Since the discovery that lithium, the classic treatment for bipolar disorder, binds and inhibits GSK3β, there have been studies devoted to understanding the role of GSK3β in stress related disorders. GSK3β is a protein kinase abundant and constitutively active in the brain. Studies show that when GSK3β is phosphorylated its activity is inhibited, while when it is not phosphorylated GSK3β participates in cellular plasticity and cell death. Our colleagues have discovered a novel phosphorylation site at serine 389 that is expressed largely in the cell nucleus and appears to play a role in DNA repair. Using a genetically modified mouse, where GSK(ser)389 is replaced with an alanine and unable to be phosphorylated we have discovered a specific pattern of apoptosis in brain regions involved in stress. Consistent with this, the genetically modified mice show reduced anxiety, suggesting nuclear GSK3β may play a role in stress-associated DNA double strand break (DSB) repair. The goal of this study is to assess the role of GSK3β in stress associated regulation following variate stress. We introduced wild type mice to 14 days of variate stress and assessed the behavioral and molecular outcomes. We hypothesized that exposure to stress would increase phosphorylation at the serine 389 site. Our results show that stress alters phosphorylation of GSK3β in areas of the brain associated with stress pathology.