

[PA1-1] [ 2003-10-10 14:00 - 17:30 / Grand Ballroom Pre-function ]

### **The Mechanism of Contraction Response to EFS in Cat Esophageal Circular Muscle**

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Low-frequency electrical field stimulation of transmural nerves of cat esophageal circular smooth muscle produces an "off contraction", which occurs after electrical field stimulation (EFS) of transmural nerves is stopped. We previously examined signal transduction pathways mediating ACh-induced contraction of circular smooth muscle of esophagus. The extracellular  $Ca^{2+}$  is needed for the contraction, results in the activation PKC. EFS-induced contraction was abolished by the pretreatments of tetrodotoxin (1 $\mu$ M) and atropine (1 $\mu$ M). In the present study, we investigated whether EFS-induced contraction would be affected by inhibitors of phospholipases and protein kinases. This contraction was not blocked by D609 (10 $\mu$ M, PC-PLC inhibitor), pCMB (10 $\mu$ M, PLD inhibitor), DEDA (10 $\mu$ M,  $PLA_2$  inhibitor). PKC inhibitor, GF109203X (10 $\mu$ M), did not reduce the response. We further studied whether extracellular  $Ca^{2+}$  may be required or not for the contraction. Nimodipine (100nM, L-type  $Ca^{2+}$  channel blocker) decreased the contractile response by 50% approximately. Our study suggests that the off contraction in cat esophageal circular smooth muscle may be mediated through different signals when compared with agonist-induced contraction.

[PA1-2] [ 2003-10-10 14:00 - 17:30 / Grand Ballroom Pre-function ]

### **Comparison of conotoxin GVIA and cilnidipine on nicotinic receptor stimulation-induced catecholamine release in the rat Adrenal Gland**

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The present study was designed to compare the effects of conotoxin GVIA, a selective blocker of N-type voltage-dependent calcium channels (VDCC) and cilnidipine, a blocker of both L- and N-type VDCC, on the secretion of catecholamines (CA) evoked by cholinergic stimulation and membrane-depolarization in the isolated perfused rat adrenal gland, and also to establish the mechanism of action. 1. The inhibition of the CA secretory response evoked by acetylcholine ( $5.32 \times 10^{-3}$  M) was stronger in cilnidipine-treated glands than in conotoxin GVIA-treated glands. However, the CA secretion evoked by high potassium ( $5.6 \times 10^{-2}$  M), a membrane depolarizer, was more significantly inhibited in conotoxin GVIA-treated glands than in cilnidipine-treated glands. 2. The secretory responses of CA evoked by DMPP ( $10^{-4}$  M for 2 min), a selective agonist of neuronal nicotinic receptors, and McN-A-343 ( $10^{-4}$  M for 2 min), a selective agonist of neuronal muscarinic receptors, were also more depressed in cilnidipine-treated glands than in conotoxin GVIA-treated glands. 3. The CA release evoked by Bay-K-8644 ( $10^{-5}$  M), a dihydropyridine-sensitive  $Ca^{2+}$  channel activator, was more significantly inhibited in cilnidipine-treated glands than in conotoxin GVIA-treated glands. The inhibition by conotoxin GVIA of the CA release evoked by cyclopiazonic acid ( $10^{-5}$  M), a selective inhibitor of  $Ca^{2+}$ -ATPase in the endoplasmic reticulum, was similar to that by cilnidipine. 4. Taken together, these experimental results demonstrate that the CA secretion evoked by stimulation of cholinergic (nicotinic and muscarinic) receptors is more strongly inhibited by cilnidipine than conotoxin GVIA, but the CA secretion evoked by membrane depolarization is rather more depressed by conotoxin GVIA. It seems that there is some difference in the inhibition of the CA release between conotoxin GVIA (a selective blocker of N-type VDCC) and cilnidipine (a blocker of both L- and N-type VDCC).

[PA1-3] [ 2003-10-10 14:00 - 17:30 / Grand Ballroom Pre-function ]

### **Mitogen-activated protein kinase signaling pathway mediates 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)-induced apoptosis in Jurkat T cells.**

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