

Cimicifugoside Inhibits Catecholamine secretion by blocking Nicotinic Acetylcholine Receptor in Bovine Adrenal Chromaffin cell.

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The medicinal plant *Cimicifuga Racemosa* (Black cohosh) has been used to treat many kinds of neuronal and menopausal symptoms, such as arthritis, menopausal depression, nerve pain, etc. Here, we examined the effect of Cimicifugoside (CF), one of triterpene glycosides which have been known as pharmacologically active ingredients of *C. Racemosa*, on nicotinic acetylcholine receptor (nAChR)-mediated catecholamine (CA) secretion in bovine adrenal chromaffin cell. Cimicifugoside inhibited calcium increase induced by 1,1-dimethyl-4-phenylpiperazinium iodide (DMPP), a nAChR agonist with a half maximal inhibitory concentration (IC₅₀) of $18 \pm 2 \mu\text{M}$. In contrast, cimicifugoside did not affect the calcium increases evoked by high K⁺, veratridine, and bradykinin. The DMPP-induced sodium increase was also inhibited by cimicifugoside with IC₅₀ of $2 \pm 0.3 \mu\text{M}$, suggesting that the activity of nAChRs is inhibited by cimicifugoside. Cimicifugoside did not effect on the KCl-induced secretion but markedly inhibited the DMPP-induced catecholamine secretion which was monitored by carbon-fiber amperometry in real time, and by high performance liquid chromatography (HPLC) through electrochemical detection. The results suggest that cimicifugoside selectively inhibits nAChR-mediated response in bovine chromaffin cells.