Ginsenosides Rg1 and Rb1 enhance glutamate release through activation of protein kinase A in rat cerebrocortical nerve terminals (synaptosomes).

Chang Y, Huang WJ, Tien LT, Wang SJ.

Abstract

We examined the effect of ginsenoside Rg1 or Rb1, the active ingredients of ginseng, on the release of endogenous glutamate from glutamatergic nerve terminals purified from rat cerebral cortex. Result showed that the Ca(2+)-dependent release of glutamate evoked by 4-aminopyridine was facilitated by ginsenoside Rg1 or Rb1 in a concentration-dependent manner. Sequential experiments reveal that ginsenoside Rg1 or Rb1-mediated facilitation of glutamate release (i) results from an enhancement of vesicular exocytosis; (ii) is not due to an alternation of synaptosomal excitability; (iii) is associated with an increase in Ca(2+) influx through presynaptic N- and P/Q-type voltage-dependent Ca(2+) channels; (iv) appears to involve a protein kinase A pathway. These results conclude that ginsenoside Rg1 or Rb1 exerts their presynaptic facilitatory effect, likely through the activation of protein kinase A, which subsequently enhances Ca(2+) entry to cause an increase in evoked glutamate release from rat cortical synaptosomes. This finding might provide important information regarding the action of ginseng in the central nervous system.