Taste Reactivity in Alcohol Preferring and Non-preferring Rats

by

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INTRODUCTION

Alcoholism represents a serious public health problem, and to attain a better understanding of this phenomenon, researchers have attempted to develop various animal models of human alcoholism. This would, of course, be of great value in understanding the cause, effects, and treatment of alcoholism. Because humans self-administer alcohol orally, it is necessary that an animal model also involve the voluntary, oral consumption of alcohol (Amit, Sutherland, Gill, & Ogren, 1984). As Li, Lumeng, McBride, and Waller (1981) have pointed out, all of the conditions in the pathogenesis of alcoholism, whatever they may be (tolerence, alcohol metabolic rate, physical dependence, etc.), are contingent upon the acquisition of ethanol through drinking, and knowledge of this drinking behavior is fundamental in understanding its aberrations (viz., addiction).

Traditional measures of alcohol drinking in animals have involved one and two bottle preference tests. Preference tests allow an animal to approach, sample, and consume a taste solution voluntarily. In the one bottle preference test, the animal is presented with the taste solution as its sole source of fluid, and water is used intermittently to establish a baseline. Acceptability of

the solution is measured relative to water consumption. In the two bottle preference test, both water and a taste solution are presented concurrently; results of the test are generally measured as a ratio of taste solution intake to total fluid intake. These types of preference tests have shown large individual variations with regard to alcohol consumption in rodents. However, most rats consume less than 3 g/kg body weight per day; only 1-3% consume 6-8 g or more (Li, Lumeng, McBride, & Waller, 1979). Therefore, most rats do not ingest alcohol in significant quantities to satisfy the requirement for an animal model of alcoholism.

There have been several different attempts to enhance alcohol self-administration in randomly-bred stock rats. For instance, oral intake of alcohol has been measured in many experimental situations including the following: food deprivation, fluid deprivation, polydipsia, periodic availability, alcohol deprivation, lateral hypothalamic stimulation, and stress-induced drinking.

The food deprivation paradigm involves presenting ethanol to animals while they are being maintained at 80% of their body weight. It appears that ethanol's caloric value is the major factor in its consumption by food-deprived rats because subsequent satiation results in

an immediate decrease in alcohol consumption (Meisch & Thompson, 1974).

Another procedure, the fluid deprivation procedure, involves depriving the animals of all fluid except for access to ethanol. Ethanol is either provided for a short period (30 min.) each day, or it is made available continuously. Preference tests are usually taken before and after the forced ethanol drinking procedure. Experimental studies using this method have shown either no significant difference in alcohol consumption following forced ethanol drinking (Cicero & Smithloff, 1973) or a decrease in ethanol consumption (Carey, 1972).

Schedule induced polydipsia, first described by Falk (1961), has been another popular method to induce voluntary fluid consumption. It refers to a situation in which rats will consume as much as half of their body weight in water within a few hours when food pellets are presented intermittently. After polydipsia is established, researchers have introduced ethanol by progressively increasing its concentration. This procedure has been shown to produce a significant increase in alcohol consumption which results in intoxication; however, it does not appear to change alcohol preference following the polydipsic regimen (Sentar & Sinclair, 1967).

The periodic availability effect occurs when rats that are given periodic opportunities to consume alcohol, display a gradual increase in their selection of alcohol relative to water. Holloway, Bird, and Devenport (1983) found that the increase in alcohol consumption produced by this method was highly concentration dependent, occurring best when the alcohol solutions were within the more preferred range. They also found that the increase was not sustained when alcohol became continuously available. The periodic availability phenomenon is also not specific to alcohol as it occurs when other sapid solutions are used as well (e.g., quinine or saccharin).

The alcohol deprivation effect is a reversal of the periodic availability effect. In this situation, rats are placed on a chronic alcohol regimen and then temporarily withdrawn from the regimen. The temporary withdrawal results in an increase in consumption when the rats are again presented with alcohol. However, this increase in alcohol consumption has been shown to be only temporary (Sinclair, 1972; Sinclair & Senter, 1967).

Another method used to increase oral consumption in rats involves lateral hypothalamic stimulation. It has been shown that during hypothalamic stimulation, rats will ingest amounts of alcohol that lead to intoxication. This

exposure, however, does not seem to change alcohol preference in a home cage, free choice situation (Wayner, Greenberg, Carey, & Nolley, 1971).

Stress induced drinking is a procedure developed to simulate in an animal the same sort of stress that may be present in human alcoholism. In an experiment by Myers and Holman (1967), it was shown that rats receiving intense shock at random for 14 days did not increase their intake of alcohol presented in concentrations varying from 3% to 20%. This lack of preference was present in both naive rats and rats that were acclimated to the alcohol. In a similar experiment, Freed (1967) found that rats subjected to daily, prolonged sessions with alcohol would not increase alcohol consumption when stressed by repeated electric shocks. Rodgers and Thiessen (1964) employed another environmental stressor in which mice were put in a high density group (10 in a single cage); the situation, however, did not produce alcohol preference as intake remained unchanged.

While it is true that many of the above methods have produced increased ingestion of alcohol, none have changed alcohol preference in the free choice, home cage situation. It is this failure to produce a long term change in voluntary oral consumption that has been the most serious

obstacle to the development of an animal model of alcoholism.

It is possible that the previous experiments failed to produce voluntary oral consumption because of the aversive postingestional effects caused by alcohol or because of the aversive taste of the alcohol. Several studies have shown that aversive postingestional effects can limit alcohol consumption. Carey (1972) found a significant decrease in ethanol preference in rats resulting from forced ethanol drinking under fluid deprivation. With the forced-drinking schedule, under fluid deprivation, rats drank fairly large doses of ethanol. It was hypothesized that the large dose of ethanol produced a conditioned aversion which led to a decrease in ethanol consumption on subsequent preference tests. Berman and Cannon (1974) demonstrated that ethanol was an effective unconditional stimulus to produce a conditioned aversion to a saccharin solution, even in doses as low as 2.0 g/kg. The conditioned aversion was found to be dose-dependent, demonstrating that the aversiveness of ethanol consumption increased as a function of the magnitude of the dose. A similar experiment by Kulkosky, Sickel, and Riley (1979) also showed that ethanol, especially at higher doses, had the ability to induce a conditioned taste aversion to saccharin.

A second possibility for the lack of voluntary oral consumption of alcohol is that the taste is aversive to animals. For example, preference tests have shown that rats do not consume large amounts of alcohol at high (10%-15%) concentrations. However, conventional preference tests (one and two bottle) which have generally been used to infer palatability do not directly measure taste; they only measure the amount of fluid ingested. Pelchat, Grill, Rozin, and Jacobs (1983) found that a decrease in preference for a solution does not mean that the solution is aversive or unpalatable; lack of intake could be due to other factors. For example, rats given taste-shock pairings and taste-illness pairings both reduced sucrose intake. However, only the rats that had sucrose paired with illness exhibited truly aversive-type behaviors on actual taste measures. This suggests that preference tests may not be an accurate measure for taste palatability, and that research based on preference tests may provide little information with regard to taste.

