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GPER agonist G1 attenuates LPS-induced inflammatory responses in murine BV-2 microglial cells

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Accumulating clinical and experimental evidence suggests that chronic neuroinflammation is associated with the progressive death of dopaminergic neurons in Parkinson's Disease (PD). The G Protein-Coupled Estrogen Receptor (GPER) was reported to be a novel membrane estrogen receptor which responds to estrogen and mediates estrogen's rapid cellular effects. Studies have shown that activation of GPER by selective agonist, G1 could account for some of the protective effects of estrogen against inflammatory responses in animal model of multiple sclerosis. The present study aimed to evaluate the protective effects of G1 on lipopolysaccharide (LPS)-induced microglia activation. We demonstrated that GPER was expressed in BV2 microglial cells. G1 treatment significantly inhibited the LPS-induced production of pro-inflammatory cytokines such as iNOS, COX2, IL-1β and TNFα. These effects could be abolished by GPER antagonist G15 and lentivirus-mediated knock-down of GPER. Moreover, LPS treatment significantly increased the phosphorylation level of ERK, JNK and p38 in BV2 microglial cells. G1 pretreatment could remarkably attenuate these changes while co-treatment with G15 could reverse the protective effects of G1. Taken together, this study demonstrated that GPER agonist G1 can prevent LPS-induced activation of microglia. GPER may be a novel therapeutic target for chronic inflammatory diseases.

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