Modulatory EFFECTS OF Panax ginseng AGAINST neuroinflammation Induced by lipopolysaccharide/interferon gamma

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Korean ginseng (*Panax ginseng* C.A. Meyer) is one of the most widely used medicinal plants, particularly in traditional oriental medicine. Recently, there is increasing evidence in the literature on the pharmacological and physiological actions of ginseng. Recent research has suggested that some of ginseng's active ingredients exert antioxidant, anti-inflammatory, anti-apoptotic, and immune-stimulatory activities all of which are mostly underlying the possible ginseng-mediated protective mechanisms. Although *Panax* ginseng exerts the beneficial effects in the wide range of actions, little is known about the mechanism by which *Panax* ginseng rescues the neuronal cells from inflammation. In the present study, we investigated whether *Panax* ginseng possesses modulatory effects against neuro-inflammation induced by lipopolysaccharide/interferon gamma. For this study, human neuronal SK-N-MC or rat C6 microglial cells were activated by treating lipopolysaccharide (10 μg/ml)/interferon gamma (100 U/ml) and were evaluated the modulatory effects of *Panax* ginseng against the expression of inflammation cytokine-specific mRNA by multiplex RT-PCR. Our results clearly demonstrate that *Panax* ginseng extracts down-regulated the expression of cytokines IL-1 receptor and IL-1α induced by LPS/INF-γ, suggesting that ginseng extracts mediated neuroinflammation induced by (pro-)inflammatory cytokines. We also evaluated potential of *Panax* ginseng components as the ligand of Peroxisome Proliferator-Activated Receptor Gamma (PPARγ), which plays in brain inflammatory conditions because various PPARγ ligands inhibit proinflammatory mediators, such as cytokines and inducible nitric oxide synthase. We suggest that both panaxdiol and panaxtriol can be used as modulators of PPARγ. These data suggest that the protective effects of *Panax* ginseng against LPS/ INF-γ-induced neuro-inflammation may be mediated by the suppression of NO synthesis via down-regulation of pro-inflammatory cytokines activation as well as by acting as the ligand of PPARγ in the neuronal cells. [Supported by a grant from Korean Ginseng Society 2005]

Key words; cytokine, Lipopolysaccharide, *Panax* ginseng, neuroinflammation, PPARγ,