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After interacting with rat demonstrators that had eaten a novel, palatable diet, many observer rats exhibited either attenuation or total blockade of their subsequent acquisition of a lithium-chloride-induced aversion to that diet. In natural circumstances, such social attenuation of aversion learning could prevent new recruits to a population (weanlings or recent immigrants) from learning maladaptive aversions ("food phobias") to tainted or spoiled samples of normally safe foods that others of their social group were eating.

The ability of rats to learn aversions to unfamiliar foods following a single pairing of taste with toxicosis has been discussed as an adaptive specialization of Pavlovian conditioning, allowing animals to avoid repeated ingestion of toxic foods in their natural habitat (Rozin & Kalat, 1971; Shettleworth, 1984; Zahorik & Houpt, 1981). Although learning an aversion to a food following a single pairing of ingestion of that food with illness may often be advantageous, rapid acquisition of flavor aversions need not always be beneficial. A single meal of an unfamiliar safe food, eaten during the hours before onset of a bout of gastrointestinal distress induced by factors (bacterial, viral, or endoparasitic) unrelated to ingestion, could result in learning of a maladaptive "food phobia" rather than an adaptive aversion to a toxic agent. Similarly, eating a tainted first sample of a normally safe, unfamiliar food could entail prolonged future rejection of an important potential source of nutrients (see Galef, in press-a, for further discussion). The ability to respond to truly toxic foods differently than to foods only happenstantially associated with illness would reduce the potential costs of one-trial, taste-toxicosis association learning while maintaining potential benefits.

In natural circumstances, Norway rats are social animals; each rat lives as a member of a colony during at least a part of its life (Telle, 1966), and colonial living gives each rat access to information concerning the foods others of its social group are exploiting (Galef, 1983, 1986b; Galef & Wigmore, 1983; Posadas-Andrews & Roper, 1983; Strupp & Levitsky, 1984). It is relatively unlikely that the foods other rats are eating are toxic. If a toxic food were lethal, rats that had eaten it would no longer be alive. If a toxic food were noxious rather than lethal, others would probably have learned to avoid ingesting the noxious food. Thus, rats that exhibit a reluctance to form aversions to foods that others of their social group were exploiting might avoid some of the costs of one-trial, taste-toxicosis association learning resulting from the chance pairing of ingestion of an unfamiliar food with illness.

The present experiments were designed as laboratory analogues of situations in which a naive rat (an observer) that had prior contact with some food only as the result of exposure to conspecifics that had eaten that food (Galef & Stein, 1985) ingested the food and became ill (perhaps because the particular sample the observer ate was tainted, perhaps because the observer was about to suffer a bout of nausea regardless of what it ate). The goal of the experiments reported below was to determine whether prior interaction with a conspecific that had eaten a food would, like actual ingestion of a food (Revusky & Bedarf, 1967; Siegel, 1974), interfere with subsequent acquisition of an aversion to the food.

EXPERIMENT 1

In Experiment 1, demonstrator rats assigned to the experimental conditions were first fed a novel diet. Each demonstrator then interacted with a naive, conspecific observer. Observers were subsequently: (1) fed the diet that their respective demonstrators had eaten, (2) poisoned, and (3) offered a choice between the food eaten by their respective demonstrators and a completely unfamiliar food. I compared the food choices of these observers with the food choices of rats in control conditions, poisoned after eating the same food observers in the experimental groups had eaten, but not previously exposed to demonstrators. I thus determined whether information acquired from demonstrators might attenuate later taste-aversion learning by observers.

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Method

Subjects

Sixty-six experimentally naive, 42-day-old, female Long-Evans rats obtained from Charles River Canada (St. Constant, Quebec) served as observers. An additional 36, 56- to 63-day-old females of the same strain served as demonstrators in the present experiment.

Apparatus

Observers were housed individually throughout the experiment in 22×24×27.5 cm wire-mesh hanging cages. Each demonstrator was housed in a plastic shoe-box cage in a room separate from observers.

Procedure

Treatment of demonstrators and observers in each of two experimental groups (1-dem, LiCl and 2-dem, LiCl) was as follows (see Figure 1 for a schematic of the procedure used with subjects in the 1-dem, LiCl group).

Step 1. Observers and demonstrators were placed on a 23-h food-deprivation schedule and were fed powdered Purina Laboratory Rodent Chow (Diet Pur) for 1 h/day for 2 consecutive days.

