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Review

Psychological processing in chronic pain: A neural systems approach

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ABSTRACT

Our understanding of chronic pain involves complex brain circuits that include sensory, emotional, cognitive and interoceptive processing. The feed-forward interactions between physical (e.g., trauma) and emotional pain and the consequences of altered psychological status on the expression of pain have made the evaluation and treatment of chronic pain a challenge in the clinic. By understanding the neural circuits involved in psychological processes, a mechanistic approach to the implementation of psychology-based treatments may be better understood. In this review we evaluate some of the principle processes that may be altered as a consequence of chronic pain in the context of localized and integrated neural networks. These changes are ongoing, vary in their magnitude, and their hierarchical manifestations, and may be temporally and sequentially altered by treatments, and all contribute to an overall pain phenotype. Furthermore, we link altered psychological processes to specific evidence-based treatments to put forth a model of pain neuroscience psychology.

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1. Introduction

As organisms, we respond to external and internal milieu. In humans, such behavioral and physiological responses may be adaptive or maladaptive. In chronic pain, alterations in physical systems can result in changes in psychological processes observed as neural-circuit defined changes in behavior. Control of these processes may be viewed as a balance of excitatory and inhibitory events. Some processes may contribute to an escalating cascade or deescalating cascade of neural systems to produce altered behaviors in a sensitized or desensitized state. Chronic pain provides an ideal model to attempt to understand these types of processes in the context of localized and integrated neural networks. Brain regions consistently implicated include the primary and secondary somatosensory cortex (S1 and S2), spinal cord, thalamus, insula, anterior cingulate cortex, prefrontal cortex (Apkarian et al., 2005; Peyron et al., 2000; Tracey, 2008); midbrain areas including the periaqueductal gray (Linnman et al., 2011) and cerebellum (Moulton et al., 2010), and subcortical structures including the hippocampus, basal ganglia, and amygdala (Borsook et al., 2010; Maleki et al., 2011; Schweinhardt and Bushnell, 2010; Simons et al., 2012a); and see (Bushnell et al., 2013) for a recent review on brain regions involved in cognitive and emotional aspects of chronic pain) (see Fig. 1). The brain's interconnected networks contribute to resting and active functions that are orchestrated with varying degrees of contributions from these and other brain regions.

Pain is a response to nociceptive stimuli, often the driving force leading individuals to seek treatment, when they ache, hurt, and/or suffer. As pain becomes chronic, there is a tendency to be different - ones psychological state of being (and mind) is altered. Physical and emotional pain exists on the same continuum (Borsook et al., 2007; Elman et al., 2011; Perl, 2007) with common brain networks involved (Bendelow and Williams, 2008), with duplication and redundancy abounding. For example, using both noxious heat and unpleasant pictures, overlapping activation was observed in the posterior cerebellum with significant inverse correlations with limbic structures (viz., anterior hypothalamus, subgenual anterior cingulate, and parahippocampal gyrus), suggesting that the cerebellum contains specific regions involved in encoding generalized aversive processing, not necessarily unique to affective or sensorimotor functions (Moulton et al., 2011). Fig. 2 conceptualizes physiological and psychological processing of nociception and

pain related processes that may lead to altered behaviors. There are two major points that need to be considered when trying to understand a brain-systems approach to psychological processes in pain: (1) experiencing pain can trigger a cascade of neurological (initially sensory) events that lead to an altered psychological state; and (2) prior psychological states can confer a heightened risk for pain chronicity due to processes such as cross sensitization, where exposure to stress in the past results in greater sensitivity to other seemingly unrelated stimuli (e.g., childhood trauma, loss of a parent, and addiction) (Elman et al., 2013; Goldberg et al., 1999; Nicolson et al., 2010).

In this review we evaluate some of the principle behavioral alterations in the context of neural circuits and how these may be altered as a consequence of chronic pain. Data extracted from original research and from seminal review articles were critically assessed and summarized within the following main sections: (1) Pain, Neural Circuits, and Maladaptive Changes that contribute to central sensitization and perturbations in allostasis (allostatic load); (2) Alteration of Psychological Processes in Chronic Pain with evidence-based treatments that map onto key areas such as cognition, reward state and fear learning; (3) Premorbid Risk Factors and Pain where vulnerabilities such as psychological trauma can confer heightened risk; and lastly, we bring together these concepts with (4) Integrating Dysfunction: Complex Behaviors with Reciprocal, or Multiple and Multiplying Effects and provide suggestions for (5) Treatment and Training Approaches aimed at psychologists' training in the basic tenets of pain neuroscience.

2. 1: Pain, neural circuits, and maladaptive changes

2.1. Chronic pain, neurocircuits and behavior

The evolution or transition to chronic pain is not obvious. For many chronic pain conditions, the transition follows a specific insult (e.g., complex regional pain syndrome, post herpetic neuralgia, diabetic neuropathy, spinal cord injury, etc.), relates to a missing enzyme (e.g., Fabry's Disease) or is the result of a genetic condition (e.g., hemiplegic migraine), while in some cases pain emerges spontaneously. In cases of a specific insult the evolution of altered behaviors that are comorbid with pain unfold. Take for example, the individual who has a traumatic amputation from

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