Leptin modulates olfactory acuity

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We have previously shown that olfactory acuity is modulated by nutritional status. This suggests that olfactory function is under the control of neuroendocrine signals that modulate feeding behavior. Although the anorectic hormone leptin is well known to regulate food intake and energy expenditure, its action on olfactory function is postulated by the presence of its receptor in the olfactory bulb (OB). In this study, we have performed histological, electrophysiological and behavioral experiments in order to determine whether leptin is implicated in the modulation of olfactory function. Firstly, we have demonstrated that the long isoform of the leptin receptor is highly expressed in the OB and is mostly localized in mitral cells (main neurons) and granular cells. Furthermore, patch-clamp experiments show that leptin modulates mitral cells spontaneous activity either through an increase or a decrease in their mean firing frequency. In addition, using a behavioral test designed to measure sensitivity to odorants, we found that CNS delivery of leptin decreases olfactory sensitivity in a dose-dependent manner. By contrast, obese Zucker fa/fa rats, carrying a non-functional leptin receptor, display higher olfactory sensitivity than their lean controls. Taken together these data suggest that leptin is able to modulate olfactory sensitivity by altering the transmission of the sensory stimulus in the OB. These results support the notion that the sense of smell participates in the regulation of ingestive behavior by responding to hormonal cues of nutritional status.

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High calcium preference is associated with low expression of the calcium-sensing receptor gene, *Casr*, in tongue epithelial tissue

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Calcium is essential for survival but high concentrations of calcium are unpalatable to humans and most animals avoid them. An exception is the PWK/PhJ (PWK) strain of mice, which in contrast to the C57BL/6J (B6) and other inbred strains, avidly and specifically ingests calcium. A genome scan of B6 × PWK F_2 hybrid mice linked a component of this strain difference to a region on chromosome 16 at microsatellite marker *D16Mit60* (32.6 Mb, 23.4 cM), with a peak LOD score of 8.0. Nearby (36.4 Mb, 26.3 cM) is the calcium-sensing receptor gene, *Casr*, which has a central role in extracellular calcium homeostasis. To evaluate *Casr* as a candidate gene responsible for the behavioral phenotype, we compared *Casr* DNA sequences between B6 and PWK mice. There were six polymorphisms in the coding region but these were synonymous and so were unlikely to influence receptor function. However, there were several polymorphisms upstream, which may affect the amount of mRNA expressed. Therefore, we measured expression of *Casr* mRNA in B6 and PWK mouse taste and non-taste tissues by real-time quantitative PCR. Using three *Casr* gene expression assays with probes targeting different parts of the gene, we found that Casr expression was 1.5–7.0-fold higher in B6 than PWK in both taste and non-taste epithelial tissues. We speculate that *Casr* expression negatively regulates calcium intake, so the PWK strain's avidity for calcium is due, at least in part, to relatively low expression of *Casr* in taste or other tissues.

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Within-meal eating rate and 1-h appetite in slow, medium, and fast paced eaters

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Eating slowly is recommended for weight management, but data are lacking on how it may influence energy intake regulation, and if it can be easily adopted. We asked 90 females (22 ± 6 yr; BMI = 22 ± 3) to rate themselves as slow ($n = 13$), medium ($n = 47$), or fast ($n = 30$) eaters. During the mid-follicular phase, after a standardized breakfast and 4 h fast, they ate an identical ad libitum pasta lunch on two occasions, once instructed to eat quickly and once slowly. They completed visual analogue scales of hunger (H), satiety (S), and desire-to-eat (DTE) before and 60 min after lunch. Eating rate (61 ± 43 kcal/min) correlated positively with 60 min H ($r = 0.207$, $p = 0.005$) and DTE ($r = 0.244$, $p = 0.001$), and negatively with S ($r = -0.151$, $p = 0.044$). Meal duration was not correlated with energy intake, but it was negatively correlated with DTE at 60 min ($r = -0.171$, $p = 0.022$). At 60 min, H (9 ± 12 vs. 13 ± 13; $p = 0.00122$) and DTE (10 ± 12 vs. 16 ± 14; $p = 0.00003$) were lower ($r = 0.00122$, $p = 0.00002$) and S higher (84 ± 16 vs. 77 ± 20; $p = 0.00021$) after slow versus quick eating. Slow eaters ate less (626 ± 220 kcal) than medium paced eaters (724 ± 169 kcal; $p = 0.036$). Whether told to eat quickly or slowly, slow eaters tended to eat slower (54 ± 42 kcal/min) than medium (60 ± 41 kcal/min) or fast eaters (66 ± 47 kcal/min; $p > .05$). These findings suggest that eating rate may be important in 60 min H, S, and DTE, slow eating may be uncommon in young women, and one-time instruction on changing habitual eating rate may not be effective enough.

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Effects of irregularity and predictability of the time of daily sessions on rats' feeding behavior

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This study tested the effects of irregularity and predictability of time of daily sessions on rats' feeding behavior. Daily 1.5 h feeding was provided in the homecages. In a regular group ($N = 6$), daily 1.5-h feeding was provided at 10:00 a.m. In an irregular group ($N = 6$), daily sessions started at 1 of 2 different times (10:00 a.m. or 5:00 p.m.) with average inter-session interval of 22.5 h. In a signaled group ($N = 6$), the time of the session was yoked to the irregular group but a signal (an empty food tray in the homecage) was provided 30 min prior to the sessions. No supplemental food was provided. The experiment lasted for 12 days. Daily food consumption increased in the regular group but not in the irregular group. Daily food consumption in the signaled group was similar to that in the regular group. Rats in the irregular lost more body weight than rats in the regular and signaled group. These results suggest that unpredictability of the feeding schedule is responsible for the deteriorating effects of irregular feeding schedule.

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