Glossopharyngeal nerve transection does not compromise the specificity of taste-guided sodium appetite in rats

STACY MARKISON, STEVEN J. ST. JOHN, AND ALAN C. SPECTOR
Department of Psychology, University of Florida, Gainesville, Florida 32611–2250

Markison, Stacy, Steven J. St. John, and Alan C. Spector. Glossopharyngeal nerve transection does not compromise the specificity of taste-guided sodium appetite in rats. *Am. J. Physiol.* 269 (*Regulatory Integrative Comp. Physiol.* 38): R215–R221, 1995.—The chorda tympani nerve (CT) has been shown to be critical in the sodium-specific drinking behavior of sodium-depleted rats, but the role of other gustatory nerves and the contribution of the major salivary glands remain to be elucidated. In this study, rats received either bilateral section of the CT (CTX) or the glossopharyngeal nerve (GLX), extirpation of the sublingual and submaxillary salivary glands (DSAL), or sham surgery. After recovery, rats were sodium depleted with furosemide and tested for their licking responses to 0.05 and 0.3 M NaCl, KCl, $CaCl_2$, and NH_4Cl, as well as distilled water in an automated gustometer. Rats that received GLX maintained a specific sodium appetite comparable to controls despite denervation of ~64% of the taste buds. In contrast, compared with control rats, CTX and DSAL rats had altered response profiles, showing much smaller differences in licking to NaCl relative to the other stimuli. This was accompanied by a substantially lower lick rate in DSAL rats, raising the possibility that general licking impairments contributed to the decreased NaCl responsiveness in these rats. These findings imply that the CT, but not the glossopharyngeal nerve, is necessary for the maintenance of normal sodium-specific, taste-guided behavior under sodium deplete conditions.

chorda tympani; sublingual and submaxillary salivary glands; sodium depletion; furosemide; gustatory nerves; salt

WHEN RATS ARE DEPLETED of sodium by adrenalectomy, subcutaneous Formalin injection, dietary sodium restriction, or acute treatment with a natriuretic, intake of concentrated sodium chloride (NaCl) solutions that are normally avoided in the sodium-replete state is increased (e.g., 19, 35). This appetite occurs the first time a rat experiences sodium depletion (15, 35). Sodium-depleted rats ingest sodium salt solutions in preference to other salts, such as potassium chloride (KCl) and ammonium chloride (NH_4Cl; see Ref. 21). This behavior has been shown to occur within the first minute of a session before postingestive events can have an influence (15). Collectively, experimental findings suggest that sodium appetite is unlearned, taste guided, and highly specific to sodium (see Ref. 35).

Breslin et al. (3) showed that normal taste-guided, sodium-specific licking behavior is dependent on the chorda tympani nerve (CT). Intact sodium-depleted rats, presented with brief trials (10 s) of 0.05 and 0.3 M salt solutions (NaCl, KCl, NH_4Cl, $CaCl_2$) and water, licked significantly more NaCl than the other solutions. In contrast, rats with bilateral CT section decreased responding to NaCl compared with intact rats and increased responding to KCl and NH_4Cl. Hence in CT-sectioned rats, the sodium appetite, although present, was blunted and less specific. These results have recently been replicated in Wistar and Fischer 344 rats (4).

Other behavioral evidence also supports the role of the CT in salt-related behaviors. When the rat CT was bilaterally sectioned, the detection threshold for NaCl was increased, in some cases by as much as two orders of magnitude, whereas the detection threshold for sucrose was relatively unaffected (22, 29). Bilateral CT section also has been shown to severely impair performance on a NaCl vs. KCl discrimination task, but glossopharyngeal nerve (GL) transection was without effect (27). Recently, O'Keefe et al. (16) found that CT-sectioned rats on a sodium-deficient diet showed a loss of sensitivity to the lowest hypotonic solutions (0.03 and 0.06 M NaCl) presented in 30-s trials.

These behavioral findings are consistent with the electrophysiological profile of the CT. Frank et al. (9) found that most taste-responsive fibers in the CT are stimulated by NaCl. One fiber type (N units), accounting for about one-half of the taste fibers reported, was narrowly tuned to respond to sodium and lithium salts. A second fiber type (H units), accounting for roughly the other one-half of fibers reported, responded well to NaCl but best to HCl. Boudreau et al. (2), recording from geniculate ganglion cells (ganglion of the CT), found results comparable with the single-fiber electrophysiology. Thus these fibers that respond specifically to NaCl are removed when the CT is transected. It follows that behaviors dependent on the input from such sodium-responsive fibers would also be disrupted.

This conclusion, however, must be tempered by results indicating that rats with CT section, under certain circumstances, can use the remaining portion of the gustatory signal or other cues to guide their behavior. For example, CT section did not alter NaCl preference in 24-h tests (10, 17, 20) or unconditioned licking to NaCl in water-deprived rats in 10-s trials (7). In the study by Breslin et al. (3), sodium-specific licking behavior was compromised in CT-sectioned rats, although it was not completely eliminated. The CT-sectioned rats responded more to NaCl than to water and did not substantially alter their responsiveness to some of the nonsodium salts. In the absence of data derived from sodium-replete rats, it is difficult to say whether the maintained sodium preference was driven by the sodium-depleted state. In any event, it is clear that CT-sectioned rats can discriminate NaCl from some nonsodium salts and water at the concentrations employed by Breslin et al.(3).

