation that manifest as hypoxemia and wheezing. These changes stimulate pulmonary peripheral nervous system receptors and central nervous system centers that lead to prominent clinical features of cough and dyspnea.

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EPIDEMIOLOGY

Approximately 300 million people are affected by asthma worldwide, with over 300,000 annual deaths, most of which occur in developing countries. Asthma may affect children or adulthood. Most children who have asthma will not have symptoms until adolescence, but symptoms may recur in adulthood, depending on environmental and lifestyle factors. In the United States and most developed countries, approximately 5% to 12% of adults and 15% of children live with asthma. The male to female ratio is 1.1 in adults and 2.1 in children. The mechanisms for persistence in childhood are not known.

Asthma:

Asthma is a phenotypically heterogeneous syndrome. Patients with asthma are more responsive to a wide range of triggers compared to people without asthma, reflecting a combination of genetic, epigenetic, and environmental risk factors. Individuals with childhood onset asthma are more likely to have a family history of asthma, atopic dermatitis (eczema), or allergic rhinitis. Genetic studies of asthma indicate that the condition can be related to alteration of both immune and epithelial barrier functions.

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Clinical Feature (example, 1 of 4): DYSPNEA

Why? Dyspnea is a symptom of breathing discomfort which is described as chest tightness, increased work of breathing, or difficulty by patients. In asthma, dyspnea is a common symptom. It is due to the microvascular (i.e., hyperperfused) and mechanical (i.e., altered airway patency) mechanisms that result from the airway inflow. Bronchospasm causes narrowing of the airways resulting in hyperinflation of the lungs. Mechanoreceptors (e.g., stretch receptors, J-fibers, etc.) located in the airways and chest wall sense hyperinflation and patients sometimes feel they can't take a deep breath. Additionally, peripheral sensory chemoreceptors detect hypoxemia while central sensory chemoreceptors detect an increase in carbon dioxide levels, which can develop from V/Q mismatching. The receptors in the lungs and brain then send signals through the vagus nerve to the respiratory center in the medulla oblongata, and then relayed to the somatosensory cortex and limbic system, where they are perceived as dyspnea and drive further activation of the respiratory system. This may be detected on physical exam as increased work of breathing, use of accessory muscles of respiration, and substernal or intercostal retractions.

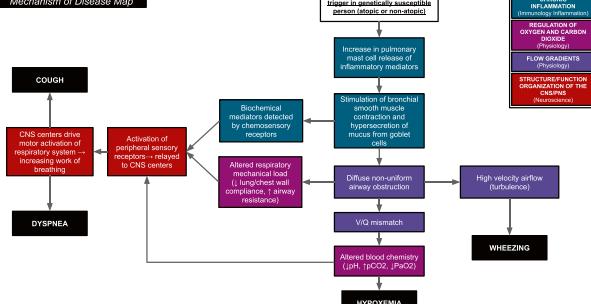
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ASTHMA: MECHANISM OF DISEASE (MOD) MAP

Asthma Mechanism of Disease Map



ASTHMA: CORE CONCEPTS

Chronic Inflammation (Immunology Inflammation)

If the initial cause of an inflammatory response persists, a chronic inflammatory response ensues. Chronic inflammation drives the pathogenesis of many acquired and congenital diseases, and is characterized as an aberrant and prolonged immunopathologic response to persistent infection (e.g., hepatitis), immune-mediated inflammatory processes (e.g., atherosclerosis), and repeated exposure to exogenous or endogenous toxins (e.g., cigarette smoke). Chronic inflammation involves complicated cytokine networks and cellular interaction and induces progressive changes in types of cells recruited to the sites of inflammation. Tissue injury occurs in the lungs in the form of airway remodeling, airway obstruction, and fibrosis. These processes, often incomplete, repair response. With regards to chronic persistent infections and premunition, the interplay between cellular processes and the microbe subverts the mechanisms of chronic inflammation and tissue injury and resulting clinical signs and symptoms.

