Behavioral Neuroscience
1994, Vol. 108, No. 1, 131-140

0735-7044/94/$3.00

Combined, but Not Single, Gustatory Nerve Transection Substantially Alters Taste-Guided Licking Behavior to Quinine in Rats

Steven J. St. John, Mircea Garcea, and Alan C. Spector

On the basis of electrophysiological studies, the glossopharyngeal nerve (GL) is far more responsive to quinine than the chorda tympani (CT) or greater superficial petrosal (GSP) nerves. The licking behavior of water-deprived rats to quinine (0.03–3.0 mM) and distilled water (10-s trials) was tested before and after various nerve transections. GL+CT section caused a substantial reduction in responsiveness. GSP+CT section had a moderate effect, and GL section alone produced only marginal impairments. Control, partially desalivated, and CT-sectioned rats were unaffected. Thus, the GL is not necessary for normal unconditioned taste-guided appetitive responsiveness to quinine, but the collective input from the GSP and CT is necessary and most likely sufficient. These data suggest that the quinine-evoked input of the GL and CT converge centrally.

Quinine, which tastes bitter to humans, is an alkaloid that is toxic at high concentrations (Garcia & Hankins, 1975). The ability to avoid harmful substances on the basis of taste is an adaptation that appears to have evolved very early in evolutionary history (Garcia & Hankins, 1975). Rats are acutely sensitive to quinine, detecting it at concentrations as low as 1.2×10^{-5} M (Koh & Teitelbaum, 1961). In the suprathreshold domain, rats reliably decrease ingestion of quinine as a function of increasing concentration (Benjamin, 1955b; Pfaffmann, 1952; Spector, 1992).

The peripheral signal representing quinine is transmitted to the brain by four nerve branches that appear to be differentially sensitive to this stimulus in neurophysiological studies. The greater superficial petrosal (GSP) branch of the facial nerve (cranial nerve VII) innervates taste buds in and around the nasoincisor ducts (NID) of the hard palate and most of the taste buds on the soft palate (accounting for approximately 17% of the total rat buds; Cleaton-Jones, 1976; Miller, 1977). Whole nerve records of the rat GSP indicated that quinine was the least potent stimulus for the GSP compared with sucrose, sodium saccharin, citric acid, and sodium chloride (NaCl; Nejad, 1986). Presently, no study has examined single units in the rat GSP. Examination of single units in the superior laryngeal branch of the vagus nerve in hamsters yielded units that produced both inhibitory and excitatory responses to quinine, but, in general, quinine was, at best, a moderate stimulus for this nerve (Dickman & Smith, 1988). The taste buds innervated by the vagus (approximately 4–10% of the total in the rat; S. P. Travers & Nicklas, 1990; see also Miller) are located on the esophagus and the laryngeal epithelium and are thought to be involved with the protection of the airways.

The chorda tympani (CT) branch of the facial nerve innervates taste buds in the fungiform papillae of the anterior two thirds of the tongue, as well as in the anterior foliate papillae (approximately 15% of the total; see Miller, 1977). The CT responded only weakly to quinine in electrophysiological studies (Nejad, 1986; Pfaffmann, 1955). Single units in the CT that responded to quinine did so in a nonspecific fashion; such units also responded to NaCl and HCl (Frank, Contreras, & Hettinger, 1983).

The glossopharyngeal nerve (GL) is by far the most electrophysiologically responsive nerve to quinine. This nerve innervates taste buds in the foliate papillae and in the circumvallate papilla on the posterior tongue (accounting for 64% of the total; see Miller, 1977). Single units in the petrosal ganglion of the GL are highly responsive to quinine, even when these units do not respond to other alkaloids such as atropine, which is another potent stimulus of petrosal ganglion cells (Boudreau, Do, Sivakumar, Oravec, & Rodriguez, 1987). Frank (1991) identified a substantial population of narrowly tuned cells in the GL (Q units, which comprised 31% of the total fibers sampled) that responded best to quinine when compared with sucrose, NaCl, and HCl. This is to be compared with units in the CT, where Frank et al. (1983) found only 1 quinine-best unit out of 44 fibers sampled, using a similar response criterion.

These findings suggest that the GL substantially contributes to the quinine-evoked neural signal, whereas the GSP and CT play a less crucial role. Nerve-transection studies have been conducted to test this hypothesis. Combined transection of the GL, CT, and vagus nerves decreased the aversion to both low and high concentrations of quinine when measured with long-term two-bottle preference tests (Jacquin, 1983; Vance, 1967). Combined transection of the GL and CT had similar effects on quinine preference (Grill & Schwartz, 1992; Pfaffmann, 1952). Despite the fact that combined GL and CT section denervated 80%–85% of the total taste buds, it did not

Steven J. St. John, Mircea Garcea, and Alan C. Spector, Department of Psychology, University of Florida.

This research was supported by U.S. Public Health Service Grant DC-00161 to Alan C. Spector. Portions of this work were presented in 1993 at the 15th Annual Meeting of the Association for Chemoreception Sciences in Sarasota, Florida. We thank Rachel Redman and Ray Cauthon for their technical assistance in this experiment.

Correspondence concerning this article should be addressed to Alan C. Spector, Department of Psychology, University of Florida, Gainesville, Florida 32611. Electronic mail may be sent to spector@webb.psych.ufl.edu.

entirely eliminate quinine responsiveness. Presumably, this results from the signal generated by the GSP (Pfaffmann, 1952) or from postingestive factors (Grill, Spector, Schwartz, Kaplan, & Flynn, 1987; Grill, Schwartz, & Travers, 1992).

Similar results have been obtained in behavioral studies designed to minimize the contribution of postingestive effects on responsiveness. Yamamoto and Asai (1986) found a significant increase in licks to 0.5 mM quinine (the only concentration tested) during 20-s trials in water-deprived rats with combined GL and CT section compared with controls, indicating a decreased behavioral responsiveness to that concentration. Grill and Schwartz (1992) found that combined GL and CT section abolished the concentration-dependent increase in stereotyped oromotor behaviors (i.e., gapes) when quinine was infused directly into the oral cavity of nondeprived rats (a technique commonly referred to as *taste reactivity*). Although these taste reactivity data suggest that certain behaviors may be mediated entirely by input from the GL and CT nerves, two-bottle preference data imply that the transection of the GL and CT does not render the rat ageusic to quinine (Grill & Schwartz; Pfaffmann, 1952).

The results of single nerve sections (either CT alone or GL alone) are more equivocal. Section of the GL did not appear to alter aversion to quinine in two-bottle preference tests (Akaike, Hiji, & Yamada, 1965; Grill et al., 1992). One study found a decrease in quinine aversion after CT section (Akaike et al., 1965), whereas others did not (Grill et al.; Pfaffmann, 1952). Rats licking a midrange suprathreshold concentration of quinine (0.5 mM) in a brief exposure test were unaffected by either GL or CT section, but effects at a wider range of concentrations were not tested (Yamamoto & Asai, 1986). Although studies of preference or licking behavior failed to show any effect of GL section on quinine responsiveness, the two studies that measured taste reactivity showed that GL section significantly decreased stereotyped aversive oromotor responses, such as gapes (Grill et al.; J. B. Travers, Grill, & Norgren, 1987). In contrast, CT section either had no effect or only a minor effect on quinine-elicited gaping (Grill et al.; J. B. Travers et al.). Thus, the effect of GL or CT transection on behavioral responsiveness to quinine varies depending on the procedure used to assess function. To date, no study has examined the effect of GSP section, either alone or in combination with other nerves, on behavioral responsiveness to quinine.