In all likelihood, both postingestional factors and taste factors influence the amount of alcohol consumed by an animal. In the end, however, these factors are probably dependent upon the genetic makeup of the organism. There has been considerable evidence for a genetic predisposition

in the development of human alcoholism, and research which has taken advantage of family, twin, and adoption studies point to the importance of these genetic factors (Schuckit, 1984). Consequently, there have been attempts to develop an animal model of alcoholism based on selective breeding programs. One example of selective breeding for an animal model of alcoholism is the alcohol preferring (P) line of rats. In conjunction with the P line of rats, an alcohol non-preferring (NP) line of rats has also been developed (Li et al., 1979).

The P and NP lines were developed by selective breeding for high and low alcohol preference, respectively, from a foundation stock of Wistar rats (Li et al., 1981). These rats were tested with an unflavored solution of 10% (v/v) ethanol which was continuously available with a second bottle of water. Solid food was provided ad lib. Consumption of the 10% ethanol, water, and food was measured daily for 3 weeks. A single pair of animals showing the highest consumption scores (g ethanol/kg body weight/day) were mated and a single pair showing the lowest consumption scores were mated. Li, Lumeng, McBride, Waller, Murphy (1986) reported that, after 20 generations, the consumption scores (g/kg/day; means $\pm$ SD) were: P males, 5.5 $\pm$ 1.2; P females, 7.3 $\pm$ 1.7; NP males, 1.1 $\pm$

0.6; NP females 1.0 ± 0.9. They have also shown that P rats consume 20-30% of the total calories as ethanol as they substitute ethanol calories for part of their food calories and gain weight at the same rate as control animals not given ethanol.

The P line of rats has been shown to meet almost all the perceived requirements of an animal model of alcoholism (Lester & Freed, 1973): These animals voluntarily drink large quantities of 10% ethanol to produce pharmacologically significant effects; they work through operant responding to obtain ethanol when food and water are freely available; they show behavioral tolerance to ethanol; and the amount consumed voluntarily approaches their apparent maximum capacity for ethanol elimination (Li et al., 1979). Waller, McBride, Lumeng, and Li (1982) have also shown that the P line of rats voluntarily consume sufficient ethanol under free-feeding conditions to develop physical dependence.

To determine the precise factor which allows P rats to ingest large quantities of alcohol, it is necessary to consider both postingestional and taste factors as these factors might differ genetically in P rats relative to NP or normal rats. It is possible that P rats do not experience the aversive postingestional effects that may

normally cause a conditioned taste aversion; this would, consequently, allow them to consume more alcohol. It may also be possible that P rats have a learning deficit that prevents them from acquiring a conditioned taste aversion. However, research by Froehlich, Harts, Lumeng, and Li (1986) has discounted both of these explanations. They found that P rats can learn a conditioned taste aversion to saccharin when ethanol is used as the unconditioned stimulus; therefore, the genetic difference is neither because of an inability to experience illness induced by alcohol or an inability to learn a conditioned taste aversion.

One possibility for a genetic explanation that has had popular support in recent studies has been the idea that ethanol acts as a positive reinforcer for P rats. A study by Waller, Murphy, McBride, Lumeng, and Li (1986) has shown that P rats, but not NP rats, exhibited a stimulatory effect to low doses of ethanol on measures of spontaneous motor activity. It has also been found that P rats differ from the NP rats in certain concentrations of monoamines. It is believed that the differences in monoamines, particularly serotonin, may be responsible for a positively reinforcing effect that mediates the drug oriented behavior in the P rat (Amit et al., 1984).

Another possibility for the alcohol consumption by P rats is that these rats find the taste of alcohol palatable. It is likely that both palatability and postingestional positive reinforcement are important in mediating the ingestive behavior of P rats to alcohol. The purpose of the present project, however, was to examine the taste factors of alcohol in P and NP rats. All other factors are contingent upon the acquisition of ethanol through drinking, and taste is the first determinant in whether alcohol will be consumed or rejected.

The role of taste in the voluntary consumption of alcohol by P rats, and the rejection of alcohol by NP rats, is uncertain because little has been done to examine actual taste factors. As previously mentioned, preference tests have shown a high intake measure for P rats versus a low intake measure for NP rats. Preference testing performed as a function of concentration has also been done. With this method, ethanol preference was defined by the intake of more than 50 percent of the total fluid volume as the ethanol solution (Li et al., 1981). Earlier work had shown that rats preferred ethanol over water when the concentration was less than 6% (Myers & Veale, 1972). The P line of rats, however, exhibited high preference for ethanol even when the concentrations was 14% or greater,

and the NP line of rat never exhibited preference for ethanol even at concentrations as low as 2% (Li et al., 1981). Because the P line of rats exhibited a high preference for alcohol, it seems possible that P rats find the taste of alcohol palatable. However, because preference tests only measure intake, the role of taste remains confounded with other factors, such as postingestional factors.

Grill and Norgren (1978) have developed a test in which taste responses to gustatory stimuli can be measured relatively independently of postingestional effects. This test, the taste reactivity test, examines stereotyped, ingestive and aversive response sequences elicited by intraoral infusions of small volumes of taste stimuli (Schwartz & Grill, 1984). Ingestive responses serve to move the fluid to the rear of the oral cavity so that it can be swallowed, and aversive responses serve to expel the fluid from the oral cavity (Grill, 1985). Sucrose and low salt concentrations have been shown to elicit the typical ingestive responses, and conversely, quinine solutions have been shown to elicit the typical aversive responses (Schwartz and Grill, 1984). Because only a small infusion of a taste solution is presented for a short time

(1 ml/min), the possibility of postingestional effects is greatly minimized.

In the present project, P and NP rats were implanted with an intraoral fistula and videotaped in a clear plexiglass chamber while being presented (intraorally) with various alcohol concentrations. The stereotypical responses which were recorded during the taste reactivity test were initially identified by Grill and Norgren (1978) and detailed further in Kiefer and Dopp (in press). All of these responses were characterized as either ingestive, aversive, or neutral. The ingestive responses consisted of tongue protrusions, and lateral tongue protrusions. Tongue protrusions were extensions of the tongue on the midline, which broke the plane of the upper incisors. Lateral tongue protrusions involved the unilateral emergence of the tongue such that a retraction of the upper lip laterally was produced.

Aversive responses consisted of gape, head shake, forelimb flail, fluid expulsion, and passive drip. A gape involved the rapid retraction of the corners of the mouth which consequently formed a triangular shape and exposed the upper and lower incisors. A head shake was the rapid side-to-side movement of the head which was frequently associated with another response: fluid expulsion.

Forelimb flail was a rapid movement of the paws from side-to-side which was also frequently associated with fluid expulsion. Passive drip occurred when the rat allowed fluid to accumulate in the mouth and eventually drip to the floor.

Neutral responses consisted of mouth movements, face washing, paw licking, and locomotion. Mouth movements were rhythmic, low amplitude, openings of the mandible. Face washing occurred when the rat wiped the front paws over the top of its head. Paw licking was scored when the rat made rhythmic extensions of the tongue along the midline toward the forepaws which were held in front of the face. Locomotion involved the quadrapedal movement in the test chamber with the stipulation that the mouth was still in view.

The taste reactivity test provided an opportunity to examine the responses of naive P and NP rats to the taste of alcohol. The P and NP rats were also tested with a sucrose solution and a quinine solution to determine the reactivity elicited by prototypical gustatory stimuli. Following initial reactivity tests, animals were given standard preference tests with 10% alcohol using a two-bottle choice procedure. These tests were done to confirm that the rats were actually P and NP rats. The

consumption tests were also used to examine the relationship between original reactivity and subsequent ingestion. A second taste reactivity test was then done to examine the taste responses of alcohol experienced P and NP rats to the same solutions used in the initial reactivity test.

