cloned and shown to function as a partial un­
coupler. The expression of UCP-2 and UCP-
3 is not limited to BAT. UCP-2 is widely ex­
pressed in many tissues, whereas UCP-3 is
specific to skeletal muscles and adipose tis­
ues. 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 resis­
tant mice (A/J). After 3 weeks feeding
cafeteria-diet or normal laboratory chow, C57
BL mice with cafeteria-diet showed a large in­
crease 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. There­
fore, 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-γ) 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 ar­
tery.
2. AVP caused concentration-dependent
contractions. In the presence of endothelium,
AVP induced rhythmic oscillatory tension su­
perimposed on a tonic contraction. In the ab­
sence of endothelium, AVP-induced contrac­
tions were accompanied with irregular tran­
sient 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 selec­
tive 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 relaxa­
tions 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 -80 mV (test pulse) from a holding potential at -80 mV evoked an inward barium current (IBa). Met-enkephalin (met-ENK) reversibly inhibited IBa and this inhibition was significantly reduced by naloxone.
3. Selective opioid receptor agonists (DAMGO; µ, DPDPE; δ, U50488; κ) also reversibly inhibited IBa. The order of the inhibitory potency was DAMGO>U50488>DPDPE.
4. The inhibitory effect of DAMGO on IBa almost disappeared in the presence of ω-conotoxin GVIA but not ω-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 IBa in response to the test pulse by about 15%. Application of prepulse partly reduced IBa inhibition induced by opioids.
6. Intracellular application of GDPβS or GTPγS and pretreatment with pertussis toxin significantly decreased IBa inhibition induced by DAMGO.
7. The amplitude of IBa was decreased by cessation of external perfusion. The decrease in IBa 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 µ, δ and κ opioid receptors in the adrenal chromaffin cells as well as cerebral cortex of the