Transection of the CT partially denervates the sublingual and submaxillary salivary glands (Young & Van Lennep, 1978). Extirpation of these glands consistently decreased quinine aversion as measured by long-term two-bottle preference tests (Brosvic & Hoey, 1990; Catalanotto & Sweeney, 1972, 1973). The effect of desalivation on quinine responsiveness has not been tested in a paradigm designed to minimize postingestive feedback.

Interpretation of the sometimes disparate effects of nerve sections on quinine responsiveness requires careful consideration of the methodology used. For example, long-term preference tests measure both taste-generated appetitive and consummatory behavior, but postingestive effects can also influence intake. Taste reactivity paradigms are designed to minimize postingestive effects, but they focus only on consummatory behavior. To combine the advantages of both paradigms, we examined the effect of various gustatory nerve sections (alone or in combination with another gustatory nerve) on appetitive licking behavior to small volumes of quinine. This paradigm is similar to that of Yamamoto and Asai (1986); however, this experiment tested responsiveness to an expansive array of quinine concentrations instead of only one and used a within-subjects design as opposed to a between-subjects design. Subtle effects of taste bud denervation on quinine responsiveness could be missed when only one concentration is employed. In addition, this study includes a group with combined GSP and CT section; thus, this is the first study to examine the relative contributions of the seventh and the ninth cranial nerves to behavioral responsiveness to quinine. The rats were tested before and after surgery in a specially designed gustometer, and a computer monitored immediate licking responses during 10-s stimulus trials. Results from previous work in this laboratory (Spector, 1992) demonstrated that such a technique provides a reliable monotonically decreasing sigmoidal quinine concentration–response curve that is sensitive to neural manipulations in the gustatory system. Furthermore, this study incorporated the advantages of both one- and two-bottle intake tests, in that a rat was not forced to consume the quinine because both the tastant and water were available during the session (two-bottle test), but the rat was forced to repeatedly sample, at least briefly, each stimulus (one-bottle test) (Benjamin, 1995a; Grill et al., 1987).

Method

Subjects

Seventy-two naive male Sprague-Dawley rats (CD stock; Charles River Breeders, Wilmington, MA), weighing 298–495 g at the start of the experiment, were individually housed in stainless steel cages. The rats had free access to Purina chow (5001) and a corn oil diet (5 parts Purina powdered chow to 2 parts Wesson corn oil; see Catalanotto & Sweeney, 1973). The corn oil diet was provided to facilitate swallowing for the desalivated and denervated rats during the water-deprived testing phase after surgery. Except where noted otherwise, the rats had free access to distilled water. The rats were run in four phases of 12–18 rats each. Throughout the experiment, body weight was monitored several times a week. The rats were maintained on a 12:12-hr light–dark cycle in a room where temperature, humidity, and lighting were automatically controlled. All manipulations were performed during the light phase.

Procedure

Spout training. Approximately 24 hr prior to training, the water bottles were removed from the home cages. On the first 2 days of training, the rats were placed individually in a specially designed gustometer (see Spector, Andrews-Labenski, & Letterio, 1990, for a detailed description) for 30-min sessions, where they licked a spout to receive distilled water. A computer monitored licking behavior through a contact circuit that delivered less than 50 nA to the animal. Throughout the experiment, each lick delivered approximately 5 μl of fluid, controlled by a computer-manipulated solenoid valve. During these sessions, the rats received their daily fluid allotment. This phase served to habituate the rats to the gustometer, to train them to lick the spout to receive fluid, and to provide a presurgical baseline of the microstructure of licking behavior (see Davis, 1989).

Presurgical testing. Presurgical testing for quinine responsiveness occurred during the next 3 days. The water-deprived rats were tested in the same apparatus in 40-min sessions. Rats were required to lick the dry spout two times within 500 ms to initiate a trial. There were two types of trials during which rats had access to 10 s of fluid: distilled water rinse trials and test trials. A distilled water rinse trial always preceded a test trial and served to rinse the oral cavity, providing a relatively constant receptor adaptation state for each test trial (see Bartoshuk, 1977).

Test trials consisted of the presentation of either distilled water or one of seven concentrations of quinine hydrochloride (0.003, 0.01, 0.03, 0.1, 0.3, 1.0, and 3.0 mM), which were prepared daily from reagent grade chemicals (Fisher) and distilled water. The presentation order of test solutions was randomized in blocks of eight. At the end of each distilled water rinse or test trial, the spout was rotated out of reach over a drainage funnel and flushed with distilled water. Two bursts of pressurized air evacuated and dried the spout before the spout was rotated back into position. This process took approximately 6 s. The rats could then initiate another trial and could instigate as many trials as possible until the 40 min expired. The rats received their daily fluid during these test sessions. After the third presurgical test session, distilled water bottles were replaced on the home cages, and surgery was performed 3–7 days later.

Postsurgical testing. On the final day of the recovery period, the water bottles were removed from the home cages approximately 24 hr prior to testing. Postsurgical testing for quinine responsiveness was conducted during the next 3 days in a manner identical to that previously described. Following the third postsurgical testing session, the rats were tested in a single 30-min session identical to the first two 30-min presurgical spout training sessions. The spout did not move during this session, and the only fluid available was distilled water. The purpose of this session was to examine the effect of surgery on the microstructure of licking behavior, most notably the interlick interval.

Surgery

Rats from all surgical groups were deeply anesthetized with ketamine hydrochloride (87 mg/kg ip) and xylazine hydrochloride (17 mg/kg ip) and prophylactically treated with a penicillin suspension (30,000 units, im). During surgery, they rested on isothermal heating pads.

The CT was bilaterally transected in 10 rats (CTx). These rats were placed in a headholder tilted approximately 80° away from the surgeon. The external auditory meatus was retracted with blunted hypodermic needles to reveal the tympanic membrane. With the aid of a surgical microscope, the tympanic membrane and ossicles were removed with no. 7 microforceps. If the CT survived the removal of the ossicles, it was avulsed with the forceps. One rat died shortly after surgery, reducing the sample size to $n = 9$.

The GL was bilaterally transected in 10 rats (GLx). These rats were placed in a supine position in the headholder. An incision was made along the midline of the ventral neck. The sublingual and submaxillary salivary glands were retracted, as were the sternohyoideus, the omohyoideus, and the posterior belly of the digastricus muscles, which revealed the hypoglossal nerve. The underlying fascia was then carefully dissected to reveal the GL close to the bulla. The GL was held with no. 7 microforceps and a 2–3 mm portion was cut with microscissors and removed. Care was taken not to disturb the nearby hypoglossal and vagus nerves. The incision was closed with sutures (4-0 silk). One rat died shortly after surgery, reducing the sample size to $n = 9$.

Nine rats received combined bilateral transection of the GL and the CT (GLx+CTx). The surgical procedure was the same as previously described. Two rats died shortly after surgery, reducing the sample size to $n = 7$.

The 22 rats that received combined bilateral transection of the GSP and CT nerves (GSPx+CTx) were placed in a headholder tilted 80° away from the surgeon. An incision was made around the dorsal side of the pinna, and the pinna was retracted. The fascia around the external auditory meatus was dissected using no. 7 microforceps, and the meatus was punctured. This opening was enlarged by retracting the surrounding musculature and by carefully removing a small part of the auditory bulla with ronguers. The tympanic membrane and ossicles were removed, and the CT sectioned in the process. The tensor tympani and a small piece of temporal bone were also removed, exposing the GSP. The nerve was avulsed with the microforceps. The incision was closed with wound clips. Three rats died shortly after surgery, and a 4th rat developed a severe eye infection and was euthanized prior to postsurgical behavioral testing. This reduced the sample size to $n = 18$.