Whether the reduction in NaCl responsiveness found by Breslin et al. (3) is specific to CT transection or whether any manipulation in the peripheral gustatory system might also cause this effect is not known.

Therefore, it is important to examine the effect of transecting a different gustatory nerve. The CT innervates only ~15% of the total taste buds in the rat. The GL innervates the greatest proportion (~64%) of rat taste buds (see Ref. 14). Obviously, if the effects found by Breslin et al. (3) were caused by any substantial denervation of taste buds, then transection of the GL would also be expected to cause these deficits. In addition, electrophysiological evidence indicates that the GL may play some role in NaCl-related behaviors. Although the GL does not contain the narrowly tuned N units like those of the CT, it does contain fibers that respond to NaCl, nonsodium salts, and acids (8).

Accordingly, the present study was conducted to test whether the GL is also necessary for the normal specificity characteristic of sodium appetite in the rat. Using the design of Breslin et al. (3), we studied the effects of GL section (as well as CT section) and partial desalivation on sodium appetite. The sublingual and submaxillary salivary glands are of interest because, in addition to the CT's innervation of the anterior tongue, it also partially innervates these salivary glands (eg., Ref. 12). Thus the effect of CT section on sodium-specific licking behavior could be due in part to the loss of the parasympathetic efferent supply to these glands.

METHODS

Subjects

Thirty-three naive male Sprague-Dawley (Charles River) rats were used in this study. Rats were ~3 mo of age at the start of the experiment and weighed between 305 and 436 g. All rats were housed in individual wire mesh cages in a room where temperature and lighting (12:12-h light-dark) were automatically controlled. All manipulations were performed during the light phase. Rats had free access to food (Purina 5001) and distilled water, except where otherwise stated.

Apparatus

The apparatus used has been described elsewhere (26). Briefly, the apparatus consisted of 12 fluid reservoirs that were filled with various solutions. The reservoirs were connected to small solenoid valves that deposited 5 μl of solution per lick into a drinking spout. The spout was situated behind a 1-cm slit in a side wall of the testing chamber. A computer recorded a lick when spout contact by the rat completed an electrical circuit that passed <50 nA through the animal.

Presurgical Training and Testing

Rats were on a restricted water schedule during the training sessions so that the only time they had access to fluid was in the gustometer. On *days 1* and *2*, the rats were trained to lick the drinking spout for distilled water (30-min sessions). On *days 3* and *4*, the session duration was increased to 40 min, and the rat was required to lick the spout twice within 500 ms to receive 10-s access to distilled water and 0.1 M sucrose presented in randomized blocks of two. After a trial the spout rotated away from the rat and was placed over a drainage funnel where it was rinsed with distilled water, evacuated with pressurized air, and then repositioned. This cleaning procedure took ~6 s. Sucrose was included during the last 2 days of training to promote stimulus sampling.

Surgery

After testing on *day 4*, rats again had distilled water on their home cage. On *days 6* and *7*, surgery was performed. All surgeries, except for the partial desalivations, were performed with the aid of an operating microscope. Rats were deeply anesthetized (86 mg/kg ketamine hydrochloride, 13 mg/kg xylazine hydrochloride, ip) and injected with Bicillin (30,000 units, im) as a prophylactic measure. Supplemental anesthesia was delivered as necessary. The incision area was then shaved and cleaned with Betadine. The rats were placed in a head holder with a specialized mouth bar.

For the CT transection (CTX), the head and body of the rat were positioned 80° away from the surgeon. The external auditory meatus was widened using five blunted, curved 25-gauge hypodermic needles. The tympanic membrane was removed, the nerve was avulsed using no. 7 microforceps, and the auditory ossicles were extracted. Two CTX rats died shortly after this surgery, reducing the sample size to $n = 7$.

For the GL transection (GLX), the rats were positioned supine in the head holder. An incision was made on the midline of the ventral neck. Blunted, curved hypodermic needles were used to retract the sublingual and submaxillary salivary glands, the sternohyoideus, the omohyoideus, and the posterior digastricus muscles, thus exposing the hypoglossal nerve. The surrounding fascia was gently peeled away to reveal the GL close to the medial external wall of the tympanic bulla. The GL was grasped with microforceps, and a 2- to 3-mm section was removed using microscissors. The retractors were removed and the incision was closed with 4–0 silk suture. One GLX rat died shortly after this surgery, reducing the sample size to $n = 6$.

The surgical procedure to remove the sublingual and submaxilary salivary glands (DSAL) was similar to the GLX procedure. Fascia was peeled away to expose the sublingual-submaxillary salivary gland complex. The complex was gripped with forceps, the blood supply and duct were ligated with 4–0 silk suture, and the salivary glands were then extirpated. The incision was closed with 4–0 silk suture. Two DSAL rats died shortly after this surgery, reducing the sample size to $n = 7$.

For the control rats (Con), the tympanic membrane was exposed in the same manner as the CTX rats; however, it was only punctured. The rat was then repositioned and the GL was exposed, as described above, but not disturbed. One Con rat died shortly after surgery, reducing the sample size to $n = 7$. All surgical procedures were performed bilaterally. Seven to eight days were allowed for recovery before the rats were depleted of sodium and again tested in the gustometer.