Step 2. Following a third 23-h period of food deprivation of both demonstrators and observers, each demonstrator was offered a weighed food cup containing powdered Normal Protein Test Diet (Diet NPT; Teklad, Madison, WI) and, immediately after feeding, was placed in the cage of a hungry observer. Each observer assigned to the 1-dem, LiCl (n = 12) group interacted for 30 min with a single demonstrator fed Diet NPT. Each observer assigned to the 2-dem, LiCl (n = 10) group interacted for 15 min with each of two demonstrators in succession, each previously fed Diet NPT for 1 h.

Step 3. At the end of the 30-min period of interaction of each observer with a demonstrator or demonstrators, each observer was offered, for 15 min, a weighed food cup containing Diet NPT.

Step 4. At the end of this 15-min feeding period, each observer received an intraperitoneal injection of .75% of body weight (b.w.), 1% wt/vol LiCl solution.

Step 5. One hour following injection, each observer was given pellets of Purina Laboratory Rodent Chow and was left undisturbed for 23 h to recover from toxicosis.

Step 6. At the end of the 23-h recovery period, food pellets were removed from each observer's cage and replaced with two weighed food cups: one containing the averted Diet NPT, the other containing a totally novel diet (Diet Coc; powdered Purina Laboratory Rodent Chow adulterated 2% by weight with Hershey's cocoa). Observers were left to feed on Diets NPT and Coc for 22 h.

At the end of the 22-h test period, the experimenter determined the amount of Diet NPT eaten by each observer as a percentage of that observer's total intake during testing (Step 6).

Observers assigned to each of four control groups were treated identically to observers in the 1-dem, LiCl and 2-dem, LiCl groups except that observers in the first two control groups (0-dem, LiCl; n = 22) did not interact with demonstrators during Step 2 of the experiment, and observers in the second two control groups (0-dem, Sal; n = 22) did not interact with demonstrators during Step 2 and were injected with .75% of b.w. physiological saline solution, rather than LiCl solution, during Step 4 of the experiment.

Because our studies required that demonstrators eat Diet NPT during Step 2, observers eat Diet NPT during Step 3, and observers eat Diets NPT and Coc during Step 6, we excluded from analysis data from any observer (1) whose demonstrator failed to eat 2.0 g of Diet NPT during Step 2 (n = 1), (2) that failed to eat 1.0 g of Diet NPT during Step 3 (n = 1), or (3) that failed to eat a total of 5.0 g during Step 6 (n = 1).

Results

The main results of Experiment 1 are presented in Figure 2, which shows the mean amount of Diet NPT eaten as a percentage of the total amount consumed during the 22-h choice test (Step 6) by observers in each of the various groups. Because there was considerable variability in the behavior of observers in both the 1-dem, LiCl and 2-dem, LiCl groups, I have also shown in Figure 2 the individual datum points collected from the subjects assigned to those two conditions. Individual datum points reveal the effects of interaction with a demonstrator on diet choice by observers more clearly than any summary statistic.

As can be seen from inspection of Figure 2, observers in both the 1-dem, LiCl and 2-dem, LiCl groups ate a greater percentage of Diet NPT than did observers in the corresponding 0-dem, LiCl groups (Mann-Whitney U tests; see Figure 2 for U and p values). Many observers in the 2-dem, LiCl group exhibited a total blockade of aversion learning; that is, their preference for Diet NPT during testing was similar to that of subjects in the 0-dem, saline groups (which were not poisoned following ingestion of Diet NPT).