The sublingual and submaxillary salivary glands were removed in 8 rats (DSAL). An incision was made in the ventral neck. Once the fascia around the sublingual and submaxillary salivary glands was dissected, the duct and vasculature were ligated with 4-0 silk suture and cut, and the glands were removed.

Thirteen rats served as surgical controls (CON). In these rats, the GL was exposed as described earlier but was not disturbed. The headholder was then tilted 80° away from the experimenter and an incision was made around the dorsal side of the pinna. Using no. 7 microforceps, the fascia around the auditory meatus was retracted, and the meatus was punctured to expose the tympanic membrane. A small hole was made in the tympanic membrane with the microforceps, but care was taken not to disturb the underlying ossicles. The incision was closed with wound clips. Four controls died shortly after surgery, reducing the sample size to $n = 9$.

The rats recovered for the next 17–21 days. During this time they had access to distilled water, Purina Chow pellets, and the corn oil diet. Body weight was monitored regularly, and, to potentially facilitate recovery, powdered chow was given to some animals as an additional alternative to the other food choices. With the exception of 6 rats from the GSPx+CTx group, all rats returned to their presurgical body weights before behavioral testing resumed. No rat weighed less than 92% of its presurgical body weight when testing resumed.

Several methods were employed to encourage recovery in the rats receiving combined GSP and CT transection. One rat was given wet mash (powdered Purina Chow 5001 mixed with water) on 2 days. Nine rats were treated with 30,000 units of penicillin for 2 days following surgery; 4 rats from this group received further penicillin treatment. No rat received penicillin treatment less than 4 days before the resumption of testing. Finally, 2 rats developed lesions on one eye. These rats were treated bilaterally with an ophthalamic ointment (gentamicin sulfate, Schein Pharmaceutical); treatment was discontinued 4 days prior to testing, after the condition had noticeably improved.

Histology

One to 4 days after the last test session (no more than 29 days after surgery), the rats were deeply anesthetized with sodium pentobarbital and perfused transcardially with isotonic saline followed by 10% buffered formalin. The tongue, soft palate, and NID were removed and stored in buffered formalin for later histological examination. Because transection of the gustatory nerves causes taste bud degeneration in the respective receptor fields (e.g., Cleaton-Jones, 1976; Ganchrow & Ganchrow, 1989; Guth, 1957; Miller, 1977), oral tissue was examined postmortem for the presence of taste buds to evaluate the efficacy of the nerve sections.

The circumvallate papilla, soft palate, and NID were embedded in paraffin, sectioned (10 μm) on a rotary microtome, mounted consecutively on glass slides, and stained with hematoxylin and eosin. The soft

palate and NID from all rats in the CON and GSPx+CTx groups were examined, as were the circumvallate papillae from all rats in the GLx, GLx+CTx, and CON groups. Circumvallate papillae were analyzed for the presence or absence of buds; bilateral GL section results in the virtual elimination of taste buds in the circumvallate papilla (Guth, 1957). The number of taste buds in the soft palate and NID were quantified; in the soft palate, only the taste buds of the Geschmacksstreifen (a dense line of taste buds separating the hard and soft palates; see Miller, 1977) were counted. To provide a conservative evaluation of the effect of nerve section, taste buds were counted whether or not taste pores were observed. It should be noted that in intact control tissue, taste pores were frequently encountered.

The anterior tongue was examined in all rats in the CTx, GLx+CTx, and CON groups. It was first immersed in distilled water for 1 hr and then dipped in 0.5% methylene blue followed by a rinse in distilled water to remove the excess stain. The tongue was then cut along the midline into left and right halves, and the underlying muscle and connective tissue removed with ronguers. The two halves were then flattened between two glass slides, revealing both the dorsal and ventral surfaces. Under the microscope, a taste pore appeared as a blue dot roughly centered in the pale staining fungiform papilla. The number of fungiform papillae with and without an intact taste pore in the anterior 5 mm of the tongue was quantified.

The oral tissue of the rats in the DSAL group was not examined. It should be mentioned, however, that previous authors have found changes in taste bud structure (e.g., alteration in size, bacteria infestation of the taste pore, disorganized structure) following sublingual and submaxilary gland extirpation (see Cano & Rodriguez-Echandia, 1980; Nanda & Catalanotto, 1981).

Data Analysis

The lick data for each rat were collapsed across trials for each concentration of quinine. The average number of licks to a given quinine concentration was standardized to the average number of licks to distilled water in the following way: ratio score = mean licks to quinine_x/mean licks to water_{test}, where x is a given concentration and *test* represents the distilled water test trial. This served to control for potential differences in lick rate that were nongustatory in origin. An analysis of variance (ANOVA) of Surgery (before vs. after) × Concentration was conducted for each surgical group. In addition, a sigmoidal two-parameter logistic function was fit to the mean concentration-response data for each group both before and after surgery (least squares, Sigmaplot 4.1): $f(x) = 1/(1 + (x/c)^b)$, where x represents quinine concentration, b represents the slope, and c represents the concentration of quinine that evoked the half-maximum response (corresponding to a 0.5 taste to water ratio score). The slope (b) and half-maximum (c) parameter from the mean concentration-response curves were tested using independent-sample t tests (before vs. after, two-tailed). An attempt was made to fit the same two-parameter curve to individual data, but several subjects in the GLx+CTx group had postsurgical concentration-response functions that were substantially flattened and no longer sigmoidal.

Software developed by this laboratory allowed the analysis of several components of licking microstructure, including interlick intervals (defined as the interval between the onset of one lick and the onset of the next lick, if separated by less than 500 ms). Additionally, the ability of a rat to maintain a steady burst of licking was examined. A burst was defined as a run of consecutive licks with interlick intervals less than 500 ms.

Matched t tests (two-tailed) were performed on mean interlick interval data obtained during the second 30-min water-only session before surgery and the single 30-min session after surgery. The longest burst in these sessions was identified for each rat, and an average maximum burst size was calculated for each group before and after surgery. Matched t tests were also performed on the mean licks recorded during water test trials in the 40-min test sessions (before vs. after surgery, two-tailed). Finally, body weight was recorded for all rats on the day of surgery, 7 days after surgery, and 14 days after surgery. A two-way ANOVA (Group × Time) was conducted on the percentage of change from surgical body weight, and paired comparisons were run to identify which groups differed from controls.

In all of the statistical procedures, the rejection criterion was set at the conventional .05 level ($\alpha = .05$).

