Chronic stress and anxiety can damage the brain, increase the risk of major psychiatric disorders

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A scientific review paper warns that people need to find ways to reduce chronic stress and anxiety in their lives or they may be at increased risk for developing depression and even dementia.

Led by the Rotman Research Institute at Baycrest Health Sciences, the review examined brain areas impacted by chronic anxiety, fear and stress in animal and human studies that are already published. The authors concluded that there is "extensive overlap" of the brain's neurocircuitry in all three conditions, which may explain the link between chronic stress and the development of neuropsychiatric disorders, including depression

and Alzheimer's disease.

The paper is posted online this month in the journal *Current Opinion in Psychiatry*.

Experiencing anxiety, fear and stress is considered a normal part of life when it is occasional and temporary, such as feeling anxious and stressed before an exam or a job interview. However, when those acute emotional reactions become more frequent or chronic, they can significantly interfere with daily living activities such as work, school and relationships. Chronic stress is a pathological state that is caused by prolonged activation of the normal acute physiological stress response, which can wreak havoc on immune, metabolic and cardiovascular systems, and lead to atrophy of the brain's hippocampus (crucial for long-term memory and spatial navigation).

"Pathological anxiety and chronic stress are associated with structural degeneration and impaired functioning of the hippocampus and the prefrontal cortex (PFC), which may account for the increased risk of developing neuropsychiatric disorders, including depression and dementia," said Dr. Linda Mah, clinician scientist with Baycrest's Rotman Research Institute and lead author of the review.

The review paper examined recent evidence from studies of stress and fear conditioning in animal models, and neuroimaging studies of stress and anxiety in healthy individuals and in clinical populations.

Dr. Mah and colleagues looked specifically at key structures in the neurocircuitry of fear and anxiety (amygdala, medial prefrontal cortex, hippocampus) which are impacted during exposure to chronic stress. The researchers noted similar patterns of abnormal brain activity with fear/anxiety and chronic stress - specifically an overactive amygdala (associated with emotional responses) and an under-active PFC (thinking

areas of the brain that help regulate emotional responses through cognitive appraisal). This see-saw relationship was first identified in a landmark study by world-renowned neurologist and depression researcher Dr. Helen Mayberg over a decade ago.

Dr. Mah, an assistant professor of Psychiatry in the Department of Geriatric Psychiatry at the University of Toronto, concluded her review on a hopeful note by suggesting that stress-induced damage to the hippocampus and PFC is "not completely irreversible". Anti-depressant treatment and physical activity have both been found to increase hippocampal neurogenesis, she said.

"Looking to the future, we need to do more work to determine whether interventions, such as exercise, mindfulness training and cognitive behavioural therapy, can not only reduce stress but decrease the risk of developing neuropsychiatric disorders," said Dr. Mah

The scientific review paper follows on the heels of a major study Dr. Mah published in the *American Journal of Geriatric Psychiatry* (first posted online in October 2014), which found some of the strongest evidence yet that anxiety may accelerate conversion to Alzheimer's disease in people diagnosed with mild cognitive impairment.

Dr. Alexandra Fiocco, a psychologist with the Institute for Stress and Wellbeing Research, Ryerson University, contributed to the review paper in *Current Opinion in Psychiatry*. The work was supported in part by the Ministry of Health and Long-Term Care AFP Innovation Fund.

More information: *Current Opinion in Psychiatry*, www.ncbi.nlm.nih.gov/pubmed/26651008

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