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Gastrodia elata Blume extract inhibits the production of nitric oxide and proinflammatory mediators in LPS-stimulated BV-2 microglia cells via JNK mitogen-activated protein kinase and NF-kappa B singnaling pathway

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실험목적 (Objectives)

A large body of evidence has suggested a strong association between neuroinflammation and the pathogenesis of many neurodegenerative diseases. Inflammatory response in the central nervous system mediated by activation of microglia is a key event in the early stages of the development of neurodegenerative diseases. Suppression of microglial activation would have therapeutic benefits, which lead to alleviation of the progression of neurodegeneration. It seems possible that treatment with antiinflammatory agents, including oriental and traditional medicinal plants, might delay the progression of neurodegeneration through the inhibition of microglial activation. The present study is focused on the inhibitory effect of GE extract on the production of proinflammatory mediators in LPS-stimulated BV-2 microglial cells.

재료 및 방법 (Materials and Methods)

○ Materials

Gastrodia elata Blume (Orchidaceae, GE) was purchased from the traditional herb market, authenticated by a taxonomist, Plant Extract bank, Korea and a voucher specimen (CA04-048) has been deposited at the institute's herbarium.

○ Methods

- Cell culture and viability assay
- Assay of NO production
- Isolation of total RNA and RT-PCR
- Western blot analysis
- Immunofluorescence assay

실험결과 (Results)

GE inhibited LPS-induced production of nitric oxide (NO) in a dose dependent fashion. The mRNA and protein levels of inducible NO synthase (iNOS) and cyclooxygenase-2 (COX-2) was also decreased by GE dose-dependently. GE also

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suppressed NF-κB activation by blocking IκB degradation. Furthermore, GE also inhibited the JNK mitogen-activated protein kinase (MAPKs) in LPS-stimulated BV-2 cells. GE have several phenolic compounds, such as Gastrodin [4-Hydroxybenzyl alcohol 4-O-bata-D-glucoside], vanillyl alcohol [VA, 4-hydroxy-3-methoxybenzaldehyde], Gastrodin metabolite [GM, 4-hydroxybenzyl alcohol], vanillin and 4-hydroxybenzaldehyde [4HBA]. Considering the results obtained, our study demonstrates that GE exhibit anti-inflammatory effects by suppressing the production of proinflammatory mediators through MAPK and NF-κB signaling pathways in activated microglial cells and might be developed as a promising candidate in treating various neuroinflammatory diseases.

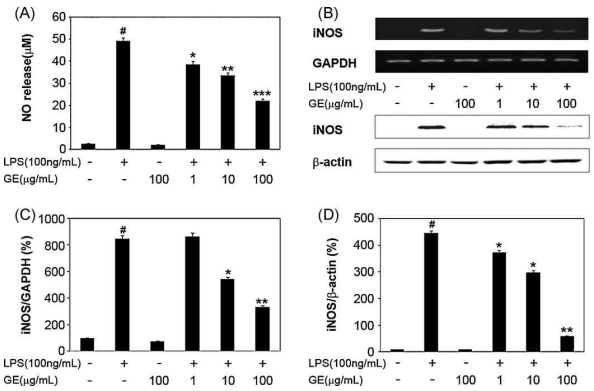

Fig. 1. Inhibition of NO release and iNOS production by GE in LPS-stimulated BV-2 microglia

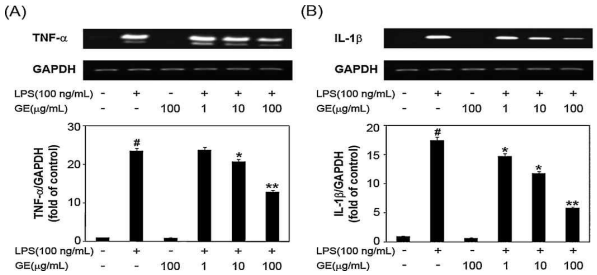

Fig. 2. Effect of GE on pro-inflammatory cytokines in LPS-stimulated BV-2 microglia.

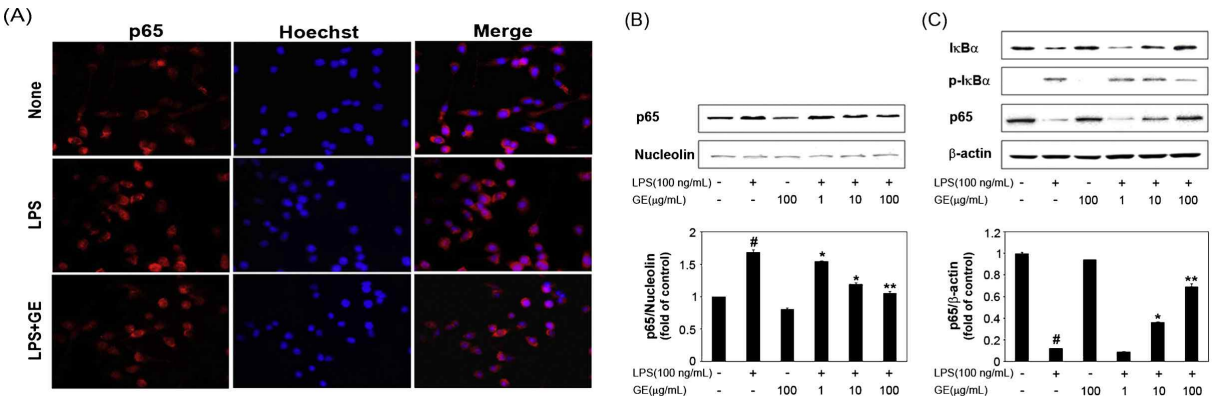

Fig. 3. Effect of GE on NF-κB activity in LPS-stimulated BV-2 microglia. Sub-cellular location of NF-B p65 subunit was determined by immunofluorescence assay (A). Total nuclear protein using anti-NF-κB p65 (B) and IκB-α(C) was measured following western blotting.