Sodium Depletion

On *day 13*, the rats were given a sodium-deficient diet (Teklad 170905) in addition to their regular chow so that food-related neophobia would be minimized during the sodium-depletion procedure. On *day 14*, the rats were injected with 30 mg/kg of the diuretic/natriuretic furosemide (Lasix) administered in two equal doses, 2 h apart. The first injection was given 24 h before testing. At the time of the first injection, rats were placed in clean metabolism cages and given ad libitum access to sodium-deficient diet and distilled water (normal laboratory chow was removed). Urine was collected over the next 24 h. The concentration of sodium and potassium excreted was determined with the aid of a flame photometer (Ciba Corning 480), and total sodium and potassium lost during this 24-h period was computed.

Postsurgical Testing

On *day 15*, the rats were tested in the gustometer for a 40-min session in which distilled water and 0.05 and 0.3 M concentrations of NaCl, KCl, NH_4Cl, and $CaCl_2$ were presented in randomized blocks of nine, with one exception: 0.3 M NaCl was always presented on the first trial of the session to alert the rats to the presence of NaCl and promote further sampling from the drinking spout. All solutions were made daily from biological grade chemicals (Fisher) and distilled water. The number of licks to each stimulus was recorded. At the end of the session, the rats were returned to their home cages and their regular sodium-sufficient chow was returned.

Water was removed from the home cage on *day 16*. The next day, the water-deprived rats were placed in the gustometer for the postsurgical licking assessment. During this 40-min session, they were randomly presented with distilled water and 0.1 M sucrose, as they were on *day 4*, and the average number of licks in a 10-s trial for each tastant was determined. Performance on *day 16* was compared with that on *day 4* to estimate each rat's maximum licking rate before and after surgery.

Histology

One to two days after the postsurgical licking assessment, the rats were deeply anesthetized with pentobarbital sodium (130 mg/kg) and perfused transcardially with isotonic saline followed by 10% buffered Formalin. The tongues were removed and stored in 10% buffered Formalin for later histological examination. It has previously been shown that after gustatory nerve transection the taste receptors on the tongue degenerate (e.g., Refs. 11, 14). Therefore, to assess the efficacy of the CTX, fungiform papillae and taste pores on the anterior tongue were counted. Taste buds and taste pores also were examined in the circumvallate papilla to determine the completeness of the GLX.

The anterior portion of the tongue was examined from the intermolar eminence to the tip on both the dorsal and ventral sides. The anterior tongues were soaked in distilled water for at least 30 min, dipped in 0.5% methylene blue, rinsed in distilled water, and cut on the midline. For each half, the lingual muscle was removed, the epithelium was flattened between two glass slides, and the tissue was immediately viewed under a microscope. The total number of fungiform papillae and taste pores were counted for each rat by an observer who was unaware of the surgical treatment and the behavioral results of each animal.

The circumvallate papillae for the GLX, Con, and DSAL rats were embedded in paraffin, cut in 10-μm slices, mounted on glass slides, and stained with hematoxylin and eosin. Because successful bilateral GLX results in a complete absence of taste buds in the circumvallate papilla (11), only the presence or absence of taste buds in the circumvallate papilla was noted.

Data Analysis

Sodium depletion. The total amounts of sodium and potassium lost were computed as a function of the concentration in the urine and the volume of urine excreted. Because the sodium-deficient chow contained ample potassium and some food may have spilled into the collection flask, the urinary potassium loss was most likely overestimated. To compute potassium state, the amount of potassium in the urine was subtracted from the amount of potassium ingested in the chow. A one-way analysis of variance (ANOVA) was used to determine differences between groups.

Histology. The number of taste pores on the anterior tongue was compared between groups using a one-way ANOVA and Dunnett's post hoc comparisons. No analyses were conducted with the number of taste buds in the circumvallate papillae because there were none in the GLX group.

Licking assessment. For each rat, the highest lick rate to either stimulus (water or sucrose) before (*day 4*) and after (*day 17*) surgery was compared using a two (before vs. after surgery) by four (group) ANOVA to assess any nonspecific effects of surgery on lick rate.

Salt responsiveness during sodium depletion. The total number of licks per 10-s trial was averaged over the testing session for each stimulus. Rats were required to have more than four trials to each stimulus during the testing session to be included in the data analysis. This somewhat arbitrary criterion was employed to ensure that the statistical means were a reliable representation of the actual behavioral phenomenon. Two rats (one each from the CTX and DSAL groups) were excluded because they did not meet this criterion. Their response profiles were, nonetheless, characteristic of their respective groups.

A one-way ANOVA for the effect of stimulus was conducted for each group. If the ANOVA revealed significant differences in lick rate among stimuli, then Bonferroni-adjusted paired comparisons were performed between water, 0.05 M NaCl, and 0.3 M NaCl with all solutions. The corresponding Bonferroni-adjusted α level was 0.002381 (0.05 divided by 21). In addition, nine one-way ANOVAs were conducted to compare the response to each stimulus between groups. If there was a significant main effect of group, then Dunnett's two-sided post hoc comparisons were computed to determine which groups were different from the Con group.

RESULTS

Sodium Depletion

The total amount of sodium excreted in the 24 h after administration of the natriuretic did not significantly differ between groups [Con = 2.7 ± 0.15 mmol, CTX = 2.4 ± 0.23 mmol, GLX = 3.1 ± 0.15 mmol, DSAL = 2.7 ± 0.15 mmol; $F(3,20) = 2.38$, $P = 0.1$]. Because the diet had negligible sodium, all of the rats were in a negative sodium state by the time taste testing began.

