Acquisition of taste avoidance habits in rats lacking gustatory neocortex

STEPHEN W. KIEFER and J. JAY BRAUN

Arizona State University, Tempe, Arizona 85281

Normal rats and rats lacking gustatory neocortex were compared in the discriminative acquisition of aversions to either a .146 M sucrose solution or a .153 M sodium chloride solution. Training involved repeated pairings of one taste solution with injections of apomorphine hydrochloride (20 mg/kg IP) during restricted periods of fluid access. Balanced, nonpaired presentations of the other taste solution and water were presented also. Rats lacking gustatory neocortex acquired discriminatively specific aversions to sucrose and sodium chloride, but compared to normal control subjects they acquired the aversions more slowly and they generalized more to the unpaired taste solution. The results confirm earlier suggestions that gustatory neocortex lesions disrupt the associative salience of specific taste stimuli. In addition, these results also clarify the nature of the deficit by showing that it appears to be relative rather than absolute. That is, the brain lesion does not prevent totally the acquisition of aversions to certain taste stimuli as suggested by earlier work. Rather, it interferes substantially with the efficiency and discriminative precision of aversion learning.

Ablations of the gustatory neocortex (GN) substantially disrupt normal associative responses to taste stimuli. Rats lacking GN are deficient relative to normal and operated control rats in acquiring aversions to representative cues from each major class of taste stimuli (Braun, Slick, & Lorden, 1972; Lorden, 1976). The reported deficiencies have ranged from decreased discriminative precision for taste stimuli such as .153 M sodium chloride, $10^{-4.0}$ M quinine hydrochloride, and $10^{-1.6}$ M hydrochloric acid (Lorden, 1976) to what appeared to be a complete lack of development of discriminatively specific aversions to .0041 M sodium saccharin (Braun, et al., 1972) or to .146 M sucrose (Lorden, 1976) solutions. However, simple preference tests have shown all of these stimuli to be readily detectable, compared to water, by untrained control groups of rats with GN lesions. In fact, a comprehensive series of one-bottle and two-bottle preference tests reported by Braun and Kiefer (1975) failed to reveal any deficiencies in basic preference-aversion thresholds in rats lacking GN for any of the basic taste categories. Thus, the deficiencies revealed in the Braun et al. (1972) and Lorden (1976) studies appeared to be specific to the rats' abilities to utilize associatively taste stimuli that otherwise were detectable. In addition, because control ablations of nongustatory neocortex failed to

produce any noteworthy changes in taste responsiveness in these experiments, it was suggested that the deficiencies following GN lesions were specific to gustatory neocortex. Taken together, these observations suggest a fundamental dissociation between detectability and associability, or "salience" (Kalat & Rozin, 1970) of taste stimuli which becomes especially apparent following GN lesions in rats.¹

Both the Braun et al. (1972) and Lorden (1976) studies used a single training trial in which a taste was paired with cyclophosphamide-induced toxicosis, and the degree of aversion learning was measured over subsequent extinction trials (resistance to extinction). But using repeated training trials with lithium chloride as the unconditioned stimulus, Hankins, Garcia and Rusiniak (1974) found that rats with GN lesions appeared capable of eventually developing significant aversions to a .0041 M saccharin solution. However, several features of the Hankins et al. experiment make it difficult to interpret. First, the saccharin solution was the only taste solution presented to the rats; thus no indication of the discriminative specificity of the learned aversion was available. Second, water consumption data were not presented; hence, it could not be adduced whether the reductions in the consumption of saccharin may have reflected simply a drug-induced overall reduction of fluid intake. In this regard, it is noted that a considerable amount of generalized suppression of drinking per se had been evident in the Braun et al. (1972) study.

The present study focused on one of the most extreme examples of dissociation observed in the previous studies: .146 M sucrose, an unquestionably

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suprathreshold concentration of sucrose for rats lacking GN as adduced from detectability tests (Braun & Kiefer, 1975), did not appear to be at all salient as an associative cue for rats lacking GN (Lorden, 1976). Using a single training trial with cyclophosphamide as the unconditioned stimulus, sucrose consumption measured in extinction subsequent to the training trial did not hint of a learned aversion to sucrose in rats lacking GN. In marked contrast, mean sucrose consumption per trial of normal rats in extinction following sucrose-toxicosis pairing was reduced almost to zero. Braun et al. (1972) had reported a similar extreme dissociation for .0041 M saccharin solutions. Thus, it was not evident from these studies whether rats lacking GN were capable of developing significant aversions to these concentrations of saccharin and sucrose.