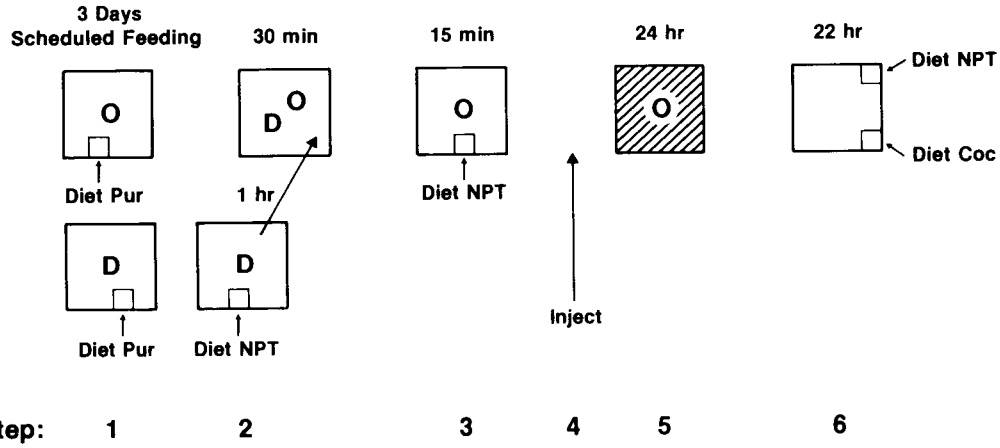

Figure 1. Schematic of the procedure of Experiments 1, 2, and 3. Cross-hatching = ad-lib access to Purina Laboratory Chow; O = observer; D = demonstrator.

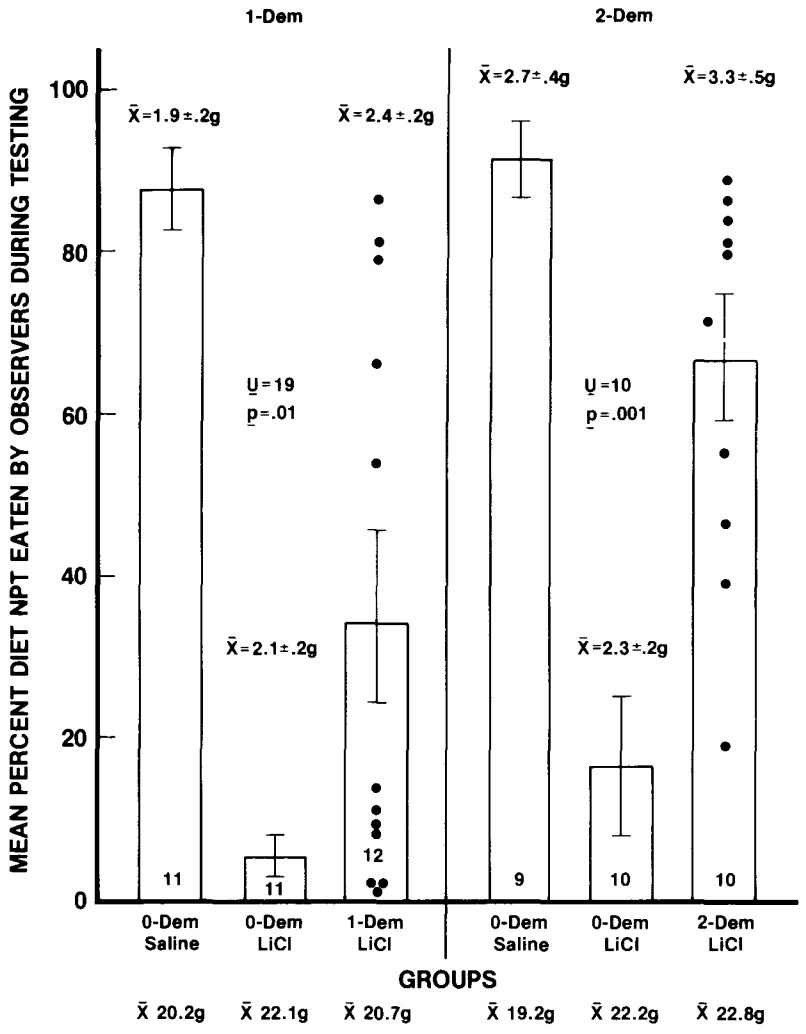

Figure 2. Mean amount of Diet NPT eaten by observers in Experiment 1 as a percentage of the total amount consumed during testing (Step 6 of procedure). Means and *SEM*s above histograms = mean amounts of Diet NPT eaten by observers during Step 3 of procedure; means below histograms = mean amounts eaten by observers during testing; numbers inside histograms = number of animals per group; points = individual datum points; flags on histograms = ±1 *SEM*.

Because animals in the 1-dem, LiCl and 2-dem, LiCl conditions were not run concurrently, it is not statistically acceptable to compare the diet preferences of observers in the two groups. However, the data do suggest that interaction with 2 demonstrators fed Diet NPT was more effective than interaction with a single demonstrator fed Diet NPT in attenuating later learning of an aversion to Diet NPT by observers (see also Galef, 1986c).

