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The Potential Role of Anxiety Sensitivity in the Risk For and Recovery From Heart Attacks: Comment on Edmondson et al. (2018)

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Abstract

Edmondson et al. (2018) provided a compelling account of the causes and consequences of patients' perceptions of enduring somatic threat following a heart attack or related cardiac event. The purpose of the current article is to place some of these observations in the context of research on anxiety sensitivity and its role as a risk factor for cardiovascular disease, posttraumatic stress disorder, and negative health behaviors. By doing so, we hope to encourage attention to anxiety sensitivity and associated prevention/intervention strategies for at-risk cardiac patients.

Keywords

anxiety sensitivity; acute coronary syndrome; posttraumatic stress disorder; negative health behaviors

A recent article by Edmondson et al. (2018) reviewed the clinical presentation, prevalence, and consequences of posttraumatic stress disorder (PTSD)-like responses to heart attacks (initial myocardial infarction or unstable angina). The authors emphasized the perception of ongoing threat in the body as a central driver of PTSD symptoms, largely due to patients' sensitization to bodily cues after their cardiac event (catastrophic interpretations of normal cardiovascular events; e.g., "It scares me when my heart beats rapidly"; Edmondson et al., 2018, p. 1163). Of particular note is the potential of these fears to derail subsequent cardiovascular disease (CVD) risk-reduction behaviors, due to avoidance of physical activity or poor medication/treatment adherence.

In reading this article, we were struck by the similarity between descriptions of an "enduring somatic threat" (EST) model of PTSD and the concept of anxiety sensitivity (AS) – the fear of anxiety-like sensations due to catastrophic interpretations of these symptoms (Reiss, Peterson, Gursky & McNally, 1986). AS and other anxiety-related concepts are frequently considered in conceptualizations of non-cardiac chest pain, but, as we argue here, AS has particular value in conceptualizations of risks for and the sequelae of documented CVD. One particular value of considering AS as a related construct to EST is that it opens the door to a broader literature that has important prevention/intervention implications. Specifically, we

argue that AS may be important for understanding risk for CVD, risk for PTSD, and risk for subsequent disability following heart attacks and other related conditions. Lastly, we review research on the modification of AS through prevention/intervention efforts.

There is now a substantial literature on the association between AS and medical conditions. A recent article by Horenstein, Potter, and Heimberg (2018) reviewed 160 studies of largely cardiac, pain, respiratory and gastrointestinal conditions and detailed the link between AS and fears of medical symptoms, avoidance of healthy behaviors, and engagement in unhealthy behaviors. Otto and associates (2016) conceptualized associations between AS and health behaviors in terms of the role of AS as an amplifying factor, enhancing the aversiveness and need to escape/avoid negative affective or somatic experiences. In regard to CVD risk factors, AS is related to coping motives for alcohol and cigarette use, mood-related eating, and avoidance of physical activity (see Otto et al., 2016). AS is also associated with reduced exercise tolerance, particularly among those with the CVD risk factor of smoking (e.g., Farris et al., 2017) or high body mass (Smits, Tart, Presnell, Rosenfield, & Otto, 2010). Furthermore, in terms of direct evidence of a linkage between AS and CVD, a cross-sectional study of 635 participants (aged 10-66 years) found that AS was associated with both carotid plaques and increased arterial stiffness, after statistical adjustment of demographic factors and specific CVD risk factors (low-density lipoprotein cholesterol and blood pressure) (Seldenrijk et al., 2013). Accordingly, AS may play a role not only in setting the stage for cardiovascular risk—with linkages to the top three preventable causes of disease and death in the United States: (1) tobacco use, (2) poor diet and physical inactivity (obesity), and (3) alcohol overuse (see Otto, Smits, Fitzgerald, Powers, & Baird, 2018)—but also in inhibiting adaptive responses to CVD, such as engagement in physical activity.

AS is also a prospective predictor of the development of PTSD following a traumatic stressor (e.g. Boffa et al., 2016). Hence, elevated AS may be a risk factor not only for CVD but also for the development of PTSD after a cardiac event. AS is also linked to the degree of reactivity to somatic symptoms in those with PTSD; accordingly, directly targeting AS in treatment may enhance recovery from PTSD (Wald & Taylor, 2008) and the likelihood of the associated negative health behaviors described above (see Otto et al., 2016).

There is a wealth of evidence that AS can be targeted with a number of short-term and focused interventions (see Otto et al., 2018). Perhaps the best studied are interoceptive exposure interventions in which individuals are systematically introduced to feared somatic/emotional sensations by using a variety of exercises to induce such sensations (e.g., spinning in a circle or hyperventilation to elicit sensations such as numbness, tingling, dizziness, and derealization). These exposures provide experiential learning that these sensations need not be feared. Programmed episodes of exercise have also been used in an interoceptive exposure format to reduce AS, offering the additional benefit of directly encouraging this health behavior. In addition, psychoeducation and cognitive-restructuring have been used to provide corrective information about feared sensations with the goal of changing how these sensations are interpreted. Also, cognitive bias modification training has been tasked to modify anxiety-related symptom interpretation via repeated practice of cognitive tasks that reinforce non-threat processing. Any of these interventions may be of use in the cardiac

clinic, where they can be implemented to reduce AS more generally as well as the specific fears of cardiac-related sensations and symptoms that lead to maladaptive avoidance as characterized by Edmondson et al. (2018). Importantly, for a CVD population, relevant cautions need to be applied, such as medical clearance for interoceptive exposure using physical exercise or hyperventilation.

In summary, AS has been shown to be a risk factor for poor cardiovascular health, a risk factor for PTSD, and a risk factor for a wide variety of negative health behaviors, including exercise avoidance. As such, we believe that AS is a potentially important treatment target for individuals with emergent PTSD-like responses to a cardiac event. In addition, attending to AS as a broad-based risk factor for negative health behaviors (including those highly relevant to CVD recovery) links the somatic threat observed by Edmondson et al. (2018) to a broader health literature and additional intervention strategies.

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