Using a repeated trials discrimination training procedure, the present experiment found that rats lacking GN can, indeed, learn a discriminatively specific aversion to .146 M sucrose. However, as predicted by Lorden's (1976) results, these rats displayed abnormal generalization between taste species.

METHOD

Subjects

Forty-two male Long-Evans hooded rats (Blue Spruce Farms, Inc., Altmont, New York), approximately 90-120 days old, were used as subjects. One rat died following surgery, leaving a total of 41 rats. The rats were housed individually in wire-mesh cages with ad-lib food in a room which was under constant illumination. Except during the training period, glass-distilled water was available ad lib from water bottles attached to the cages.

Surgery

Twenty-four rats were selected randomly for surgery and given ablations of the gustatory neocortex according to procedures detailed elsewhere (Wolf, DiCara, & Braun, 1970). Under sodium pentobarbital anesthesia (60 mg/kg IP), a bone defect was made in the anterolateral portion of the skull. Tissue then was aspirated with a fine-tipped glass pipette. The area ablated was intended to include the "taste nerve area" defined by Benjamin and Akert (1959) as a 1×3 mm area of tissue bridging the middle cerebral artery on the anterolateral surface of the rat brain adjacent to the rhinal sulcus. Control lesions were not employed because previous studies from our laboratories had used such groups and had found no evidence for an effect of the control lesions on taste aversion learning (Braun et al., 1972; Lorden, 1976) or on other measures of taste responsiveness (Braun & Kiefer, 1975).

Following surgery, wet mash made from Wayne Blox food pellets and distilled water was presented periodically to the operated rats. These procedures were necessary to assist the rats in recovering from the temporary anorexia that typically is found following anterolateral neocortex ablations (Braun, 1975). Postoperative weight measures were taken daily until each rat began eating dry pellets, at which time weights were measured every 3rd day. All surgically treated rats were given at least 4 weeks to recover.

Histology

For histological preparation, all operated rats were given lethal doses of sodium pentobarbital and perfused intracardially with physiological saline followed by 10% formal-saline. The brains were removed and stored in a buffered Formalin solution.

Surface diagrams of the neocortical ablations were drawn with the aid of a dissecting microscope. One eyepiece of the microscope was equipped with a 10×10 mm grid divided into .5-mm squares. Using the reticle as a guide, each lesion was sketched to scale by filling in a grid diagram especially prepared for this purpose. The completeness of each lesion with respect to removal of gustatory neocortex was rated by two independent raters using the procedure described by Kiefer and Braun (1977). With the aid of a template constructed from the details provided by Benjamin and Akert (1959), the lesions were rated on the following scale: 1 (complete), 2 (probably complete), 3 (uncertain), 4 (probably incomplete), and 5 (incomplete). Estimates of the percentage of neocortex removed were calculated by counting the number of squares encompassed by the lesion and dividing this by the total number of squares encompassed by the entire neocortex. The ratings and estimates of lesion size were conservative with respect to the topography of the lesions because tissue tends to move into the ablated area from surrounding regions.

Following the drawing and analysis of the surface diagrams, the brains were embedded in celloidin and sectioned coronally at 30 μ m. Every 10th section through the forebrain and every 5th section through the lesion and the gustatory thalamus were taken. The sections were stained with cresyl violet and examined to verify the lesion placement and to determine the pattern of retrograde degeneration in the gustatory subnucleus of the ventral thalamic nucleus.

Training

The basic procedures for aversion training were similar to those originally used by Braun et al. (1972) and by Lorden (1976). The rats were adapted to a drinking schedule of 15 min fluid access every 12 h (8:00 a.m. and 8:00 p.m.). Fluids were presented in inverted calibrated 50-ml centrifuge tubes fitted with one-hole rubber stoppers and stainless steel drinking spouts. At the end of a drinking session, the amount of fluid consumed was recorded to the nearest .5 ml. Distilled water was presented for 7 days. At the end of this time, three groups of normal rats and three groups of rats lacking gustatory neocortex were formed and matched on the basis of mean consumption over the last four water presentations.

On the 8th day, a schedule of fluid presentation was begun by which each rat received three aqueous solutions over a 36-h period, a single bottle per trial every 12 h. The solutions were .146 M sucrose, .153 M sodium chloride, and distilled water. Earlier preference tests had shown these concentrations of sucrose and salt to be approximately equivalent hedonically for normal and brain-operated rats (Braun & Kiefer, 1975). The order of fluid presentation was balanced within each group, with slight asymmetries due to uneven group sizes, and matched to the other groups. Four groups of rats, two normal and two with GN lesions, received a repeated trials training regimen in which they were injected with the emetic drug apomorphine hydrochloride (20 mg/kg IP) immediately following every presentation of one of the taste solutions. The remaining two groups, one normal and one with GN lesions, were given immediate injections of an equivalent volume of physiological saline following every presentation of the sucrose solution.

