

The biology of anxious temperament may lie with a problem in an anxiety "off switch"

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Persistent anxiety is one of the most common and distressing symptoms compromising mental health. Most of the research on the neurobiology of anxiety has focused on the generation of increased anxiety, i.e., the processes that "turn on" anxiety.

But what if the problem lay with the "off switch" instead? In other words, the dysfunction could exist in the ability to diminish anxiety once it has begun.

A new report in the current issue of *Biological Psychiatry* by researchers at the University of Wisconsin at Madison suggests that deficits in one of the brain's off switches for anxiety, neuropeptide Y receptors, are decreased in association with anxious temperament.

To conduct their work, the researchers studied 24 young rhesus monkeys to examine expression of the neuropeptide Y system in relation to anxious temperament. Neuropeptide Y is a neurotransmitter that helps regulate the body's response to stress. Anxious temperament is a trait that presents early in life and increases the risk of developing anxiety and depressive disorders.

They found that elevated anxious temperament is associated with decreased messenger RNA expression of two neuropeptide Y receptors, Y1R and Y5R, in the central nucleus of the amygdala, a region of the brain that plays an important role in regulating fear and anxiety.

"This finding is very important as it focuses our thinking about treatment on promoting recovery after stress rather than suppressing the normal adaptive reaction to threatening situations. Fear, at times, is the best possible reaction to life events. However, persistent fear can be destructive. This new finding points us in the direction of new treatments that aim to promote resilience rather than blunting one's life experiences," said Dr. John Krystal, Editor of *Biological Psychiatry*.

The authors agree, with first author Dr. Patrick Roseboom noting that "extreme anxiety in children is a prominent predictor of the later development of [anxiety disorders](#) and other illnesses such as depression and substance abuse. Using young rhesus monkeys in our model of anxious temperament is critical as brain structure and function in non-human primates closely resembles that of humans."

"Identifying the molecular underpinnings of why some individuals are at-risk for developing [anxiety](#) and depression has the potential to identify new treatment targets," added Roseboom. "The current findings suggest that focusing on a system that provides resilience may be an important strategy at the molecular level."

More information: "Neuropeptide Y Receptor Gene Expression in the Primate Amygdala Predicts Anxious Temperament and Brain Metabolism", Patrick H. Roseboom, Steven A. Nanda, Andrew S. Fox, Jonathan A. Oler, Alexander J. Shackman, Steven E. Shelton, Richard J. Davidson, and Ned H. Kalin ([DOI: 10.1016/j.biopsych.2013.11.012](https://doi.org/10.1016/j.biopsych.2013.11.012)), *Biological Psychiatry*, Volume 76, Issue 11 (December 1, 2014).

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