

Enhancing of learning activity and modulation of neuronal function by red ginseng

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We assessed the effect of ginseng on the learning and memory impairments induced by scopolamine. The cognitive-enhancing effect of ginseng extract was investigated using a passive avoidance test. Drug-induced amnesia was induced by treating animals with scopolamine (1mg/kg, i.p.) administration significantly reversed scopolamine-induced cognitive impairments in mice by passive avoidance test. In addition, ginseng improved the A β -induced memory impairment and oxidative stress.

We determined the antagonism of the ginseng total saponin (GTS) on the development of nalbuphine-induced tolerance and physical dependence. GTS is known to have antinarcotic action with a dose of 100mg/kg (i.p.) in rats. GTS significantly inhibits the development of nalbuphine-induced physical dependence as well as the tolerance. The level of pCREB was elevated in the striatum by the chronic treatment with nalbuphine or GTS, however, the elevation of pCREB was inhibited by the GTS co-treatment. It has been suggested that NMDA receptor and/or NO is involved in the phenomena of opioid dependence and withdrawal. However, the level of nNOS and NR1 was not modulated by the treatment with nalbuphine or GTS on the cortex, hippocampus and striatum in the rat brain. These results suggest that the GTS could be used to ameliorate the nalbuphine tolerance and withdrawal symptoms.

Ginseng saponins are transformed by intestinal microflora and the transformants would be absorbed from intestine. In the present study, we have investigated the effects of biotransformed ginsenoside Rg3, Rh2 and compound K on the modulation of NMDA receptor and GABA_A receptor binding in rat brain. The NMDA receptor binding was analyzed by quantitative autoradiography using [³H]MK-801 binding, and GABA_A receptor bindings were analyzed by using [³H]muscimol and [³H]flunitrazepam binding in rat brain slices. Ginsenoside Rg3, Rh2 and compound K were infused (10 μ g/10 μ l/ hr) into rat brain lateral ventricle for 7 days, through pre-implanted cannula by osmotic minipumps (Alzet, model 2ML). Ginsenoside Rh2 and compound K induced the downregulation of the [³H]MK-801 binding as well as upregulation of the and [³H]muscimol binding in a region-specific manner after prolonged infusion into lateral ventricle. However, ginsenoside Rg3 did not show the significant changes of ligand bindings. In

addition, ginsenoside Rh2 and compound K modulated the expression of nNOS in hippocampus and striatum. These results suggest that prolonged infusion of ginsenosides could differentially modulate [³H]MK-801 and [³H]muscimol binding in a region-specific manner and ginsenoside Rh2 and compound K could play an important role in the biological activities in the central nervous systems and neurodegenerative disease.

In summary, ginseng could be used to enhance the brain memory function and ameliorate the opioid tolerance and withdrawal symptoms.

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