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Research Report

Unconditioned oromotor taste reactivity elicited by sucrose and quinine is unaffected by extensive bilateral damage to the gustatory zone of the insular cortex in rats

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ABSTRACT

Rats display stereotypical oromotor and somatic responses to small volumes of intraorally infused taste solutions. These behaviors, known as taste reactivity, are categorized by their association with ingestion or rejection and are thought to reflect the palatability of the stimulus. Because supracollicular decerebrate rats display normal taste reactivity responses, it would appear that forebrain structures are not necessary for generating them. However, because moving the plane of transection rostrally, or damaging or manipulating specific ventral forebrain sites disrupts normal taste reactivity behavior, lesions of the gustatory cortex, a region that has been suggested to be involved with palatability processing, may do the same. In the current study, rats received two injections of either ibotenic acid (N=12) or vehicle (N=8), targeting the conventionally defined gustatory cortex in each hemisphere, and were implanted with intraoral cannulae. Following recovery, their responses to intraoral infusions (0.23 ml in 1 min) of dH$_2$O, sucrose (1.0 M and 0.1 M), and quinine hydrochloride (3 mM and 0.3 mM) were video recorded. Analysis of brains with sufficient bilateral lesions (N=10) revealed that, on average, approximately 94% of the gustatory cortex was destroyed. These extensive bilateral lesions had no significant effect on taste reactivity; the numbers of ingestive and aversive responses to sucrose and quinine were similar between groups. Though these findings do not rule out involvement of the gustatory cortex in palatability processing, they make evident that the region of insular cortex destroyed is not necessary for the normal expression of unconditioned affective behavioral responses to taste stimuli.

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1. Introduction

The behavioral responses a taste stimulus elicits can be divided into two functional subclasses: appetitive and consummatory (see Berridge, 2000; Spector, 2000). Craig (1918) described appetitive responses as those behaviors which bring the animal into contact with a taste stimulus (e.g., foraging, approaching a drinking spout) and consummatory responses as reflex-like actions that are elicited once a taste stimulus makes contact with oral sensory receptors (e.g., swallowing, oromotor responses). One- and two-bottle intake tests, commonly used assays of taste palatability, involve both appetitive and consummatory behavior because the animal must approach and make contact with the licking spout (the appetitive components) upon which oromotor responses (the consummatory component) are elicited by the stimulus once it engages the receptors of the oral cavity. Purely consummatory responses can be measured via the delivery of taste solutions through a surgically implanted intraoral cannula (Berridge, 1996; Grill and Norgren, 1978a; Grill et al., 1987). When taste stimuli are delivered in this way, rats elicit stereotypical affective behavioral responses referred to as taste reactivity (TR, Grill and Norgren, 1978a). These unconditioned reflex-like behaviors are thought to reflect the palatability evaluation of taste stimuli (Berridge, 2000; Grill and Berridge, 1985). Normally preferred stimuli (e.g., sucrose) elicit responses that promote ingestion (i.e., ingestive behaviors) while taste stimuli that are normally avoided (e.g., quinine) elicit responses that promote rejection (i.e., aversive behaviors; Grill and Norgren, 1978a).