Structure/Function Organization of the CNS/PNS (Neuroscience)

Neurons and axons are organized into functional groups and tracts/nerves, respectively, that when disrupted will lead to characteristic cognitive, emotional, motor, somatosensory or special sensory deficits.

Regulation of Oxygen and Carbon Dioxide (Physiology)

Oxygen and carbon dioxide are transported by coordinated functions of the circulatory system, respiratory system and oxygen carrying capacity (red blood cells). Alterations in any of these areas can cause problems with gas exchange and thus are highly regulated. Oxygen is required for aerobic cellular metabolism and is highly regulated by peripheral chemoreceptors. CO₂ is a byproduct of metabolism that is primarily regulated by the central chemoreceptors in the medulla oblongata as well as peripheral chemoreceptors to a smaller degree. Hypoxia can lead to cellular and tissue dysfunction. Changes in the amounts of CO₂ can lead to pH changes which can affect cellular function.

Flow Gradients (Physiology)

Substances in the body move passively from high concentrations to low concentrations according to laws of diffusion. Air and blood also use flow-down gradients but also abide by scientific principles based on mass and energy, such as Poiseuille law, Laplace Law, the Law of Diffusion and Fick's Law. Changes in the number of ions and action potentials, molecules or nutrients such as glucose, amino acids, vitamins and other minerals also can be absorbed/secreted and movement is affected by physics and chemical properties.

Fig. 1 Integrated Illness Script (IIS) and Mechanism of Disease (MOD) map for asthma

support for inductive reasoning from the observed features back through the relevant mechanisms and basic science concepts to the originating insult. The MOD map, on the other hand, provides a holistic and deductive visual representation of the clinical path: from the original insult, through the causal mechanisms and their corresponding concepts, to the resulting clinical features seen at presentation. The Overview section of the IIS provides a brief clinical definition, a description of the initiating pathophysiological insult, and a concise summary of the most salient basic science concepts impacted by the underlying disease. The Epidemiology section describes risk factors and their underlying mechanisms, highlighting pathology and genetics concepts. The Key Clinical Features articulate the most common presenting clinical findings as well as the basic science causal mechanisms that explain why each feature occurs in the condition. Links to core basic science concepts underlying the mechanisms are included (www.aquifersciences.org). The MOD map flows consistent with the manner a basic scientist would explain the occurrence of a given feature in a known disease and provides an integrated scientific view of the condition. In each section, annotated references are provided to supply supporting evidence for emerging and cutting-edge concepts and mechanisms.

The structure of the IIS-MOD map was created through an iterative multidisciplinary and multi-institutional

consensus process of leading clinical and basic science educators and validated through multiple national workshops over a 3-year period. The design was advanced by six nationally selected pilot schools with teams of basic science and clinical educators and senior medical students working in rapid cycle prototypes to create content for fifty exemplar IIS-MOD maps. To date, six medical schools are utilizing the IIS and MOD map models as pedagogical tools to educate learners at all levels.

For novice learners, the IIS and MOD map can provide a cognitively integrated framework with which to develop, apply, and elaborate basic science knowledge in a manner that supports emerging clinical decision-making skills. For experienced basic science and clinical faculty, the IIS and MOD map facilitate the difficult task of “unpacking” and making transparent deeply encapsulated knowledge to learners and collaborating colleagues. Importantly, the development and use of the IIS have enabled more effective collaborative instructional design and teaching between scientists and clinicians, and between scientists across disciplines. We believe the use of IIS-MOD maps has the potential to enhance curriculum development, teaching, and learning and to advance the value and safety of patient care by both novice and experienced clinicians.

Declarations

Conflict of Interest On behalf of all authors, the corresponding author states that there is no conflict of interest. Aquifer, Inc. is a federally recognized non-profit health professions education organization, and therefore, I have no commercial or financial interest to disclose. Aquifer's courses are developed and maintained by faculty and student contributors, and in partnership with other national health professions education organizations and sustained by subscriptions. Leslie Fall, Jackie Short, and Amy Wilson-Delfosse receive salary support from Aquifer. The remaining authors receive an honorarium for their contributions.

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