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Social Blockade of Taste-Aversion Learning in Norway Rats (*Rattus norvegicus*): Is It a Social Phenomenon?

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In Experiment 1, hooded rats (*Rattus norvegicus*) were exposed to a novel diet in a food dish or on a conspecific; they were allowed to consume the same diet and then were injected with a toxin LiCl. Later both groups ate more of the novel diet than animals that had not been exposed, and the conspecific-exposed group ate more than the dish-exposed group. Reducing aversion learning by exposure on a conspecific is known as *social blockade.* We examined if this effect is because a conspecific intensifies dietary cues and thereby increases latent inhibition. Experiment 2 failed to show that diet on a conspecific is a more effective conditioned stimulus for taste-aversion learning than diet in a dish, and Experiment 3 showed that diet in a dish is an effective overshadowing stimulus in aversion learning but diet on a conspecific is not. These results suggest that social blockade cannot readily be assimilated to a latent-inhibition model and may be a distinctly social form of learning.

In a series of studies motivated by consideration of the natural ecology of the Norway rat, Galef and his associates have provided laboratory demonstrations of a range of behavioral processes whereby the dietary preferences and foraging behavior of both preweanling juvenile and adult rats may be influenced by interaction with a conspecific that is consuming or has recently consumed a novel diet (e.g., Galef, 1977, 1983; Galef & Clark, 1972). The most recent studies have focused of diet in favor of that eaten by the conspecific. Galef and Wigmore found that this social effect on dietary preferences is disrupted by rendering observers anosmic before the interaction and by separation of the observer and demonstrator with a Plexiglas screen, but the effect is sustained when the demonstrator is anesthetized after it has eaten the target diet and when the demonstrator and the observer are separated by a hardware cloth screen. They concluded that information

group interacted with a demonstrator that had eaten a novel diet. When the interaction had ceased, the observer was allowed to eat some of the diet that the demonstrator had eaten, was immediately poisoned, and then after a recovery period, was offered a choice between the food eaten by their demonstrator and another, completely novel, diet. During the choice test these experimental subjects consumed a significantly greater proportion of the diet they ate immediately before toxicosis than control rats that did not interact with a demonstrator during the initial phase of the procedure.

The aim of the present series of experiments was to replicate Galef's (in press) social blockade effect and to investigate the mechanism through which it occurs. Specifically, we sought to establish whether cues provided by another animal are an essential or an incidental component of this mechanism.

Experiment 1

The first experiment was conducted in an attempt to replicate Galef's (in press) social blockade effect, that is, to show that preexposure to a conspecific that has eaten a novel diet can reduce subsequent aversion learning to that diet. It also compared the extent to which aversion learning is reduced by preexposure to dietary cues in a social context (on a demonstrator) and in an asocial context (in a dish).

Method

Subjects

Sixty-four, experimentally naive, male, Long-Evans rats (*Rattus norvegicus*) obtained from Charles River UK (Margate, Kent, United Kingdom) served as observers, and an additional 32 males of the same strain served as demonstrators. When the experiment began, the observers' weights ranged from 115 g to 170 g.

Apparatus

Observers and demonstrators were housed individually and in separate rooms throughout the experiment, on a 12:12-hr light/dark cycle (lights on at 900 hr), and in plastic and wire mesh hanging cages that measured 28 cm wide × 22 cm high × 41 cm deep. At certain stages in the procedure, one or two metal food dishes were screwed to the grill floor of each cage.

Two novel diets were used. They were powdered laboratory maintenance diet adulterated either 1% by weight with Schwartz ground cinnamon (Cin) or 2% by weight with Sainsbury's cocoa (Coc).

Procedure

The experiment had a 2 × 2 factorial design. The variables were: exposure, in which animals were preexposed to traces of a novel diet, the target diet (EXP) or to traces of the familiar laboratory maintenance diet (NEXP); and carrier, for which the preexposed diet was carried either by a conspecific demonstrator that had recently consumed some of that food (DEM) or by a familiar food dish (DISH). The counterbalanced variables were target diet (Cin or Coc) and the position of the target diet during the choice test (left or right).

A period of scheduled feeding preceded the experiment proper for both observers and demonstrators. They were given familiar laboratory maintenance diet in powdered form for 1 hr each day in a single dish in their home cages. This dish remained in the rat's cage throughout the preexperimental period. The experiment began on the 5th day of scheduled feeding for demonstrators and on the 3rd day of scheduled feeding for observers. The experimental procedure was as follows.

