Taste Responses in the Greater Superficial Petrosal Nerve: Substantial Sodium Salt and Amiloride Sensitivities Demonstrated in Two Rat Strains

Suzanne I. Sollars and David L. Hill
University of Virginia

Gustatory input to the brain is relayed through four peripheral nerves: the chorda tympani, greater superficial petrosal, glossopharyngeal, and superior laryngeal nerves. Two of these nerves, the CT and the GSP, are branches of the facial nerve and have their cell soma in the geniculate ganglion. Although a plethora of information exists regarding gustatory response properties of the CT, which innervates taste buds on the anterior tongue, relatively little is known about the response properties of the GSP, which innervates taste buds on the palate.

Electrophysiological responses of the CT to chemical stimulation have been studied extensively in rats (Beidler, 1953), in hamsters (Frank, 1973), during development (Hill, Mirstretta, & Bradley, 1982), and after various experimental manipulations (Contreras & Frank, 1979; Hill & Phillips, 1994). The CT in normal adult rats is characterized as being highly responsive to salts and has a large number of fibers particularly sensitive to sodium stimuli (Frank, Contreras, & Hettinger, 1983). Importantly, sodium responses in the CT are substantially suppressed by lingual application of the epithelial sodium channel blocker, amiloride. The indirect assessment of receptor function by recording salt responses from the CT before and after amiloride has provided insights into the mechanisms of salt transduction on the anterior tongue (Avenet & Lindemann, 1988; Brand, Teeter, & Silver, 1985; Formaker & Hill, 1988; Ye, Heck, & Desimone, 1993). In contrast, the limited number of studies on GSP responses, which can be used to assess palatal receptor cell function, present a much different pattern of effective stimuli. For example, the prevailing view is that sugars are more effective taste stimuli for GSP responses compared with CT responses (Harada & Smith, 1992; Nejad, 1986), that salts are more effective in eliciting CT responses compared with GSP responses (Harada, Yamamoto, Yamaguchi, & Kasahara, 1997), and that amiloride is much more effective in suppressing sodium responses in the CT compared with the GSP (Harada et al., 1997). Therefore, previous work suggests that taste information originating on the anterior tongue is quite different than that originating on the palate. As such, cells in the geniculate ganglion that send peripheral processes into the CT compared with those that send processes into the GSP should receive different gustatory information. This distinctly different information from the central processes of geniculate ganglion cells should then converge onto gustatory neurons in the first synaptic relay in the brain, in the rostral pole of the nucleus of the solitary tract (NTS). In fact, convergence of information originating from the anterior tongue and from the nasoincisor duct on the palate, carried by the CT and the GSP respectively, has been documented in rat NTS neurons (Travers, Pfaffmann, & Norgren, 1986).

The limited information that exists regarding response properties of the GSP was obtained solely from standard adult rats and hamsters that experienced no prior experimental manipulation. As a result, the information available regarding GSP taste stimulation is severely lacking, especially when compared with the abundance of information on CT taste responses in various species, strains, and experimental treatments. A useful approach to study mechanisms of gustatory function is to compare animal models that differ genetically or have distinctly different taste-related behaviors. This allows the opportunity to make hypotheses about the underlying neural substrate for taste. In one such model,
the Fischer 344 (F344) rat, there have been dramatic differences identified in salt-related behavioral taste responses relative to more widely used strains. F344 rats are unusual in that they do not prefer any concentration of sodium chloride \((\text{NaCl})\) to water in long-term intake tests and are averse to concentrations of \(\text{NaCl}\) that other rat strains strongly prefer (Midkiff, Fitts, Simpson, & Bernstein, 1985). Additionally, they demonstrate a substantial aversive response to \(\text{NaCl}\) in short-term taste reactivity tests (Grill & Bernstein, 1988).