Results

Histological Analysis

Examination of the circumvallate papilla confirmed the efficacy of GL section for all 7 GLx+CTx rats and for 8 of the 9 GLx rats. The circumvallate papilla of 1 rat of the GLx group was only partially denervated, appearing to have approximately one third of the taste buds normally found in control tissues; this rat was discarded from data analysis. Successfully denervated animals had virtually no taste buds in the circumvallate papilla. Examination of anterior tongues stained with methylene blue revealed that rats of the GLx+CTx ($n = 7$), CTx ($n = 9$), and GSPx+CTx groups received successful CT section. Animals of the CTx group averaged 9.6 fungiform papillae with taste pores ($SE = 1.53$) and 69.7 ($SE = 3.9$) fungiform papillae without taste pores in the anterior 5 mm of the tongue. Rats of the GLx+CTx group had a mean 9.6 ($SE = 2.04$) fungiform papillae with taste pores and 72.0 ($SE = 2.60$) fungiform papillae without taste pores. Rats of the GSPx+CTx group averaged 5.7 ($SE = 1.10$) fungiform papillae with taste pores and 68.4 ($SE = 4.40$) fungiform papillae without taste pores. By comparison, control rats had an average 90.9 ($SE = 4.40$) fungiform papillae with pores and 3.33 ($SE = 0.83$) fungiform papillae without pores. The persistence of a few intact taste buds in CT denervated animals is in agreement with previous reports (Whitehead, Frank, Hettinger, Hou, & Nah, 1987; see also Ganchrow & Ganchrow, 1989).

Examination of the Geschmacksstreifen and NID confirmed the efficacy of GSP transection in 11 of 18 rats in the GSPx+CTx group. Successfully transected rats had a mean 3.2 ($SE = 1.68$) taste buds in the NID and 1.36 ($SE = 0.41$) taste buds in the Geschmacksstreifen; CON group rats averaged 68.0 ($SE = 5.02$) and 60.6 ($SE = 3.63$), respectively. Because of poor histological preparation, full counts of Geschmacksstreifen taste buds could not be obtained for two controls. No successfully transected rat had more than 18 taste buds collectively in the NID and Geschmacksstreifen. The 7 rats with substantial numbers of taste buds remaining in the palatal fields were discarded from the behavioral analysis.

Body Weight

The mean body weight for each group was recorded 1 and 2 weeks following surgery and is plotted as a percentage change from presurgical body weight (see Figure 1). A two-way ANOVA revealed a significant main effect of both group, $F(5, 46) = 52.71$, $p < .0005$, and days, $F(1, 46) = 2{,}148.98$, $p < .0005$, as well as a significant Group × Day interaction, $F(5,$

46) = 4.312, p = .003. The GSPx+CTx group, $F(1, 46)$ = 170.6, p < .0005, and the GLx+CTx group, $F(1, 46)$ = 19.2, p = .0001, were significantly different from controls on Day 7. The GSPx+CTx group, $F(1, 46)$ = 188.7, p < .0005; the GLx+CTx group, $F(1, 46)$ = 26.2, p < .0005; and the GLx group, $F(1, 46)$ = 12.5, p = .001, were significantly different from controls on Day 14.

Concentration Response Data

In general, the only manipulations to have profound and statistically significant effects on quinine responsiveness were groups that received transection of more than one gustatory nerve (GLx+CTx and GSPx+CTx). The GLx group showed a tendency to be impaired in quinine responding, but in only one analysis was the effect statistically significant and in that case the effect was small. There was no evidence suggesting the quinine responsiveness changed after surgery in the CTx, DSAL, and CON groups (see Figure 2).

The two-way ANOVA (Surgery × Concentration) revealed a significant effect of concentration in all groups (all p values < .0005). In addition, the GLx+CTx group showed a significant effect of surgery, $F(1, 6)$ = 29.92, p = .002, and a significant Surgery × Concentration interaction, $F(6, 36)$ = 7.25, p < .0005. The GSPx+CTx group also showed a significant effect of surgery, $F(1, 10)$ = 6.238, p = .032, and a significant interaction of Surgery × Concentration, $F(6, 60)$ = 8.840, p < .0005. The other groups did not show a significant effect of surgery (all p values > .13) or a significant Surgery × Concentration interaction (all p values > .26).

Independent t tests on the parameters of the group mean curves basically agreed with the ANOVA results. The one-half maximum parameter (parameter c) of the GLx+CTx curve significantly shifted 1.183 log units to the right, $t(10)$ = 3.54, p < .01, and the slope (parameter b) significantly flattened from b = 1.147 to b = 0.629, $t(10)$ = 4.184, p < .002. Parameter c of the GSPx+CTx group mean curve shifted 0.337 log units to the right, $t(10)$ = 2.664, p < .05, and parameter c of the GLx curve shifted 0.176 log units to the right, $t(10)$ = 4.27, p < .002. No significant effect on parameter b was found in either case, and no significant changes were found in the parameters defining the other group mean curves.

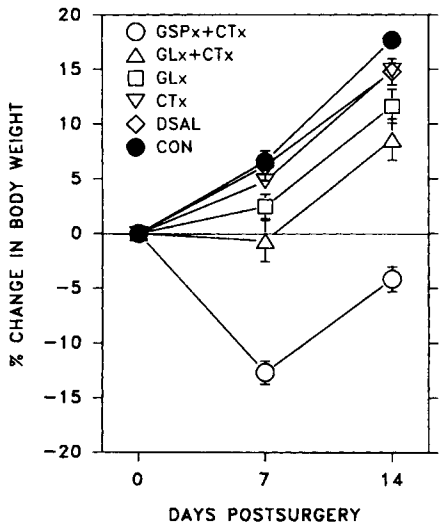

Figure 1. Mean percentage change in body weight from day of surgery plotted for postsurgical Days 7 and 14. GSPx+CTx = greater superficial petrosal and chorda tympani transected rats; GLx+CTx = glossopharyngeal and chorda tympani transected rats; GLx = glossopharyngeal transected rats; CTx = chorda tympani transected rats; DSAL = partially desalivated rats; CON = control rats.

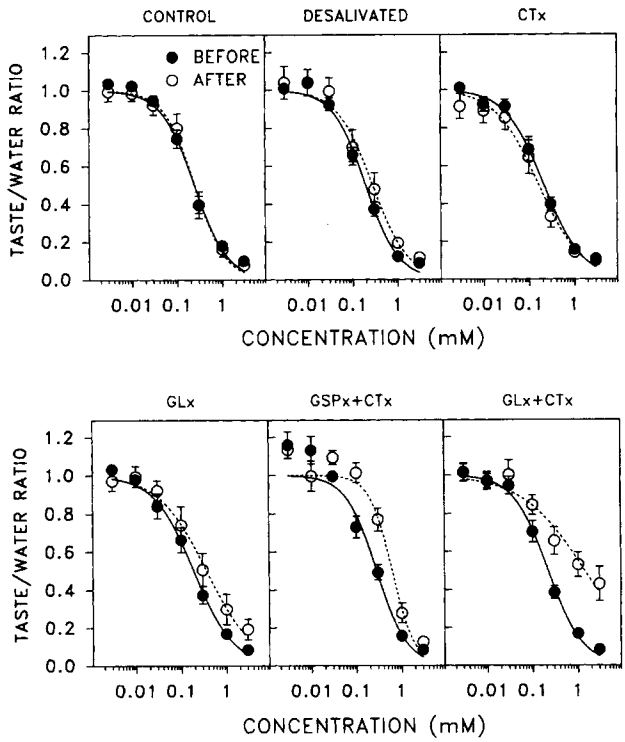

Figure 2. Semilogarithmic plot of the mean (±SE) number of licks to quinine divided by the number of licks during water test trials as a function of quinine concentration. Filled circles and solid lines represent presurgical concentration-response data, and open circles and dashed lines represent postsurgical data. Curves were fit to the sigmoidal logistic function: $f(x) = 1/(1 + (x/c)^b)$ using the least squares method. CTx = chorda tympani transected rats; GLx = glossopharyngeal transected rats; GSPx+CTx = greater superficial petrosal and chorda tympani transected rats; GLx+CTx = glossopharyngeal and chorda tympani transected rats.

