

Social stress and the inflamed brain

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Depression is the leading cause of disability with more than 350 million people globally affected by this disease. In addition to debilitating consequences on mental health, depression predisposes an individual to physiological disease such as heart disease, and conversely heart disease increases the risk of depression. According to the World Health Organization by the year 2020 heart disease and depression will be the number one and number two leading causes of disability in developed countries. While the co-occurrence of these disorders is well recognized, an understanding of the underlying mechanisms that lead to this relationship are lacking.

Dr. Susan K. Wood, a Research Associate at the Children's Hospital of Philadelphia, investigates brain-related biomarkers for depression-heart disease comorbidity. She uses a <u>rodent model</u> of social stress likened to bullying in people that she has found to produce depressive-like behaviors and dysfunctional <u>cardiovascular changes</u> in a susceptible subset of rodents. Her previous work highlighted a role for the stressrelated neurohormone corticotropin-releasing factor in rendering an individual vulnerable to stress-induced depression and heart disease. Intrigued by what other biomarkers may be distinct her latest study is the first to identify gene and protein expression differences in the brains of rodents that are either vulnerable or resilient to developing stressinduced depressive-like behaviors and cardiovascular dysfunction.

The study, conducted in male rats, compared expression of 88 genes involved in signaling within the brain between socially stressed and nonstressed rats. It revealed more than 35 genes in stressed rats that had



altered expression compared with non-stressed controls. Many of the genes that were differentially expressed were related to inflammation. Follow-up studies measuring protein levels revealed that Interleukin-1 β and Monocyte chemotactic protein-1, inflammatory markers known to play a role in depression and heart disease, were suppressed in the brains of the resilient subset of rats and Interleukin-1 β was increased in the vulnerable group. Dr. Wood measured the gene and protein levels under resting conditions 24 hours after just 5 daily 30-minute exposures to social stress.

The identification of factors in the brain that distinguish susceptibility and resiliency to depression and heart disease comorbidity would be a major advance in predicting, preventing and treating these disorders. Dr. Wood is continuing these studies as an Assistant Professor at the University of South Carolina School of Medicine with the hope that these findings will uncover new targets to treat the mind and body.

Her findings will be presented April 21st, 2013 during Experimental Biology 2013 in Boston, MA.

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