Study identifies co-factors critical to PTSD development

April 3 2013

Research led by Ya-Ping Tang, MD, PhD, Associate Professor of Cell Biology and Anatomy at LSU Health Sciences Center New Orleans, has found that the action of a specific gene occurring during exposure to adolescent trauma is critical for the development of adult-onset Post-Traumatic Stress Disorder (PTSD.) The findings are published in *PNAS* Online Early Edition the week of April 1-5, 2013.

"This is the first study to show that a timely manipulation of a certain neurotransmitter system in the brain during the stage of trauma exposure is potentially an effective strategy to prevent the pathogenesis of PTSD," notes Dr. Tang.

The research team conducted a series of experiments using a specific strain of transgenic mice, in which the function of the gene can be suppressed, and then restored. The model combined exposure to adolescent trauma as well as an acute stressor. Clinically PTSD may occur immediately following a trauma, but in many cases, a time interval may exist between the trauma and the onset of disease. Exposure to a second stress or re-victimization can be an important causative factor. However, the researchers discovered that exposure to both adolescent trauma and to acute stress was not enough to produce consistent PTSD-like behavior. When exposure to trauma and stress was combined with the function of a specific transgene called CCKR-2, consistent PTSD-like behavior was observed in all of the behavioral tests, indicating that the development of PTSD does not depend only on the trauma itself.

As a predominant form of human anxiety disorders, PTSD affects 7.8% of people between 15-54 years in the United States. PTSD can cause feelings of hopelessness, despair and shame, employment and relationship problems, anger, and sleep difficulties. Additionally, PTSD can increase the risk of other mental health conditions including depression, substance abuse, eating disorders, and suicidal thoughts, as well as certain medical conditions including cardiovascular disease, chronic pain, autoimmune disorders, and musculoskeletal conditions.

A favored current theory of the development of anxiety disorders, including PTSD, is a gene/environment interaction. This study demonstrated that the function of the CCKR-2 gene in the brain is a cofactor, along with trauma insult, and identified a critical time window for the interaction in the development of PTSD.

"Once validated in human subjects, our findings may help target potential therapies to prevent or cure this devastating mental disorder," Dr. Tang concludes.

Provided by Louisiana State University