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Microglia shape synaptic development and maintenance. Here, we propose that SRGAP2 regulates microglial morphology by adjusting cytoskeletal dynamics, and in turn, controls neuronal synaptic density by modulating synaptic pruning.

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NEUROINFLAMATION ASSOCIATED WITH NON-MOTOR SYMPTOMES IN PARKINSON'S DISEASE- INSIGHTS FROM THE A53T MOUSE MODEL

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The molecular mechanisms behind non-motor symptoms of Parkinson's disease are poorly understood. In literature, the connection between neuroinflammation and depressive symptoms was observed in PD patients, however the implications of such findings are limited due to cerebrospinal fluid and blood derivatives being the only obtainable samples for analysis. The aim of this preliminary study was to assess neuroinflammation, memory deficits and depression like symptoms associated with PD and to detect pro-inflammatory mediators associated with it using a transgenic A53T mouse model. The transgene consists of the human α-synuclein sequence with the A53T mutation driven by the mouse prion protein (PrP) promoter resulting in over-expression of human α-synuclein in key structures associated with PD. Although it was determined that microglial activation is increased in these mice, the specific cytokine profile driving the inflammation has not been characterized in literature. We have examined the expression of three key inflammatory mediators (tumor necrosis factor alpha (TNFα), interleukin-1beta (IL-1β) and interleukin-6 (IL-6) described as markers associated with the microglial pro-inflammatory phenotype in the prefrontal cortex, hippocampus, and striatum of 8 male 6 months old A53T mice. Additionally, depressive like symptoms and anhedonia were assessed using Tail Suspension Test and Sucrose Preference Test respectively, while cognitive status and memory deficits were assessed using Novel Object Recognition Test. Non-transgenic litter mates were used as controls. Obtained results depict a characteristic