Stage 1. At the time when the observers had begun to eat on the 2 previous days, either a demonstrator or a trace of diet was placed in each of their home cages. Immediately before being presented to the observers, the demonstrators had been allowed 45 min in which to eat either a novel diet (EXP–DEM) or powdered laboratory maintenance diet (NEXP–DEM). Those observers that were not presented with a demonstrator had 0.1 g of a novel diet (EXP–DISH) or of powdered laboratory maintenance diet (NEXP–DISH) placed in their food dish. This amount was chosen because it was the minimum quantity of food that could be measured with accuracy. Each observer or observer–demonstrator pair was left undisturbed for 30 min.

Stage 2. The demonstrators were removed from the cages of animals in the EXP–DEM and NEXP–DEM groups, and 20 g of diet were placed in each observer's food dish. The observers that had been preexposed to a novel diet at Stage 1 were given the same novel diet to eat at Stage 2, and equal numbers of observers that had not been preexposed to a novel diet at Stage 1 were given Cin or Coc at Stage 2. The subjects were left to eat the diet that they had been given for 15 min.

Stage 3. Each observer was given an ip injection (1% of body weight) of 1% lithium chloride (LiCl) solution and returned to its home cage from which the food dish and its contents had been removed. One hr later each animal was given food pellets in the cage hopper and left undisturbed to recover from toxicosis until the next day.

Stage 4. Twenty-four hr after the observers had been injected, the food pellets were removed from the observers' hoppers, and each animal was presented with two food dishes, one that contained 30 g of Cin and the other, 30 g of Coc. Twenty-four hr later the dishes were removed, and the amount of each diet consumed was measured.

Results and Discussion

The main results of Experiment 1 are presented in Figure 1, which shows for each group the mean amount of the target diet eaten at Stage 4 (during the 24-hr choice test) as a percentage of the total amount consumed during that period. The results indicate that preexposure to the diet that was paired with toxicosis reduced subsequent aversion learning, and they provide some evidence that the magnitude of this effect was greater when the preexposed diet was carried by a demonstrator than when it was carried by a dish.

Animals that were preexposed to the target diet ate a greater percentage of that diet (EXP $M = 46.9\%$) than nonpreexposed animals (NEXP $M = 24.3\%$), $F(1, 10) = 63.79$, $p = .00001$, and animals that were preexposed to the target diet on a demonstrator ate a greater percentage of that diet (EXP–DEM $M = 51.1\%$) than animals that were preexposed to the target diet in a dish (EXP–DISH $M = 42.8\%$), $F(1, 10) = 5.93$, $p = .035$.

Figure 2 shows the mean weight of the target diet eaten by each group at Stage 2 in the 15 min after the preexposure phase. These data were examined to find out whether the observed effects of preexposure on test performance were mediated by its effects on the amount of target diet eaten before poisoning. Animals that had been preexposed to the

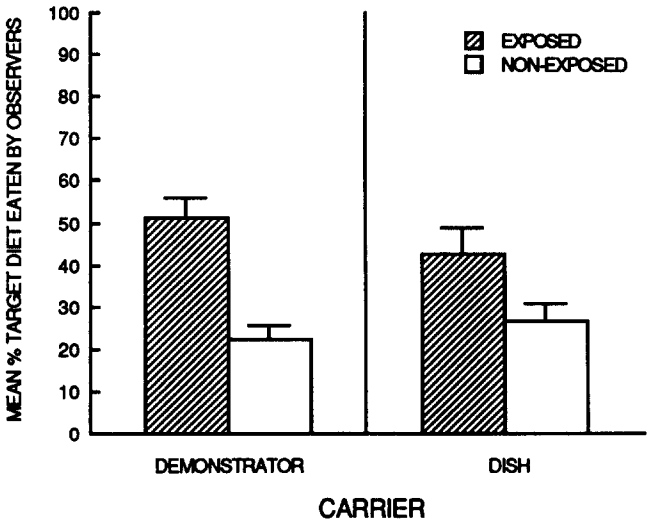

Figure 1. Mean amount of target diet eaten as a percentage of total amount eaten during choice test in Experiment 1. (Bars indicate *SE*. $N = 16$.)

target diet ate more before they were poisoned (EXP $M = 2.62$ g) than animals that had not been preexposed (NEXP $M = 2.2$ g), $F(1, 10) = 20.29$, $p \leq .001$, but there was no evidence that EXP–DEM and EXP–DISH groups differed in this respect. This suggests that although the main effect of exposure on postinjection consumption of the target diet may be attributable to its effect on preinjection intake, the difference between EXP–DEM and EXP–DISH groups in postinjection consumption is not similarly reducible.

