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Fibromyalgia: The Learning of an Illness and its PNI Correlates

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ABCTRACT

Fibromyalgia is presented as an example of how an illness is learned along with its PNI correlates. In this work I argue that fibromyalgia, as well as most illnesses are learned patterns that have an initial preservation function. In the case of fibromyalgia, the patient may learn a functional hypervigilance in order to cope with a potential threat during sleep (possible physical threats during the day and/or sexual abuse while asleep). Initially, it was adaptive learning to sleep lightly (with the consequence of delta sleep deprivation, pro inflammatory products etc.), but as the contextual conditions change (i.e. no longer a need to be hypervigilant), the behavior becomes dysfunctional, and eventually its PNI damage reaches a critical mass where the symptoms, rather than the cause, emerge as an "illness".

A biocognitive theory and treatment model (cognition and biology within a cultural context) is presented based on clinical evidence that fibromyalgia, as well other illnesses, are curable when the learned PNI correlates can be replaced by behavioral patterns that are incompatible with the illness.

Key Words: biocognitive theory, learned illness, initially-adaptive function

Biocognitive Theory

Cognition, emotions and their biological correlates interact within inseparable culturally-influenced contexts. In addition to genetic and environmental factors, illnesses have an initially-adaptive function. An illness begins when its *initially-adaptive function* loses contextual relevance. The implicit language of an illness is learned with *coauthors* who contribute to the initial contextual conditions.

Causality in Biocognitive Theory

Upward and downward causalities are necessary but not sufficient to encompass the complexity of human behavior. *Contextual coemergence* causality is introduced in biocognitive theory to postulate that *psycho-cultural* biological processes coemerge within a contextual field, rather than "emerge" independent of covariant and context. Contextual coemergence causality is coauthored in bidirectional and culturally-influenced contexts (e.g. teacher/student in a classroom, physician/patient in a hospital, antibody/pathogen in an infection etc.).

The Cause and Effect Confusion

Pathogens and biochemical deregulations are effects, rather than causes of illnesses. The *cause and effect confusion* is perpetuated by the ascending causality model of the life sciences that attributes pathology to emerging conditions (e.g. cancer caused by cancerous cells). The contextual coemergence model attributes pathology to the coauthoring of pathogens and/or biochemical deregulations, with a chronically disempowered psychoneuroimmunoendrocrinological *bioinformational field* (e.g. cancer caused by the coauthoring of precancerous cells with a weak immune system). A bioinformational field encompasses the coauthoring of PNIE processes within a culturally-influenced context.

How Symbols Become Biosymbols

Symbols (words, images, concepts) and their biological correlates are inseparably learned in culturally-influenced clusters (*biocognitions*). The strength of their associations is shaped by contextual authorities (cultural editors). Cultural editors include parents, health professionals, clergy, teachers, and any other authorities in their respective domains.

The contributing effects of cultural editors increase with the degree of control, authority, and credibility ascribed to them by a shared cultural context. The effects can be psychoneuroimmunologically empowering or disempowering. The admonitions, praises, beliefs, rewards and punishments, dispensed by cultural editors, as well as by significant environmental events, are experienced and archived in *clusters* of cognitive, affective, sensorial and neuroimmunoendocrinological interpretations.

A Learning Path of an Illness

Disempowering behavior that has an initially-adaptive function (e.g. a child suppressing anger with an abusive parent) is culturally learned in psychoneuroimmunoendocrinological clusters (a biocognition). When disempowering behavior is no longer adaptive (e.g. an adult suppressing anger with an abusive adult), all of the components of the biocognitive cluster are adversely affected.

Learned maladaptive disempowerment coauthors psychoneuroimmunological helplessness (i.e. a condition where the necessary resources are not accessed to overcome a challenge). Chronic maladaptive disempowerment can trigger genetic predispositions and psychoneuroimmunological deregulations that eventually reach a critical mass and become an illness.

Fibromyalgia Syndrome (FMS)

FMS is a diffuse musculoskeletal pain and fatigue disorder without clear etiology. More than 9 million American adults have the disorder, predominantly women. Pain is the cardinal symptom, emanating from muscles, tendons, ligaments, bursa, and joints. The pain is usually described as steady, radiating, burning, and spreading over large areas of the body. Fatigue, lethargy, depression, sleep disturbances, bowel irregularities and cognitive difficulties may also be present.

In addition to genetic predispositions and environmental factors, fibromyalgia, as well as most non-congenital illnesses may have a learning process that had an initially-adaptive function. The initial wisdom of that function can be utilized to reverse or decelerate the deleterious path of an illness.

A Learning Path of Fibromyalgia

There is a high incidence of early emotional and physical trauma in fibromyalgia patients. Fibromyalgia has comorbidity with chronic pain, sleep disorders, PTSD, “sickness response” syndrome, chronic fatigue syndrome, and depression. The initially-adaptive functions of fibromyalgia include: hypervigilance, unassertiveness, and suppression of defensive behavior in response to disempowering contexts.

PNIE Correlates in the Learning Path of Fibromyalgia

Learned Hypervigilance:

Initially-adaptive function: to sleep lightly, to maintain a state of alarm, to suppress anger, pain, and defensive behavior in threatening contexts.

PNIE correlates: Delta sleep deprivation, hyper-reactive SNS, hyper-reactive HPA axis, and sensitization of primary nociceptors.

PNIE consequences of chronicity: Reduced serotonin and human growth hormone (HGH), reduced cortisol, increased norepinephrine, increased proinflammatory cytokines IL1 and IL-2, decreased concentration of natural killer cells (NK)

Biocognitive Treatment Model

Reversal of the dysfunctional learning path of fibromyalgia:

1. Identification of the initially-adaptive function, the coauthors, and the aversive context.

2. Assessment of the PNIE correlates chronically triggered by the loss of adaptive function.

3. Biocognitive interventions to replace the dysfunctional learning path with corrective behavior that can reverse the pathological psychoneuroimmunological manifestations.

Treatment Strategies

Premise:

Fibromyalgia research suggest the interplay of chronic pain and hypervigilance in fibromyalgia patients evokes a chronic sympathetic hyperactivity that can sensitize primary nociceptors, weaken innate immune response, as well as increase proinflammatory and/or decrease anti-inflammatory cytokines as reflected by a blunted cortisol/MIF ratio and Th1/Th2 imbalance.

Treatment:

(*Hypervigilance*) Biocognitive desensitization of the hyper-reactive SNS to reduce high epinephrine levels. Teach delta waves sleep to stimulate serotonin and HGH production. Incorporate "burst training" exercises to increase HGH while the patient learns to enter delta sleep.

(*Disempowering Behavioral Patterns*) Teach modulated expression of anger and assertive limit-setting to decrease levels of proinflammatory cytokines IL1 and IL2 and to increase NK concentration.

Conclusions

1. A biocognitive treatment model proposes illnesses have initially-adaptive functions to cope with disempowering conditions that are culturally influenced.

2. When the initially-adaptive function is generalized, it can promote a chronically deregulated PNIE process that may result in illness.

3. A contextual coemergence causality is introduced to conceptualize a coauthored and contextual attribution of PNIE processes.

4. The dysfunctional learning path of illnesses may be reversed or decelerated.

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