Method

Subjects

Naive, male, alcohol-preferring rats from the 29th generation (P; n=10) and alcohol-nonpreferring rats from the 28th generation (NP; n=11) were obtained from the laboratory of T.K. Li at The University of Indiana School of Medicine. The rats were individually caged in a room with a normal 12 hour light/dark cycle beginning at 0700 hr. Food and water were available ad libitum.

Surgery

All animals were food and water deprived 24 hr before surgery. Each rat was anesthetized with sodium pentobarbital (Nembutal, 55 mg/kg, ip) and mounted in a nontraumatic headholder (Kopf) which immobilized the head during sugery. All animals were implanted unilaterally with an intraoral fistula made of polyethylene tubing. The tubing was placed anterolateral to the first maxillary molar and threaded subcutaneously to exit on top of the

skull. A metal fistula was connected to the polyethylene tubing and both were secured to the skull with dental acrylic (see Phillips & Norgren, 1970, for details). Following surgery, each rat was given 30,000 units of bicillin (Depo penicillin) im (15,000 units Benzathine and 15,000 units Procaine). On postoperative days 1-4, rats were given wet mash twice a day with water available ad lib; then each rat received standard rat chow and water for the remainder of the experiment. Each animal's fistula was flushed with water daily to maintain its viability.

Postoperative Habituation

Postoperative habituation lasted eight days and occurred in the experimental room. On the first six days, each animal was removed from its home cage and placed in the test chamber (a cylinder of clear plexiglass, 22.2 cm in inside diameter and 25.4 cm high) for 3 min. On the seventh and eighth day of postoperative handling, each animal received a 1 ml infusion of distilled water (rate = 1 ml/min) into the oral cavity while in the test chamber. This allowed acclimation to the infusion process.

Taste Reactivity

Taste reactivity testing took place in the test chamber. The chamber rested on a glass base and was

mounted over a mirror so that the animal's ventral side could be videotaped during infusion.

Polyethylene tubing was connected to a 10 cc glass barrel syringe which was mounted in an infusion pump. The tubing was first filled with solution to minimize deadspace and then attached to the animal's fistula. The animal was placed in the test chamber for a 1 min adaptation period before testing and videotaping began.

Taste reactivity testing consisted of videotaping each animal's response to the infusion of 1 ml of a solution delivered at the rate of 1 ml/min. Videotaping was done with a D5000 Panasonic camera outfitted with a 8:1 autofocus zoom lens, and connected to a BR-7700U JVC video cassette recorder with a time code generator. The infusion pump and electronic timer were turned on simultaneously so that the start of infusion marked the start of the trial. The rat's first response was noted and videotaping continued until 60 seconds of responses had been obtained.

All rats received distilled water on the first day of testing to establish a baseline. Over the next seven days all rats received trials with the following solutions: 5%, 10%, 20%, 30%, 40% (v/v) ethanol made from 95% ethanol and distilled water; .3 M sucrose; and .0005 M quinine hydrochloride. Order of taste solution presentation was

randomly determined for the P rats. Each NP rat was yoked to one P rat and given the same order of solution presentation.

Two-Bottle Preference Tests

After the completion of initial reactivity testing, alcohol consumption was measured using a two-bottle choice procedure. For a period of three weeks, each rat was given two bottles, one with distilled water and the other with 10% ethanol (v/v). The bottles were refilled and weighed every 48 hr, and the positions of the bottles were switched every 24 hr to control for position bias. Food was continuously available throughout the preference tests. At the end of the three week period, rats were placed on ad lib water.

Post-Consumption Taste Reactivity

The final taste reactivity test began one day after the completion of the two bottle tests. On the first day of testing, all animals were given a 1 ml infusion of distilled water to reacclimate them to the infusion process. On the following seven days, animals were again tested with 5%, 10%, 20%, 30%, 40% ethanol (v/v); .3 M sucrose; and .0005 M quinine hydrochloride. Order of solution presentation was identical to that used in the initial reactivity tests.

Taste Reactivity Scoring

Responses to all solutions were videotaped, and the resulting trials were analyzed frame-by-frame by advancing the videotape and simultaneously recording each response on a scoring sheet. These scoring sheets broke the 60 sec trial into 600 blocks; each block was filled with the response that occurred at that .1 second. All scoring was done without knowledge of the solution to eliminate bias. Further, tapes were scored without knowledge of whether the rat was P or NP. When the animal was not making a response, "No Response" was recorded, and when the animal's mouth was not visible on the videotape, "No Data" was recorded. The data from all of the scoring sheets were transferred into microcomputer files. The raw data were then summed, and these sums were entered into a mainframe computer for statistical analysis.

Statistical Analysis

Statistical analysis of the data included analysis of variance (ANOVA) and Pearson product-moment correlations. A 2x2x5 (Group x Exposure x Concentration) ANOVA was used to determine if there were significant differences in taste reactivity to alcohol between the P and NP lines of rats and to see if taste reactivity changed from Exposure 1 to Exposure 2. Separate 2x2 (Group x Exposure) analyses were

used for the water, sucrose, and quinine solutions. Pearson product-moment correlations were computed between Exposure 1 taste reactivity and alcohol consumption during the first six days of two-bottle alcohol access. Similar correlations were calculated between alcohol consumption on the last six days of two-bottle alcohol access and Exposure 2 taste reactivity. Alcohol consumption data were expressed as g/kg body weight/ 48 hr period. Finally, correlations were computed between Exposure 1 and Exposure 2 taste reactivity responses to determine if the rats' reactivity remained consistent.

Results

There were no significant differences in ingestive or aversive responses found between the P rats and NP rats on the initial taste reactivity tests. However, there was a significant difference in the number of mouth movements (considered a neutral response). NP rats made significantly more mouth movements than the P rats on virtually all solutions tested. During the two-bottle tests, consumption of alcohol by P rats was consistently higher than that of NP rats across all test days. On the final taste reactivity tests, significant differences in ingestive and aversive responses emerged between the two groups of rats. The P rats made more ingestive responses

and fewer aversive responses to alcohol while the NP rat's ingestive and aversive responses did not change from Exposure 1 to Exposure 2. Mouth movements did increase for both groups, but NP rats continued to make more mouth movements than P rats.

Verification of P and NP Status

The two-bottle fluid consumption test served to verify that the rats were actually P and NP rats. As Figure 1 shows, mean alcohol consumption by the P rats was higher than that of the NP rats for the entire three week period. P rats consumption scores increased gradually whereas NP rats scores remained similar throughout the three week period. Consumption scores for the last 48 hr period (g/kg/48 hr; mean $\pm$ SD) were: P rats, 8.61 $\pm$ 2.15; NP rats, 2.87 $\pm$ 1.60. No overlap occurred in consumption between P and NP rats during the last 4 days. There were three rats in the NP group that consumed alcohol above the normal criterion to be considered NP rats. One rat drank 17.4 g/kg bw during the first 48 hr period; its consumption decreased after this but still varied from 1.72 to 8.42 g/kg bw. Two other rats consumption scores varied from 1.50 to 8.0 (g/kg bw). To be conservative, taste reactivity data from these three rats were included with the NP group.

