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Representation of Taste Stimuli in the Brain

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Introduction

Ingestive behavior is controlled by various neural systems in the central nervous system such as oromotor and taste neural systems. Since the taste system is the final arbiter by which an animal determines whether a chemical or food will be acceptable (Travers and Norgren, 1986), the taste system is one of the main neural systems for ingestive behavior. The brainstem contains a basic neural system for ingestive behavior, that includes brainstem taste areas such as the nucleus of the solitary tract (NTS) and the pontine parabrachial nucleus (PBN), by which the animal can manifest different oromotor responses to four basic tastes (Grill and Norgren, 1978). It has been suggested that the forebrain, including the amygdala (AM) and hypothalamus, which receives taste information from the brainstem taste areas, is a higher center which modulates activity of this basic neural system by its descending projections (Norgren, 1995). The AM and hypothalamus, which receives massive afferent fibers from the AM, have been reported to be important in motivation, feeding behavior and evaluation of the biological significance of sensory stimuli (Rolls, 1976; Nishijo *et al.*, 1988). Lesions of the AM and hypothalamus altered food preference in monkeys and rats (Murray *et al.*, 1996; Isaacson, 1982) and attenuated behavioral responses to both preferred and aversive taste stimuli in rats (Kemble and Schwartzbaum, 1969). Furthermore, decerebration and electrical stimulation or inactivation of the forebrain modulated activity of taste neurons in the NTS and PBN (Matsuo *et al.*, 1984; Mark *et al.*, 1988; Di Lorenzo, 1990).

In the present study, to investigate the nature of taste information processing in the AM, neuronal activity was recorded from the AM during discrimination of sensory stimuli associated with various taste solutions and ingestion of taste solutions.

Materials and methods

Male Wistar rats weighing 280–350 g were used. After recovery from surgery to attach a cranioplastic cap and two intraoral catheters to the skull, a rat was placed painlessly in a special stereotaxic apparatus equipped with devices for sensory stimulation (Nishijo and Norgren, 1990, 1991, 1997; Nishijo *et al.*, 1998). Each AM neuron was tested with various conditioned stimuli, including auditory, visual, somatosensory and olfactory stimuli associated with reward. Some AM neurons were further tested with taste stimuli through intraoral cannulae; 0.1 M NaCl, 0.3 M sucrose (Suc), 0.01 M citric acid (CA), 0.0003 M quinine HCl (QHCl), 0.1 M monosodium glutamate (MSG) and 0.2 M lysine HCl.

Results

Of the 420 cells that responded to one or more sensory stimuli, 108 responded to oral-sensory stimulation. Of these 108 oral-sensory neurons, 84 could be further classified as taste and non-taste oral-sensory based on the data from intraoral infusions. Twenty-four cells were classified as taste-sensitive because they responded more strongly to gustatory stimuli than to water and 60 neurons were classified as non-taste oral-sensory neurons.

Figure 1A shows an example of a unimodal taste neuron responding during intraoral infusions. The neuron responded selectively to QHCl. Of the 24 taste oral-sensory neurons, 21 were tested at least with four standard taste solutions. Based on the magnitudes of their responses to these four standard stimuli, the taste neurons were classified as follows: 4 NaCl-best, 7 sucrose-best, 3 citric acid-best and 6 QHCl-best. The remaining neuron responded significantly only to lysine HCl and MSG. Pearson's correlation coefficients between sapid chemicals were analyzed using these 21 taste neurons (Figure 1B). Sucrose was correlated with NaCl (r = 0.440) most among the four basic chemicals and least with QHCl (r = 0.138). NaCl was most correlated with citric acid (r = 0.672) and less with sucrose (r = 0.440) and QHCl (r = 0.573). Citric acid was most correlated with QHCl (r = 0.905) and less with NaCl (r = 0.672). QHCl was most correlated with citric acid (r = 0.905) and least with sucrose (r = 0.138). This pattern of interstimulus correlation coefficients suggest that taste quality is organized based on palatability; taste stimuli could be arranged in one dimension in that sucrose (most palatable), NaCl, citric acid and QHCl (least palatable) are sequentially plotted along a one-dimensional line. If this one-dimensional arrangement of taste chemicals is true, pairs of neighboring chemicals should be highly correlated as noted above.