The CT has been implicated as the gustatory nerve critical to the maintenance and development of the salt aversion in F344 rats, and it is suggested that the transduction process responsible for strain-related differences in these rats is the amiloride-sensitive sodium channel. This conclusion is based on (a) the large strain-related differences in CT function in which F344 rats have significantly larger relative taste responses to sodium than their controls and a greater sensitivity to amiloride (Bernstein, Longley, & Taylor, 1991); (b) the dependence of the CT to determine taste-related behaviors in F344 rats, as concluded from behavioral studies in which the CT is sectioned (Sollars & Bernstein, 1994b; Sollars, Sollars, & Bernstein, 1991); and (c) the large nerve-related differences in taste function between the CT and GSP, in that the CT is viewed as primarily transmitting information about salts and the GSP transmitting information about sugars.

To provide further examination of salt and sugar responses in the GSP and the ability of amiloride to inhibit sodium salt responses, neurophysiological recordings were obtained from the GSP while stimulating the entire palate with chemical stimuli. In addition, the current study was designed to examine the potential functional differences of the GSP in F344 and Sprague-Dawley (SD) rats that display very different gustatory-related responses.

**Method**

**Subjects**

Adult (42–70 days of age) SD (2 male and 3 female) and F344 (4 male and 1 female) rats (Harlan Sprague-Dawley, Dublin, VA) were maintained on a 12-hr light-dark cycle with ad libitum access to chow and water. Recordings were made from the right or left GSP in five rats of each strain.

**Surgery**

Rats were anesthetized with sodium pentobarbital (Nembutal), 50 mg/kg intraperitoneally; additional doses were given throughout the procedure to maintain a surgical level of anesthesia. After a tracheotomy, PE 190 tubing that was flanged at approximately the midpoint was placed through the esophagus into the oral cavity. The tubing exited the esophagus in the neck through a small incision. The flap was placed to cover the opening to the esophagus in the oral cavity, and perforated tubing extended the length of the palate. In this way, taste solutions could be applied through the esophageal tubing to bathe the posterior palatine field, the geschmacksstreifen and nasoincisor ducts of the palate innervated by the GSP (Miller, 1977; Miller & Spangler, 1982). In one rat, a solution of 5% (weight/volume) methylene blue was applied to the palate through the esophageal tubing to verify the stimulus application technique and to ensure that stimuli contacted all palatal taste receptor fields. Muscle around the tube was sutured tightly to prevent backflow of solution into the esophagus. The hypoglossal nerves were sectioned bilaterally to eliminate tongue movement.

Rats were placed in a supine position in a stereotaxic device with a modified headholder, which anchored the mouth just rostral to the nasoincisor duct but did not obscure the duct. Body temperature was maintained with a water-circulating heating pad. The juncture between the anterior and posterior digastricus muscle was sectioned and the posterior muscle retracted away from the tympanic bulla. A small hole was cut into the muscle that directly overlies the tympanic bulla, and muscles were retracted to expose the ventral surface of the tympanic bulla. A hole was cut into the tympanic bulla, and the cochlea, tensor tympani muscle, and part of the temporal bone overlying the geniculate ganglion and GSP were removed. The ganglion was transected proximal to the GSP; the GSP was freed of connective tissue and desheathed. The cut end of the GSP was placed on a platinum recording electrode, and a mixture of petroleum jelly (Vaseline) and mineral oil was placed into the bulla to surround the nerve.

**Neurophysiology**

Multifiber neural activity from the whole nerve was amplified, displayed on an oscilloscope, and monitored with an audio amplifier. For data analysis, the amplified signal was passed through an integrator with a time constant of 0.5 sec (Beidler, 1953; Harper & Knight, 1987), and the summed electrical activity was led to and analyzed with MacLab Scope software (ADInstruments).