Some years ago, Pfaffmann et al. (1977) speculated that taste hedonics were mediated by the ventral forebrain pathway that arises from the gustatory zone of the parabrachial nucleus (PBN), whereas sensory-discriminative functions (e.g. qualitative identification) were associated with the gustatory thalamocortical pathway. The extent of forebrain involvement in affective behavioral responses, specifically TR responses, to taste stimuli however was seriously challenged by Grill and Norgren (1978b) by demonstrating that chronic supracollicular decerebrate rats were able to elicit TR responses to sucrose and quinine that did not differ from intact controls. In this preparation, only two gustatory relays in the brainstem, the nucleus of the solitary tract (NST) and the PBN, remain neurally connected to motor output circuits. Although this finding precludes the necessity of the forebrain in triggering unconditioned affective oromotor and somatic behaviors, several lines of investigation have demonstrated forebrain control over them. For instance, when Grill and Norgren (1978b) moved their plane of transection rostrally to just anterior of the thalamus, rats displayed enhanced aversive TR behaviors to all taste stimuli. In fact, in the chronic thalamic preparation, TR responses associated with ingestion were completely absent. These findings indicated that neural mechanisms rostral to the midbrain somehow modulate the hedonic impact of a taste stimulus. Candidate forebrain sites for such affective processing of taste input include the central nucleus of the amygdala (Touzani et al., 1997) and the ventral pallidum/substantia innominata (Cromwell and Berridge, 1993) as localized destruction of these structures leads to increases in the aversive impact of taste stimuli, findings that buttress Pfaffmann et al. (1977)'s hypothesis that the ventral forebrain pathway stemming from the PBN is involved with the mediation of taste hedonics. Moreover, pharmacological manipulations of ventral forebrain structures have provided further support for their role in affective taste processing. Infusions of mu opioid agonists into the ventral pallidum or nucleus accumbens, for example, have been shown to enhance ingestive responses to oral infusions of sucrose and attenuate aversive responses to oral infusions of quinine (Peciña and Berridge, 2005; Peciña et al., 2006; Smith and Berridge, 2005, 2007). The results from the aforementioned studies highlight the fact that the lack of an effect of a neural insult at lower levels of the ascending gustatory system (i.e., the decerebrate rat preparation) is not necessarily emulated by more targeted manipulations (pharmacological or lesions) at higher levels.

Clearly, the ventral forebrain plays a role in affective taste processing, but evidence for the gustatory cortex (GC), too, has also been accumulating. In fact, very early research showed elevations in quinine avoidance thresholds in rats with large ablations of the GC (Benjamin, 1955a, 1955b; Benjamin and Akert, 1958). Much more recently, based on electrophysiological recordings from awake rats presented with intraorally delivered taste stimuli, some investigators have suggested that GC neurons are involved in processing the palatability of taste stimuli (e.g., Grossman et al., 2008; Katz et al., 2001; Sadacca et al., 2012). That said, relatively normal preference-avoidance functions for a variety of taste stimuli measured by intake tests in rats with substantial GC damage have been demonstrated (Benjamin, 1955a, 1955b; Braun et al., 1982; Dunn and Everitt, 1988). Thus, the necessity of the GC for expression of taste affect remains unclear. Intake tests, however, can be influenced by other factors such as postingestive events, thereby providing only a partial analysis of affective gustatory responsiveness. To overcome this limitation, Hashimoto and Spector (2014) employed a brief-access taste test, which minimizes postingestive factors (e.g., Davis, 1973; Smith, 2001; Spector, 2003), to assess the effects of GC lesions on unconditioned licking to sucrose and quinine. Still, no differences were observed between the animals with and without GC lesions, suggesting that the damaged region of the GC was unnecessary for the normal expression of taste-triggered unconditioned licking responses and their suppression. However, brief-access taste tests (as well as intake tests) rely in part on appetitive behavior because they require the animal to voluntarily approach the drinking spout. Moreover, the rats in the Hashimoto and Spector (2014) study were either food- or water-deprived during testing to promote stimulus sampling, and this may have modulated the responsiveness of the animals.

The TR procedure, on the other hand, is well suited to test taste-related palatability unencumbered by postingestive stimulation and physiological need states. Because in this paradigm the stimulus is directly infused into the oral cavity under explicit experimenter control, no appetitive behavior is involved and thus it represents a relatively pure assessment of consummatory responsiveness. In addition, the infusion of a very small volume of the taste stimulus under study over a relatively brief period of time obviates the contribution of postingestive events to the responses observed. Thus, in the current study, to further examine the role of the GC in palatability processing in the rat model, we tested whether large neurotoxic lesions of the GC would disrupt unconditioned TR to two prototypical tastants: sucrose, a normally preferred stimulus that is sweet to humans