Preventing Pain Requires Translating Biology into Social Change

Post-traumatic stress disorder (PTSD) is a frequent cotraveller with chronic pain [1,2]. Beyond epidemiological studies, however, investigation into mechanistic links between these conditions have been lacking in both number and appropriate design. It is logical to examine the role of a sensitized central nervous system as a biomarker of this putative link. Previous studies, however, have not utilized a model of deep muscle pain, likely necessary to activate both A-delta and C fibers [2] which may be required for an ecologically valid test of central sensitization. The small but elegant psychophysiological study by Moeller-Bertram in this issue of Pain Medicine suggests that veterans who have been exposed to combat and are living with PTSD may have a unique physiology which increases their risk for developing chronic pain [3]. The main observations of the study include: 1) no differences in sensory detection and pain thresholds between the veteran warfighters with and without PTSD and 2) that veteran warfighters with PTSD experienced increased sensory and emotional pain over time and increased temporal summation of pain. The lack of baseline differences between the groups for sensory detection and pain thresholds between the veteran warfighters with and without PTSD and 2) that veteran warfighters with PTSD experienced increased sensory and emotional pain over time and increased temporal summation of pain. The “activating” symptoms of PTSD include re-experiencing and hyperarousal and are consistent with a state of high emotional and physiological stress. PTSD is linked with perturbed hypothalamic-pituitary-adrenal axis activity [4], cardiovascular hyper-reactivity [5], impaired immune response [6], dysregulated chronobiology [7], and abnormal functioning of the amygdala, insula, and anterior cingulate cortices [8,9]. PTSD is a complex disorder with genetic, epigenetic, and social etiologies [10,11], and it makes sense that these dysregulated physiological and social systems contribute to impaired descending inhibition of pain. This study advances the fields of pain medicine, psychiatry, and public health because it provides evidence that PTSD (in a sense a proxy diagnosis for a more general condition of a dysregulated response to stress) increases the risk for both hypersensitivity and enhanced temporal summation (two experimentally measured dimensions of central sensitization) [12]. The logical next clinical research questions in need of experiments which could translate these findings into the clinic are: 1) Does effective treatment of PTSD prevent or correct experimentally evoked evidence of central sensitization? and 2) Does prevention or effective treatment of PTSD prevent conversion of acute to chronic pain?

A clinical implication of these observations for pain medicine physicians is that they should be more rigorously screening for a history of trauma and PTSD. When PTSD is diagnosed, they should then be referring patients to psychiatry and psychology colleagues for further evaluation and treatment. The clinical implications for psychiatry and public health are the need for indicated prevention [13] for individuals at risk for PTSD, so that early interventions can be implemented in the immediate aftermath of trauma. These early interventions and indicated prevention may ultimately prevent the development of chronic pain and other conditions associated with a stressed central nervous system such as depression, anxiety, and cognitive impairment [14].

However, if we view, as stated above, that PTSD is a proxy for a “dysregulated response to stress,” it may not be a stretch to suggest that the prevention of PTSD and an abnormal stress response extends to the prevention of childhood neglect and abuse, poor nutrition, poverty, and racism. Reducing these chronic social stressors, more common than acute trauma and perhaps no less devastating to the maintenance of “stress homeostasis” [15], may go a long way to reducing the epidemic of chronic pain, high use of opioids, and increased use of expensive and invasive interventions. The links among pain, psychiatric disorders, and both acute and chronic environmental stressors are rich and complex. To positively impact both clinical and social change, the results of controlled clinical experiments such as this need to be disseminated to a broad, multidisciplinary audience. Translating basic science and psychophysiological observations into an improved practice of pain medicine requires that we prevent and reduce the experiences of trauma and chronic social stressors and improve access to care for PTSD and psychiatric distress early and in conjunction with the management of chronic pain conditions.

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References


Editorial