Press Release

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PTSD Increases Risk for Metabolic Syndrome and Reduced Cortical Thickness

Reports new study in Biological Psychiatry

Philadelphia, PA, August 31, 2016 – Metabolic syndrome, a cluster of cardiometabolic conditions, may be a biological mechanism linking posttraumatic stress disorder (PTSD) to structural brain abnormalities, according to a new study in *Biological Psychiatry*. The findings highlight the need to develop effective interventions for PTSD to treat not only the symptoms associated with the disorder, but also potential ensuing metabolic and neurodegenerative consequences, which may be suggestive of premature aging.

"The results of this study have important implications for our newest cohort of veterans returning from the conflicts in Iraq and Afghanistan," said first author Erika Wolf from the National Center for PTSD, VA Boston Healthcare System in Massachusetts. "They suggest that it might be appropriate to view PTSD as a risk factor for metabolic disease and as such, to screen young veterans with PTSD for metabolic problems."

Stress has been thought to be a contributing factor to the development of metabolic syndrome, which occurs about twice as often in patients with PTSD than in the general population. Additionally, metabolic syndrome increases risk for cardiovascular disease, type 2 diabetes, and other medical conditions that often accompany PTSD, and is associated with neurodegeneration.

In the study, jointly funded by the National Institute of Mental Health and the United States Department of Veterans Affairs, senior author Mark Miller, also from the National Center for PTSD, and colleagues examined the associations between PTSD, metabolic syndrome, and structural integrity of the brain. They assessed 346 United States military veterans deployed to Iraq and Afghanistan who participated in the Translational Research Center for TBI and Stress Disorders (TRACTS) for PTSD and metabolic syndrome, of which 274 also had magnetic resonance imaging measures of cortical thickness, an index of the neural integrity of the brain.

Consistent with previously published rates, the prevalence of metabolic syndrome among veterans with PTSD was nearly twice as high as those without PTSD. Structural brain images revealed an association between greater metabolic syndrome severity and reduced cortical thickness. In an analysis with multivariate statistical models, the researchers then found an indirect effect of PTSD on cortical thickness via metabolic syndrome severity.

"Our finding that PTSD-related metabolic syndrome was associated with reduced thickness in large regions of the cortex of the brain is alarming, particularly given that veterans in this study were, on average, quite young and in their early 30s," said Wolf.

The question of how PTSD and metabolic syndrome affect brain structure remains unanswered and additional research will be needed to rule out the possibility that reductions in cortical thickness are actually a risk factor, rather than consequence, of PTSD and metabolic syndrome.

Still, according to Wolf, this association raises concern about the possibility of subsequent neurocognitive decline in this population. “The effects observed in this study may be part of larger PTSD-related accelerated cellular aging process that is manifested in premature health decline,” she said.

“This important study suggests a link between PTSD, metabolic syndrome, and brain health,” said John Krystal, Editor of *Biological Psychiatry*. “By implication, this study suggests that effective treatment for PTSD is needed to reduce emotional distress and to preserve overall health.”

Notes for editors

The article is "Posttraumatic Stress Disorder as a Catalyst for the Association Between Metabolic Syndrome and Reduced Cortical Thickness," by Erika J. Wolf, Naomi Sadeh, Elizabeth C. Leritz, Mark W. Logue, Tawni B. Stoop, Regina McGlinchey, William Milberg, and Mark W. Miller (doi:10.1016/j.biopsych.2015.11.023). It appears in *Biological Psychiatry*, volume 80, issue 5 (2016), published by Elsevier.

Copies of this paper are available to credentialed journalists upon request; please contact Rhiannon Bugno at +1 214 648 0880 or biol.psych@utsouthwestern.edu. Journalists wishing to interview the authors may contact Pallas Wahl at +1 857 203 5879 or Pallas.wahl@va.gov.

The authors’ affiliations, and disclosures of financial and conflicts of interests are available in the article.

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