Furosemide causes the excretion of both sodium and potassium. Potassium balance, however, was restored by the presence of potassium in the diet. Despite the fact that urinary potassium excretion was most likely overestimated, all of the rats were in a positive potassium state; there were no significant differences between the four groups [$F(3,20) = 1.86$, $P = 0.168$].

Histology

The rats in the CTX group each had less than 12 anterior tongue taste pores compared with a mean of 135 (± 13) in the Con group. The persistence of a few intact taste pores after CTX has been previously reported (e.g., Refs. 3, 27, 31, 34). There was a main effect of group [$F(3,21) = 94.08$, $P < 0.0001$], and a post hoc comparison indicated that only the CTX group had significantly fewer taste pores ($P < 0.05$). None of the other groups differed from the Con group. No rat in the GLX group had any taste buds in the circumvallate papillae, confirming the efficacy of the nerve transections.

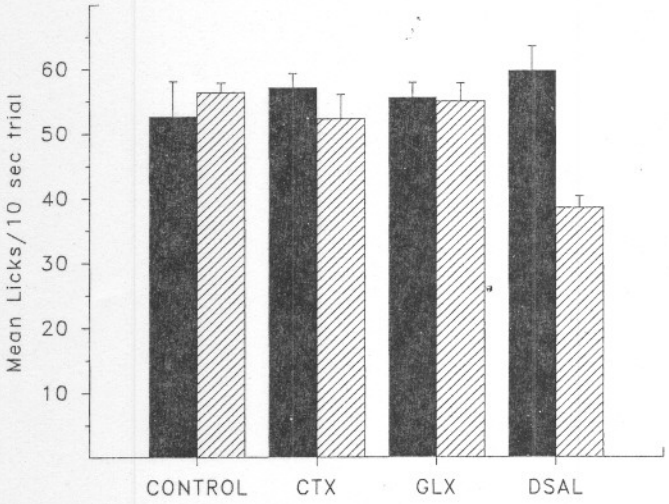

Fig. 1. Mean (±SE) licks in 10-s trials to most preferred stimulus (0.1 M sucrose or distilled water) measured during the presurgical (solid bars) and postsurgical (hatched bars) licking assessment. CTX, chorda tympani nerve section; GLX, glossopharyngeal nerve section; DSAL, sublingual and submaxillary salivary glands extirpation.

Licking Assessment

Assessment of the maximum lick rates for the water-deprived DSAL rats indicated that the surgery induced a general licking impairment (Fig. 1). A two (before vs. after) by four (group) ANOVA indicated a significant main effect of time [$F(1,21) = 6.17$, $P = 0.02$] and a significant interaction [$F(3,21) = 6.39$, $P = 0.003$]. Simple effects revealed that only the DSAL group had significantly lower licking rates after surgery ($P < 0.05$). Accordingly, salt responsiveness in the DSAL group must be considered with caution.

Salt Responsiveness During Sodium Depletion

The profiles of salt responsiveness were sharp and specific to NaCl for the Con and GLX groups. In contrast, the profiles for the CTX and DSAL groups were more broad (Fig. 2). Separate one-way ANOVAs revealed significant differences between stimuli within each group (Table 1.)

The Con group licked 0.05 M NaCl significantly more than all other stimuli (all P values < 0.0012) except 0.3 M NaCl. This group also licked more 0.3 M NaCl than all other stimuli (all P values < 0.0006) and licked more water than 0.05 M $CaCl_2$ ($P < 0.0017$). Paired comparisons within the GLX group were statistically similar to the Con group (all P values < 0.0008), except that the GLX group did not lick significantly more water than 0.05 M $CaCl_2$ ($P = 0.015$). In contrast, the CTX group and the DSAL group showed few significant differences, indicating broadened salt responsiveness profiles. The CTX group licked 0.3 M NaCl significantly more than 0.05 and 0.3 M $CaCl_2$ (all P values < 0.0016). The DSAL group licked 0.05 M NaCl significantly more than 0.05 and 0.3 M $CaCl_2$ (all P values < 0.0015). Mean licks to water did not significantly differ from that to NaCl in either the CTX or DSAL group.

As expected, differences between groups occurred primarily for NaCl, with the DSAL and CTX groups showing lower responsiveness than the Con group (Table 2). The CTX group had significantly fewer licks than the Con group for 0.05 M NaCl ($P < 0.05$), although the reduction in licks to 0.3 M NaCl did not meet the 0.05 criterion for significance. The DSAL group had significantly fewer licks than the Con group for 0.05 M NaCl, 0.3 M NaCl, and 0.05 M $CaCl_2$ (all P values < 0.05). The DSAL group also had a significantly lower lick rate to water than the Con group ($P < 0.05$), indicating that desalivation may have a general effect on licking rate. None of the nine comparisons revealed any significant differences between the Con and GLX groups.

DISCUSSION

It is clear that an intact GL is not necessary for the rat to demonstrate specificity in its taste-based behavioral responsiveness to NaCl when challenged by acute sodium depletion. In striking contrast, the typical robust and selective licking of NaCl that is characteristic of the rat's sodium appetite depends on an intact CT.