Lick Rate and Water Licks Analysis

Matched t tests revealed significant increases in interlick intervals (corresponding to a decrease in local lick rate) in all groups (all p values < .03) except the CON and GLx+CTx groups (see Figure 3). The effect, however, was small except in the DSAL group (an increase of 29.7 ms) and the GSPx+CTx group (an increase of 22.4 ms). In 10-s trials, the 29.7-ms increase in the mean interlick interval after desalivation corresponds to a mean decrease of nine licks in a 10-s trial. Although this effect is nominal at best, its magnitude was nonetheless surprising and deserves some attention.

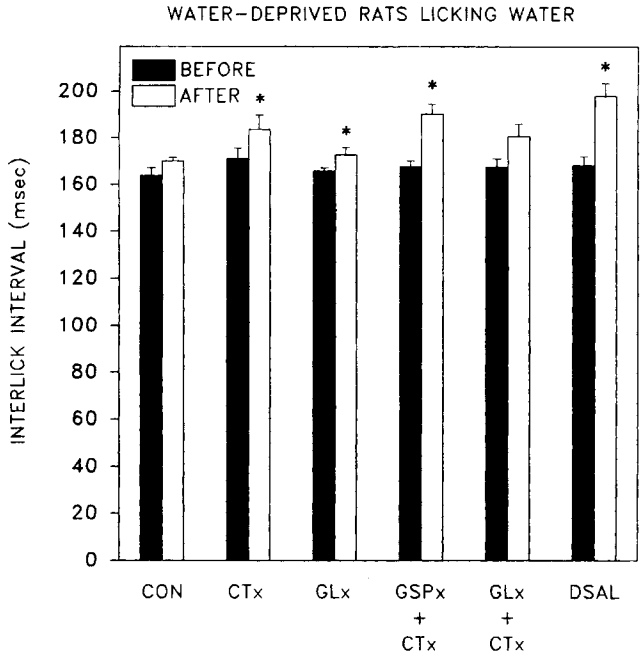

Figure 3. The mean interlick interval (in ms) for each surgical group, both before (filled) and after (open) surgery. Asterisks represent a significant difference after surgery (matched t tests, two-tailed, $p < .05$). CON = control rats; CTx = chorda tympani transected rats; GLx = glossopharyngeal transected rats; GSPx+CTx = greater superficial petrosal and chorda tympani transected rats; GLx+CTx = glossopharyngeal and chorda tympani transected rats; DSAL = partially desalivated rats.

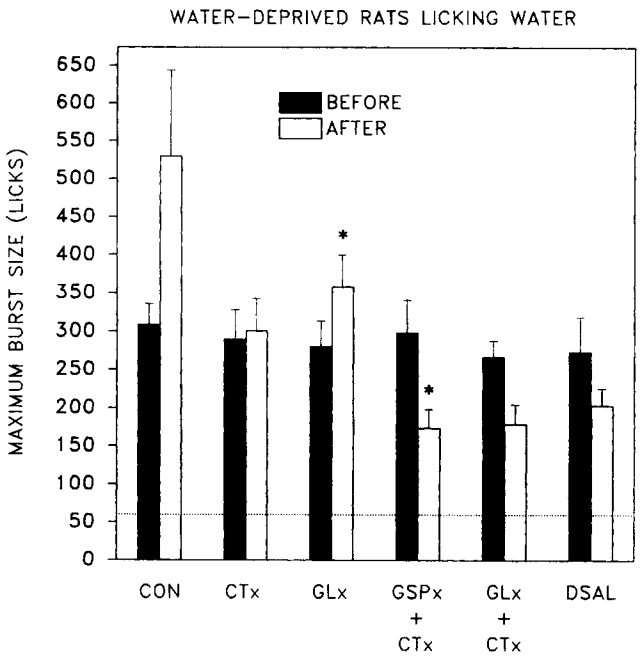

Figure 4. Mean of the maximum burst size (number of licks, $\pm SE$) in 30-min sessions when water-deprived rats licked a spout for water before (filled) and after (open) treatment. A burst was defined as a consecutive run of licks with interlick intervals less than 500 ms. The dashed line at 60 licks represents the size of a burst expected (at 6 licks/s) if licking were continuous during an entire 10-s test trial. Asterisks represent a significant change after surgery (matched t tests, two-tailed, $p < .05$). CON = control rats; CTx = chorda tympani transected rats; GLx = glossopharyngeal transected rats; GSPx+CTx = greater superficial petrosal and chorda tympani transected rats; GLx+CTx = glossopharyngeal and chorda tympani transected rats; DSAL = partially desalivated rats.

The mean maximum burst duration before and after surgery is illustrated in Figure 4 for each group. Several groups showed an increase or decrease in the size of their maximum burst following surgical manipulation, including controls. However, we were primarily interested in whether these groups could sustain a burst throughout a 10-s trial (approximately 60 licks, the dashed line in Figure 4). All groups maintained this ability after surgery.

Table 1 shows the results of matched t tests on the licks to water test trials. There were significant decreases in the GLx+CTx ($p = .002$), GSPx+CTx ($p = .001$), CTx ($p = .001$), and DSAL ($p = .001$) groups. This decrease in licks to the vehicle highlights the importance of standardizing the quinine licks to water licks.

Discussion

Combined GL and CT section produced a profound decrease in the rats' responsiveness to suprathreshold quinine (i.e., a decrease in the normal avoidance). Section of the electrophysiologically responsive GL produced little if any behavioral change in quinine responsiveness. These findings are in agreement with some previous behavioral work but are contrary to expectations based on neurophysiological studies. Combined GSP and CT section also caused a decreased responsiveness to quinine, but not to the same degree as when the GL was cut along with the CT.

Although some nerve sections clearly influenced licking behavior, the possibility that the apparent effects on quinine responsiveness were strictly motor in origin is not supported by the data. First, all groups demonstrated the capacity to maintain a steady burst of greater than 60 licks when licking a spout for water (see Figure 4). Second, the measurement of

Table 1
Group Mean Licks to 10-s Water Trials and Results of Matched t Tests

Group	10-s trials: Before ($M \pm SE$)	10-s trials: After ($M \pm SE$)	t	p
Controls	56.98 ± 2.08	54.07 ± 1.20	1.209	.261
GSPx + CTx	50.53 ± 2.65	35.85 ± 2.60	4.85	.001
GLx + CTx	57.15 ± 1.75	46.96 ± 2.44	5.348	.002
GLx	57.09 ± 2.31	53.90 ± 1.66	1.547	.166
CTx	56.45 ± 2.03	44.96 ± 2.92	4.781	.001
DSAL	53.56 ± 1.60	41.02 ± 2.59	5.124	.001

Note. GSPx = rats with bilaterally transected greater superficial petrosal nerves. CTx = rats with bilaterally transected chorda tympani nerves. GLx = rats with bilaterally transected glossopharyngeal nerves. DSAL = partially desalivated rats.

interlick intervals before and after surgery demonstrated that although in some groups the rate of licking was slower, in all groups it was not substantially compromised. For example, rats with combined GSP and CT section were capable of 52.5 licks on average in a 10-s trial compared with 59.6 licks before surgery. Third, the possibility that a motor impairment obscured decreases in quinine responsiveness is unlikely, because differences in lick rate were controlled by standardizing quinine licks to water licks in the form of a ratio score. The increase in interlick interval in the desalivated group was somewhat more profound and demonstrates the need for standardizing responses to water. It remains unclear exactly how desalivation altered lick rate. This effect was unexpected and deserves further study. The finding that combined GSP and CT section seemingly impairs lick rate is in contrast with the data of Krimm, Nejad, Smith, Miller, & Beidler (1987) who reported that the lick rate of rats with combined GSP and CT transection was not different than that of controls. This disparity may be due to methodological differences; Krimm and colleagues measured licks in 30 s to water following hypertonic NaCl injection, whereas we measured the average interlick interval of water-deprived rats licking water over an entire 30-min session.