Discussion

The present results indicate that exposure of a rat to conspecifics that have eaten a diet can, like actually eating a diet (Revusky & Bedarf, 1967; Siegel, 1974), attenuate or block subsequent learning of an aversion to that diet. Diet-identifying cues experienced during social interaction act, like actual ingestion of a food, to reduce subsequent learning of an aversion to the food.

Although both eating a food and exposure to a demonstrator that has eaten a food attenuate subsequent aversion learning, the results of recent studies using the basic procedure described here indicate that social attenuation or blockade of subsequent aversion learning involves more than simple latent inhibition. Heyes and Durlach (in press) have found significantly greater attenuation of aversion learning following exposure to a demonstrator that had eaten a diet than following actual ingestion of the same diet.

EXPERIMENT 2

The results of Experiment 1, although consistent with the view that interaction of an observer with demonstra-

tor(s) that had eaten a diet attenuates subsequent learning of an aversion to that diet, are open to other interpretations. It is, for example, possible that interaction with conspecifics immediately before eating a food in a taste-toxicosis conditioning situation might interfere with a subject's subsequent aversion learning, even if the conspecifics had not eaten the food to which their observers were about to learn an aversion.

The present experiment was undertaken to determine whether the attenuation of aversion learning exhibited by observers assigned to the 1-dem, LiCl and 2-dem, LiCl groups in Experiment 1 was the result of a simple disruption of learning caused by 30 min of prior social interaction with demonstrators, rather than a specific inhibition of learning of an aversion to the particular diet previously eaten by the demonstrators.

Method

Subjects

Thirty-six experimentally naive, 42-day-old, female Long-Evans rats from the McMaster vivarium, born to breeding stock acquired from Charles River Canada, served as observers. An additional 36, 56- to 63-day-old females from the same source, which had served as observers in previous experiments, served as demonstrators in the present experiment.

Procedure

The procedure of Experiment 2 was identical to that of Experiment 1 (see Figure 1) except that an additional group of subjects was examined. The subjects in this additional group were treated exactly like the subjects in the 2-dem, LiCl group of Experiment 1 except that the two rats that interacted with each observer during Step 2 of the present experiment were not fed any food prior to interacting with their respective observers.

Results and Discussion

The main results of Experiment 2 are presented in Figure 3, which shows the mean amount of Diet NPT eaten by observers as a percentage of the total amount they ingested during the 22-h test period. As inspection of Figure 3 reveals, observers interacting with demonstrators fed Diet NPT exhibited an attenuation of their subsequent aversion learning to Diet NPT. Observers that interacted with demonstrators fed nothing (those in the Nothing-LiCl group in Figure 3) did not exhibit a similar attenuation of their subsequent aversion learning.

The results of the present experiment are not consistent with the hypothesis that the attenuated aversion learning exhibited by observers in the 1- and 2-dem, LiCl groups of Experiment 1 was the result of simple disruption of taste-toxicosis conditioning by immediately antecedent social contact with conspecifics.

EXPERIMENT 3

If social attenuation of taste-aversion learning plays a role in diet selection by rats outside the laboratory, then the effects on observers' aversion learning of interaction with a demonstrator that has eaten a diet should last for many hours. In the present experiment, I introduced a delay of 24 h between the interaction of observers with demonstrators fed Diet NPT and the conditioning of an aversion to Diet NPT in those observers. Such a procedure also provides a second test of the hypothesis rejected on the basis of the results of Experiment 2, that social attenuation of aversion learning is the result of social interaction simply disrupting subsequent taste-aversion learning.

