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Sunggaltang(升葛湯) suppresses LPS-induced NO production and iNOS expression in microglial cells

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It have been reported that Spleen-Stomach (脾胃) system has power to control total human body and spirit in Traditional Korean Medicine. In other words, it is that treats diseases through to supply energy to deficiency other organism. And so it is regarded totally as to improve weakness and to remove foreign invader, including inflammatory factors and viral infection. Seunggaltang (SKT: 升葛湯) had been made to as purpose to remove the damp-heat (濕熱) of the Spleen-Stomach (脾胃) from Seungmagalgeuntang (升麻葛根湯) prescription in the Dongeui Oriental Hospital. Therefore this herbal drug may affect to the key parameters of inflammation as production of nitric oxide (NO), cyclooxygenase (COX)-2, prostaglandin E_2, and proinflammatory cytokines. We studied the effect of SKT on NO production. SKT inhibited the secretion of NO and expression level of inducible nitric oxide synthase (iNOS) in BV2 microglia, without affecting cell viability. And SKT also reduced production of PGE_2 and expression of COX-2 mRNA. Proinflammatory cytokines such as TNF-α, IL-1β, IL-12 were inhibited by SKT in a dose-dependent manner.

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Inhibitory Effect of Hwanggigunjungtang (黃耆健中湯) on the secretion of PGE2 and NO in LPS-stimulated BV2 microglial cells

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Hwanggi-gunjungtang (HGT: 黃耆健中湯) has been used to as purposes to tonify the spleen and replenish Ki (補脾益氣) effects in Traditional Korean Medicine. HGT has been used for treatment of chronic diseases in the folk medicine recipe. Since nitric oxide (NO) is one of the major inflammatory parameter, we firstly studied the effect of aqueous extracts of HGT on NO production in lipopolysaccharide (LPS)-stimulated BV-2 microglia. HGT inhibited the secretion of NO in BV-2 microglia, without affecting cell viability. The protein level of inducible nitric oxide synthase (iNOS) was decreased by HGT. And HGT also inhibited production of PGE_2 and expression of COX-2. Proinflammatory mediators such as TNF-α, IL-1β, IL-12 were inhibited by HGT in a dose-dependent manner.

This results indicate that HGT could exert its anti-inflammatory effects by suppressing the synthesis of NO and proinflammatory mediators.