In this study, CTX disrupted the specificity of sodium appetite. Responsiveness to NaCl in CTX rats was not different from that of water, KCl, or NH_4Cl. Breslin et al. (3) found that the CTX rats appeared to retain partial competence with regard to the specificity of depletion-induced sodium appetite. In that study, CTX rats maintained a preference for NaCl compared with water, NH_4Cl, and $CaCl_2$ but did not prefer NaCl to 0.05 M KCl. It should be noted, however, that in the Breslin et al. (3) study, CTX rats discriminated NaCl from water and some nonsodium salts, but licking to NaCl was substantially decreased relative to controls. Thus section of the CT caused a broadened response profile. Although our study employed virtually an identical design, our results indicated a more profound degree of impairment after CTX in that CTX rats did not statistically prefer NaCl to water. Nevertheless, the shapes of the salt response profiles for the CTX rats from the two studies are very similar.

The fact that the GL and CT can be functionally distinguished by this test of salt responsiveness in a sodium-deplete state reveals some fundamental properties concerning the organization of the gustatory system. First, the magnitude and specificity of unconditioned responsiveness to NaCl in sodium-depleted rats are not simply related to the total number of taste buds present. If this were true, then GLX should have caused deficits much greater than CTX by virtue of the fact that the GL innervates four times as many taste buds as does the CT. This does not, however, rule out that the number of taste buds located in a particular receptor field, such as the anterior tongue, might bear some relationship to salt discriminability. In fact, there is evidence that within the anterior tongue receptor field, taste bud number does relate to salt discriminability (28, 32). The relatively small sample size of the control group in this study precluded a comprehensive test of this hypothesis.

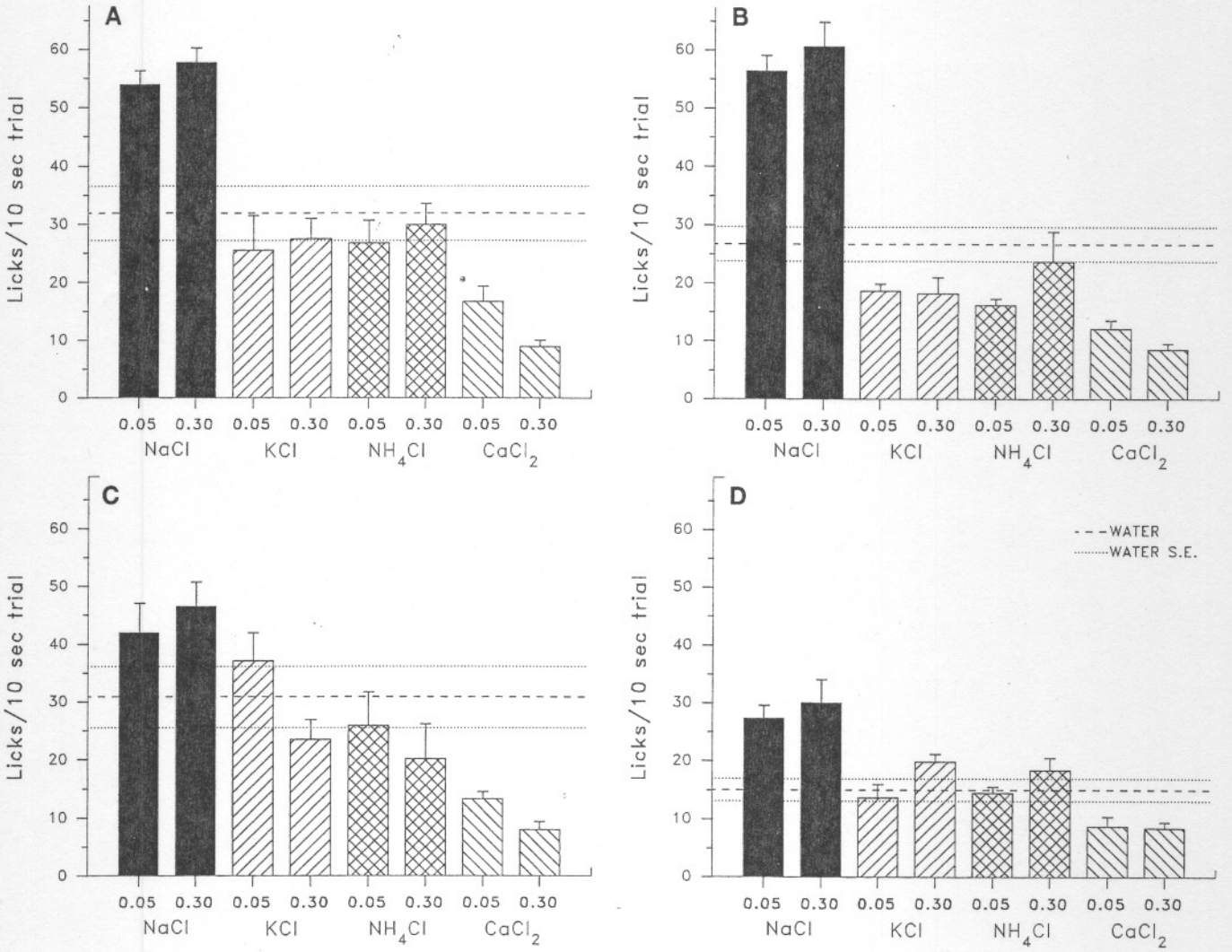

Fig. 2. Mean (±SE) licks taken during 10-s trials to distilled water (dashed line ± dotted lines) and 0.05 and 0.3 M sodium chloride (NaCl, solid bars), potassium chloride (KCl, left-hatched bars), ammonium chloride (NH_4Cl, crosshatched bars), and calcium chloride ($CaCl_2$, right-hatched bars) when rats were sodium depleted. Individual graphs are presented for sham-operated control (*A*, $n = 7$), GLX (*B*, $n = 6$), CTX, (*C*, $n = 6$), and sublingual and submaxillary desalivation (*D*, $n = 6$) groups.

