[PA1-8] [04/21/2000 (Fri) 10:30 - 11:30 / [1st Fl, Bldg 3]]

The influences of magnetic fields on clonidine-induced sleep in two-day-old chicks

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It has been shown that magnetic fields (MFs) affect a variety of biological effects in animal brain. There have been few experiments on the effects of MFs on sleep. Therefore, we investigated whether MFs affect the sleep induced by clonidine, central α_2 adrenergic receptor agonist. clonidine produced dose-related increase the sleeping time and dose-related decrease onset time in two-day-old chicks. Exposure of MFs (5, 10, 20 gauss; for 3, 6, 9, 12hrs) to chicks significantly increased the clonidine-induced sleep time in intensity and exposure duration-dependent manners of MFs. To determine whether the $GABA_A$/benzodiazepine receptor system is involved in the decrease in clonidine sleep caused by activation of central α_2 adrenergic system, we examined in chicks the effects of the benzodiazepine receptor antagonist flumazenil (0.5mg, i.p.) and $GABA_A$ antagonist bicuculline (0.1mg, i.p.) on clonidine-induced sleep. Bicuculline and flumazenil inhibited the increase of clonidine-induced sleep by MFs. These results suggest that MFs can increase clonidine-induced sleep via GABA and benzodiazepine receptor system.

[PA1-9] [04/21/2000 (Fri) 10:30 - 11:30 / [1st Fl, Bldg 3]]

Facilitatory effect of cytisine on catecholamine secretion

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The present study was attempted to examine the characteristics of cytisine on catecholamine secretion in the isolated perfused rat adrenal gland and to clarify the mechanism of its action. Cytisine (5 to 20 mM) injected into an adrenal vein evoked a dose-dependent significant secretory response of catecholamines (CA) from the rat adrenal gland. However, upon the repeated injection of cytisine (10 mM) at 15 min intervals, CA secretion was rapidly decreased after third injection of histamine. CA release evoked by the continuous infusion of cytisine was also gradually reduced from 15 min after the initiation of cytisine infusion. Tachyphylaxis to releasing effects of CA evoked by cytisine was observed by the repeated administration. The cytisine-induced CA secretion was markedly inhibited by the pretreatment with chlorisondamine, nicardipine, TMB-8, and perfusion of Ca^{+2}-free Krebs solution, while was not affected by pirenzepine and diphenhydramine. Moreover, the CA secretion evoked by acetylcholine was greatly potentiated by the prior perfusion of cytisine (5 mM). Taken together, these experimental data suggest that cytisine causes CA secretion in a calcium-dependent fashion from the perfused rat adrenal gland through activation of neuronal nicotinic receptors located in adrenomedullary chromaffin cells. It also seems that cytisine-evoked catecholamine release is not relevant to activation of cholinergic muscarinic or histaminergic receptors.

[PA1-10] [04/21/2000 (Fri) 10:30 - 11:30 / [1st Fl, Bldg 3]]

Effects of DMSO (dimetyl sulfoxide) on degranulation and tyrosine phosphorylation of the FcεRI signaling components in RBL-2H3 cells

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