Age-Related Effect of Serotonin Transporter Genotype on Amygdala and Prefrontal Cortex in Adolescence

Reported by: Dijla Alsaigh, Research Assistant

The serotonin transporter-linked promoter region (5-HTTLPR) polymorphism of the serotonin transporter gene is associated with amygdala response during negative emotion. These low expressing alleles are related to increased risk for depression and brain activation patterns found in depression (increased amygdala activation and decreased amygdala-prefrontal cortex connectivity). Researchers at the University of Michigan wanted to see if there was a significant relationship between increased amygdala activation with age in the low expressing genotype group relative to the higher expressing group and decreased amygdala-prefrontal connectivity with age in the low expressing group.

The Brain

The amygdala is a small structure in the brain that is responsible for the response and memory of emotions. The prefrontal cortex is a part of the brain located at the front of the frontal lobe. It is involved in a variety of behaviors, including decision making, and greatly contributes to personality development and expression. The ventral medial prefrontal is involved in the processing of risk and fear. Lastly, The corticolimbic system is made up of the prefrontal cortices, amygdala and hippocampus and processes a large span of behavioral and cognitive functioning, including decision making and emotional regulation.



According to the NIH, one of the most common mental health disorders in the United States is Depression and the current research suggests that depression is caused by a combination of factors that include, but are not limited to, genetics and environmental factors. What does this mean?

The researchers confirmed both of their hypotheses that there was significantly greater amygdala activation with age in the lowexpressing genotype group and lowered amygdala to ventromedial prefrontal cortex connectivity with age in the low expressing genotype group. These findings suggest that low expressing genotypes may not result in the corticolimbic system associated with depression risk until later adolescence. This means that those individuals with the low expressing gene are more likely to develop depression, however the risk may not be there until later adolescence. This allows for the possible explanation that genetic exposures may establish in functional brain differences during adolescence because of an interaction between stress and genes.