Second, these findings suggest that the taste responsive fibers of the GL, unlike those of the CT, do not provide unique information that enables distinctions of NaCl from other salt stimuli in sodium-depleted rats. In other words, the taste input from the GL is either irrelevant with regard to this behavioral task or it is redundant to the signals contained in other gustatory nerves. Evidence supporting the former comes from electrophysiological studies that demonstrate that the CT contains a subpopulation of fibers that are narrowly tuned to respond to sodium; such fibers do not appear to be present in the GL (8, 9).

Table 1. *Statistical results comparing licks taken to each taste stimulus*

Group	*F* Value	*P* Value
Con	$F(8,48) = 33.12$	< 0.001
CTX	$F(8,40) = 12.44$	< 0.001
GLX	$F(8,40) = 46.62$	< 0.001
DSAL	$F(8,40) = 12.64$	< 0.001

Con, control; CTX, chorda tympani nerve section; GLX, glossopharyngeal nerve section; DSAL, sublingual and submaxillary salivary glands extirpation.

Table 2. *Between groups statistical results comparing licks to each stimulus*

Stimulus	*F* Value	*P* Value
Distilled water	$F(3,21) = 3.64$	$= 0.03$
0.05 M NaCl	$F(3,21) = 16.07$	$= 0.001$
0.3 M NaCl	$F(3,21) = 13.21$	$= 0.001$
0.05 M KCl	$F(3,21) = 5.39$	$= 0.007$
0.3 M KCl	$F(3,21) = 2.03$	$= 0.14$
0.05 NH_4Cl	$F(3,21) = 2.96$	$= 0.06$
0.3 NH_4Cl	$F(3,21) = 1.39$	$= 0.27$
0.05 M $CaCl_2$	$F(3,21) = 3.07$	$= 0.05$
0.3 M $CaCl_2$	$F(3,21) = 0.11$	$= 0.96$

The rats had their sublingual and submaxillary salivary glands extirpated had a profile of salt responsiveness that mimicked that observed after CTX. There are at least two possible explanations for this effect. First, partial desalivation could have caused a change in taste sensibility. It is possible that the DSAL rats exhibited decreased ability to discriminate between the different salt solutions when depleted of sodium. In support of this, Catalanotto and Sweeney (6) demonstrated that when the sublingual and submaxillary salivary glands are removed, rats exhibit a more flattened NaCl preference-aversion function as measured by long-term, two-bottle (NaCl vs. water) tests. In addition, Cauthon et al. (7) found that water-deprived desalivated rats increased their lick rate to midrange NaCl concentrations after surgery. Thus it is possible that the altered salt responsiveness in sodium-depleted CTX rats was caused by depriving the sublingual-submaxillary gland complex of their parasympathetic efferent supply.

Several points, however, challenge this conclusion. First, CTX does not completely denervate the sublingual and submaxillary salivary glands (eg., Ref. 12); thus extirpation is more extreme compared with CTX. Second, although Brosvic and Hoey (5) showed that partially desalivated Long-Evans rats decreased their preference for NaCl compared with controls, detectability and discriminability of NaCl were not affected. Third, evidence suggests that DSAL and CTX do not produce the same effects. Sollars and Bernstein (24) found that removal of the sublingual and submaxillary salivary glands did not change salt preference in Fischer 344 rats, whereas CTX did. Additionally, desalivation does not alter detection thresholds for NaCl (5), whereas CTX does (22, 29).

The second possible explanation for the effect of partial desalivation is that it caused a motor or mechanical impairment. Desalivated rats in this study significantly decreased licking to sucrose and water when motivated by water deprivation (Fig. 1). It is noteworthy that changes in licking behavior have been observed after interference with the salivary glands in rats. Wong and Kraintz (36) reported that the interlick interval was longer in rats with their salivary ducts ligated compared with control rats. Experiments from our laboratory have also revealed decreases in local lick rate (rate of liking within bursts) in water-deprived rats after desalivation (18, 31). Possibly a lack of lubrication caused by the removal of the glands underlies these impairments, but it seems implausible that this would appreciably affect the ingestion of fluid. In general, it is unlikely that surgery in and of itself produces the impairment. With respect to the location and size of the incision, amount of fascia dissected, and sutures remaining, the GLX and Con surgeries were very similar to the DSAL surgery, yet there were no impairments in these other groups. In addition, the DSAL rats appeared to be just as healthy as the other groups as measured by percentage of presurgical body weight 7–8 days after surgery; an ANOVA indicated no significant differences between any of the groups [$F(3,24) = 1.86$, $P = 0.31$]. Damage to the CT as a result of the DSAL surgery might have occurred; this disturbance, however, would likely result in a reduced number of taste pores on the anterior tongue, which was not observed. In the absence of relevant data, it is difficult to speculate further on what the mechanism of these alterations in general licking behavior would be.