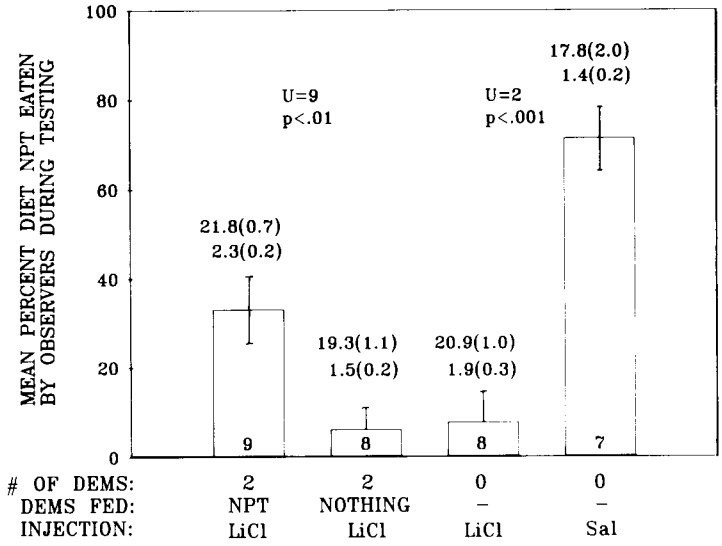

Figure 3. Mean amount of Diet NPT eaten by observers in Experiment 2 as a percentage of total intake during testing (Step 6 of procedure). Means and *SEMs* (in parentheses) above histograms = mean total amounts eaten by observers during testing (upper number) and mean amounts of Diet NPT eaten by observers during Step 3 of procedure (lower number); numbers inside histograms = number of animals per group; flags on histograms = ±1 *SEM*.

It is very unlikely that simple interaction with a conspecific would disrupt taste-aversion learning 24 h later.

Method

Subjects

Sixteen experimentally naive, 42-day-old, female Long-Evans rats from the McMaster vivarium served as observers. An additional 16, 56- to 63-day-old females from the same source, which had served as observers in previous experiments, served as demonstrators in the present experiment.

Procedure

The procedure of Experiment 3 was identical to that of Experiment 1 (see Figure 1) except that (1) only 0-dem, LiCl and 2-dem, LiCl groups were examined, and (2) a 24-h delay was imposed between the interaction of observers with demonstrators fed Diet NPT (Step 2) and Diet NPT-aversion conditioning (Steps 3 and 4). At the beginning of the 24-h delay, observers were fed powdered Purina Laboratory Rodent Chow for 2 h. They were food-deprived for the remaining 22 h of the delay period.

Results and Discussion

The main results of Experiment 3 are presented in Figure 4, which shows the mean amount of Diet NPT eaten by observers as a percentage of the total amount ingested by observers during Step 6, the 22-h test period. As can be seen in Figure 4, a delay of 24 h between interaction of demonstrators fed Diet NPT and their observers did not eliminate the effects of interaction with Diet NPT-fed demonstrators on subsequent learning of an aversion to Diet NPT by their observers ($U = 4$, $p < .002$).

The present results, like those of Experiment 2, are not consistent with the hypothesis that social interaction per se was responsible for the socially induced attenuation of aversion learning seen in Experiments 1, 2, and 3. Furthermore, the finding that interaction with a demonstrator fed a diet attenuates subsequent learning of an aversion to that diet for at least 24 h increases the probability that such social effects on aversion learning might occur in natural circumstances.

EXPERIMENT 4

The observers in Experiments 1-3 above can be thought of as caught up in a conflict situation; the observers' own experiences with respect to ingestion of Diet NPT lead them to avoid further ingestion of that diet, whereas the observers' social interactions with demonstrators fed Diet NPT lead them to eat Diet NPT. If such a conceptualization is useful, then the magnitude of social attenuation of aversion learning should decrease as the severity of induced aversions increases; social attenuation of aversion learning should be more pronounced with relatively mild than with relatively strong aversions. In the present experiment, I examined the effects of the magnitude of the aversive US to which observers were exposed during taste-toxicosis conditioning on social attenuation of subsequent aversion learning. My purpose was to determine whether the severity of the US might provide a boundary condition on social attenuation of aversion learning.

Method

Subjects

Sixty experimentally naive, 42-day-old, female Long-Evans rats obtained from Charles River Canada served as observers. An additional 84, 56- to 63-day-old females of the same strain, which had served as observers in previous experiments, served as demonstrators in the present experiment.

