Colorado Psychological Association
Spring Conference 2006
Research Poster

Environmental Origins of Disease

The Role of Stress, Trauma, and Timing

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Research

No cure
Environment affects risk
Paradoxical findings

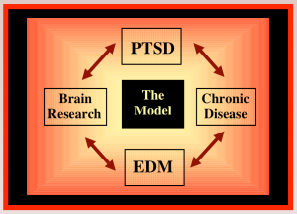
Abstract

Background

There are no cures for chronic disease and existing treatment is often ineffective or associated with significant side-effects. Non-genetic risk factors significantly affect risk for disease, however, and present an opportunity for the development of successful prevention and treatment strategies. Despite hypotheses that events in early life initiate chronic disease and that stressful events often precede diagnosis or exacerbate symptoms, the role of environmental factors is poorly understood and findings are frequently conflicting.

Methods

Through the process of *Grounded Theory*, a comprehensive model for understanding the role of environmental events in risk for disease is being developed and refined through an iterative process [1-3]. Research from the "Decade of the Brain" provides powerful new insights for understanding the role of life events in the origins and expression of chronic illness. Insights drawn from research in traumatic stress (reviewed in [4-7]) and experience-dependent maturation (reviewed in [8-11]) suggest that 1) traumatic events in early life initiate disease during critical periods of organ development, 2) exposure to stressful events related to these original traumatic events perpetuate and may precipitate disease, and 3) stressful events that trigger symptom exacerbations relate to previous traumatic experiences unique to each individual. This information is utilized to examine findings from clinical, physiological and epidemiological research in chronic disease.



Grounded Theory

Objectives

- Clarify the role of environmental events in the initiation, perpetuation, precipitation, and expression of chronic disease and disease complications.
- Integrate perspectives from existing studies in brain research with clinical, physiological, and epidemiological studies in chronic disease.
- Identify and evaluate effective treatment and prevention strategies.

Develop a model

Understand origins
Integrate brain research
Work from a new paradigm

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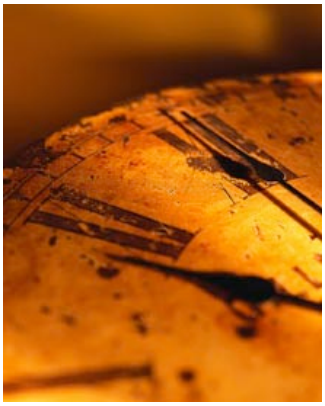
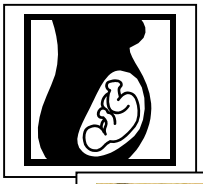


H1: Timing determines disease specificity

H2: Intensity affects risk
H3: Stressors are idiosyncratic



Findings

A comprehensive model has been developed that appears capable of explaining and predicting the impact of environmental events in association with risk for disease. Similar trajectories leading to the development of chronic illness have been elucidated for physical disease as well as mental illness.

Hypothesis 1: Timing of traumatic stress during critical periods of organ system development initiates risk and determines specificity of structural or functional abnormalities [8] [12-22] leading to disease.

Findings: Stressful events in early life, including emotional stress and obstetrical complications during pregnancy, labor, and delivery, are associated with increased risk for asthma [23-33], autism [34], eating disorders [35], inflammatory bowel disease [36], multiple sclerosis [37], schizophrenia [38], and type 1 diabetes [39-44]. Low birth weight, which can be caused by prenatal stress, is associated with increased risk for the metabolic syndrome (increases in blood pressure, cholesterol, glucose, obesity, type 2 diabetes, heart disease, and stroke) [45-48] as well as asthma [23] [24] [33] [49-53], autism [54], celiac disease [55], inflammatory bowel disease [36], osteoporosis [22], and osteoporotic fractures [56].

Hypothesis 2: Intensity of stressful events [4-7] influences rates of disease progression and age of onset.

Findings: Stressful events are more frequent or more severe, or both, in inflammatory bowel disease [57-59], multiple sclerosis [60] [61], schizophrenia [38], and type 1 diabetes [62] [63] [64].

Hypothesis 3: Idiosyncratic stressors affect symptom variability because they relate to contextual cues associated with previous traumatic events, which are experiences that are unique to each individual [4-7].

Findings: Individual responses to stress, including variation in symptom expression and exacerbation, are highly variable and are unique to the individual rather than to the stressful event. Responses to stress in type 1 diabetes [65] [66] are idiosyncratic and similar findings have been noted in asthma [67] [68] [69], multiple sclerosis [61], and inflammatory bowel disease [57-59].

Implications

The impact of life events on brain plasticity has been grossly underestimated. Cultural, societal, and global traumatic events, such as the Holocaust on the one hand, and the rapidly expanding implementation of technologically assisted births (cesarean section [70] [71], labor induction, routine parent-infant separation in the first hours postpartum [72]) with Westernization on the other, appear capable of affecting risk for disease in the individual and across multiple generations. Such events are having large and essentially unrecognized effects on society and health [3] [73].

Brain plasticity appears capable of facilitating recovery, and body-oriented trauma therapies appear to be uniquely effective in treating illnesses such as PTSD [4-7]. Treatment of trauma-related bonding disruptions in mothers has been found to reverse asthma in the child on the same day that recovery is achieved in the parent [74-78].

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