In balancing the groups for order of presentation of the taste solutions, the to-be-paired taste was presented before the unpaired taste for half of the rats and vice versa for the other half. This had an impact on the variance of the results for rats with GN lesions, as will be discussed later.

Each solution was presented five times during acquisition for a total of 15 trials. This was followed by two presentations of each solution in extinction. In the results and discussion sections, the groups are identified by three letters: The first letter indicates that the group was either normal (N) or lacking gustatory neocortex (G); the second identifies the solution paired with injection, either sucrose (S) or sodium chloride (T); the last letter indicates

that the injection paired with a particular taste was either the drug, apomorphine hydrochloride (D), or physiological saline (P).

The sucrose and salt solutions were made immediately prior to a drinking period from reagent grade chemicals and distilled water and were presented at room temperature (24°C). Following a watering session, all tubes, stoppers, and glassware were washed in hot Alconox solution, rinsed thoroughly with tap water, and finally rinsed with distilled water. The apomorphine was prepared ½ h before each watering session by dissolving the 6-mg apomorphine hydrochloride tablets (Lilly) in distilled water.

RESULTS

Histology

As determined from an analysis of the surface drawings, all brain lesions except two were rated as at least "probably complete" by both independent raters; the two rats sustaining incomplete GN ablations were both in Group GSP. The mean amount of neocortex removed was relatively consistent across groups (GSD, 28.8%; GSP, 22.7%; GTD, 25.6%). As portrayed in Figure 1, the lesions included and extended well beyond, especially in the dorsal direction, the "taste nerve area" defined by Benjamin and Akert (1959). Lesions rated "probably complete" were those which tended to fall on a line of the template used to judge the completeness of a lesion. In view of the general massiveness of the lesions, the tendency for healthy tissue to spill into the space created by the ablation, and our inability to distinguish behaviorally between rats whose lesions were rated "complete" as compared to those rated "probably complete," it is concluded that these lesions involved no functional sparing. This was essentially confirmed by examination of the cross sections showing the great majority of lesions to extend from the genu of the corpus callosum to the anterior commissure. Subcortical damage was limited to minor encroachments of the caudate-putamen, but this was not represented disproportionately across groups.

Clear evidence of retrograde degeneration was found in the medial subdivision of the ventral thalamic nucleus, the region which projects to the gustatory neocortex (Benjamin & Akert, 1959; Wolf, 1968). However, as in other studies (Ables & Benjamin, 1960; Braun, 1975; Kiefer & Braun, 1977; Wolf et al., 1970), the degeneration was not complete. It is believed that remaining, healthy-appearing cells in the extreme medial tip of the ventral thalamic nucleus have sustaining or essential projections to the lateral subthalamus (Norgren & Wolf, 1975).

Behavioral Results

Figure 2 portrays the overall results of the experiment by comparing mean consumption of the paired taste to that of the unpaired taste across acquisition and extinction trials for each group of rats. The results of Groups NSD and NTD (Panel C) and

Figure 1. Representative surface diagrams of the largest (top), median (middle), and smallest (bottom) lesions employed in the experiment. Both hemisphere lesions (solid lines) are combined in a single lateral view. The taste-nerve area as defined by Benjamin and Akert, 1959, is outlined by the dashed line. Numbers to the left are rat identifications. Abbreviations: MCA = middle cerebral artery; OB = olfactory bulbs; RS = rhinal sulcus.

Groups GSD and GTD (Panel D) were combined in this figure because there were no obvious differences between the groups that were combined.

The overall results can be verbally summarized as follows: Both normal rats and rats with GN lesions significantly reduced intake of the taste paired with drug injection, either sucrose or sodium chloride, and they drank significantly less of the paired taste on the fifth and final presentation than did their saline-injected control groups. In addition, the conditioned aversions were discriminatively specific in that the rats eventually drank significantly less of the taste paired with the drug than they did of the un-

Figure 2. Mean milliliters consumed of paired taste and unpaired taste across five acquisition trials and two extinction trials (Presentations 6 and 7). Note that the normal-drug Group is made up of Groups NSD and NTD. Similarly, the gustatory-drug Group is composed of Groups GSD and GTD.

paired taste. However, acquisition of the taste aversions proceeded significantly more slowly for rats with GN lesions, and these rats showed abnormal generalization to the unpaired taste solution.

