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GPER is involved in the anti-inflammatory effects of genistein in BV2 microglial cells

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Abstract: Objective Genistein is an isoflavonoid phytoestrogen extracted from leguminous plants exerting both estrogen agonist and antagonist activities. The present study hypothesized that the anti-inflammatory effects of genistein against lipopolysaccharide (LPS)-induced BV2 microglial activation might be mediated by membrane estrogen receptor (G-protein coupled estrogen receptor, GPER). Methods The anti-inflammatory effects of genistein were investigated in LPS-induced microglial activation in murine BV2 cells. G1, the specific agonist of GPER, was used as a positive control. The pharmacological blockade and lentivirus-mediated siRNA knockdown of GPER were used to study the underlying mechanism. Results Genistein mimicked the effects of G1 treatment by inhibiting the LPS-induced gene expressions of inflammatory cytokines, including TNF-α, IL-1β, COX-2 and iNOS in BV2 microglial cells. Pre-treatment with GPER antagonist G15 or siRNA knockdown of GPER could significantly block the anti-inflammatory effects of genistein and G1. Furthermore, pretreatment with G15 could also block the inhibitory effects of genistein on LPS-induced phosphorylation of JNK, p38, ERK and IκB. Conclusion Genistein exerts anti-inflammatory effects in BV2 microglial cells possibly by the activating GPER. Keywords: genistein; G protein-coupled estrogen receptor; insulin-like growth factor 1 receptor; microglia