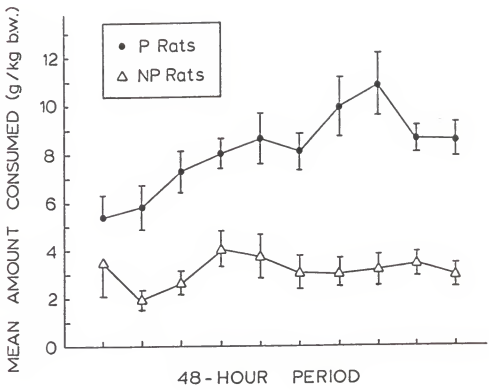

Figure 1. Mean (± SEM) amount of 10% alcohol consumed (g/kg bw) during the two-bottle preference tests. Each point represents the mean of the total amount of alcohol consumed over a 48 hr period by alcohol preferring (P) rats and alcohol non-preferring (NP) rats.

Taste Reactivity

Mouth movements. Trials with all solutions typically began with mouth movements (considered neutral responses); statistical analysis of mouth movements during alcohol trials revealed a significant Group difference, F (1,19) = 28.64, $p<.001$. As Figure 2 shows, NP rats made more mouth movements than did P rats on both exposures. There was also a significant Exposure difference, $F(1,19)$ = 17.32, $p<.001$; the mean number of mouth movements increased during Exposure 2 for both the P and NP rats. A significant effect of Concentration, $F(4,76)$ = 13.27, $p<.001$, was also found; the mean number of mouth movements increased as the concentration of alcohol increased. Finally, analysis of mouth movements showed a significant Group difference, $F(1,19)$ = 7.25, $p<.02$, to sucrose; again, the NP rats made more mouth movements than the P rats on both exposures. No significant differences were found between groups or exposures for either water or quinine.

The fact that the NP rats made more mouth movements to alcohol than the P rats could be accounted for, in part, by three rats in the NP group whose alcohol consumption had been high during the two-bottle tests. Separate analysis of mouth movements, without the three NP outliers, continued to show a significant Group difference to

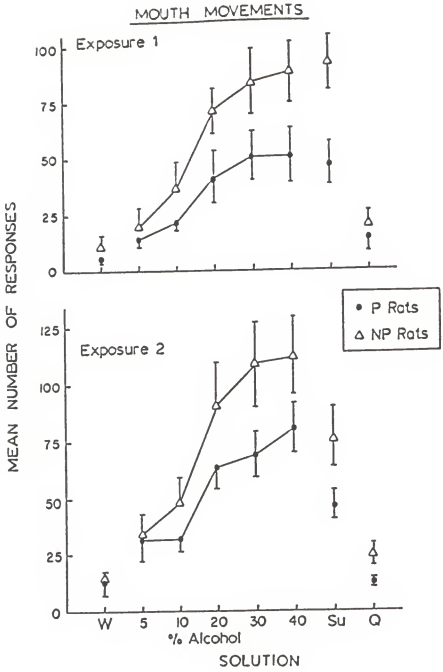

Figure 2. Mean number of mouth movements elicited by water, alcohol, sucrose, and quinine on Exposure 1 (top) and Exposure 2 (bottom) taste reactivity for alcohol preferring (P) rats and alcohol non-preferring (NP) rats. Bars indicate SEM. Abbreviations: W = water; Su = .3 M sucrose; Q = .0005 M quinine.

alcohol, $F(1,16) = 5.90$, $p<.05$; on Exposure 1. NP rats made more mouth movements than the P rats; however, the difference between the two groups was considerably smaller. On Exposure 2, there were no significant differences found between the P and the NP rats after the exclusion of the three NP outliers. The outliers had accounted for more than half of the mouth movements made during the second exposure by the NP rats.

Tongue Protrusions. The mean number of tongue protrusions (responses which were considered ingestive) by each group of rats is shown in Figure 3. Analysis of tongue protrusions during alcohol trials revealed a Group x Exposure interaction $F(1,19) = 49.47$, $p<.001$. P and NP rats did not differ significantly in the number of tongue protrusions made to any of the solutions on Exposure 1; however, there was a significant difference between the P and NP rats to alcohol on Exposure 2. On Exposure 2, P rats showed a significant increase in the number of tongue protrusions to alcohol whereas NP rats remained virtually the same on both exposures. There was also a significant increase in the number of tongue protrusions as the concentration of alcohol increased from 5% to 40%. This was reflected in a significant effect of concentration, $F(4,76) = 2.61$, $p<.05$. There were no significant Group or

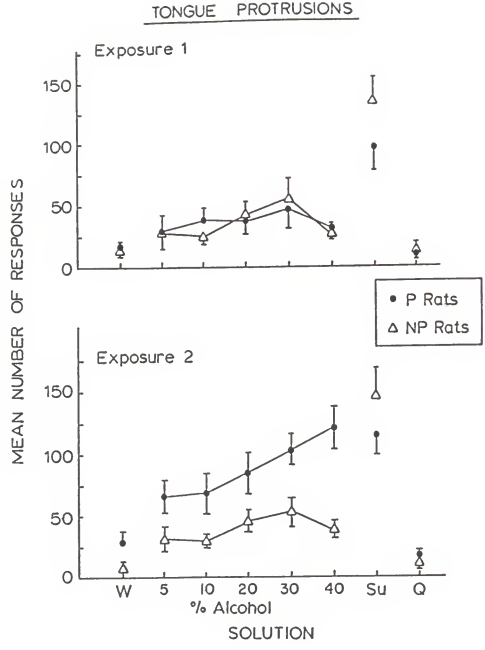

Figure 3. Mean (± SEM) number of tongue protrusions produced by alcohol preferring (P) rats and alcohol non-preferring (NP) rats on Exposure 1 (top) and Exposure 2 (bottom) taste reactivity. See Figure 1 for explanation of solutions and abbreviations.

Exposure differences found for either sucrose or quinine. On Exposure 1, both the P and the NP rats made a large number of tongue protrusions to sucrose and a small number of tongue protrusions to quinine. Analysis of tongue protrusions during water trials showed a significant Group x Exposure interaction $F(1,19) = 5.87$, $p<.03$. P rats showed a significant increase in the number of tongue protrusions to water on Exposure 2.

Lateral Tongue Protrusions. The mean number of lateral tongue protrusions can be seen in Figure 4. Analysis of lateral tongue protrusions, an ingestive response similar to tongue protrusions, also showed a significant Group x Exposure interaction, $F(1,19) = 24.39$, $p<.001$, when the alcohol data were analyzed. Again, P and NP rats showed no differences in the number of lateral tongue protrusions to any of the solutions on Exposure 1; however, there was a significant difference between the P and NP rats on Exposure 2 as the P rats made more lateral tongue protrusions than the NP rats. There was also a Group x Concentration interaction, $F(4,76) = 2.61$, $p<.05$. P and NP rats were not significantly different at the lower concentrations but became significantly different as the concentration increased. There were no significant differences between P rats and NP rats in the number of

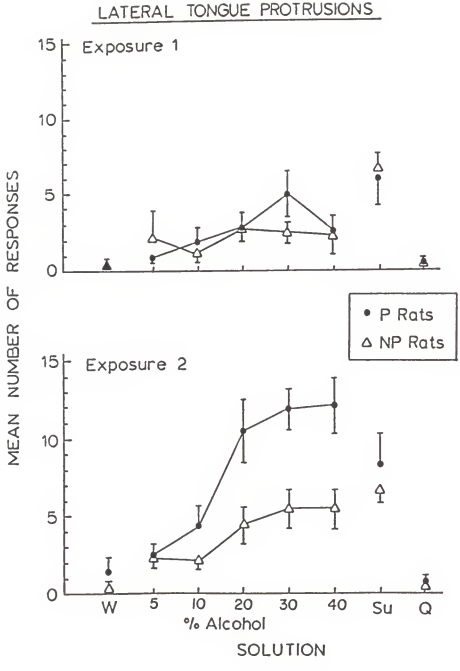

Figure 4. Mean (± SEM) number of lateral tongue protrusions produced by alcohol preferring (P) rats and alcohol non-preferring (NP) rats on Exposure 1 (top) and Exposure 2 (bottom) taste reactivity. See Figure 1 for explanation of solutions and abbreviations.

lateral tongue protrusions to water, sucrose, or quinine during Exposure 2. On both Exposures 1 and 2, sucrose elicited a large number of lateral tongue protrusions for both the P and NP rats whereas quinine and water elicited practically none.

