Multiple sclerosis (MS) is a neurodegenerative disease characterized by inflammation in the central nervous system, leading to demyelination and subsequent impairment of neural communication. While MS disproportionately affects females and is associated with a shorter lifespan, the underlying mechanisms, particularly regarding the role of tumor necrosis factor-alpha (TNF-α) and microglia, remain unclear. In this study, we aimed to investigate the expression of TNF-α in female rodent microglia within the thalamus after inducing an inflammatory response with lipopolysaccharide (LPS), mimicking systemic inflammation. Immunohistochemistry was used to analyze colocalization of TNF-α and microglia in LPS-induced rats compared to controls. Results indicated no significant differences in microglia soma area or processes length between LPS-induced and control rats. However, a significant increase in colocalization of TNF-α and microglia was observed in LPS-induced rats. These findings suggest a potential role of microglial TNF-α in neuroinflammatory responses associated with MS. Limitations include a small sample size and sex bias towards female rodents. Future research should explore sex differences and investigate therapeutic interventions targeting TNF-α in MS treatment. Understanding the complex interplay between TNF-α, microglia, and neuroinflammation is crucial for developing effective therapies to mitigate MS progression and improve patient outcomes.