Thus, Experiment 1 replicated Galef's (in press) social-blockade effect by showing that preexposure to a conspecific that has consumed a novel diet can reduce subsequent aversion learning to that diet. It also provided some evidence that preexposure to a novel diet on a conspecific reduces subsequent aversion learning more than preexposure to a 0.1 g of the novel diet in an asocial context, that is, in a familiar food dish.

Experiment 2

Galef (in press) suggested that there is a component of rat breath that makes simultaneously presented diets attractive. This implies that a component of rat breath can act as an appetitive unconditioned stimulus and that associative interference (Scavio, 1974) may be the mechanism that mediates social blockade of taste-aversion learning. That is, an association between a diet and rat breath may be formed during preexposure, and this conditioning may interfere proactively with the establishment of an association between the same diet and toxicosis. Alternatively, social blockade of aversion learning may be simply an instance of latent inhibition, or a decline in the associability of a stimulus as a result of unreinforced presentation (Best & Gemberling, 1977; Lubow, 1973). According to this interpretation, specifically social cues do not play an important role in social blockade, and the same mechanism is responsible for a reduction in aversion learning after preexposure to the diet on a demonstrator and in a dish.

Experiment 1 provided evidence that demonstrator preexposure has a greater effect on subsequent aversion learning than does dish preexposure, but this does not justify the conclusion that the demonstrator preexposure, or social-blockade, effect was mediated by a different mechanism, that is, associative interference rather than latent inhibition. The magnitude of latent-inhibition effects increases with stimulus salience. (*Salience* is defined in relation to the intensity of the preexposed stimulus, the extent to which it is processed, or the duration of the interstimulus interval.) A number of factors may have contributed to making demonstrator-presented diet more salient in Experiment 1: (a) A demonstrator may have carried more particles of the diet than a dish; (b) more of the particles that it did carry may have been convected toward the observer as a consequence of the demonstrator's respiration and body temperature; (c) because a trace of diet presented in a dish may be rapidly eaten, demonstrators may have provided a more durable source of dietary cues; and (d) the presence of the demonstrator may have heightened the observer's arousal or made it attend more to the dietary stimuli so that they were processed more thoroughly.

However, two considerations argue against the salience hypothesis, that is, the view that latent inhibition is responsible for the reduction of aversion learning after preexposure to a diet on a demonstrator and in a dish, but that the former effect was stronger in Experiment 1 because the demonstrators were a source of more salient dietary stimuli than the dishes. First, latent inhibition tends to be context specific (Hall & Channell, 1986; Lovibond, Preston, & Mackintosh, 1984), and the preexposure and conditioning contexts were more alike for dish-preexposed animals than for demonstrator-preexposed animals. Thus, if latent inhibition alone were occurring, one would have expected the dish-exposed animals to show less, not more, aversion learning. Second, dish and demonstrator preexposure had equivalent effects on preinjection consumption of the target diet, which suggests that the

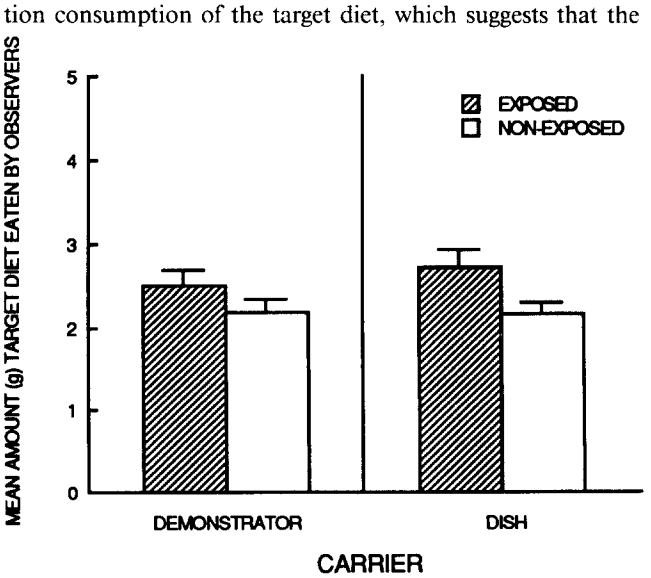

Figure 2. Mean amount in g of target diet eaten prior to LiCl injection in Experiment 1. (Bars indicate *SE*. $N = 16$.)

preexposed stimuli were equally salient. However, these considerations are not conclusive. If demonstrators provide a source of substantially more salient cues, then the effects of context change might be submerged, and failure to find a difference between dish and demonstrator groups in the amount consumed immediately after preexposure may be due to insensitivity of the test.

