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Letter to the Editor

PTSD is a risk factor for cardiovascular disease: Time for increased screening and clinical intervention☆

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Research has suggested that posttraumatic stress disorder (PTSD) is associated with increased risks for cardiovascular diseases (Boscarino, 2008, Boscarino and Chang, 1999, Kubzansky, et al., 2007, Ahmadi, et al., 2011, Kubzansky and Koenen, 2009, Coughlin, 2011). While most studies in the past have been with US war veterans (Boscarino, 2004), others have focused on different groups, including women in the community (Kubzansky, et al., 2009), civilians exposed to warzone stressors (Sibai et al., 1989), victims of nuclear disaster (Cwikel, et al., 1997), and national population samples in non-US countries (Glaesmer, et al., 2011). Recently, patients with PTSD in a Framingham coronary heart disease (CHD) study were also found to have an increased risk of CHD, with PTSD-positive cases having an increase in Framingham CHD risk scores (Jin, et al., 2011). The study by Jordan et al. published in *Preventive Medicine* adds to this growing PTSD-cardiovascular literature (Jordan, et al., 2011). This literature now suggests that, regardless of the study population, the type of stressor exposure, or the cardiovascular measure used, there is a positive association between having PTSD and the onset of cardiovascular disease. In fact, of the nearly 100 disease studies published in PUBMED, virtually none report negative associations with PTSD (Kubzansky and Koenen, 2009, Coughlin, 2011, Boscarino, 2004). The study by Jordan et al., which is based on a prospective study of 39,324 World Trade Center (WTC) Health Registry participants, found that men and women with PTSD at study enrollment had an elevated risk of heart disease and that this risk had a dose–response association with PTSD symptoms. Given this body of research, the question now is not if there is a link between PTSD and cardiovascular disease, but why this association exists and can this outcome be prevented (Boscarino, 2011)? Indeed, in this same issue of *Preventive Medicine*, the Editors noted that the Framingham risk score may have to be adapted to include PTSD as a CHD risk factor in the future (Morabia and Costanza, 2011).

Studies suggest that PTSD might result in inflammatory injuries through over-activation of the hypothalamic-pituitary-adrenal stress axis, subsequently followed by hypocortisolism related to molecular down regulation of this system (Boscarino, 2004, Heim, et al., 2000). Consistent with this, research suggests that systemic inflammatory activity appears common in PTSD cases (von Kanel, et al., 2007). This PTSD-disease link also could be related adverse health behaviors, such as cigarette smoking and substance misuse related to self-regulation of aversive psychological states brought on by PTSD psychopathology (Vlahov, et al., 2002, Boscarino, et al., 2011a). There are other variables that might also explain this association (Boscarino, 2004). At this time, not one causal pathway has been identified and ultimately this may involve multiple pathways, including behavioral risk factors (Boscarino, et al., 2011c). Recently our research indicated that *FKBP5*, *COMT*, and *CHRNA5* genetic loci involving pathways associated with inflammation, addiction, sleep, and anxiety circuitry, were associated with PTSD (Boscarino, et al., 2011b). Interestingly, it has been reported that the *CHRNA* gene, which encode components of the nicotinic acetylcholine receptor, was associated with peripheral arterial disease (Thorgeirsson, et al., 2008). Recently, this gene was also associated with cigarette smoking, nicotine dependence, opioid misuse, as well as PTSD (Erlich, et al., 2010, Boscarino, et al., 2011b). Thus, one biologic pathway for cardiovascular disease might include nicotine addiction associated with having the genetic variant of the *CHRNA* gene, without which there would be insufficient exposure to cigarette smoking to result in disease. Additionally, cigarette smoking is also associated with PTSD, in part, because PTSD victims often attempt to self-medicate aversive arousal symptoms and smoking (and other psychoactive substance use) seems to ameliorate this psychological state (Boscarino, et al., 2011c). Other genetic components may also be associated with the pathophysiology of heart diseases following PTSD onset (Boscarino, 2011).

Fortunately, from a prevention point of view, our studies suggest that PTSD screening can be effectively conducted among at-risk populations (Boscarino et al., 2012; Boscarino et al., 2011d). Research also suggests that those who received counseling shortly after the World Trade Center event, not only had better PTSD outcomes, but also improved outcomes in a number of different clinical areas (Boscarino, et al., 2011a). Evidence currently suggests that these early interventions may prevent PTSD memory consolidation and, thus, chronic PTSD (Boscarino, et al., 2011a). These PTSD findings may have implications for cardiovascular disease prevention. Future data collection planned for the WTC Health Registry should consider including mental health treatment data, as we have previously done (Boscarino, et al., 2004, Boscarino, et al., 2011a). While these data might be confounded, propensity score matching could be used to examine the unbiased impact of these reported treatments (Boscarino, et al., 2011a). To be sure, the study by Jordan et al. represents an important contribution to the field. The next step now should be to prevent the onset of cardiovascular disease following PTSD, if at all possible.

Conflict of interest statement

None.

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