Passive Drip. Passive drip was the primary aversive response made during taste reactivity testing. During alcohol trials, it accounted for 69% of the total aversive responses made by the P rats on Exposure 1 and 79% of the total aversive responses made by the P rats on Exposure 2. Passive drip accounted for 62% of the total aversive responses made by the NP rats on Exposure 1 and 55% made by the NP rats on Exposure 2. The mean number of passive drips can be seen in Figure 5. Analysis of passive drips during alcohol trials revealed a significant Group x Exposure interaction, $F(1,19) = 12.90$, $p<.003$. Both the P and the NP rats made more passive drips to alcohol on Exposure 1 than on Exposure 2. During Exposure 2, however, the number of passive drips decreased significantly, $F(1,19) = 3.82$, $p<.05$, for the P rats but did not change significantly for the NP rats. There were no significant differences in the number of passive drips to water; however, quinine did reveal a significant group difference,

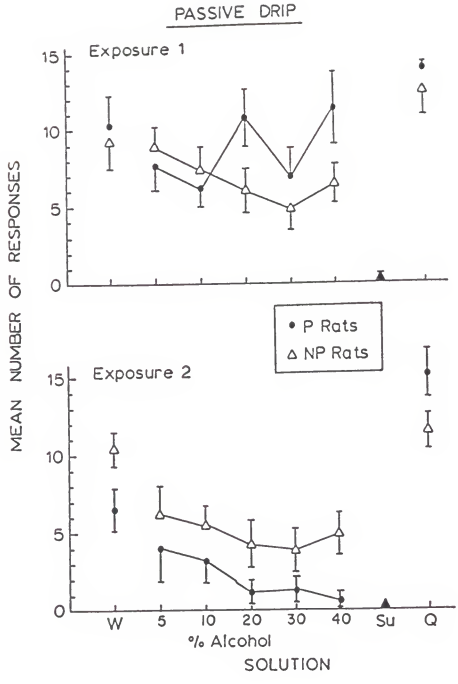

Figure 5. Mean (± SEM) number of passive drips produced by alcohol preferring (P) rats and alcohol non-preferring (NP) rats on Exposure 1 (top) and Exposure 2 (bottom) taste reactivity. See Figure 1 for explanation of solutions and abbreviations.

$F(1,19) = 4.75$, $p<.05$, as the P rats made more passive drips than the NP rats.

Gape. The frequency of gapes was low for both the P and the NP rats on both exposures (see Table 1). However, statistical analysis of gapes to alcohol did reveal a significant Exposure x Concentration interaction, $F(4,76) = 5.16$, $p<.001$. On Exposure one, both P and NP rats made significantly more gapes as the alcohol concentration increased from 5% to 40%. There were no differences in the number of gapes across alcohol concentration on Exposure 2.

Head Shake, Forelimb Flail, and Fluid Expulsion. The means and standard errors for the remainder of the aversive responses (head shake, forelimb flail, and fluid expulsion) can be seen in Tables 2, 3, and 4 respectively. Analysis of head shakes to alcohol revealed a significant Group difference, $F(1,19) = 19.61$, $p<.001$, with the NP rats making more head shakes than the P rats on both exposures. There was also a significant Group difference, $F(1,19) = 5.93$, $p<.05$, found for head shakes to water; again, NP rats made more head shakes than the P rats on both exposures. No significant Group or Exposure differences were found for either sucrose or quinine.

Statistical analysis of forelimb flails showed a significant Group difference to both alcohol, $F(1,19) =$

Table 1

Mean number of Gapes (+ SE)

Exposure	Group	SOLUTION							
		H2O	5%	10%	20%	30%	40%	SU	Q
Exposure 1	P	0.0 ±0.0	0.0 ±0.0	0.6 ±0.0	1.8 ±0.5	2.5 ±0.4	8.2 ±1.8	0.0 ±0.0	4.4 ±0.4
	NP	0.0 ±0.0	0.0 ±0.0	0.0 ±0.0	0.7 ±0.5	0.8 ±0.4	3.6 ±1.8	0.0 ±0.0	1.2 ±0.7
Exposure 2	P	0.1 ±0.1	0.5 ±0.3	0.1 ±0.1	0.0 ±0.0	0.0 ±0.0	0.1 ±0.1	0.0 ±0.0	3.4 ±1.2
	NP	0.6 ±0.6	0.0 ±0.0	0.2 ±0.2	0.0 ±0.0	0.1 ±0.1	0.9 ±0.4	0.3 ±0.3	0.8 ±0.6

Table 2

Mean number of Head Shakes (+ SE)

Exposure	Group	SOLUTION							
		H2O	5%	10%	20%	30%	40%	SU	Q
Exposure 1	P	0.0 ±0.0	0.4 ±0.3	0.5 ±0.3	0.3 ±0.2	0.5 ±0.2	0.0 ±0.0	0.0 ±0.0	0.5 ±0.3
	NP	0.6 ±0.2	0.6 ±0.3	1.1 ±0.4	1.2 ±0.4	0.6 ±0.3	1.3 ±0.4	0.0 ±0.0	0.7 ±0.3
Exposure 2	P	0.1 ±0.1	0.0 ±0.0	0.0 ±0.0	0.1 ±0.1	0.2 ±0.2	0.1 ±0.1	0.0 ±0.0	0.5 ±0.3
	NP	0.7 ±0.4	1.0 ±0.3	0.9 ±0.3	1.1 ±0.4	0.4 ±0.2	1.0 ±0.4	0.0 ±0.0	1.4 ±0.4

Table 3

Mean number of Forelimb Flails (+ SE)

Exposure	Group	SOLUTION							
		H2O	5%	10%	20%	30%	40%	SU	Q
Exposure 1	P	0.1 ±0.1	0.7 ±0.4	0.5 ±0.3	0.2 ±0.2	0.0 ±0.0	0.0 ±0.0	0.0 ±0.0	0.1 ±0.1
	NP	0.6 ±0.3	0.6 ±0.3	1.0 ±0.5	0.6 ±0.4	0.3 ±0.3	1.4 ±0.4	0.1 ±0.1	1.9 ±0.7
Exposure 2	P	0.1 ±0.1	0.5 ±0.3	0.2 ±0.1	0.0 ±0.0	0.1 ±0.1	0.0 ±0.0	0.1 ±0.1	0.4 ±0.4
	NP	0.8 ±0.6	2.0 ±1.2	1.0 ±0.6	1.7 ±1.2	0.4 ±0.3	0.6 ±0.4	0.4 ±0.3	2.7 ±1.5