Experiment 2 was designed as a direct test of the salience hypothesis. Instead of being preexposed to the target diet, eating it, and then being poisoned, as they were in Experiment 1, the subjects in Experiment 2 were exposed to the diet in a dish or on a demonstrator and then immediately injected with LiCl. Thus, the dietary cues presented on a dish or a demonstrator acted as the conditioned stimulus for conditioning, and it was predicted that if those presented on a demonstrator were more salient, then demonstrator-exposed animals would subsequently show a stronger aversion to the target diet than dish-exposed animals.

Method

Subjects and Apparatus

Thirty-two, experimentally naive, male, hooded, Lister rats obtained from Harlan Olac Limited (Bicester, Oxfordshire, United Kingdom) served as observers, and an additional 16 males of the same strain served as demonstrators. When the experiment began, the observers' weights ranged from 340 g to 440 g. The same apparatus as that described in Experiment 1 was used.

Procedure

The design and procedure were similar to those of Experiment 1 except that Stage 2 was omitted from the procedure. Thus, when each observer had been exposed to traces of either a novel target diet (EXP) or familiar laboratory maintenance diet (NEXP) for 15 min, it was immediately injected with LiCl. One day later the subjects were given a 24-hr choice test.

Results and Discussion

Figure 3 shows for each group the mean amount of the target diet eaten during the choice test as a percentage of the total amount eaten. If in the paradigm used in Experiments 1 and 2, demonstrators were sources of more salient dietary cues than dishes, then one would expect the DEM groups to show more aversion learning than the DISH groups. In fact, it appears that the difference between the DEM–EXP and DEM–NEXP groups is no greater than that between the DISH–EXP and DISH–NEXP groups.

These impressions were confirmed by a 3-way analysis of variance that had diet as a within-subjects variable and exposure and carrier as between-subjects variable. The only significant effect was that of exposure (EXP M = 21.6% and NEXP M = 56.9%), $F(1, 6) = 13.83$, $p \leq .01$. Subsequent analysis of the simple effects provided evidence of aversion learning in both the EXP–DISH and EXP–DEM groups by showing that on average the animals in each of these groups ate a

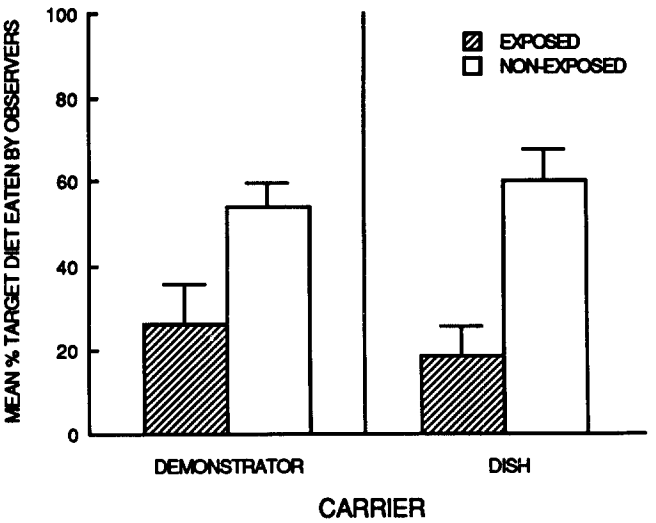

Figure 3. Mean amount of target diet eaten as a percentage of total amount eaten during choice test in Experiment 2. (Bars indicate *SE*. $N = 8$.)

significantly smaller percentage of the target diet than their NEXP controls (DISH–EXP M = 17.2%, and DISH–NEXP M = 60.0%), $F(1,6) = 12.72$, $p \leq .05$, and (DEM–EXP M = 25.9%, and DEM–NEXP M = 53.9%), $F(1, 6) = 11.37$, $p \leq .05$. The latter effect confirms the observation that "cues emitted by one rat, reflecting the identity of the diet that rat has recently eaten, form an adequate conditional stimulus for toxicosis-based aversion learning" (Galef, Wigmore, & Kennett, 1983, p. 362).

