

Post-traumatic stress disorder is a systemic illness, not a mental disorder: is Cartesian dualism dead?

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Mind and body are intimately linked, in health and in disease

Descartes' notion of dualism, which argues for the distinction between the mind and the body,¹ has underpinned and subtly driven much of the confused thinking in medicine about psychiatric disorders. A substantial and still accumulating body of evidence about the extensive psychophysiological and somatic comorbidities of post-traumatic stress disorder (PTSD),^{2,3} however, now challenges this notion, suggesting the need to reconceptualise PTSD as a systemic disorder rather than one confined to the mind. The somatic pathologies range from metabolic syndrome and related cardiovascular conditions to autoimmune diseases, including rheumatoid arthritis.^{2,4} Such disorders have been associated with a range of quantifiable abnormalities, including inflammatory cascades, altered psychophysiological reactivity and neuroendocrine function, and shortened telomere lengths.⁵

The study of a convenience sample of Australian Vietnam War veterans published in this edition of the *MJA*⁶ explores in detail the somatic comorbidities of PTSD. McLeay and her co-authors found that the relationship between PTSD, gastrointestinal disorders and abnormal respiratory function in veterans remained statistically significant even after controlling for factors known to be associated with chronic disease and early mortality in the absence of PTSD, such as higher body mass index, smoking, alcohol dependence, anxiety, and depression. These direct somatic comorbidities highlight the fact that the pathophysiological burden of PTSD cannot be attributed to other comorbidities, but indicate the biological dysregulation inherent to this disorder,⁷ a factor not systematically addressed by current treatments.⁸ Many of the physiological and immune abnormalities in PTSD are also present in the sub-syndromal form of the disorder, and therefore provide potential targets for early intervention.⁵ One important question not explored by McLeay and colleagues is the relationship between combat exposure and physical disorder when full-blown PTSD is absent but subsyndromal symptoms are present.

Chronic pain and disability resulting from traumatic injury³ constitute another domain of somatic pathology in PTSD, with longitudinal studies indicating how important PTSD is for the long term outcome.⁹ The foundations of this relationship are the shared neurobiology of pain and PTSD, and their complex interaction.¹⁰ These findings are of particular importance for managing injuries in people such as emergency service workers and veterans, for whom there are significant risks of physical injury as well as of PTSD caused by trauma exposure. Physical health outcomes in these populations, particularly for older members with their cumulative burden of trauma exposure, are underpinned to a



significant degree by the somatic pathology, pain and disability that is driven by PTSD. The refining of the stressor criterion for PTSD in the fifth edition of the *Diagnostic and statistical manual of mental disorders* (DSM-5) to include “experiencing repeated or extreme exposure to aversive details of ... traumatic event(s)”¹¹ as a category of exposure highlights the salience of the effects of cumulative trauma exposure for the pathophysiology of the disorder. Treatment plans for PTSD, including those for preventing “burnout” in emergency service personnel, have largely failed to adopt an integrated approach or to develop management strategies that recognise the common roots of the physical and psychological dimensions of the health of these individuals.

The failure to attend to the somatic pathology of PTSD has not served patients well. People with PTSD frequently also present with somatic symptoms of a non-specific nature⁸ that represent an integral part of the patient's sense of ill-health. Medical journals, as well as the general media, frequently attest to the fierce controversies and battles in academia and among advocacy groups regarding conditions linked with military service, such as the effects of Agent Orange exposure in Vietnam War veterans and Gulf War syndrome. These conditions arise from veterans' preoccupation with their sense of somatic ill-health and its possible causation.¹² The various editions of the DSM of the American Psychiatric Association have failed to incorporate this central component of the patients' illness experience by not including somatic symptoms as one of the axes of distress in their diagnostic criteria for PTSD. As a consequence, the biological mechanisms of the symptoms, their prevalence, and their relationship with later somatic pathology have all been inadequately explored.

The limited effectiveness of evidence-based psychological interventions in people with PTSD, particularly in veteran populations,¹³ highlights the need to develop biological therapies that

address the underlying neurophysiological and immune dysregulation associated with PTSD. It is possible that these neurobiological dimensions may drive the relatively poor outcomes of psychological interventions. One important strategy for better understanding the sequence of the emergence of the psychological symptoms and somatic pathology in PTSD is to adopt a staging model that distinguishes the emerging matrix of the early patterns of biological dysregulation from the neurobiology of chronic, longstanding PTSD, which may reflect the secondary consequences of prolonged pathophysiological dysregulation.⁵ It is only by effectively collating such evidence that we will realise that Descartes' views on dualism are completely outmoded.

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