**Stimuli and Stimulation Procedures**

Responses were recorded to concentration series (0.05–2.0 M) of \(\text{NaCl}\), sodium acetate \((\text{NaAc})\), and ammonium chloride \((\text{NH}_4\text{Cl})\); 0.05 to 1.0 M sucrose and maltose; and 0.01 M hydrochloric acid \((\text{HCl})\) and 0.01 M quinine hydrochloride mixed in distilled water. Stimuli flowed over the palate through the esophageal tubing (see prior discussion). Five milliliters of each stimulus were applied at a rate of 0.5 ml per second. Thirty seconds after stimulus-flow offset, the palate was rinsed for a minimum of 40 sec with distilled water. Palatal stimulation included high concentrations of salts because the responses of the GSP, unlike those of the CT, do not plateau at approximately 0.5 M, but continue to increase in magnitude. After the initial stimulus series in which water was used as the rinse and solvent, the palate was preadapted to 100 \(\mu\text{M}\) amiloride hydrochloride, and responses were recorded to a series of 0.05 to 2.0 M \(\text{NaCl}\) and \(\text{NaAc}\), all of which were mixed in 100 \(\mu\text{M}\) amiloride. During this series, 100 \(\mu\text{M}\) amiloride served as the rinse. Throughout the recording session, the stability of neural activity was monitored by periodic application of 0.5 M \(\text{NH}_4\text{Cl}\). Recordings were considered stable and included in the analysis only if the preceding and subsequent \(\text{NH}_4\text{Cl}\) responses deviated by less than 10%.

**Data Analysis**

To be consistent with earlier reports involving whole-nerve gustatory responses (e.g., Hill, 1987; Hill & Bour, 1985), 0.5 M \(\text{NH}_4\text{Cl}\) was used as the standard measure to which all other solutions were compared. In addition, responses to \(\text{NH}_4\text{Cl}\) showed little change in magnitude after amiloride. Thus, an accurate comparison of response magnitudes could be made for solutions before and after amiloride. Response magnitudes were calculated...
as the averaged height of the steady-state response that occurred between 20 and 35 sec after stimulus onset (Figure 1). Ratios relative to 0.5 M NH₄Cl were calculated from these averages, which provided a basis to compare magnitudes (i.e., relative magnitude) across conditions. For each solution presented in amiloride, suppression ratios were calculated as follows:

Percent suppression = \([1 - (RM_{after}/RM_{before})] \times 100\),

where \(RM_{after}\) is the relative magnitude of a response to a solution presented in amiloride, and \(RM_{before}\) is the relative magnitude of a response to a solution presented before amiloride. Thus, 100% suppression indicates a response reduced by amiloride to baseline neural activity, and 0% suppression indicates no suppressive effect of amiloride.

Statistical analyses using Fisher's exact tests indicated no significant \((p > .10)\) differences in taste response relative magnitudes between male and female rats within each strain. Thus, subsequent statistical analyses were based on the combined data from both sexes within a strain. Relative magnitudes and percent suppression ratios were compared with repeated measures analysis of variance (ANOVA). Statistically reliable ANOVA results were followed with post hoc analysis with Bonferroni or independent t tests, with the alpha level set at \(p < .05\), and adjusted to compensate for the number of statistical tests conducted within a series (e.g., \(p < .05/7\); Kirk, 1968).

Results

Salt Responses

Relative GSP response magnitudes to salts were dependent on the stimulus and on the rat strain. As seen in Figure 1, NaCl was an effective stimulus for the GSP in SD and F344 rats. In both strains, the intensity of response to NaCl increased as the concentration of the stimulus increased.

Figure 1. An example of typical greater superficial petrosal nerve integrated neurophysiological responses to stimuli applied to the palates of Fischer 344 (F344) and Sprague-Dawley (SD) rats. NH₄Cl = ammonium chloride; NaCl = sodium chloride.
(Figure 2A). This is in contrast to results from CT electrophysiology in which neural responses plateau at concentrations at or below 0.5 M (Beidler, 1953). There were also clear strain differences that emerged in the response to NaCl. In SD rats, the GSP response magnitude to 1.0 M NaCl was approximately equivalent to the response to the NH₄Cl standard (stimulus response/0.5 M NH₄Cl response = 1.0). In contrast, F344 rats had an overall relative heightened response to NaCl, $F(1, 8) = 34.05, p < .001$; the response magnitude to 0.5 M NH₄Cl was matched at 0.25 M NaCl (Figure 2A). Post hoc analysis indicated that relative responses to 0.05 through 1.0 M NaCl were higher for F344 rats than SD rats ($p < .05$).

