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 obesety between prone (C57BL) versus resistant mice (A/J). After 3 weeks feeding cafeteria-diet or normal laboratory chow, C57 BL 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

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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 by0.1nM 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 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 about15%. Application of prepulse partly reduced I_{Ba} inhibition induced by opioids.

6. Intracellular application of GDPβS or GTPγS 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