Table 4

Mean number of Fluid Expulsions (+ SE)

Exposure	Group	SOLUTION							
		H2O	5%	10%	20%	30%	40%	SU	Q
Exposure 1	P	0.1 ±0.1	0.8 ±0.4	0.9 ±0.6	0.6 ±0.3	0.4 ±0.2	0.6 ±0.3	0.0 ±0.0	0.6 ±0.3
	NP	1.0 ±0.4	1.1 ±0.6	1.9 ±0.6	0.6 ±0.2	0.6 ±0.4	1.8 ±0.6	0.0 ±0.0	2.1 ±0.6
Exposure 2	P	0.1 ±0.1	0.3 ±0.2	0.2 ±0.1	0.1 ±0.1	0.2 ±0.2	0.0 ±0.0	0.0 ±0.0	0.8 ±0.4
	NP	1.4 ±1.0	2.6 ±1.1	1.9 ±0.8	2.4 ±1.4	0.6 ±0.3	1.4 ±0.5	0.0 ±0.0	3.6 ±1.6

8.05, $p<.02$, and quinine, $F(1,19) = 5.54$, $p<.03$. In both cases, NP rats did more forelimb flailing than the P rats. Analysis of fluid expulsion, a response that sometimes accompanies head shakes and forelimb flails, revealed a significant Group difference, $F(1,19) = 10.90$, $p<.004$. Here again, the NP rats made more responses of fluid expulsion than the P rats. There was also a significant Group difference for fluid expulsion to quinine, $F(1,19) = 4.50$, $p<.05$, and water, $F(1,19) = 4.85$, $p<.04$. For both, NP rats had higher amounts of fluid expulsion than the P rats.

Neutral responses. Neutral responses that were analyzed included locomotion, grooming, and paw licking. In analysis of locomotion, significant differences generally showed that P rats locomoted more than the NP rats. For instance, significant Group differences were found for both alcohol, $F(1,19 = 10.07$, $p<.01$, and quinine, $F(1,19) = 11.51$, $p<.004$. In both cases, the P rats locomoted more than the NP rats (see Figure 6). There was also a significant Exposure difference, $F(1,19) = 6.76$, $p<.02$, to quinine; both the P and the NP rats locomoted more on Exposure 1 than they did on Exposure 2. Analysis of locomotion to sucrose revealed a significant Group x Exposure interaction, $F(1,19) = 10.66$, $p<.005$. In this

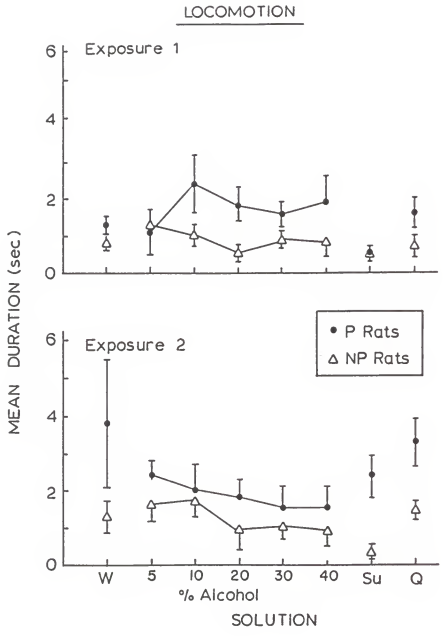

Figure 6. Mean (± SEM) duration in sec for locomotion produced by alcohol preferring (P) rats and alcohol non-preferring (NP) rats on Exposure 1 (top) and Exposure 2 (bottom) taste reactivity. See Figure 1 for explanation of solutions and abbreviations.

case, no difference existed between the P and NP rats on Exposure 1; however, the P rats locomoted more than the NP rats on Exposure 2.

There were no significant differences in paw licking except for a Group effect with quinine, $F(1,19) = 10.06$, $p<.01$; the NP rats did more paw licking than the P rats during both exposures. Analysis of grooming showed only a significant Concentration effect, $F(4,76) = 4.78$, $p<.003$. Both the P and NP rats did more grooming at the lower concentrations. The means and standard errors for paw licking and grooming are located in Tables 5 and 6 respectively.

Relationship Between Consumption and Reactivity

Significant positive correlations were found between ingestive responses made on the initial taste reactivity tests and the first 6 days of 10% alcohol consumption for both P and NP rats (see Table 7). This indicated that those rats that made more ingestive orofacial responses on the taste reactivity tests also consumed more alcohol during the first six days of the two-bottle tests. There were also significant positive correlations found between mouth movements (a neutral response) and alcohol consumption during the first 6 days.

Table 5

Mean (+ SE) duration of Paw Licking in sec

Exposure	Group	SOLUTION							
		H2O	5%	10%	20%	30%	40%	SU	Q
Exposure 1	P	15.5 ±13.6	39.8 ±20.1	20.4 ±12.1	8.5 ±7.0	6.5 ±5.4	4.6 ±3.1	26.6 ±14.4	4.6 ±4.4
	NP	6.9 ±3.6	5.6 ±3.1	14.9 ±10.8	17.7 ±7.5	3.2 ±1.9	21.4 ±4.4	20.3 ±15.1	14.9 ±4.1
Exposure 2	P	16.8 ±16.8	31.4 ±19.6	22.7 ±15.5	15.3 ±10.8	3.5 ±2.1	0.0 ±0.0	13.9 ±8.5	1.7 ±1.2
	NP	10.4 ±10.0	27.1 ±20.3	21.4 ±15.7	6.4 ±4.9	1.1 ±0.8	5.6 ±2.4	20.0 ±13.7	9.6 ±3.2

Table 6

Mean number of Grooming responses (+ SE)

		SOLUTION							
Exposure	Group	H2O	5%	10%	20%	30%	40%	SU	Q
Exposure 1	P	0.6 ±0.6	2.7 ±1.4	2.2 ±1.3	0.4 ±0.3	0.2 ±0.2	0.0 ±0.0	1.0 ±0.7	0.4 ±0.4
	NP	0.4 ±0.3	0.7 ±0.5	0.6 ±0.4	0.1 ±0.1	0.1 ±0.1	0.0 ±0.0	0.6 ±0.5	1.4 ±0.7
Exposure 2	P	1.1 ±1.1	1.5 ±1.1	0.7 ±0.7	0.4 ±0.4	0.0 ±0.0	0.0 ±0.0	0.2 ±0.2	0.1 ±0.1
	NP	0.8 ±0.6	1.6 ±1.1	1.6 ±1.1	0.2 ±0.2	0.0 ±0.0	0.0 ±0.0	0.4 ±0.3	1.1 ±0.9

Table 7

Correlations between total reactivity responses across alcohol concentrations on Exposure 1 and first six days of 10% alcohol consumption (g/kg bw)

Group	MM	TP	LT	A	PD	HS+FF+FE
P (n=10)	.72**	.28	.67*	-.69*	-.70*	-.46
NP (n=11)	.77**	.69**	.76**	-.18	-.57*	-.16

*p<.05. **p<.01.

