Oh, how sweet it is: Focus on *Altered Pontine Processing in a Rat Model of Obesity* by Kovacs and Hajnal (2008)

Andre T. Roussin and Patricia M. Di Lorenzo*

Binghamton University

*To Whom Correspondence should be addressed

Dept. of Psychology
Binghamton University
Box 6000
Binghamton, NY 13902-6000

Ph: 607-777-2055
FAX: 607-777-4890
Email: diloren@binghamton.edu

Copyright © 2008 by the American Physiological Society.
Obesity is an increasing problem in the United States and is responsible for a host of associated health problems, including hypertension and diabetes. It is therefore important to identify factors contributing to overeating and consumption of unhealthy foods; taste perception and preference are obvious factors to consider. Kovacs and Hajnal have uncovered a relationship between obesity and taste processing in the central nervous system in this issue of the Journal of Neurophysiology, demonstrating that obesity alters brainstem responses to sweet stimuli.

There is evidence that obese people do in fact process taste differently from others, particularly with respect to their responses to sweet tastes. For example, obese people are less sensitive to sweet tastes, but like them more than lean individuals (Bartoshuk et al. 2006). Differences in behavioral responses to sweet tastes also exist between populations with high levels of obesity and related maladies and the general population; African Americans (Salbe et al. 2004) and Pima Indians (Bacon et al. 1994; Desor et al. 1975; Schiffman et al. 2000), both show increased liking or preference for sweet tastes. The central mechanisms that underlie differences in both perception and the hedonics of sweet tastes between obese and non-obese individuals remain poorly understood, however.

To study these mechanisms, Kovacs and Hajnal (2008) investigated differences in single-neuron pontine taste responses between two strains of rat: the Otsuka Long Evans Tokushima Fatty (OLETF) and the Long Evens Tokushima Otsuko (LETO). Gustatory responses in OLETF rats resemble those of obese humans in that, compared with LETO rats, they fail to regulate their meal size, display heightened consumption of sweet substances, and progressively develop obesity and non-insulin-dependent diabetes
mellitus (Bi and Moran 2002; Kawano et al. 1992). OLETF rats also display heightened consumption of sucrose solutions compared to LETO controls as well as a greater preference for highly concentrated sucrose solutions (De Jonghe et al. 2005).

In their recent paper *Altered Pontine Processing in a Rat Model of Obesity*, Kovacs and Hajnal (2008) demonstrated that obesity may alter the way sweet stimuli are processed at an early level of taste processing, the parabrachial nucleus of the pons (PbN). The PbN is home to third-order gustatory neurons, and influences the motivational and hedonic aspects of taste via dopaminergic mesolimbic pathways (Norgren et al. 2006). Kovacs and Hajnal (2008) found that neurons responsive only to sucrose were more spontaneously active in OLETF rats than in lean LETO controls. Interestingly, sucrose-specific cells responded less vigorously to dilute sucrose solutions in OLETF rats compared with LETO controls while responding more to highly concentrated sucrose solutions. The net effect of these changes was that sucrose-specific cells in obese rats were sensitive to differences in concentration over a wider range of concentrations than those in lean rats (Kovacs and Hajnal 2008). These results are highly consistent with observations that obese humans tend to be less responsive to mildly sweet stimuli and prefer sweeter stimuli, when compared with the non-obese (Bartoshuk et al. 2006).

Both central and peripheral effects may partially underlie the difference between pontine taste responses in OLETF and LETO rats. As with sucrose specialist cells in the PbN, responses to concentrated sucrose solutions are heightened in taste afferents carried within the chorda tympani nerve (CT, innervating taste buds on the rostral 2/3 of the tongue) of OLETF rats (Tsunoda et al. 1998). Central effects are also very likely to
influence this change in taste responsiveness, however. Responses in broadly tuned OLETF cells were actually less vigorous than in lean controls, an effect that is opposite to what one would expect based on CT responses alone. This central modulation may arise at least in part from descending inputs to the PbN; for example, perturbing activity of the central nucleus of the amygdala, the lateral hypothalamus, and the gustatory cortex all affect taste responses in the PbN (Di Lorenzo 1990; Li et al. 2005; Lundy and Norgren 2001). A decrease in descending tonic inhibition is certainly consistent with the increase in spontaneous activity of sucrose-specialist neurons in OLETF rats observed by Kovacs and Hajnal (2008).

Several changes in an animal's acute homeostatic state are known to affect both behavior and taste responses in the brain stem. In the nucleus of the solitary tract, the first central relay for taste, both salt deprivation (Nakamura and Norgren 1995; Tamura and Norgren 1997) and the administration of satiety factors (Giza et al. 1992) alter both taste responses and taste preferences. Intra-duodenal infusion of nutrients decrease PbN taste responses, especially in cells that respond only to sucrose (Hajnal et al. 1999); this change is the opposite of that which accompanies obesity (Kovacs and Hajnal, 2008). The fact that the consumption of concentrated sweet solutions increases along with obesity in OLETF rats (Hajnal et al. 2005) and corresponds to changes observed in OLETF PbN responses (Kovacs and Hajnal 2008) suggests that obesity and/or chronic metabolic states may affect the descending projections underlying this homeostatic gustatory modulation. By showing that PbN responses to sweet tastes are selectively altered by chronic obesity, Kovacs and Hajnal (2008) may have uncovered a
critical link between the pathological association of body weight and consumption that characterizes obesity.


**Lundy RF, Jr. and Norgren R.** Pontine gustatory activity is altered by electrical stimulation in the central nucleus of the amygdala. *J Neurophysiol* 85: 770-783, 2001


