

## How Experiencing Traumatic Stress Leads to Aggression

Strengthened amygdala pathways increase aggression, may be targets for PTSD treatment. Traumatic stress can cause aggression by strengthening two brain pathways involved in emotion, according to research recently published in the Journal of Neuroscience. Targeting those pathways via deep brain stimulation may stymie aggression associated with post-traumatic stress disorder.

The consequences of traumatic stress linger long after the stress ends. People suffering from post-traumatic stress disorder often display heightened aggression, caused by unknown changes in the amygdala. An almond-shaped structure nestled deep inside the brain, the amygdala plays an essential role in emotion, social behaviors, and aggression.

Nordman et al. examined how different amygdala circuits changed in male mice after traumatic stress. Two connections strengthened, resulting in more attacks on other mice: the circuitry connecting the amygdala to the ventromedial hypothalamus and the bed nucleus of the stria terminalis. The former modulates the frequency of attacks, while the latter controls the length of attacks. The research team then used low frequencies of light to stop the pathways from strengthening, preventing an increase in aggressive behavior. Deep brain stimulation may elicit the same effect in humans.

*Materials provided by Society for Neuroscience.*

## New Pathways in Brain's Amygdala

Researchers at The University of Texas at San Antonio (UTSA) are pioneering an innovative brain study that sheds light on how the amygdala portion of the brain functions and could contribute to a better understanding of post-traumatic stress disorder, anxiety, depression and Alzheimer's disease.

The researchers, Alfonso Apicella, an associate professor in the Department of Biology, research associate Alice Bertero, postdoctoral fellow Paul Luc Caroline Feyen, and graduate student Hector Zurita, published their study, "A non-canonical cortico-amygdala inhibitory loop," in the Journal of Neuroscience, the flagship journal of the Society for Neuroscience.

Conditions such as PTSD, anxiety and depression are thought to be linked to the abnormal functioning of the amygdala, which is located within the temporal lobes and plays a key role in processing emotions, actions and cognition.

"This novel research paper provides anatomical and physiological evidence for the existence of a long-range inhibitory pathway from the auditory cortex to the amygdala in the mouse brain," Apicella said. "For the first time, in our paper we show this emotional pathway."

Apicella explained that advances in the techniques for labeling individual neuronal cells made it possible to study the individual neurons extending from the auditory cortex to the amygdala.

He added that the inhibitory cortical neurons can alter the activity of the amygdala's principal neurons and can therefore directly control the output of the amygdala. The neurons contain a substance called somatostatin, and it regulates physiological functions and forms a connection with principal neurons that project to other brain regions outside the amygdala that are involved in fear and aversive behavior.

"The discovery that the amygdala receives both excitatory and inhibitory inputs from that cortex suggests that the timing and relative strength of these inputs can affect the activity of the amygdala," Acipella said.

Apicella and his research team noted that future experiments should examine whether this is a general mechanism by which sensory stimuli can influence the processes controlled by the amygdala, such as fear/aversive behavior and how the disruption of this pathway can lead to several neurological and psychiatric disorders, such as, Alzheimer's, anxiety, depression and PTSD.

Research related to this topic will continue in Apicella's lab. His research group investigates the neural basis of perception. More specifically, the researchers want to understand how cortical microcircuits process sensory information leading to behavioral outcomes.

*Materials provided by University of Texas at San Antonio.*

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