

Approximately 25% of individuals diagnosed with posttraumatic stress disorder (PTSD) will not display full symptoms for at least six months following a traumatic event; a condition known as delayed onset PTSD. McAllister and McAllister (1967) proposed that conditioned emotional responses such as fear after a long delay can increase with time; a phenomenon described as incubation. Recent studies suggest a strong relationship between fear expression and amygdala levels of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA; Rea et al., 2009). We hypothesize that the inhibitory influence of GABAergic neurons within the basolateral amygdala normally gates the expression of a fear memory, and that emergence of fear incubation results from decreased GABA synthesis. To test this, two groups of male C57BL/6J mice were context fear conditioned and tested in the same context at a recent (REC; 3 day), or remote (REM; 28 day) interval. A third within subject group (WIS) was tested at both recent and remote intervals. Behavioral data for the REC and REM groups were consistent with prior research showing that delayed testing at the REM interval yields an incubation-like increase in freezing. However, mice in the WIS group showed mixed results with some mice showing decreases and others showing increases in freezing across the REC and REM test intervals. More subjects will be tested under the same conditions to confirm these findings. To understand neural mechanisms underlying these conditions, we will use immunohistochemistry to determine cells positive for c-Fos, a marker for neural activity, and glutamic acid decarboxylase 65 (GAD-65) in the BLA and hippocampus. Both of these structures play an important role in fear memory retrieval, and have high concentrations of GABAergic cells. Our prediction is that mice that show delayed increases in freezing will have less GAD-65 positive cells than mice that fail to display fear incubation.