This study found no changes in behavioral responsiveness to quinine following the removal of the sublingual and submaxillary salivary glands (DSAL). Catalanotto and Sweeney (1972, 1973) and Brosvic and Hoey (1990) did find that extirpation of the sublingual and submaxillary salivary glands in rats decreased quinine avoidance in two-bottle (quinine vs. water) preference tests. There are several differences in methodology that may account for the discrepancy. First, as mentioned, these investigators used 24-hr tests, whereas we measured immediate responses to small volumes of the stimulus. Second, our experiment used Sprague-Dawley rats, whereas the other experimenters used Holtzman, Wistar, or Long-Evans rats. Third, quinine hydrochloride was used in the present experiment whereas quinine sulfate was used in the other studies. Fourth, we tested the desalivated rats 3 weeks after surgery. Brosvic and Hoey tested rats 7–8 weeks postsurgery, and Catalanotto and Sweeney tested rats 3 months or 7 months after surgery. Because it is possible that saliva contains trophic factors for taste receptor cells, some length of time may be required before desalivation interferes with normal gustatory function (Cano & Rodriguez-Echandia, 1980; Nanda & Catalanotto, 1981).

Rats that received bilateral GSP and CT transection tended to lose weight into the second week of recovery, and then slowly regained presurgical body weight. The rats with combined GL and CT transection were also slower to recover than controls, although they were less affected than the GSP and CT transected animals. These data, in principle, support the observations of Jacquin (1983), who found that transection of the GL, CT, and pharyngeal branch of the vagus severely interfered with normal food intake and body weight gain, unless rats were given a highly palatable diet (pablum). The magnitude of this effect, however, does not appear to correspond to the number of taste buds deafferented, but perhaps it depends upon which gustatory afferents are disrupted. Rats in this experiment were most severely affected after bilateral

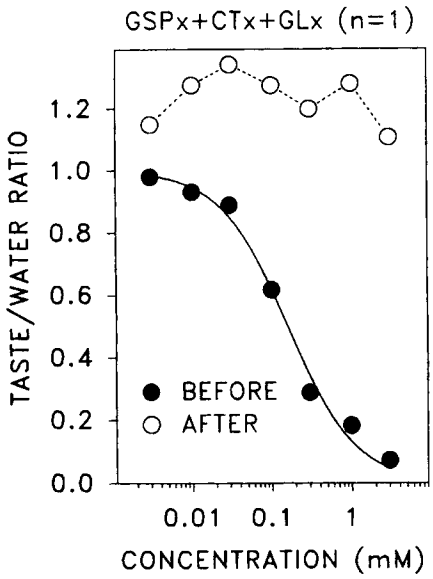

Figure 5. The concentration-response data of a single rat with bilateral transection of the GSP, CT, and GL. Filled circles and solid lines represent presurgical concentration-response data, and open circles and dashed lines represent postsurgical data. The after points are connected by straight lines due to the nonsigmoidal shape of this curve. GSPx+CTx+GLx = greater superficial petrosal, chorda tympani, and glossopharyngeal transected rat.

GSP and CT transection, which denervates only approximately one third of the rat taste buds, whereas those rats receiving a quantitatively greater denervation of taste buds (i.e., GL or GL and CT transected animals) were not as affected. The fact that CT section had no obvious effect on body weight, yet combined GSP and CT transection significantly retarded body weight gain postsurgery, suggests that portions of the facial nerve, perhaps the GSP alone, are critical for the maintenance of normal ingestive behavior in the rat.

This study is the first to report the effects of transecting the collective gustatory branches of the facial nerve (GSP and CT) on quinine responsiveness. Following this treatment, rats were somewhat impaired in their responsiveness to quinine, although the effect was small. This appears to agree with the relatively unresponsive nature of the facial nerve to quinine in electrophysiological studies (Nejad, 1986; Ogawa, Sato, & Yamashita, 1968; Pfaffmann, 1955). What is surprising, however, is the competence of the rat after GL transection, when the only gustatory information available to the animal is transmitted by the facial nerve and the vagus. The contribution of the vagus nerve to gustatory processing is considered negligible, a hypothesis that is supported by preliminary data from a single rat receiving combined bilateral GSP, CT, and GL transection (see Figure 5). The only gustatory information available to this rat was transmitted by the vagus nerve, but the rat behaved as though it were aguesic to quinine. Note, too, that if trigeminal or olfactory signals contributed to the rat's behavior in this paradigm, they were not sufficient to produce any suppression of licking over the concentrations tested. Although more animals with combined transection of the GSP,

CT, and GL must be examined, it appears as though GL-transected animals rely exclusively on the facial nerve. Assuming no contribution from vagal taste fibers, the facial nerve is surprisingly sufficient for the rat to retain normal or near-normal behavioral responsiveness to suprathreshold concentrations of quinine in this paradigm.

The failure to find effects following GL transection is in contrast to studies that have measured oromotor and somatic responses to intraorally infused taste stimuli (referred to as *taste reactivity*) (Grill et al., 1992; Travers et al., 1987). Those studies, like this one, employed a wide range of concentrations and measured immediate responses to small stimulus volumes. Grill et al. reported a significant reduction in gapes and aversive taste reactivity responses to intermediate and high concentrations of quinine in rats with bilateral GL sections compared with controls. Taste reactivity paradigms focus on consummatory behaviors that presumably have a large reflexive component. This study, on the other hand, in addition to having a consummatory component, measured appetitive licking—the behavior associated with an animal approaching and bringing the stimulus into the oral cavity. Each paradigm, by virtue of its methodological characteristics, may be measuring different aspects of how taste influences the ingestive control system. In this experiment, water deprivation promoted the appetitive behavior; in studies of taste reactivity, rats were tested in the nondeprived state. The lack of a decrease in quinine responsiveness in the present study is in agreement with the findings of others who employed procedures that involve both appetitive and consummatory components (i.e., long-term preference tests or brief-access licking behavior in water-deprived rats; Akaike et al., 1965; Grill et al.; Yamamoto & Asai, 1986).

Transection of the CT slightly increased responsiveness at all concentrations, but this effect was not statistically significant and thus corresponds to the majority of the previous behavioral data (Grill et al., 1992; Pfaffmann, 1952; Yamamoto & Asai, 1986). Despite the fact that transection of either the GL or the CT alone does not cause a decrease in quinine responsiveness, combined transection results in a profound decrease. In no condition (with the exception of the one rat in Figure 5), however, was quinine responsiveness completely eliminated; regardless of treatment, the rats were still able to decrease licking as a function of quinine concentration. Grill and Schwartz (1992) found that combined GL and CT section abolished the concentration-dependent increase in aversive oromotor behaviors to intraorally infused quinine. Again, this contrary finding may be the result of the different methodological characteristics of the taste reactivity paradigm compared with the present study. In other studies of brief-access licking behavior, as well as in long-term preference tests, quinine responsiveness was not completely eliminated after combined section of the GL and CT (Grill & Schwartz; Pfaffmann; Yamamoto & Asai), even if a branch of the vagus nerve was sectioned additionally (Jacquin, 1983; Vance, 1967).