Statistical analyses concentrated first on results of the four groups which received sucrose-injection pairings (NSP, GSP, NSD, and GSD—the groups of principal interest in this study): A 2 by 2 factorial analysis (Lesion by Injection) of sucrose consumption of Presentation 1 (which occurred before the rats had experienced a drug injection) revealed no significant differences between groups; this indicates that the groups were matched in terms of initial consumption of the sucrose solutions. A 2 by 2 by 4 repeated measures analysis of variance of sucrose consumption over Acquisition Presentations 2-5 revealed significant effects of lesion [$F(1,23) = 5.69$, $p = .02$], injection [$F(1,23) = 80.23$, $p < .001$], and trial [$F(3,69) = 17.80$, $p < .001$]. A similar analysis for consumption of the unpaired taste (sodium chloride) over Presentations 2-5 for these groups disclosed a Lesion by Injection interaction [$F(1,23) = 5.46$, $p = .02$] as the only significant effect which suggested that the rats in Group GSD generalized the sucrose aversion to the safe salt solution, a point which is elaborated below.

In Figure 2, Panels A and B, it can be seen that pairing sucrose with injections of physiological saline was not associated with a change in sucrose consumption. Both normal rats and rats lacking GN drank approximately equal amounts of the paired and unpaired taste solutions and continued to do so

throughout the presentations. In addition, Group GSP displayed a significant preference for both taste solutions as compared to water [paired $t(6) \geq 5.16$, $ps < .01$], thus confirming that the aversion acquisition deficits found in Group GSD did not reflect merely a sensory deficit for the detection of taste stimuli.

Panel C in Figure 2 compares consumption of paired and unpaired taste solutions over presentations by the normal groups that were drug injected. Note that they drank equivalent amounts of both solutions on Presentation 1, before the first drug injection, and continued to drink about the same amount of the unpaired taste solution throughout the ensuing presentations. By Presentation 4, no normal rat consumed a measurable amount of the paired taste.

Panel D compares consumption of paired and unpaired taste solutions over presentations by rats that lacked gustatory neocortex and received drug injections. In contrast to the saline-injected control group portrayed in Panel B, the drug-injected groups drank less of the unpaired taste than of the paired taste on Presentation 1. This was because the subgroup of these rats that experienced the first presentation of the paired taste before the first presentation of the unpaired taste drank significantly less of the unpaired taste (mean $5.6 \text{ ml} \pm 1.8 \text{ S.E.}$) than did the subgroup which experienced the unpaired taste first (mean $18.1 \text{ ml} \pm 1.5 \text{ S.E.}$). Thus, substantial generalization to the unpaired taste was apparent in groups lacking GN on the first presentation. In marked contrast, the normal groups (see Panel C of Figure 2) did not show a significant generalization. When the unpaired taste solution followed the paired taste on the first presentation, the mean consumption of the unpaired taste solution was $13.8 \text{ ml} \pm 1.2 \text{ S.E.}$, and mean consumption of the unpaired taste was $15.3 \text{ ml} \pm 1.4 \text{ S.E.}$ when it preceded the paired taste on the first presentation.

The significant features of the results to be emphasized are best portrayed by an analysis of performances on the extinction trials. Focusing on the first extinction trial for Groups GSD and GTD, it is noted that each of these groups drank significantly less of the paired than of the unpaired taste. Mean consumption of sucrose ($2.7 \text{ ml} \pm 1.3 \text{ S.E.}$) was significantly less than the mean consumption of sodium chloride ($11.4 \text{ ml} \pm 2.4 \text{ S.E.}$) for Group GSD. And for Group GTD, this was reversed with the mean consumption of sucrose ($14.6 \text{ ml} \pm 2.6 \text{ S.E.}$) being significantly greater than the mean consumption of sodium chloride ($2.2 \text{ ml} \pm 1.1 \text{ S.E.}$). Thus, Groups GSD and GTD displayed discriminative specificity for the taste cue that was paired with drug injection; however, the degree of specificity was weak relative to that displayed by the normal groups. That is, the rats with GN lesions generalized more to the unpaired taste cue.

DISCUSSION

The present results clarify the earlier findings of Braun et al. (1972) and Lorden (1976) with regard to the nature of the involvement of gustatory neocortex in the acquisition of aversions to taste stimuli. Using a single training trial and cyclophosphamide as the US, Lorden found no evidence of conditioning when .146 M sucrose was used as a CS. Braun et al. (1972) also found an apparent lack of conditionability of a mild saccharin solution in rats lacking GN. The results of these two studies led to the suspicion that the relationship of gustatory neocortex to the processing of information concerning "sweet" stimuli was qualitatively different from its relationship to other categories of taste stimuli. Lorden had shown all of the other qualitative taste categories to be salient conditioning stimuli for rats lacking GN. The present study plus the observation that a mild saccharin solution was salient for rats with GN lesions using a repeated trials training regimen (Hankins et al., 1974) suggest that apparent salience differences between qualitative taste categories for rats lacking GN are differences of degree rather than differences of kind.

