INFORMATION

Hokkaido University confered the degree of Bachelor of Veterinary Medicine to the following 39 graduates of the School of Veterinary Medicine on March 25, 1997.

The authors summaries of their theses are as follows:

Comparison between inhibitory effects of substance P and of hexamethonium on nicotinic acetylcholine receptor in bovine adrenal chromaffin cells

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1. It has well been documented that substance P inhibits catecholamine secretion induced by nicotinic agonists in adrenal chromaffin cells. In the present study, the mode of inhibitory effect of substance P on the nicotine-induced cytosolic Ca^{2+} concentration ($[Ca^{2+}]_c$) was analyzed in isolated bovine adrenal chromaffin cells.

2. A transient $[Ca^{2+}]_c$ rise was recorded during continuous stimulation with nicotine in Fura-2-loaded chromaffin cells. A quantitative relation was found between the peak levels of the nicotine-induced $[Ca^{2+}]_c$ rise and the concentrations of nicotine over the range 10–100 μM. The most satisfactory theory which explains the cellular mechanism of nicotine-induced $[Ca^{2+}]_c$ rise, and which fits the experimental data, requires the dominant activity of binding of two molecules of nicotine with two binding sites of nicotinic receptor in the sequence of event resulting in a $[Ca^{2+}]_c$ rise.

3. The addition of substance P (10 μM) to the extracellular solution reversibly reduced the peak levels of nicotine-induced $[Ca^{2+}]_c$ rise. The inhibitory effect of substance P on nicotine-induced $[Ca^{2+}]_c$ rise could quantitatively be explained by a kinetic scheme based on noncompetitive inhibitory action of substance P on the nicotinic receptor molecule.

4. The addition of hexamethonium to the extracellular solution reversibly reduced the peak levels of $[Ca^{2+}]_c$ response to nicotine at lower concentration (10 μM), but caused little, if any, inhibition of the peak $[Ca^{2+}]_c$ level in response to nicotine in a high concentration range (20, 50 or 100 μM). The inhibitory effect of hexamethonium on the nicotine-induced $[Ca^{2+}]_c$ rise could quantitatively be explained by a kinetic scheme based on competitive inhibitory action of hexamethonium on the binding of nicotine with the nicotinic receptor molecule.