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arteries precontracted with UTP and 5-HT.

7. AVP-induced oscillatory tension was not affected by the V2 antagonist. The V1 receptor agonist also elicited rhythmic oscillatory tension. L-NAME, charybdotoxin and ouabain inhibited AVP-induced oscillatory tension. 5-HT and ET-1, but not UTP, caused oscillatory response similar to AVP.

8. These results suggest that AVP causes

contractions by stimulation of V1 receptors on vascular smooth muscle via Ca^{2+} released from intracellular stores and Ca^{2+} influx through voltage-dependent Ca^{2+} channels and non-selective cation channels. The endothelium seems to have an inhibitory effect on AVP-induced contraction in the rat basilar artery.

Inhibitory effects of opioids on voltage-dependent calcium channels in cultured porcine adrenal chromaffin cells

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1. Inhibitory effects of opioids on voltage-dependent calcium channels were studied in cultured porcine adrenal chromaffin cells using a whole-cell patch clamp technique. The effect of opioid on catecholamine release induced by high K^+ was also examined. We identified opioid receptor subtypes expressed in porcine adrenal chromaffin cells using a RT-PCR method.

2. A depolarizing pulse to 0 mV (test pulse) from a holding potential at -80mV evoked an inward barium current (I_{Ba}). Met-enkephalin (met-ENK) reversibly inhibited I_{Ba} and this inhibition was significantly reduced by naloxone.

3. Selective opioid receptor agonists (DAMGO; μ , DPDPE; δ , U50488; κ) also reversibly inhibited I_{Ba} . The order of the inhibitory potency was DAMGO > U50488 > DPDPE.

4. The inhibitory effect of DAMGO on I_{Ba} almost disappeared in the presence of ω -

conotoxin GVIA but not ω -agatoxin IVA plus nifedipine.

5. Application of a depolarizing pulse to +100mV (prepulse) prior to a test pulse caused increases in the amplitude of I_{Ba} in response to the test pulse by about 15%. Application of prepulse partly reduced I_{Ba} inhibition induced by opioids.

6. Intracellular application of GDP β S or GTP γ S and pretreatment with pertussis toxin significantly decreased I_{Ba} inhibition induced by DAMGO.

7. The amplitude of I_{Ba} was decreased by cessation of external perfusion. The decrease in I_{Ba} was not affected by naloxone and depolarizing prepulse.

8. Met-ENK did not produced a significant inhibition of catecholamine release induced by high K^+ .

9. The RT-PCR revealed the expression of μ , δ and κ opioid receptors in the adrenal chromaffin cells as well as cerebral cortex of the

pig.

10. These results indicate that porcine adrenal chromaffin cells possess μ , δ and κ opioid receptors. It is suggested the activation of

opioid receptors inhibits N-type voltage-dependent calcium channels mainly via pertussis toxin sensitive G-proteins.

Catecholamine and ATP metabolites released from perfused adrenal glands of guinea-pig

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1. I investigated relationship between catecholamine and ATP metabolites released from perfused guinea-pig adrenal glands.

Adenine nucleotide and adenosine were measured with HPLC after conversion of these purine compounds to eteno-derivatives.

2. In HPLC analysis, ATP, ADP, AMP and adenosine were increased linearly with increasing their concentrations. The detection limits were 10-30nM.

3. The effluent from the adrenal glands contained a small amount of ADP under the resting condition. There is no relationship between the amounts of ADP and catecholamine in the effluent.

4. KCl and acetylcholine caused the release of catecholamines and ATP metabolites in dose-dependent manners. The molar ratio of catecholamine to ATP metabolites appearing in the effluent (CA/AM) was about 10 for both stimuli.

5. Veratridine and sustained application of 40mM KCl for 30 min caused long-lasting secretion of catecholamine and ATP metabolites.

The time course of secretion of catecholamine was somewhat different from that of ATP metabolites. When the amounts of ATP and ADP were subtracted from those of ATP metabolites, the time course of release of ATP metabolites (AMP and adenosine) was consistent with that of catecholamine. The CA/AMP plus adenosine was about 12 for both stimuli.

6. These results suggest that the time course of ATP secretion from the adrenal gland is consistent with that of catecholamine secretion. The molar ratio (10) of catecholamine to ATP metabolites appearing in the adrenal effluent was significantly different from those found in chromaffin vesicles (4). The released ATP seems to be significantly metabolized in the blood vessels of the perfused guinea-pig adrenal glands.