cloned and shown to function as a partial uncoupler. The expression of UCP-2 and UCP-3 is not limited to BAT. UCP-2 is widely expressed in many tissues, whereas UCP-3 is specific to skeletal muscles and adipose tissues. However, physiological function of these new UCPs remains to be determined.

In the present study, I examined UCPs mRNA expression in a model of diet-induced obesity between prone (C57BL) versus resistant mice (A/J). After 3 weeks feeding cafeteria-diet or normal laboratory chow, C57BL mice with cafeteria-diet showed a large increase in body weight compared to control diet group. In contrast, there was no difference in body weight increase between control and cafeteria-diet groups in A/J mice. From these results it is confirmed that A/J mice strain is resistant to cafeteria-diet induced obesity. In the control diet group, expression levels of UCP-1 and UCP-2 in BAT, and UCP-2 in white adipose tissue (WAT) were higher in A/J mice than in C57BL. After feeding cafeteria-diet, UCP-2 expression in BAT and WAT tended to increase in A/J mice, but no change was observed in C57BL mice. Therefore, I suggest that there is a possible relation between the induction of UCP-2 in adipose tissues and resistance to diet-induced obesity in A/J mice. Supporting this idea, peroxysome-proliferator activating receptor gamma (PPAR-g) mRNA in BAT was induced by cafeteria-diet in A/J mice; PPARs have been proposed as an inducer of UCPs expression.

Contractile effects of vasopressin on isolated rat basilar artery

Eriko KATORI

Laboratory of Pharmacology, Department of Biomedical Sciences, School of Veterinary Medicine, Hokkaido University, Sapporo, 060-0818, Japan

1. The contractile effects of arginine-vasopressin (AVP) were investigated in ring preparations isolated from the rat basilar artery.
2. AVP caused concentration-dependent contractions. In the presence of endothelium, AVP induced rhythmic oscillatory tension superimposed on a tonic contraction. In the absence of endothelium, AVP-induced contractions were accompanied with irregular transient relaxations. Removal of endothelium shifted a concentration-response curve to AVP leftward and upward.
3. In endothelium-denuded preparation, a concentration-response curve to AVP was shifted rightward and downward by a selective antagonist to V1, but not V2 receptor. A V1, but not V2 receptor agonist, caused concentration-dependent contractions with a similar potency to AVP.
4. Under Ca$^{2+}$-free conditions, contractions by 0.1nM of AVP were abolished, but 10nM of AVP induced transient contractions. AVP-induced contractions were inhibited by nifedipine, SK&F96365 and niflumic acid.
5. AVP-induced irregular transient relaxations in endothelium-denuded preparation were inhibited by charybdotoxin.
6. In the presence of endothelium, AVP, V1 and V2 agonists did not produce relaxation in
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 chromafin cells

Go KITAMURA

Laboratory of Pharmacology, Department of Biomedical Sciences, School of Veterinary Medicine, Hokkaido University, Sapporo060-0818Japan

1. Inhibitory effects of opioids on voltage-dependent calcium channels were studied in cultured porcine adrenal chromafin 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 chromafin cells using a RT-PCR method.
2. A depolarizing pulse to 0 mV (test pulse) from a holding potential at -80 mV evoked an inward barium current (I\(_{\text{ba}}\)). Met-enkephalin (met-ENK) reversibly inhibited I\(_{\text{ba}}\) and this inhibition was significantly reduced by naloxone.
3. Selective opioid receptor agonists (DAMGO; \(\mu\), DPDPE; \(\delta\), U50488; \(\kappa\)) also reversibly inhibited I\(_{\text{ba}}\). The order of the inhibitory potency was DAMGO > U50488 > DPDPE.
4. The inhibitory effect of DAMGO on I\(_{\text{ba}}\) almost disappeared in the presence of \(\omega\)-conotoxin GVIA but not \(\omega\)-agatoxin IVA plus nifedipine.
5. Application of a depolarizing pulse to +100 mV (prepulse) prior to a test pulse caused increases in the amplitude of I\(_{\text{ba}}\) in response to the test pulse by about 15%. Application of prepulse partly reduced I\(_{\text{ba}}\) inhibition induced by opioids.
6. Intracellular application of GDP\(\beta\)S or GTP\(\gamma\)S and pretreatment with pertussis toxin significantly decreased I\(_{\text{ba}}\) inhibition induced by DAMGO.
7. The amplitude of I\(_{\text{ba}}\) was decreased by cessation of external perfusion. The decrease in I\(_{\text{ba}}\) was not affected by naloxone and depolarizing prepulse.
8. Met-ENK did not produce a significant inhibition of catecholamine release induced by high K\(^{+}\).
9. The RT-PCR revealed the expression of \(\mu\), \(\delta\) and \(\kappa\) opioid receptors in the adrenal chromaffin cells as well as cerebral cortex of the