The question still remains as to what roles each of the taste nerves play in channeling gustatory information to the brain. Growing evidence indicates that the effects of gustatory nerve transection depend heavily on the task used to assess taste function. For example, large impairments after CTX have been demonstrated in the discrimination of NaCl from KCl (27, 32); the detectability of NaCl (22, 29); oral motor ingestive responses to NaCl, magnesium chloride, and quinine (10); cation-specific ingestion of NaCl in sodium-depleted rats (3, 4); the unconditioned aversion to NaCl in Fischer 344 rats (23); and the expression of a presurgically conditioned taste aversion to NaCl and monosodium glutamate in hamsters (1, 37). In contrast, 24-h two-bottle preference for NaCl and sucrose (10, 23); 30-min single-bottle intake of sucrose and polycose (33); sucrose detection threshold (29); taste-guided discrimination of sucrose from quinine (27); unconditioned licking during brief exposures of NaCl (7), quinine (31), sucrose (25, 30), or maltose (18); and oral motor-taste reactivity to sucrose (10) are relatively unaffected by CTX.

The functional role of the GL in taste-guided behavior remains in large part to be elucidated. Although recent studies have revealed that CTX results in profound performance deficits in certain taste-related tasks involving NaCl, GLX appears to have much less severe consequences, regardless of the taste stimulus used. For example, GLX has no effect on unconditioned licking during brief access trials with sucrose (30), maltose (18), NaCl (7), or quinine (31); quinine preference in a 24-h two-bottle test (10); 30-min intake of sucrose in a single-bottle test (33); a presurgically conditioned salt-discrimination task (27); or, as demonstrated by this study, the expression of a specific taste-guided sodium appetite. It would appear that much, but not all, of the gustatory information contained in the GL is redundant to that provided by other nerves. Bilateral transection of the GL, however, is not entirely without effect. After such denervation, rats decrease their intake of midrange polycose solutions during a 30-min single-bottle test (33). Moreover, NaCl, magnesium chloride, and quinine all elicit fewer aversive oral motor responses (e.g., gapes, chin rubs, etc.) when intraorally infused in GLX rats compared with the intake of controls (10). Nevertheless, given that the GL innervates close to 65% of the taste buds in the rats oral cavity (14), responds well to several different classes of chemical compounds (8), and consists of some rather narrowly tuned fibers (8), it is remarkable that its removal appears to be relatively benign with respect to taste.

We thank Mircea Garcea for assistance with the sodium depletion procedure and Dr. M. J. Fregly, Thomas Connor, and Charlotte Edelstein for the use of, and assistance with, the flame photometer.

A. C. Spector is a recipient of a Research Career Development Award from the National Institute on Deafness and Other Communication Disorders (K04-DC-00104) and also is a recipient of Grant DC-01628.

A portion of this work was presented at the 16th annual meeting of the Associations for Chemoreception Sciences in Sarasota, FL (13).

Address reprint requests to A. C. Spector.

Received 10 November 1994; accepted in final form 30 January 1995.