Similar to the responses to NaCl, the GSP responded to NaAc with increasing response magnitude as the concentration of solute increased, although relative responses were

![Figure 2A](image1)

![Figure 2B](image2)

**Figure 2.** Averaged greater superficial petrosal nerve response ratios ($M \pm SE$) to (A) sodium chloride (NaCl) and (B) sodium acetate (NaAc) in Fischer 344 (F344) and Sprague-Dawley (SD) rats. Responses were obtained from solutions mixed in distilled water or 100 μM amiloride. NH₄Cl = ammonium chloride.
lower than those to comparable concentrations of NaCl (Figure 2A and B). Strain differences in the relative response emerged across concentrations, $F(1, 8) = 15.49, p < .004$; the magnitude of F344 responses was significantly higher for 0.1 to 1.5 M NaAc than responses in SD rats (Figure 2B). The NaAc concentrations at which response magnitudes equaled the 0.5 M NH$_4$Cl standard were higher for SD rats (2.0 M) than for F344 rats (1.0 M).

Amiloride Suppression

Surprisingly, amiloride had strong suppressive effects on the GSP response to sodium salts. SD rats' responses to 0.05 M through 1.5 M NaCl and 0.1 to 1.0 M NaAc after lingual application of amiloride were significantly suppressed compared with the respective responses before amiloride (Figure 3). Quantitatively, the amount of suppression ranged from approximately 83% for 0.05 M NaCl to approximately 24% for 1.5 M NaCl (Figure 4). Amiloride was also effective in reducing the GSP salt responses in F344 rats to all concentrations (0.05–2.0 M) of both NaCl and NaAc (see Figure 3). As seen in Figure 4, the percent suppression was significantly greater in F344 rats than SD rats at 0.25 through 2.0 M NaCl and at 0.25 through 1.5 M NaAc. This greater degree of amiloride suppression for F344 rats resulted in postamiloride responses to NaCl, $F(1, 8) = 2.11, p > .10$, and NaAc, $F(1, 8) = 0.19, p > .10$, such that responses no longer differed between strains (see Figure 4). Therefore, amiloride eliminated the strain-related differences in salt response magnitudes between SD and F344 rats.

Nonsodium Salts and Nonsalt Stimuli

In both strains, NH$_4$Cl produced the largest magnitude of response compared with all the other stimuli (Figures 2 and 5A). Concentrations of 1.5 M and 2.0 M NH$_4$Cl produced responses that were at least twice the magnitude of the 0.5 M NH$_4$Cl standard. Unlike the strain differences that emerged for sodium solutions, there were no differences between the strains for NH$_4$Cl. As seen in Figure 5B, 0.01 N HCl and 0.01 M quinine were equally effective in producing responses in the GSP of F344 and SD rats. Therefore, unlike

![Figure 3](image_url)

Figure 3. Integrated greater superficial petrosal nerve neural responses to stimuli applied to the palates of Fischer 344 (F344) and Sprague-Dawley (SD) rats. Stimuli were mixed either in distilled water (Before) or 100 μM amiloride (After). NH$_4$Cl = ammonium chloride; NaCl = sodium chloride; NaAc = sodium acetate.
salt responses, there were no strain-related response differences to HCl or to quinine.

As expected, the GSP responded robustly to sucrose in both SD and F344 rats (see Figure 5C). In SD rats, the response to 0.25 M sucrose was equivalent to the 0.5 M NH₄Cl response and increased slightly at higher concentrations. The F344 response to sucrose was never as effective as the response to 0.5 M NH₄Cl (i.e., the ratio is less than 1.0); however, responses plateaued at 0.5 M sucrose (see Figure 5C). Consequently, the relative response magnitudes to sucrose in SD rats were significantly greater than those in F344 rats for all concentrations, F(1, 8) = 5.22, p = .05. By comparison with responses to sucrose, the GSP was not as responsive to maltose; increasing maltose concentrations resulted only a small increase in relative response magnitude (see Figure 5D). Moreover, there were no strain-related differences in response magnitudes to maltose.