These correlation coefficients in the AM were compared with those in the PBN (Nishijo and Norgren, 1990, 1997) (Figure 1B). Correlation coefficients between basic sapid stimuli with similar degrees of palatability were larger than those in the PBN, while correlation coefficients between basic sapid stimuli with different degree of palatability were low in both the AM and PBN. For example, correlation coefficients between NaCl and sucrose (palatable solutions) were –0.129 and –0.126 in the PBN, which were significantly smaller than that (0.440) in the present study (two-tailed t-test after Fisher's Z-transformation, $P < 0.05$). Correlation coefficients between citric acid and QHCl (aversive solutions) were 0.769 and 0.386 in the PBN, which were also significantly smaller than that (0.905) in the present study (two-tailed t-test after Fisher's Z-transformation, $P < 0.05$). Furthermore, correlation coefficients between sucrose (most palatable) and QHCl (most aversive) was low (0.138) in the AM, which was statistically not different from those (0.03 and 0.019) in the PBN (two-tailed t-test after Fisher's Z-transformation, $P > 0.05$). These results strongly suggest a difference in taste coding between the PBN and AM, i.e. taste quality versus palatability.

Discussion

Analyses using correlation coefficients suggest that taste is encoded in the AM based on the palatability of sapid chemicals. Lesions of the central nucleus of the AM do alter the relationship between oromotor responses to taste and the actual consumption of the stimuli (Seeley *et al.*, 1993). Large lesions of the AM attenuate behavioral responses to both preferred and aversive sapid stimuli

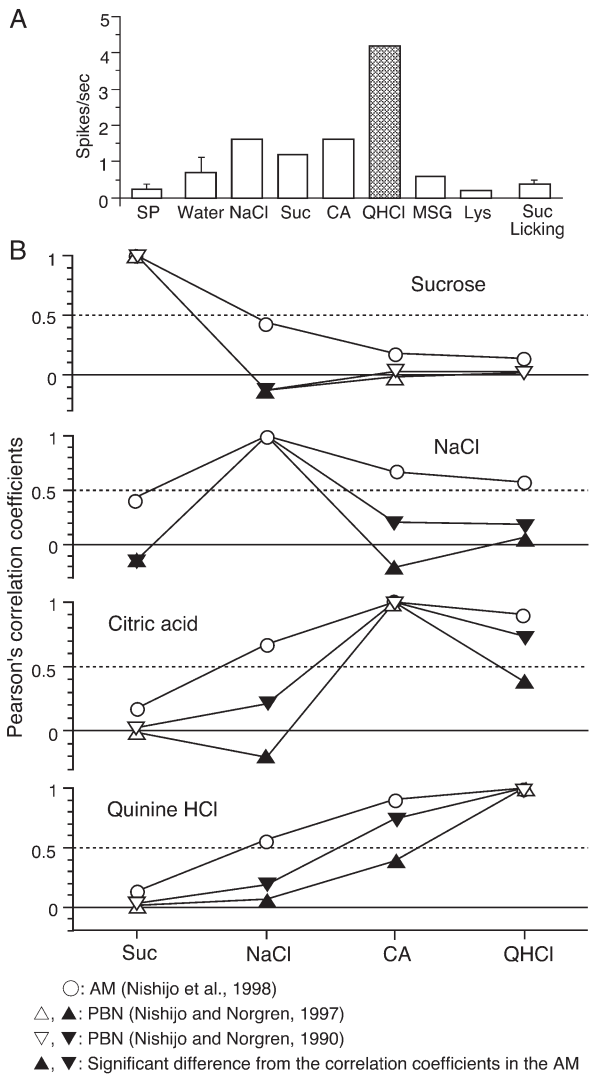

Figure 1 Gustatory responses in the amygdala (AM). **(A)** Response profile of an AM neuron to intraoral infusions of sapid solutions and to a sucrose solution licked from a spout in a conditioned task (Suc licking). Cross-hatched column deviated >2.0 SD from responses to water. **(B)** Comparison of average across-stimulus correlation coefficients between the AM and PBN in awake rats among the four basic stimuli.

and alter conditioned taste aversion (Kemble and Schwartzbaum, 1969; Yamamoto *et al.*, 1995). Finally, fiber-sparing lesions of the AM in monkeys changed their food preferences (Murray *et al.*, 1996). Thus, gustatory sensory activity reaches the AM and this information may be used in the ongoing process of evaluation. The strong reciprocal connections between the central nucleus of the AM and the brainstem taste nuclei imply that whatever information is added in the AM, it is likely to be involved in modifying ascending gustatory neuronal activity.

Previous studies reported lesions and electrical stimulation of the forebrain altered activity of brainstem taste neurons and suggested the importance of interactions between the brainstem (NTS, PBN) and forebrain (hypothalamus, AM). The AM and hypothalamus have intimate reciprocal connections and both regions send descending projections to the NTS and PBN. The results strongly suggest that these forebrain areas are higher centers that can modulate feeding behavior.

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