

**Abstract: Neuroinflammatory Response to LPS Challenge in Microglia via the NF- κ B
Pathway: Implications in Depression and Sex Differences**

Depression is a disorder that plagues our modern society, often initiated and exacerbated by stress. NF- κ B, the master regulator of innate immune responses in microglia and the mediator of pro-inflammatory cytokine release, has been seen to have significantly increased activation in response to stress, so what role does NF- κ B have in the pathogenesis of depression? In studies modeling depression, lipopolysaccharide (LPS) has long been regarded as an effective model. LPS exposure in rodents leads to sickness behaviors, such as reductions in locomotion and food intake, as well as increased levels of pro-inflammatory cytokines. This study aims to understand the physical, emotional, and cognitive effects of stress on female rats at the protein level by identifying the effects of LPS exposure on NF- κ B and microglial cell activation in the striatum, thalamus, and dentate gyrus. Across all brain regions, the striatum showed significant difference in microglial activation, with decreased cell length processes and increased cell size in the presence of an LPS challenge. However, when examined within the context of NF- κ B expression, although the striatum showed significant increase in colocalization as well, a followed fold increase analysis showed that this result was even more prominent in the dentate gyrus of the hippocampus. This implicates that depression may have a significant response on microglial inflammation response, however this effect may not be isolated to the NF- κ B pathway. This research will act as an emerging framework for further investigation of sex differences in depression, both in retrospective analysis and prospective study.