Discussion

One of the goals of the current study was to examine and analyze the responses of the GSP in a manner similar to that used for many studies of CT responses. Using this paradigm, we demonstrated that the GSP in both SD and F344 rats responds strongly to salts as well as to sugars. Because the GSP is the only gustatory nerve in rats in which sucrose is a potent stimulus, it has long been considered the “sweet-sensitive” nerve of the gustatory system. The finding that the CT responds minimally to sucrose compared with salts has reinforced the notion that palatal taste receptors mediate the taste of sugars, whereas anterior tongue receptors mediate the taste of salts (Harada & Smith, 1992; Nejad, 1986). Although the GSP is certainly responsive to sucrose, the present results indicate that it is also highly responsive to salts. In fact, relative responses to higher (≥1.0 M) concentrations of NaCl and NH₄Cl exceed the relative responses normally exhibited by the CT in rats (e.g., Beidler, 1953). This finding does not contradict previous reports of GSP neurophysiology. Others also show robust responses to salts; however, they appear not to be emphasized in such reports (Nejad, 1986; Harada et al., 1997).

Although there is some agreement with others about the effectiveness of salts in producing GSP responses, the result shown here that amiloride is an effective blocker of sodium salt responses is novel. A report by Harada and colleagues (1997) indicated minimal amiloride sensitivity of the GSP. There was only a 30% reduction in 0.1 M NaAc response and no reduction in NaCl response after amiloride application. In the current study, the robust response to sodium was effectively reduced by as much as 80% to 90% after amiloride application to the palate. Indeed, our results indicate that amiloride is as effective in suppressing sodium salt responses in the GSP as it is in suppressing sodium salt responses in the CT (e.g., Brand et al., 1985; Hill, 1987).

The discrepancy between our findings and those of Harada et al. (1997) may be explained in several ways. First,
GSP SALT AND AMILORIDE SENSITIVITY

Figure 5. Average (±SE) greater superficial petrosal response ratios of Fischer 344 (F344) and Sprague-Dawley (SD) rats calculated for (A) ammonium chloride (NH₄Cl); (B) hydrochloric acid (HCl) and quinine hydrochloride; (C) sucrose; and (D) maltose, all presented in distilled water.

data analyses were quite different between the two reports. One of the primary intentions of the statistical methods used in that report was to make response magnitude comparisons between CT and GSP recordings. In the present report, we recorded only from the GSP, and our goal was to analyze GSP responses exclusively. Thus, we used procedural and statistical methods that have been used frequently by researchers who have examined CT responses exclusive of comparisons with other nerves. Another possibility for the discrepancy between reports is a difference in the palatal fields stimulated. In the present study, we stimulated all three areas innervated by the GSP: the nasoincisor duct, the geschmacksstreifen, and the posterior palatine field (Miller, 1977; Miller & Spangler, 1982). In the Harada et al. (1997) study, the nasoincisor duct was covered by the headholder and sealed with petroleum jelly. It is possible that taste receptors in the nasoincisor duct are especially sensitive to sodium salts and to amiloride compared with posteriorly located palatal taste receptors. Another difference between studies was the concentration of amiloride. Harada and colleagues (1997) used 50 µM amiloride, whereas we used a 100 µM concentration. On the basis of results of CT suppression by amiloride, 50 µM should be sufficient to observe the full effect of amiloride. However, the dynamics
of palatal taste receptors have yet to be determined and could differ from those of anterior tongue receptors.