The majority of aversive responses from the initial exposure were found to have a significant negative correlation with the first 6 days of alcohol consumption for both P and NP rats (see Table 7). This suggested that those rats that made more aversive responses during the initial taste reactivity test drank less alcohol during the initial phase of the two-bottle tests. Table 8 shows that there were no significant correlations found between the initial reactivity results and the last 6 days of alcohol consumption for either P or NP rats.

Consumption of alcohol during the last 6 days of two-bottle testing showed significant correlations with ingestive and aversive responses made during Exposure 2 taste reactivity for the NP rats (see Table 9). A positive correlation was also found for NP rats between mouth movements and consumption. There was no significant relationship found for the P rats except for a positive relationship between mouth movements and consumption. The positive correlations indicate that those NP rats that drank more alcohol during the two-bottle test also made more ingestive responses during Exposure 2 taste reactivity. The significant negative correlations indicate that those NP rats whose consumption was low during the

Table 8

Correlations between total reactivity responses across alcohol concentrations on Exposure 1 and last six days of 10% alcohol consumption (g/kg bw)

Group	MM	TP	LT	A	PD	HS+FF+FE
P (n=10)	.37	-.06	.25	-.42	-.30	-.46
NP (n=11)	.04	-.16	.02	.24	.43	-.10

*p<.05. **p<.01.

Table 9

Correlations between last six days of 10% alcohol consumption (g/kg bw) and total taste reactivity to all alcohol concentrations on Exposure 2

Group	MM	TP	LT	A	PD	HS+FF+FE
P (n=10)	.56*	-.19	-.23	-.14	-.14	.01
NP (n=11)	.52*	.11	.66*	-.72**	-.54*	-.57*

*p<.05. **p<.01.

last 6 day of testing made a lot of aversive responses during Exposure 2 taste reactivity.

In Table 10, correlations between taste reactivity to alcohol on Exposure 1 and Exposure 2 showed few significant relationships. There were positive correlations between the number of mouth movements made on Exposure 1 and the number made on Exposure 2 for both P and NP rats. This suggested that mouth movements remained consistent from Exposure 1 to Exposure 2. There was also a positive correlation between the number of passive drips made on Exposure 1 and the number made on Expsoure 2 for the P rats. This meant that those P rats that passive dripped during the first exposure continued to passive drip during the second exposure.

Discussion

It was hypothesized that the differences in alcohol consumption between the P and NP rats may, in part, be related to an innate difference in their taste preference for alcohol. This, however, was not found to be the case. Except for NP rats making a greater number of mouth movements than the P rats, there were no significant differences found between the two groups during the initial taste reactivity tests. This indicated that the responses of naive P and NP rats' to the taste of alcohol were not

Table 10

Correlations between total taste reactivity to alcohol on Exposure 1 and total taste reactivity to alcohol on Exposure 2.

Group	MM	TP	LT	A	PD	HS+FF+FE
P (n=10)	.78**	.39	.40	.25	.71*	.18
NP (n=11)	.64*	.32	.10	.04	.13	.21

*p<.05. **p<.01.

different. The data also indicated that there was no difference between P and NP rats in response to either sucrose, a prototypical ingestive stimulus, or quinine, a prototypical aversive stimulus.

In the two-bottle preference tests, mean alcohol consumption was higher for the P rats than for the NP rats on all test days. P rats' consumption scores increased gradually whereas the NP rats' consumption leveled off almost immediately. Li et al. (1979) had previously suggested that the phenotypes of the two lines would become readily definable by using a standard two-bottle preference test that employed a single 10% ethanol concentration. Although the phenotypes of the two lines were apparent from the two-bottle preference tests used in the present experiment, mean alcohol consumption by the P rats did not reach a level consistent with that found by Li et al. (1979). In their experiments, P rats consumed between 10 and 12 g/kg/day by the end of the third week. In the present experiment, P rats' consumption never exceeded 11 g/kg/48 hr. It is likely, however, that this occurred because animals were never given forced choice alcohol (see Lumeng, Hawkins, & Li, 1977) prior to the three week two-bottle testing period. According to Lumeng et al. (1977), the procedure that is generally used to test the

drinking behavior of naive P and NP rats consists of housing the animals individually after the onset of puberty and giving the animals forced choice 10% ethanol for four days. Thereafter, the animals are given free-choice ethanol and water for three weeks. It is possible that without the forced choice period, P rats may require more time to achieve as high a level of alcohol consumption.

There were three rats in the NP group whose alcohol consumption exceeded the normal criterion to be considered true NP rats. For these rats, consumption was high from the beginning of the two-bottle tests and generally remained high throughout the testing period. Had selective testing been performed before the experiment, these rats would not have been selected as NP rats. Their data, however, were included with the data from the NP group to be conservative in the statistical analyses. If significant differences existed between the two groups with the inclusion of the three outlier rats, those differences must be substantial.

After two-bottle testing with 10% ethanol, taste reactivity to alcohol for the P rats showed a significant increase in the number of ingestive responses and a significant decrease in the number of aversive responses. Tongue protrusions and lateral tongue protrusions increased

whereas passive drips, gapes, head shakes, forelimb flails, and fluid expulsion decreased. This suggested that P rats' preference for alcohol increased after experience with alcohol. It appears that this change is due to the P rats' genetic predisposition for alcohol because the NP rats' preference did not change. In fact, ingestive and aversive responses of the NP rats remained virtually the same from Exposure 1 to Exposure 2. Mouth movements continued to be higher for the NP rats; however, this could be accounted for by the three rats in the NP group that had high levels of alcohol consumption during the two-bottle tests. There were few changes in response to either sucrose or quinine on Exposure 2. This indicated that the differences between the P and NP rats were relatively specific to alcohol. Future tests involving extensive experience with other tastants such as quinine would be necessary to confirm this hypothesis.

Correlations were computed to determine the relationship between the responses during the taste reactivity tests and fluid consumption during the two-bottle access period. Significant correlations were found between the initial reactivity tests and the first 6 days of fluid consumption for both the P and the NP rats. Rats that made a large number of ingestive responses during

Exposure 1 taste reactivity drank more alcohol during the first 6 days of fluid consumption. Rats that made a large number of aversive responses drank less alochol during the first six days of fluid consumption. Significant correlations were also found between the last 6 days of fluid consumption and Exposure 2 taste reactivity but only for the NP rats. The NP rats that drank more alcohol during the last 6 days of fluid consumption made more ingestive responses on Exposure 2 taste reactivity. Conversely, the NP rats that drank little alcohol during the last 6 days of fluid consumption made many aversive responses on Exposure 2 taste reactivity. In a previous experiment from the same laboratory (Kiefer & Dopp, in press) significant correlations between consumption and reactivity were not found. This may be because of the variance involved with using randomly bred stock rats as opposed to the more homogeneous P and NP rats. It may also be because the other experiments used various alcohol solutions during the two-bottle consumption tests whereas the present experiment used only a single (10%) ethanol concentration. Using a variety of concentrations may generate a great deal of variance in the amount of alcohol consumed during the two-bottle consumption test and, as a

consequence, decrease the likelyhood of obtaining a significant correlation.