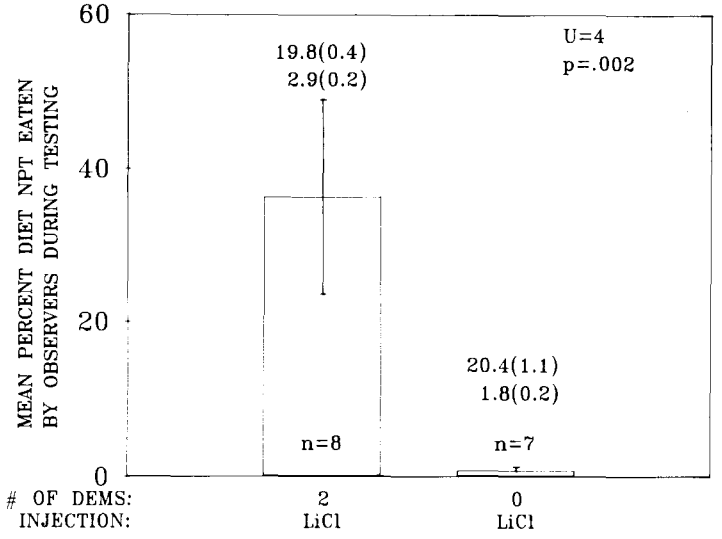

Figure 4. Mean amount of Diet NPT eaten by observers in Experiment 3 as a percentage of total intake during testing (Step 6 of procedure). Means and *SEMs* (in parentheses) above histograms = mean total amounts eaten by observers during testing (upper number) and mean amounts of Diet NPT eaten by observers during Step 3 of procedure (lower number); flags on histograms = ±1 *SEM*.

Procedure

The procedure was identical to that of Experiment 1 except in the quantities of LiCl and saline solutions injected into observers during Step 4. In the present experiment, the aversion learning of subjects in three experimental conditions and three control conditions was examined. Each observer in each experimental group interacted with 2 Diet NPT-fed demonstrators during Step 2. The three experimental groups differed only in the percentage of body weight of 1% w/vol LiCl solution with which observers were injected during Step 4: .75% (Group 2-dem, .75% LiCl; n = 12), 1.0% (Group 2-dem, 1% LiCl; n = 12), and 2.0% (Group 2-dem, 2% LiCl; n = 9). The subjects in the three control conditions interacted with no demonstrators during Step 2 and differed in the substances with which they were injected during Step 4: (1) .75% b.w. of 1% w/vol LiCl solution (Group 0-dem, .75% LiCl; n = 12), (2) 1.0% b.w., 1.0% w/vol LiCl solution (Group 0-dem, 1% LiCl; n = 9), or (3) 1.0% b.w. saline solution (Group 0-dem, saline; n = 9). I did not examine the behavior of any subjects interacting with no demonstrators and injected with 2.0% b.w. 1.0% w/vol LiCl solution (Group 0-dem, 2% LiCl) because I knew from past experiments that even subjects in Control Group 0-dem, 1% LiCl would eat essentially no Diet NPT, and exposure of additional animals to toxicosis could, therefore, not be justified. The experimenter determined the amounts of Diet NPT and of Diet Coc eaten by observers 22 h after initiation of the choice test (Step 6).

Results and Discussion

The main results of Experiment 4 are presented in Figure 5, which shows the mean amount of Diet NPT eaten as a percentage of the total amount ingested by observers in the three experimental and the three control groups during the 22-h test period. As inspection of Figure 5 reveals, the observers in Experimental Group 2-dem, .75% LiCl ate more Diet NPT during testing than did the subjects in Control Group 0-dem, .75% LiCl ($U = 26, p < .01$). Similarly, the observers in Experimental Group 2-dem, 1% LiCl ate a greater percentage of Diet NPT during testing than did the subjects in Control Group 0-dem, 1% LiCl ($U = 13, p < .01$). As was the case in Experiment 1, interaction with demonstrators fed Diet NPT attenuated aversion learning to Diet NPT.

As can also be seen in Figure 5, the effectiveness of interaction with two demonstrators fed Diet NPT in reducing aversion learning to Diet NPT by observers depended on the amount of LiCl observers received during Step 4 of the experiment. The greater the dosage of LiCl injected into observers in experimental groups during Step 4, the