The fact that neither a GL transection nor a CT transection causes a significant change in quinine responsiveness, but a combined transection does, seems to imply that the GL and CT carry redundant information that converges centrally. In other words, normal responding will be maintained if either the GL or CT is sectioned, provided the other remains intact. Using a similar behavioral methodology, Spector and colleagues (Spector, Travers, & Norgren, 1993) found that neither bilateral CT section nor NID cautery alone produced profound deficits in sucrose responsiveness, but combined treatment produced significant effects. Based on these data and electrophysiological findings (S. P. Travers & Norgren, 1991), they suggested that the respective sucrose-evoked signals from the NID and the anterior tongue taste receptors may converge centrally and contribute synergistically to sucrose responsiveness.

The convergence hypothesis does not appear to correspond in any simple way with anatomical and electrophysiological data pertaining to the GL and CT in intact rats. Transection of the GL denervates 64% of rat taste buds, compared with only 15% denervated by CT section. Furthermore, quinine is not a particularly potent stimulus for the CT, and those units that it does stimulate are also responsive to HCl and NaCl (Frank et al., 1983). In contrast, the GL contains relatively specific quinine responsive units, and quinine is a very effective stimulus for this nerve (Boudreau et al., 1987; Frank, 1991). The fact that the response properties of these two nerves markedly differ, along with the differential number of taste buds that they respectively innervate, is difficult to reconcile with the behavioral data that suggest that these nerves carry the same information. It is possible, however, that nerve damage provokes central reorganizational changes in the gustatory system (McGlathery & Whitehead, 1993) similar to those reported for the somatosensory system (see Kaas, Merzenich, & Killackey, 1983; Kaas, 1991).

Over and beyond the possibility of nerve damage–induced reorganizational effects, the resolution of this lack of correspondence between the anatomical and electrophysiological data and the behavioral data requires, in part, a careful evaluation of the behavioral methodology. First, in the present case, the response measure was the licking behavior of water-deprived rats to quinine in 10-s trials. The failure to find deficits after GL transection in this paradigm compared with the results of taste reactivity experiments in nondeprived rats (Grill et al., 1992; Travers et al., 1987) may reflect a differential contribution of the GL to appetitive and consummatory behavior, or it may reflect an interaction between this nerve and the physiological state of the animal. Previous investigators have found that deficits in preference or taste-guided licking behavior following cortical or parabrachial nucleus lesions may vary with deprivation state (Benjamin, 1955b; Spector, Grill, & Norgren, 1993). Second, considering the high motivation to rehydrate, the present technique may not be sensitive to changes at the lowest concentrations in the range employed. Techniques more sensitive to effects at low concentrations may reveal deficits in this range, which might occur if, for example, the narrowly tuned units in the GL were exceptionally sensitive to low quinine concentrations.

Third, the behavioral response measured in this experiment, which presumably reflects the affective judgment of the stimulus, was not substantially altered by GL section. However, it is possible that GL section alters the taste quality of quinine or raises the quinine detection threshold without altering its motivational properties at suprathreshold concentrations. The failure to find effects of GL section on behavioral responsive-

ness to quinine in brief-access and long-term preference tests may be analogous to a similar difficulty in finding effects of CT section on behavioral responsiveness to NaCl when such methods are used. Despite a population of Na-specific units in the rat CT (Frank et al., 1983), several investigators have failed to find profound effects on NaCl preference following CT section (Akaike et al., 1965; Grill et al., 1992; Pfaffmann, 1952; Richter, 1939; Yamamoto & Asai, 1986). The importance of the CT in guiding the animal's behavior toward NaCl became evident when the rat was forced to discriminate NaCl from KCl (Spector & Grill, 1992), when Na-depleted rats were forced to identify Na salts in an array of chloride salts (Breslin, Spector, & Grill, 1993), or when NaCl detection thresholds were measured in a shock-avoidance paradigm (Spector, Schwartz, & Grill, 1990). In the psychophysical procedures taste served as a signal for some other event (i.e., shock or punishment). Consequently, sensory function could be assessed independent of the hedonic characteristics of the taste stimulus.

Pfaffmann, Norgren, and Grill (1977) suggested that the hedonic and motivational properties of a taste stimulus may be processed by neural circuitry separate from that processing its sensory/discriminative features. If this is the case, inputs into these circuits may be dichotomized in the periphery. The narrowly tuned quinine units in the GL may be channeled into the sensory/discriminative circuit, and the more broadly tuned units of all the taste nerves may be channeled into the affective/motivational circuitry. The broadly tuned cells of the CT do seem to respond on an ingestive/aversive basis; that is, units that respond to quinine rarely respond to sucrose, and vice versa. The present experimental design does not distinguish between the sensory/discriminative and motivational properties of the stimulus. It will be important to test whether or not GL transection alone impairs the ability of the rat to discriminate quinine from other stimuli or raises detection thresholds when quinine serves as a signal for shock or reward to ascertain whether this dichotomous circuitry might underlie quinine sensitivity specifically and gustatory processing in general.

References

Akaike, N., Hiji, Y., & Yamada, K. (1965). Taste preference and aversion in rats following denervation of the chorda tympani and the IXth nerve. *Kumamoto Medical Journal, 18,* 108–109.

Bartoshuk, L. M. (1977). Water taste in mammals. In J. A. Weijnen & J. Mendelson (Eds.), *Drinking behavior* (pp. 317–339). New York: Plenum.

Benjamin, R. M. (1955a). Cortical taste mechanisms studied by two different test procedures. *Journal of Comparative and Physiological Psychology, 48,* 119–122.

Benjamin, R. M. (1955b). The effect of fluid deprivation on taste deficits following cortical lesions. *Journal of Comparative and Physiological Psychology, 48,* 502–505.

Boudreau, J. C., Do, L. T., Sivakumar, L., Oravec, J., & Rodriguez, C. A. (1987). Taste systems of the petrosal ganglion of the rat glossopharyngeal nerve. *Chemical Senses, 12,* 437–458.

Breslin, P. A. S., Spector, A. C., & Grill, H. J. (1993). Chorda tympani section decreases the cation specificity of depletion-induced sodium appetite in rats. *American Journal of Physiology, 264,* R319–R323.

Brosvic, G. M., & Hoey, N. E. (1990). Taste detection and discrimination performance of rats following selective desalivation. *Physiology & Behavior, 48,* 617–623.

Cano, J., & Rodriguez-Echandia, E. L. (1980). Degenerating taste buds in sialectomized rats. *Acta Anatomica, 106,* 487–492.

Catalanotto, F. A., & Sweeney, E. A. (1972). The effects of surgical desalivation of the rat upon taste acuity. *Archives of Oral Biology, 17,* 1455–1465.

Catalanotto, F. A., & Sweeney, E. A. (1973). Long-term effects of selective desalivation on taste acuity in rats. *Archives of Oral Biology, 18,* 941–952.

Cleaton-Jones, P. (1976). A denervation study of taste buds in the soft palate of the albino rat. *Archives of Oral Biology, 21,* 79–82.

Davis, J. D. (1989). The microstructure of ingestive behavior. In L. H. Schneider, S. J. Cooper, & K. A. Hami (Eds.), *Annals of the New York Academy of Sciences: Vol. 575. The psychobiology of human eating disorders: Preclinical and clinical perspectives* (pp. 106–121). New York: New York Academy of Sciences.