Although it is not possible to make direct comparisons of CT and GSP taste responses, many studies of CT function have used 0.5 M NH4Cl as a standard to compare stimuli in a manner similar to the methods in the present study. Thus, it is possible to make indirect comparisons between the GSP responses from the current study with CT responses from previous studies. In a study by Bernstein et al. (1991), the relative magnitude of CT responses in F344 rats was slightly higher than the degree of response we observed from the GSP. For example, Bernstein and colleagues (1991) showed that the relative magnitude of response to 0.25 M NaCl from the CT of F344 rats was approximately 1.4, and the degree of suppression by amiloride was approximately 75%. In the present study, the relative response of the GSP in F344 rats to 0.25 M NaCl was 1.04, and the degree of suppression by amiloride was 83%. Similarly, SD relative responses were only slightly higher for the CT in a study by Hill (1987) compared with the GSP responses in the present report. Hill (1987) showed that the relative response of the CT to 0.25 M NaCl was approximately 0.85 and suppressed by amiloride approximately 54%. The GSP relative response of SD rats to 0.25 M NaCl in the present study was 0.53, and responses were suppressed 53% by amiloride.

It must be stressed that equivalent relative magnitudes of taste responses do not necessarily indicate equivalence in the neural signal. Because only relative responses can be obtained from whole nerve recordings, it is not clear whether nerve-related differences are due to differences in the test stimulus or to the standard stimulus or both. Nerve-related response comparisons can only be made by comparing absolute measures (e.g., action potentials/unit time) obtained from single neuron recordings. In addition to the qualifications for comparing relative responses from taste nerves, it is important to note that the similarity in the amplitude of relative response does not necessarily translate into similarity in the central processing of that response. The converse is also true; differences in the magnitude of peripheral neural responses do not necessarily indicate differences in central coding of taste. Thus, we cannot currently make broad statements about central coding of taste qualities, despite strong evidence of similarities in salt responses and dissimilarities in sucrose responses between the CT and GSP.

In addition to the neurophysiological results presented here, there is also a growing literature of behavioral studies that suggests amiloride sensitivity exists in palatal taste receptors. During discrimination tasks, rats with bilateral CT section (CTX) were able to distinguish NaCl from potassium chloride (Spector & Grill, 1992; St. John, Markison, Guagliardo, Hackenberg, & Spector, 1997), but when intact rats were treated with amiloride, they were no longer able to make the discrimination (Spector, Guagliardo, & St. John, 1996). The authors concluded that there must be an amiloride-sensitive component to the gustatory system other than that transmitted through the CT. Because studies have demonstrated a lack of amiloride sensitivity in the glossopharyngeal nerve (Formaker & Hill, 1991), and the superior laryngeal nerve appears unlikely to be involved in early detection of salts (Bradley, Stedman, & Mistretta, 1983; Stedman, Bradley, Mistretta, & Bradley, 1980), the GSP was implicated as the other nerve most likely to be amiloride sensitive. Indeed, work in hamsters has shown that 71% of taste receptor cells isolated from the soft palate were sensitive to amiloride (Gilbertson, Zhang, Schuber, DeBenedetto, & Fontenot, 1997). Therefore, there is converging evidence that the GSP and the CT transmit information about salt, and that one component of sodium transduction by both receptor cell populations is via amiloride-sensitive pathways. As such, the geniculate ganglion, where the cells of the GSP and CT are located, receives responses from salt that originate on the anterior tongue and on the palate. Consequently, as Travers et al. (1986) demonstrated, collective information from the periphery converges onto central nervous system cells in the NTS. Therefore, the results presented here provide further support that salt-elicited interactions (excitatory and inhibitory) occur in NTS neurons that receive inputs from both GSP and the CT. This convergent input has important implications for central processing of salt taste responses because both lingual and palatal taste receptors are stimulated simultaneously during an ingestive bout.