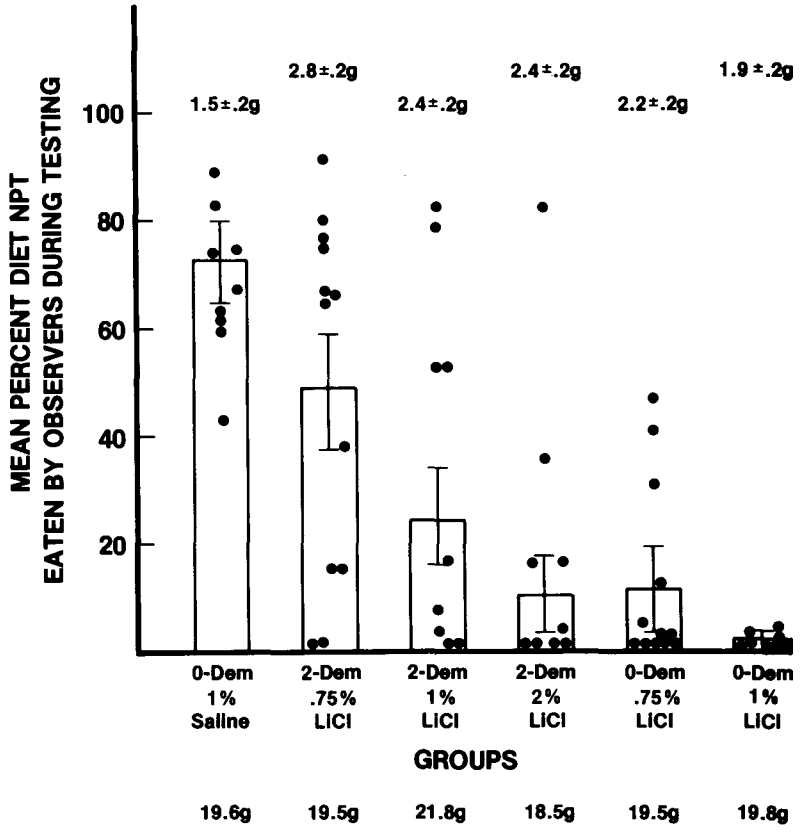

Figure 5. Mean amount of Diet NPT eaten by observers in Experiment 4 as a percentage of the total amount consumed during testing (Step 6 of procedure). Means and *SEMs* above histograms = mean amounts of Diet NPT eaten by observers during Step 3 of procedure; means below histograms = mean amounts eaten by observers during testing; points = individual datum points; flags on histograms = ±1 *SEM*.

less Diet NPT they ate during Step 6 [extension of the median test, $\chi^2(2) = 10.9$, $p < .01$]. Relatively mild aversions were attenuated by prior interaction with demonstrators fed Diet NPT more effectively than were relatively strong aversions. I detected no effect of interaction with demonstrators on intake of Diet NPT by observers in Group 2-dem, 2% LiCl; during testing, these observers did not exhibit measurable intakes of Diet NPT.

GENERAL DISCUSSION

The results of the experiments described above indicate that interaction with conspecifics previously fed a diet can attenuate subsequent learning of an aversion to that diet. The data also show that the extent of such socially mediated attenuation of aversion learning increases with a decrease in the strength of toxicosis. Thus, within a restricted range of conditions, a naive rat could be protected from learning food phobias to safe, novel foods by prior interaction with conspecifics that had eaten those foods. If a rat should happen to experience a relatively mild malaise after its first meal of a novel food, it can use its previous experience with others that ate that novel food to avoid learning a maladaptive food phobia.

The most important question unanswered by the present experiments concerns the behavioral processes supporting the observed social attenuation of later aversion learning: Are observers simply showing latent inhibition resulting from exposure to residual, diet-related cues passively carried by demonstrators, or is the presence of a demonstrator carrying the residual, diet-related cues somehow critical to the observed attenuation of aversion learning?

In a series of published studies investigating the behavioral processes supporting enhancement of observers' preferences for foods eaten by their respective demonstrators (see Galef, 1986a, for review), my co-workers and I have repeatedly found that simple exposure to a food does not enhance preference for that food nearly to the extent that interaction with a demonstrator that has eaten a food does (Galef, in press-b; Galef & Kennett, 1987; Galef, Kennett, & Stein, 1985; Galef, Mason, Preti, & Bean, 1988; Galef & Stein, 1985). If one assumes, as seems likely, that the behavioral processes mediating social enhancement of food preferences also mediate social attenuation of later aversion learning, then one would expect exposure to a demonstrator that had eaten a food to be more effective than simple exposure to the food itself in attenuating later aversion learning to that food, and this is what Heyes and Durlach (in press) have found. The weight of evidence thus suggests that something more than simple latent inhibition is responsible for social attenuation of aversion learning of the sort described in Experiments 1–4 above.

The present paper was, however, intended to provide evidence of a significant social contribution to the prevention of the acquisition of useless food aversions by rats rather than as an analysis of the behavioral processes underlying such social influences on behavior. The data presented above are consistent with the view that social interaction of a naive rat with a more experienced conspecific can reduce the probability that the naïf will acquire phobic aversions to safe foods.

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