Dickman, J. D., & Smith, D. V. (1988). Response properties in the hamster superior laryngeal nerve. *Brain Research, 450,* 25–38.

Frank, M., Contreras, R. J., & Hettinger, T. P. (1983). Nerve fibers sensitive to ionic taste stimuli in chorda tympani of the rat. *Journal of Neurophysiology, 50,* 941–960.

Frank, M. E. (1991). Taste-responsive neurons of the glossopharyngeal nerve of the rat. *Journal of Neurophysiology, 65,* 1452–1463.

Ganchrow, J. R., & Ganchrow, D. (1989). Long-term effects of gustatory neurectomy on fungiform papillae in the young rat. *Anatomical Record, 225,* 224–231.

Garcia, J., & Hankins, W. G. (1975). The evolution of bitter and the acquisition of toxiphobia. In D. A. Denton & J. P. Coghlan (Eds.), *Olfaction & taste V* (pp. 39–45). San Diego, CA: Academic Press.

Grill, H. J., & Schwartz, G. J. (1992). The contribution of gustatory nerve input to oral motor behavior and fluid intake-based preference: II. Effects of combined chorda tympani and glossopharyngeal nerve section in the rat. *Brain Research, 573,* 105–113.

Grill, H. J., Schwartz, G. J., & Travers, J. B. (1992). The contribution of gustatory nerve input to oral-motor behavior and intake-based preference: I. Effects of chorda tympani or glossopharyngeal nerve section in the rat. *Brain Research, 573,* 95–104.

Grill, H. J., Spector, A. C., Schwartz, G. J., Kaplan, J. M., & Flynn, F. W. (1987). Evaluating taste effects on ingestive behavior. In F. M. Toates & N. E. Rowland (Eds.), *Feeding and drinking* (pp. 151–188). New York: Elsevier Science.

Guth, L. (1957). The effects of glossopharyngeal nerve transection on the circumvallate papilla of the rat. *Anatomical Record, 128,* 716–731.

Jacquin, M. F. (1983). Gustation and ingestive behavior in the rat. *Behavioral Neuroscience, 97,* 98–109.

Kaas, J. H. (1991). Plasticity of sensory and motor maps in adult mammals. *Annual Review of Neuroscience, 14,* 137–167.

Kaas, J. H., Merzenich, M. M., & Killackey, H. P. (1983). The reorganization of somatosensory cortex following peripheral nerve damage in adult and developing mammals. *Annual Review of Neuroscience, 6,* 325–356.

Koh, S. D., & Teitelbaum, P. (1961). Absolute behavioral taste thresholds in the rat. *Journal of Comparative and Physiological Psychology, 54,* 223–229.

Krimm, R. F., Nejad, M. S., Smith, J. C., Miller, I. J., & Beidler, L. M. (1987). The effect of bilateral sectioning of the chorda tympani and the greater superficial petrosal nerves on the sweet taste in the rat. *Physiology & Behavior, 41,* 495–501.

McGlathery, S. T., & Whitehead, M. C. (1993). Transganglionic degeneration in the gustatory system. *Society for Neuroscience Abstracts, 18,* 1041.

Miller, I. J. (1977). Gustatory receptors of the palate. In Y. Katsuki, M.

Sato, S. Takagi, & Y. Oomura (Eds.), *Food intake and chemical senses* (pp. 173–186). Tokyo: University of Tokyo Press.

Nanda, R., & Catalanotto, F. A. (1981). Long-term effects of surgical desalivation upon taste acuity, fluid intake, and taste buds in the rat. *Journal of Dental Research, 60,* 69–76.

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.

Ogawa, H., Sato, M., & Yamashita, S. (1968). Multiple sensitivity of chorda tympani fibers of the rat and hamster to gustatory and thermal stimuli. *Journal of Physiology, 199,* 223–240.

Pfaffmann, C. (1952). Taste preference and aversion following lingual denervation. *Journal of Comparative and Physiological Psychology, 45,* 393–400.

Pfaffmann, C. (1955). Gustatory nerve impulses in rat, cat, and rabbit. *Journal of Neurophysiology, 18,* 429–440.

Pfaffmann, C., Norgren, R., & Grill, H. J. (1977). Sensory affect and motivation. In B. M. Wenzel & H. P. Zeigler (Eds.), *Annals of the New York Academy of Sciences: Vol. 290. Tonic functions of sensory systems* (pp. 18–34). New York: New York Academy of Sciences.

Richter, C. P. (1939). Transmission of taste sensation in animals. *Transactions of the American Neurological Association, 65,* 49–50.

Spector, A. C. (1992). Parabrachial gustatory lesions impair taste-guided quinine avoidance in rats. *Society for Neuroscience Abstracts, 18,* 1040.

Spector, A. C., Andrews-Labenski, J., & Letterio, F. C. (1990). A new gustometer for psychophysical taste testing in the rat. *Physiology & Behavior, 47,* 795–803.

Spector, A. C., & Grill, H. J. (1992). Salt taste discrimination after bilateral section of the chorda tympani or glossopharyngeal nerves. *American Journal of Physiology, 263,* R169–R176.

Spector, A. C., Grill, H. J., & Norgren, R. (1993). Concentration-dependent licking of sucrose and sodium chloride in rats with parabrachial gustatory lesions. *Physiology & Behavior, 53,* 277–283.

Spector, A. C., Schwartz, G. J., & Grill, H. J. (1990). Chemospecific deficits in taste detection after selective gustatory deafferentation in rats. *American Journal of Physiology, 258,* R820–R826.

Spector, A. C., Travers, S. P., & Norgren, R. (1993). Taste receptors on the anterior tongue and nasoincisor ducts of rats contribute synergistically to behavioral responses to sucrose. *Behavioral Neuroscience, 107,* 694–702.

St. John, S., Garcea, M., & Spector, A. C. (1993). The effects of gustatory denervation on quinine responsiveness in rats [Abstract]. *Chemical Senses 118,* 635.

Travers, J. B., Grill, H. J., & Norgren, R. (1987). The effects of glossopharyngeal and chorda tympani nerve cuts on the ingestion and rejection of sapid stimuli: An electromyographic analysis in the rat. *Behavioural Brain Research, 25,* 233–246.

Travers, S. P., & Nicklas, K. (1990). Taste bud distribution in the rat pharynx and larynx. *Anatomical Record, 227,* 373–379.

Travers, S. P., & Norgren, R. (1991). Coding the sweet taste in the nucleus of the solitary tract: Differential roles for the anterior tongue and nasoincisor duct gustatory receptors in the rat. *Journal of Neurophysiology, 65,* 1372–1380.

Vance, W. B. (1967). Hypogeusia and taste preference behavior in the rat. *Life Sciences, 6,* 743–748.

Whitehead, M. C., Frank, M. E., Hettinger, T. P., Hou, L. T., & Nah, H. D. (1987). Persistence of taste buds in denervated fungiform papillae. *Brain Research, 405,* 192–195.

Yamamoto, T., & Asai, K. (1986). Effects of gustatory deafferentation on ingestion of taste solutions as seen by licking behavior in rats. *Physiology & Behavior, 37,* 299–305.

Young, J. A., & Van Lennep, E. W. (1978). *The morphology of the salivary glands.* San Diego, CA: Academic Press.

Received July 14, 1993
Revision received September 1, 1993
Accepted September 1, 1993 ■