

Origins of Chronic Illness: The Role of Trauma

Excerpt adapted from:

*A New Model for Understanding the Role of Environmental Factors in the Origins of
Chronic Illness: A Case Study of Type 1 Diabetes Mellitus*
Medical Hypotheses (2004) Vol 63, issue 6, pp 1035-1046.

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Elsevier B. V., Science Direct: Medical Hypotheses
Available online June 2004 at <http://www.sciencedirect.com/mehy>

Abstract

This article describes research on the role of environmental factors affecting autonomic regulation and predisposition for states of sympathetic and parasympathetic dominance. Perspectives regarding the role of environmental factors in physiological regulation contribute to our increasing understanding of the impact of gene-environment interactions in the origins of chronic illness such as type 1 diabetes. This excerpt emphasizes research on the role of traumatic stress.

II. TRAUMATIC STRESS

Definition and physiology

The definition of traumatic stress has been summarized as the *experience of an event that is perceived by a relatively helpless individual to be both life threatening and inescapable* [1, 2]. Although the delineation between different types of stress has been difficult to fully elucidate, useful factors in the characterization of traumatic stress are that it is associated with extreme fear and high emotional charge [1, 3, 4].

Highly emotional experiences promote the imprinting of an event [5], along with associated contextual

environmental and physiologic cues, into procedural (unconscious) memory [1, 6]. In comparison with other types of conditioning, fear conditioning can occur following one trial and can be extremely long-lasting [1]. The imprinting of highly charged experiences creates conditioned responses that even when extinguished remain stored in memory and available for activation following exposure to an environmental trigger [5]. These characteristics of the nervous system represent innate survival strategies that serve as internal references to promote survival by enabling the organism to respond to future experiences with enhanced rapidity and efficiency [7].

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Traumatic stress is capable of reprogramming the HPA axis and affects ANS regulatory function, resulting in states of exaggerated autonomic cycling and arousal [8], especially in response to stress [1]. Traumatic stress can also result in states of prolonged sympathetic arousal [1, 9] as well as disruption in the interdependence between the two branches of the ANS [7].

Risk for ANS dysfunction

Kindling

The term kindling dates from studies of rats in which the repeated use of low intensity electrical stimulus produces or kindles seizures that become self-perpetuating and that require no further stimulus for seizure activity to occur [6, 10]. The seizures are the result of new circuits that are activated and kindled [1] by physiologic and psychological responses to internal and external cues [6, 11] increasingly distinct from the original learned trigger or conditioned stimulus [5]. The conditioned responses that imprint during traumatic stress appear to be mediated by changes in the hippocampus occurring at the time of high arousal [1]. They may also take place through the creation of new cell assemblies, in which neurons that initially fire spontaneously together in response to certain stimuli are more likely to eventually fire without obvious stimulation, a Hebbian model referred to by Ledoux [5]. The concept of *kindling* is utilized in the neurological literature [1, 6, 10] and in models of traumatic stress [12, 13].

Reinstatement

Conditioned responses occurring as a consequence of traumatic stress may be quiescent or inaccessible for long periods of time, but appear to remain in procedural memory [5]. Following exposure to an

event similar to the original stimulus [14] or to some unrelated stressful experience [5, 6] these memories can be kindled and the conditioned responses “reinstated”. Conditioned responses associated with PTSD such as hyperarousal become activated with increasing ease following exposure to adverse environmental cues. The effects of traumatic stress are consequently considered to be intrinsically self-perpetuating [1, 13].

The role of perception

Although certain types of events such as earthquakes or the witnessed murder of a loved one are more likely to be experienced as traumatic because of their overwhelming nature, it is the perception of an event rather than the specific nature of an event that influences kindling, reinstatement, and risk for ANS dysregulation [1, 2]. Risk is influenced by a number of factors, including intensity [15] and degree of life threat perceived during the original traumatic stressor [16], previous experience of trauma [17], and subsequent exposure to seemingly irrelevant as well as related stressors [3]. ANS function is also affected by the perceived availability and quality of social supports [3, 18] and buffers [3], history of bonding and attachment [1, 3, 16], and stage of emotional and physiological development, all of which influence the capacity to regulate arousal and to cope with stress [19]. Because experiences are unique to each individual, the determination of whether an event is experienced as traumatic, as well as risk for kindling, reinstatement, symptomatology, and disease [7, 20] appears to be idiosyncratic [1].

Risk in early life

Stress in early life influences the immune and nervous systems, among others [21]. Children are at increased risk

of perceiving stressful events as intense and life threatening because of their inherent dependence and relative helplessness [22], and are consequently particularly vulnerable to PTSD [1]. Risk for adverse physiologic consequences is believed to be due to the high degree of nervous system development [23] that occurs during childhood and to the fact that immature nervous systems are incapable of regulating states of high arousal [7]. A strong bond has been found to promote while a disrupted bond to interfere with the ability of a child to regulate arousal [24] and early separation from parents has been identified as an important risk factor for PTSD [25]. The experience of traumatic stress in the context of relationship with another human being is one of the most influential risk factors for ANS dysregulation and PTSD [1, 3]. Secure attachment, on the other hand, is one of the most protective factors against the development of symptoms following exposure to traumatic stress [22].

Prenatal stress

Early exposure to non-genetic factors such as stress in prenatal life stimulates the fetal HPA axis [26], can permanently affect the number and sensitivity of glucocorticoid receptors, and can program the HPA axis for life [26, 27]. Number of glucocorticoid receptors has been found to be proportional to the severity of symptoms of PTSD [28]. Maternal exposure to prenatal stress has also been found to predict birth size and gestational age independent of biomedical risk [29-31], and to influence physiological as well as psychological development postpartum [32]. Size at birth appears to be influenced by the timing [29] and quality [26, 29] of emotional stress experienced by the mother during

pregnancy, as well as by her perceived availability of social support [30].

Rate of progression

The rate at which reinstatement occurs following traumatic stress is idiosyncratic and symptoms of PTSD can occur weeks, months or years following the original event [1, 3]. High intensity initial and subsequent exposure to relevant stressors promotes a more rapid response of neurophysiological hyperreactivity [22] and increases the rate with which reinstatement can occur. Exposure to insufficient stimuli may allow symptoms to resolve after reinstatement or enable a dormant conditioned response to remain inaccessible [5]. Although the development, progression, and resolution of symptoms of PTSD are variable and occur in relatively few individuals following traumatic stress [33], symptoms are often remarkably refractory to treatment once they develop [3].

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