In addition to providing normative data on GSP response profiles with the recordings from SD rats, the present study provides the first report of GSP responses in animals that differ from those that are normally used. The GSP relative response to a range of NaCl and NaAc concentrations was higher in F344 rats than in SD rats. Additionally, the heightened responsivity of F344 rats was reduced to a greater degree by amiloride. Therefore, even though sodium and amiloride are highly effective in both rat strains, there are clear strain-related differences in the magnitude of the effectiveness. Because of the differences in salt responses between SD and F344 rats, it is tempting to compare the possible behavioral correlates that could be attributed to differences in peripheral function.

Unlike most strains of rat, F344 rats do not have a preference for any concentration of NaCl and demonstrate an aversion toward concentrations of NaCl that other strains of rat prefer (Midkiff et al., 1985). The amiloride-sensitive sodium channel and the resultant neural signal carried by the CT have been implicated as mediators of F344 NaCl aversion for a number of reasons. The electrophysiological response of the CT in F344 rats is significantly larger than the response to NaCl by the CT of Wistar rats, a salt-preferring strain of rat (Bernstein, Longley, & Taylor, 1991). Bilateral transection of the CT in F344 rats dramatically reverses their aversion, such that the preference-aversion curve produced from two-bottle preference tests after CTX is remarkably similar to that of intact Wistar rats (Sollars & Bernstein, 1994b; Sollars et al., 1991). Furthermore, the aversion to NaCl is not manifest in early postnatal F344 rats, but emerges coincident with the maturation of the heightened electrophysiological response to NaCl (Schafe & Bernstein, 1997). Because the normal development of the CT includes the addition of functional amiloride-sensitive sodium channels (Hill & Bour, 1985; Sollars & Bernstein, 1994a), and amiloride sensitivity of the CT is greater in F344 rats.
than in Wistar rats (Bernstein et al., 1991), this suggests that an altered signal to NaCl produced by a greater number or a heightened affinity of amiloride-sensitive sodium channels in fungiform taste receptors was responsible for the salt aversion in F344 rats. Therefore, converging results from electrophysiological and nerve cut studies provide evidence that the CT is the mediator of the strain-related differences. However, the present results challenge that hypothesis because components that are dissimilar between salt-prefering and salt-avoiding strains in the CT response (e.g., larger response to sodium and stronger amiloride suppression) are also dissimilar between strains in the GSP response.

A variety of explanations might reconcile this seemingly straightforward description of the mechanism of F344 salt aversion with the results from the present study. First, it is possible that the GSP signal is irrelevant to F344 salt aversion and the signal carried by the CT is that which underlies the aversion. Second, it could be that the combination of sodium signals from the CT and GSP is the factor that signals the behavioral aversion. Elimination of one component of the afferent signal by CTX, for example, results in normalization of the overall signal to the brain, resulting in typical preference behavior. This implies the untested possibility that bilateral transection of the GSP would also result in normalization of salt preference. However, Chappell, St. John, and Spector (1998) have shown that mixing amiloride in NaCl did not significantly reduce the degree of F344 salt aversion in a two-bottle test. Therefore, the amiloride-sensitive component of the afferent signal appears to be irrelevant to the manifestation of the aversion. Finally, the peripheral differences in taste receptors or nerves in F344 rats may be secondary to altered components of central neuroanatomy or neurophysiology. That is, the site of response alterations occurs centrally and not in peripheral receptors and neurons.

In summary, the current study provides further evidence that the GSP is highly responsive not only to sugars but also to salts. The results also show that the sodium salt response is highly sensitive to amiloride, suggesting that similar transduction processes are used by taste receptor cells on the anterior tongue and on the palate. Finally, there are quantitative differences in GSP responses between SD and F344 rats to sodium salts and to sucrose. Further characterization of the response profiles of individual GSP neurons is necessary to better determine the neural code for salt and sweet taste and may provide insights into the mechanisms responsible for strain-related differences in salt preferences.

References


Nejad, M. S. (1986). The neural activities of the greater superficial
petrosal nerve of the rat in response to chemical stimulation of the palate. Chemical Senses, 11, 283–293.


Received December 4, 1997
Revision received March 5, 1998
Accepted March 17, 1998