Experiment 3

Experiment 2 found no difference in the extent of aversion learning that results from presentation of a novel diet in a dish and on a demonstrator immediately before administration of LiCl. This suggests that the demonstrators in Experiment 2 were not a source of more salient dietary cues than the dishes. Given that the parameters used in Experiments 1 and 2 were so similar, this in turn makes it unlikely that the demonstrator-preexposed animals in Experiment 1 showed less aversion learning because the demonstrators made the preexposed stimuli more salient. However, as a null result, the outcome of Experiment 2 does not conclusively disconfirm the salience hypothesis as an account of social blockade of aversion learning.

Seeking more conclusive evidence, we conducted Experiment 3 to test the salience hypothesis by using an overshadowing design (Mackintosh, 1971). Overshadowing occurs when two stimuli are conditioned in compound and each acquires less associative strength than when it is conditioned alone. In Experiment 3, animals ate a novel diet (the target) and then after a delay were injected with LiCl. During the latter part of the delay, half of the animals were exposed to a trace of another novel diet, either in a dish (EXP–DISH) or on a demonstrator (EXP–DEM). The remaining animals were exposed to familiar laboratory maintenance diet in a dish (NEXP–

DISH) or on a demonstrator (NEXP–DEM). In effect, this procedure provided animals in exposed groups with two sets of dietary stimuli that could compete for association with toxicosis. On the basis of existing studies of overshadowing, one would predict that the result would be that the exposed animals would subsequently show less aversion to the target diet than would nonexposed controls, and because the extent of overshadowing is a function of the intensity of the additional stimulus (Mackintosh, 1976), one would expect this effect to be more pronounced in the demonstrator groups if the salience hypothesis is correct.

Method

Subjects and Apparatus

Thirty-two, experimentally naive, male, hooded, Lister rats obtained from Harlan Olac Limited served as observers, and an additional 16 males of the same strain served as demonstrators. The observers' weights ranged from 385 g to 525 g when the experiment began. The apparatus was the same as that described in Experiment 1.

Procedure

The design was similar to that of Experiment 1 except that diet was not counterbalanced. All observers had Cin as their target diet and those in the EXP groups were subsequently exposed in a dish or on a demonstrator to 4% Coc. The design was simplified in this way because the previous experiments had failed to show a three-way interaction between diet, exposure, and carrier variables.

Stage 1. At the time when the observers had begun to eat on the 4 previous days, each was presented with 20 g of Cin in the dish in its home cage and allowed to eat for 15 min. At the end of this period, the dish was replaced with a clean one, and the observers were left undisturbed for 30 min.

Stage 2. After the delay either a conspecific demonstrator or a trace of diet was introduced into each observer's cage. The demonstrators had been allowed to eat either Coc (EXP–DEM) or familiar laboratory maintenance diet (NEXP–DEM) for 45 min immediately before they were presented to the observers. The traces were either of Coc (EXP–DISH) or of familiar laboratory maintenance diet (NEXP–DISH).

Stage 3. Fifteen min after they had been presented, the demonstrators and the traces were removed from the observers' cages. Each observer was given an ip injection (1% of body weight) of 1% lithium chloride (LiCl) solution and left to recover for 4 hr before being given access to food pellets in its cage hopper for 1 hr.

Stage 4. Next day, at the time when they were usually fed and after 18 hr of food deprivation, each observer was presented with one food dish that contained 20 g of Cin diet. This dish was removed 15 min later, and the amount of diet consumed was measured.

Results and Discussion

Figure 4 shows the mean amount of Cin diet consumed by each group at the 15-min test in Stage 4 of the procedure.

Inspection of Figure 4 suggests that counter to the prediction based on the salience hypothesis, exposure to Coc in a dish before poisoning reduced aversion learning to Cin, whereas exposure to Coc on a demonstrator did not. Thus, it would appear that the DISH groups, but not the DEM groups, provided evidence of overshadowing. Two-way analysis of variance followed by an analysis of the simple effects of exposure confirmed these impressions by showing that although there was a significant main effect of exposure (EXP M = 3.1 g and NEXP M = 2.4 g), $F(1, 7) = 36.12$, $p = .0005$, the EXP–DISH group (M = 3.3 g) ate significantly more than the NEXP–DISH group (M = 2.1 g), $F(1, 7) = 12.97$, $p \leq .01$, whereas the EXP–DEM group (M = 2.9 g) and NEXP–DEM group (M = 2.7 g) did not eat significantly different amounts, $F(1, 7) = 0.80$, *ns*.