REFERENCES

1. **Barry, M. A., D. C. Larson, and M. E. Frank.** Loss and recovery of sodium-salt taste following bilateral chorda tympani nerve crush. *Physiol. Behav.* 53: 75–80, 1993.
2. **Boudreau, J. C., N. K. Hoang, J. Oravec, and L. Do.** Rat neurophysiological responses to salt solutions. *Chem. Senses* 8: 131–150, 1983.
3. **Breslin, P. A. S., A. C. Spector, and H. J. Grill.** Chorda tympani section decreases the cation specificity of depletion-induced sodium appetite in rats. *Am. J. Physiol.* 264 (*Regulatory Integrative Comp. Physiol.* 33): R319–R323, 1993.
4. **Breslin, P. A. S., A. C. Spector, and H.-J. Grill.** Sodium specificity of salt appetite in Fischer-344 and Wistar rats is impaired by chorda tympani nerve section. *Am. J. Physiol.* (*Regulatory Integrative Comp. Physiol.*). In press.
5. **Brosvic, G. M., and N. E. Hoey.** Taste detection and discrimination performance of rats following selective desalivation. *Physiol. Behav.* 48: 617–623, 1990.
6. **Catalanotto, F. A., and E. A. Sweeney.** Long term effects of selective desalivation on taste acuity in rats. *Arch. Oral Biol.* 18: 941–952, 1973.
7. **Cauthon, R., M. Garcea, and A. C. Spector.** Taste-guided unconditioned licking to suprathreshold sodium chloride solutions is unaffected by selective lingual denervation (Abstract). *Chem. Senses* 19: 452, 1994.
8. **Frank, M. E.** Taste-responsive neurons of the glossopharyngeal nerve of the rat. *J. Neurophysiol.* 65: 1452–1463, 1991.
9. **Frank, M. E., R. J. Contreras, and T. P. Hettinger.** Nerve fibers sensitive to ionic taste stimuli in chorda tympani of the rat. *J. Neurophysiol.* 50: 941–960, 1983.
10. **Grill, H. J., G. J. Schwartz, and J. B. Travers.** 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 Res.* 573: 95–104, 1992.
11. **Guth, L.** The effects of glossopharyngeal nerve transection on the circumvallate papilla of the rat. *Anat. Rec.* 128: 715–731, 1957.
12. **Hellekant, G., and K. Kasahara.** Secretory fibers in the trigeminal part of the lingual nerve to the mandibular salivary gland of the rat. *Acta Physiol. Scand.* 89: 198–207, 1973.
13. **Markison, S., S. J. St. John, and A. C. Spector.** Glossopharyngeal nerve transection does not compromise cation specificity of depletion-induced sodium appetite in rats (Abstract). *Chem. Senses* 19: 513, 1994.
14. **Miller, I. J.** Gustatory receptors of the palate. In: *Food Intake and Chemical Senses*, edited by Y. Katsuki, M. Sato, S. Takagi, and Y. Oomura. Tokyo: Univ. of Tokyo Press, 1977, p. 173–186.
15. **Nachman, M.** Taste preferences for sodium salts by adrenalectomized rats. *J. Comp. Physiol. Psychol.* 55: 1124–1129, 1962.
16. **O'Keefe, G. B., J. Schumm, and J. C. Smith.** Loss of sensitivity to low concentrations of NaCl following bilateral chorda tympani nerve section in rats. *Chem. Senses* 19: 169–184, 1994.
17. **Pfaffmann, C.** Taste preference and aversion following lingual denervation. *J. Comp. Physiol. Psychol.* 45: 393–400, 1952.
19. **Richter, C. P.** Increased salt appetite in adrenalectomized rats. *Am. J. Physiol.* 115: 157–165, 1936.
20. **Richter, C. P.** Transmission of taste sensation in animals. *Trans. Am. Neurol. Assoc.* 65: 49–50, 1939.
21. **Richter, C. P.** Salt appetite of mammals: its dependence on instinct and metabolism. In: *L'Instinct Dans le Comportement des Animaux et de l'Homme* [*Feeding Instincts in Animals and Humans*]. Paris: Masson, 1956, p. 578–629.
22. **Slotnick, B. M., S. Sheelar, and H. Rentmeister-Bryant.** Transection of the chorda tympani and insertion of ear pins for stereotaxic surgery: equivalent effects on taste sensitivity. *Physiol. Behav.* 50: 1123–1127, 1991.
23. **Sollars, S. I., and I. L. Bernstein.** Sodium appetite after transection of the chorda tympani in Wistar and Fischer 344 rats. *Behav. Neurosci.* 106: 1023–1027, 1992.
24. **Sollars, S. I., and I. L. Bernstein.** Gustatory deafferentation and desalivation: effects of NaCl preference of Fischer 344 rats. *Am. J. Physiol.* 266 (*Regulatory Integrative Comp. Physiol.* 35): R510–R517, 1994.
25. **Spector, A. C.** The effect of gustatory nerve transections on sucrose responsiveness in the rat. In: *Olfaction & Taste XI*, edited by K. Kurihara, N. Suzuki, and H. Edge. Tokyo: Springer-Verlag, 1994, p. 118–119.
26. **Spector, A. C., J. Andrews-Labenski, and F. C. Letterio.** A new gustometer for psychophysical taste testing in the rat. *Physiol. Behav.* 47: 795–803, 1990.
27. **Spector, A. C., and H. J. Grill.** Salt taste discrimination after bilateral section of the chorda tympani or glossopharyngeal nerves. *Am. J. Physiol.* 263 (*Regulatory Integrative Comp. Physiol.* 32): R169–R176, 1992.
28. **Spector, A. C., G. J. Schwartz, and H. J. Grill.** Chemospecific deficits in taste detection following bilateral chorda tympani section in rats (Abstract). *Chem. Senses* 13: 738–739, 1988.
29. **Spector, A. C., G. J. Schwartz, and H. J. Grill.** Chemospecific deficits in taste detection after selective gustatory deafferentation in rats. *Am. J. Physiol.* 258 (*Regulatory Integrative Comp. Physiol.* 27): R820–R826, 1990.
30. **Spector, A. C., S. P. Travers, and R. Norgren.** Taste receptors on the anterior tongue and nasoincisor ducts of rats contribute synergistically to behavioral responses to sucrose. *Behav. Neurosci.* 107: 694–702, 1993.
31. **St. John, S. J., M. Garcea, and A. C. Spector.** Combined, but not single, gustatory nerve transection substantially alters taste-guided licking behavior to quinine in rats. *Behav. Neurosci.* 108: 131–140, 1994.
32. **St. John, S. J., S. Markison, and A. C. Spector.** Salt discriminability is related to number of regenerated taste buds after chorda tympani nerve section in rats. *Am. J. Physiol.* 269 (*Regulatory Integrative Comp. Physiol.* 38): R141–R153, 1995.
33. **Vigorito, M., A. Sclafani, and M. F. Jacquin.** Effects of gustatory deafferentation on polycose and sucrose appetite in the rats. *Neurosci. Biobehav. Rev.* 11: 201–209, 1987.
34. **Whitehead, M. C., M. E. Frank, T. P. Hettinger, L. T. Hou, and H. D. Nah.** Persistence of taste buds in denervated fungiform papillae. *Brain Res.* 405: 192–195, 1987.
35. **Wolf, G.** Innate mechanisms for regulation of sodium intake. In: *Olfaction and Taste. Proceedings of the Third International Symposium*, edited by C. Pfaffmann. New York: Rockefeller Univ. Press, 1969, p. 548–553.
36. **Wong, R., and L. Kraintz.** Interlick interval distribution of desalivated and control rats. *Behav. Biol.* 21: 141–145, 1977.
37. **Yamamoto, T., R. Matsuo, Y. Kiyomitsu, and R. Kitamura.**