Lorden's experiment, like the present one, employed a discrimination procedure by which rats' responses to taste solutions paired with drug treatment were compared with responses to hedonically matched unpaired taste solutions. It was noted that while normal rats seemed to discriminate quite precisely between the paired and unpaired taste stimuli, rats lacking GN tended to generalize to the unpaired taste solution. In other words, the discriminative grain of the learned aversions appeared to be much coarser for rats lacking GN. This was confirmed clearly by the present study. Normal rats in the drug-injected groups responded to the unpaired taste in a virtually identical manner to that of normal rats in the saline-injected group. In contrast, rats lacking GN displayed substantial generalization to the unpaired taste, a tendency apparent even on the first presentation of the unpaired taste.

Impairments in the ability to respond discriminatively to stimuli like those reported for rats lacking GN may be a general effect of sensory neocortex lesions over a broad variety of species. For example, in a comprehensive study of stimulus generalization to tones in cats lacking auditory neocortex, Thompson (1962) found that normal cats showed a clear gradient of generalization as test tones became more remote from a training tone of 250 Hz. However, cats lacking auditory neocortex continued to respond vigorously to test frequencies beyond 2,000 Hz. A tendency to show abnormal generalization between stimuli within the sensory mode relevant to a specific ablation of sensory neocortex was shown also in early studies of sensory neocortex and associative processes. Wing

(1947) noted this in dogs that lacked visual neocortex and were trained to respond differentially to changes in background illumination. Allen (1947), also using dogs, noted it following somatosensory neocortex ablations. The indiscriminate responding by Thompson's operated cats probably was not due to an inability to discriminate frequencies, because cats lacking auditory neocortex display such an ability when trained explicitly in a discrimination paradigm (Butler, Diamond, & Neff, 1957). Likewise, as shown in the present study, rats lacking gustatory neocortex show a capacity to learn to discriminate between sucrose and sodium chloride solutions with repeated training trials.

Relative to the comparative wealth of data concerning modality-specific deficits following ablations of other sensory neocortical areas, interest in the relationship of gustatory neocortex to associative taste processes is relatively recent (Braun et al., 1972) and sparse. However, enough data have been gathered to allow some interesting comparisons, beyond those mentioned in the last paragraph, to be made between the effects of gustatory and other sensory neocortex ablations. One example is a comparison with the effects of removal of the visual neocortex on brightness discrimination habits in rats: The rat's initial normal aversion to the brighter of two alternative stimuli is substantially reduced following removal of visual neocortex (Altman, 1962; Krechevsky, 1936). Likewise, rats lacking gustatory neocortex do not display the normal aversion to novel taste stimuli as compared to familiar, experienced taste stimuli (Kiefer & Braun, 1977). In addition, just as preoperatively trained rats appear to lose a brightness discrimination habit following visual neocortex ablation (Lashley, 1935), rats lose a preoperatively instated taste aversion habit following gustatory neocortex ablation (Ouellet, Kower, & Braun, Note 1). And, perhaps most interesting, just as rats lacking visual neocortex do not display impaired thresholds for brightness discrimination (LeVere & Mills, 1977), rats lacking gustatory neocortex display no obvious changes in detection thresholds for any of the basic taste stimuli (Braun & Kiefer, 1975).

Finally, in comparing deficits following sensory neocortex ablations, it should be noted that rats lacking specific sensory areas of neocortex remain capable of acquiring (or reacquiring) habits based on fundamental cues in the sensory mode relevant to the missing cortical area. Rats lacking auditory cortex remain capable of learning associative responses to tones (e.g., French, 1942), rats lacking visual neocortex discriminate lights (e.g., Lashley, 1935), and rats lacking somatosensory cortex learn roughness discrimination habits (e.g., Zubeck, 1951). Likewise, the recent work cited in this paper on associative processes involving the gustatory mode reveals that gus-

tatory neocortex is not essential for learning new responses to taste cues. Thus, mechanisms for instatement of the engram are clearly intact in these cases. What appears to be different following ablation of specific sensory neocortical areas are animals' abilities to relate precisely a specific stimulus to a contingent event; that is, their learned responses tend to be less selective.

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NOTE

1. See Braun and Rosenthal (1976) for a discussion of the salience issue and Kiefer and Braun (1977) for a more thorough discussion of the evidence for a dissociation between systems underlying reflexive vs. associative responses to taste stimuli.

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