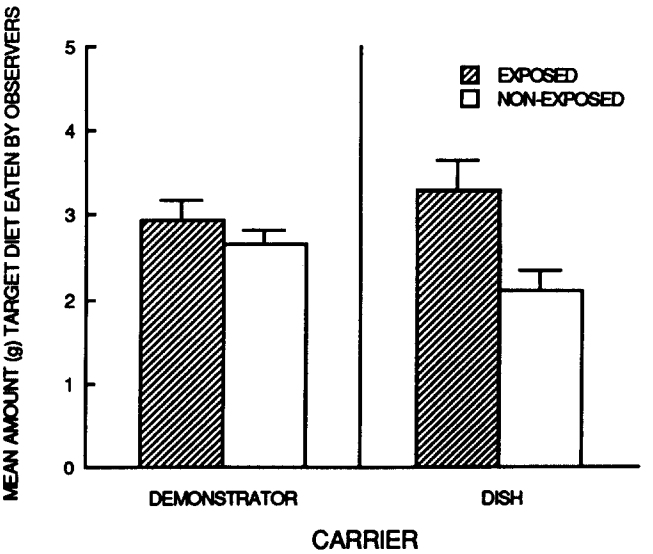

Figure 4. Mean amount in g of target diet eaten on test in Experiment 3. (Bars indicate *SE*. N = 8.)

The observed effects on the amount of Cin diet eaten on test cannot be attributed to differences in consumption of that diet during training because none were reliable (EXP–DISH M = 3.5 g, NEXP–DISH M = 3.0 g, EXP–DEM M = 3.4 g, and NEXP–DEM M = 3.5 g).

General Discussion

Experiment 1 showed that preexposure to a novel diet can reduce subsequent aversion learning to that diet both when it is presented on a demonstrator and in a dish and provided evidence that preexposure on a demonstrator results in a greater reduction than preexposure in a dish. Experiments 2 and 3 tested the salience hypothesis; the hypothesis that demonstrator preexposure had a stronger effect in Experiment 1 because demonstrators make dietary stimuli more salient and thereby increase latent inhibition. With parameters very similar to those of Experiment 1, Experiment 2 confirmed that diet on a demonstrator is a sufficient conditioned stimulus for aversion learning (Galef et al., 1983) but failed to find any evidence that diet on a demonstrator supports more aversion learning than diet in a dish; Experiment 3 provided evidence that diet in a dish is an effective overshadowing stimulus for taste-aversion learning, although it failed to find evidence that diet on a demonstrator has the same property. The results of both experiments are inconsistent with the

salience hypothesis that predicts that as compared with diet in a dish, diet on a demonstrator would support more aversion learning when it is a conditioned stimulus in a simple conditioning paradigm and more overshadowing when it is presented after the target diet and before reinforcement. Thus, the results of the present series of studies replicate Galef's (in press) social blockade of taste-aversion learning effect and render implausible an account of that effect in terms of a familiar, asocial learning phenomenon, namely, latent inhibition.

If, as we have suggested, presentation on a demonstrator does not make dietary cues more salient, then it is possible that it reduces subsequent aversion learning by making the preexposed diet attractive. One way in which such an effect may be mediated is through the process of associative interference (Scavio, 1974). Thus, a component of rat breath may act as an unconditioned stimulus, which when it is presented in conjunction with dietary stimuli, supports excitatory conditioning. This conditioning can then interfere proactively with the establishment of an association between the diet and toxicosis, which results in the formation of a milder aversion. Compared with latent inhibition, associative interference is relatively context independent (Kaye, Preston, Szabo, Druiff, & Mackintosh, 1987). Consequently, associative interference offers an account of social blockade of aversion learning that is more compatible with the observation, made in Experiment 1, that preexposure on a demonstrator reduces aversion learning more than preexposure in a dish, even though the former treatment involves a greater context shift between preexposure and conditioning.

The possibility that social blockade of aversion learning is due to associative interference is of interest both because rat breath is not widely recognized as having reinforcing properties and because cases of associative interference in which prior appetitive conditioning reduces subsequent aversive conditioning have been elusive (Mackintosh, 1983). Konorski and Szwejkowska (1952) reported that leg flexion to a conditioned-stimulus-signalling shock was retarded if that conditioned stimulus had previously signalled food, but their finding has not been directly confirmed. In advance of further studies to investigate the possibility that social blockade of taste-aversion learning is due to associative interference, we can conclude that it is a phenomenon of potential ecological

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