The only response consistently correlated with consumption for both the P and the NP rats was mouth movements. For both groups of rats, mouth movements showed significant correlations between Exposure 1 reactivity and the first 6 days of fluid consumption, and also between the last 6 days of fluid consumption and Exposure 2 reactivity. Characterization of mouth movements as either ingestive or aversive, however, was found to be problematic. Rats that made the highest number of mouth movements consistently made mouth movements during both ingestive or aversive sequences. In other words, in certain rats, mouth movements occurred simultaneously with aversive responses such as passive drip or with ingestive response such as tongue protrusions and lateral tongue protrusions. Therefore, in the present experiment, mouth movements were considered to be neutral responses because they seemed to be made primarily to the tactile properties of the alcohol. Had mouth movement been considered an ingestive response, the basic premise that has been developed thus far for the P and NP rats would not change. Although NP rats made a greater number of mouth movements on Exposure 1, it was the only response that was significantly different for the two

groups. During Exposure 2 taste reactivity, the NP rats continued to make more mouth movements. However, as previously mentioned, the higher number of mouth movements was accounted for primarily by three rats in the NP group whose alcohol consumption had been high during the two-bottle tests. The true NP rats, as a whole, did not show any significant changes from Exposure 1 to Exposure 2.

At this point, it is clear that the phenotypes of the two lines became apparent after the animals had experience with alcohol: the P rats not only consumed more alcohol than the NP rats during the two-bottle preference test but also showed an increased preference for alcohol during Exposure 2 taste reactivity. The preference for alcohol by the P rats and lack of change by the NP rats must be due to genetic differences between the two lines of rats because genetic strain was the main independent variable under study. However, because the P and NP rats were found to have no innate taste response differences, some other genetic factor must account for the consumption difference found during the two-bottle tests. The question, now, is whether the genetic differences between the two lines give rise to differences in taste preferences indirectly.

There has been much research suggesting that ethanol acts as a positive reinforcer for P rats. Perhaps positive

reinforcement indirectly influences the P rats' taste for alcohol. It is well known that P rats will work through operant responding to obtain ethanol when food and water are freely available (Li et al., 1979). A study by Waller et al. (1986) showed that P rats, but not NP rats, exhibited a stimulatory effect to low doses of ethanol on measures of spontaneous motor activity, and this has been taken as an indication of positive reinforcement. It has also been found that P rats are insensitive to the sedative or hypnotic effects of alcohol; in a sense, they have an innate tolerence to alcohol (Li et al., 1981). It was suggested by Li et al. (1986) that the combination of low-dose stimulation and acute tolerence development to the high-dose effects offer a plausible hypothesis for alcohol abuse. This hypothesis suggests that low-dose stimulation reflects positive reinforcement and subsequent ingestion of alcohol whereas high-dose stimulation reflects depressant, aversive effects that inhibit ingestion of alcohol. It is possible that P rats develop tolerence to progressively higher doses of alcohol and, therefore, the rewarding effects of ethanol become extended into the higher dose range.

If alcohol is positively reinforcing, this would explain why the P rats consumed more alcohol during the

two-bottle test. It may also indirectly explain why the high ingestive scores and low aversive scores were found for P rats during Exposure 2 taste reactivity. Green and Garcia (1971) have shown that a distinct fluid paired with recuperation from illness (a reinforcement) resulted in elevated consumption of that fluid. In the present experiment, drinking alcohol (US) may have resulted in positive reinforcement (UR). Because the taste (CS) of alcohol was associated with its unconditional effects, and became a conditioned reinforcer, this may explain why the P rats exhibited higher ingestive and lower aversive responses to alcohol during Exposure 2 taste reactivity. Because NP rats did not make this association, reactivity would not be expected to change from Exposure 1 to Exposure 2.

One issue raised by the present experiment is how normal rats' consumption and reactivity would compare to P and NP rats'. In a similar experiment from the same laboratory (unpublished observations), taste reactivity tests were used to examine the orofacial responses of naive, randomly bred stock rats (Sprague Dawley) to 10%, 20%, 30%, and 40% ethanol. Rats were then exposed to 10% ethanol in a two-bottle preference test for four weeks. Finally, the alcohol experienced rats were given taste

reactivity tests. It was found that normal rats' initial reactivity responses were not different from the P and NP rats. On the two-bottle tests, the level of alcohol consumed by the normal rats was found to be between that of the P and NP rats. The consumption score for the last 48 hr period (g/kg/48 hr; mean $\pm$ SD) was 5.31 $\pm$ 2.52. During Exposure 2 taste reactivity, normal rats' ingestive responses increased slightly but not to the level of the P rats'. For example, the number of tongue protrusions by the P rats increased 400% whereas the number of tongue protrusions by the normal rats increased only about 50%. Further, the normal rats' aversive responses decreased slightly, but the decrease was not as great as that shown by the P rats. Because normal rats consumed less alcohol than the P rats during the two-bottle test, it might be expected that their preference change during Exposure 2 taste reactivity (more ingestive responses and fewer aversive responses) would not be as great as that of the P rats, especially if the preference change was indirectly influenced by positive reinforcement.

Because P rats have been shown to meet virtually all of the requirements for an animal model of alcoholism (Li et al., 1979), an understanding of the precise factors that allow P rats to consume large quantities of alcohol would,

no doubt, lead to a greater understanding of the cause, effects, and treatment of alcoholism. It was hypothesized that the differences in alcohol consumption between the P and NP rats would, in part, be related to an innate taste difference to alcohol. However, because there were no taste reactivity differences found between naive P and NP rats on the initial test, it was concluded that an innate taste response difference did not exist. After having access to alcohol during the two-bottle test, the P rats' taste reactivity to alcohol changed. P rats began to show a preference for alcohol whereas the preference of NP rats did not change. It is possible that the P rats showed a change in preference for alcohol during Exposure 2 reactivity because they associated the taste of alcohol with its positively reinforcing effects. If this is true, it could have important implications for human alcoholism. If taste becomes associated with the unconditional effects of alcohol, and as a result, its hedonic value changes and becomes more positive, this may be an important factor in maintaining or even facilitating alcohol consumption and abuse.

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Taste Reactivity in Alcohol Preferring and Non-preferring Rats

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ABSTRACT

Taste reactivity tests were used to examine the orofacial responses of alcohol preferring (P) rats and alcohol non-preferring (NP) rats to the taste of alcohol. In the initial exposure, naive rats were tested for reactivity to five concentrations of alcohol (5%, 10%, 20%, 30%, and 40% v/v), water, and one solution each of sucrose and quinine. A two-bottle consumption test was then given for a three week period to allow the rats access to 10% alcohol. After the two-bottle preference test, a second taste reactivity test was used to examine the taste responses of alcohol experienced P rats and NP rats to the same solutions used in the initial reactivity test. The results indicated no significant differences between P rats and NP rats on the initial exposure, except that NP rats made significantly more mouth movements. During the two-bottle tests, consumption of alcohol by P rats was consistently higher than that of NP rats across all test days. P rats consumption of alcohol increased gradually, whereas NP rats consumption leveled off almost immediately. On the second taste reactivity test following alcohol access, P rats showed an increase in the number of ingestive responses and a decrease in the number of aversive responses to alcohol. NP rats taste reactivity to alcohol remained virtually the same from Exposure 1 to Exposure 2. The only exception was that NP rats made

significantly more mouth movements to alcohol on the second exposure. P rats' and NP rats' responses to sucrose and quinine did not change from Exposure 1 to Exposure 2; this indicated that the differences in reactivity between the two lines were specific to alcohol. It was concluded that there were no innate taste differences between the P and NP rats to alcohol but that other genetic factors influenced taste reactivity indirectly.