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8. Since the electric organs of most weakly electric fish, excluding the apteronotids, are modified muscles, the normal discharge can be eliminated with curarelike drugs such as Flaxedil or Alloferin.
9. An *S2/S1* ratio of 0.5 was chosen for this figure; with this large *S2* intensity, the steepness of the slopes of the phase function is significantly different in the regions of 0 and π. Since the direction of the phase shift in the regions of 0 and π is also opposite for opposite signs of ΔF, the fish could theoretically decode the sign of the ΔF from the phase function alone. Behavioral experiments (*7*) show that the phase information alone is insufficient for evoking the JAR.
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11. It is likely that these torus sign-sensitive cells are the same as the torus cells previously shown to respond to beats of opposite sign with large (160°) phase shifts of peak activity within the beat cycle when quasi-natural (slant-clipped sine-wave) stimulation is used (*6*).
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Recall (Versus Recognition) of Taste and Immunization Against Aversive Taste Anticipations Based on Illness

Abstract. *Two experiments show that, after taste-aversion conditioning, rats can use external retrieval cues to recall or anticipate the aversive taste solution and avoid its location without making contact with the flavor. They also show that the rat's avoidance of a conditioned aversive taste and its consumption of the aversive flavored solution can be attenuated by giving it prior runway training in which taste reward is given inconsistently on a partial reinforcement schedule.*

It is well known that animals can learn to suppress the intake of flavored solutions associated with x-irradiation (*1*) and poisons (*2*). It has not been demonstrated that animals can avoid a flavor on the basis of the anticipation of its conditioned aversive taste. With few exceptions (*3*), the measurement of a taste aversion has been in terms of the suppression of fluid intake. The animals have first to make a direct contact with or "recognize" the taste or odor of the fluid; then they show suppression. We have found that rats could anticipate the upcoming aversive taste, or, in more cognitive terms, that rats could use alley cues to recall the memory of a taste. In a second experiment, we found that the learning or expression (or both) of a taste aversion could be attenuated by prior behavioral procedures. [Radiation-induced (*4*) and poison-induced (*5*) aversions can be blocked or attenuated by chemical agents.] The prior behavioral treatment in experiment 2 was partial reinforcement (PRF) training using as reward the particular flavored solution to which the aversion is conditioned.

The general experimental sequence of both experiments had three phases: (i) thirsty rats were trained to run in an alley for a flavored solution as reward; (ii) that taste was then made aversive through a conditioning procedure in the home cages; and (iii) the approach response to the flavored solution, learned in phase 1, was extinguished. The experiment was conducted during the light phase (0800 to 2200 hours) of a light-dark cycle. The training apparatus was a straight alley runway with a clear Plexiglas top and a black interior. It was 194 cm long, 7 cm wide, and 7.3 cm high. The first 35 cm comprised the start box and the last 35 cm comprised the goal box. Both compartments were separated from the run segment (alley) by guillotine doors. A round metal cup, 2.7 cm in diameter, 1 cm deep, and 1.5 cm above the floor, was attached to the end wall of the goal box. The time-scoring system began when the start door was mechanically lowered by pressing a button. Three light beams, positioned at 30 cm, 122 cm, and 152 cm from the start door, controlled three electric clocks that recorded start, run, and goal times to 0.01 second.

In experiment 1, 48 60- to 70-day-old female Sprague-Dawley rats bred in our laboratory were run in three squads of 16. Deprived of water for 24 hours, the rats were placed four times in the goal box with 1 ml of a 1.5 percent solution of vinegar (by volume in tap water) as a reward. (i) Phase 1 runway acquisition training, initiated 24 hours after goal box training, consisted of 30 continuous reinforcement (CRF) trials over 3 days, with 6, 12, and 12 trials per day. Reward was 1 ml of the vinegar solution placed in the goal box cup. The intertrial interval was 20 minutes. (ii) Phase 2, conducted 24 hours after the runway acquisition phase of training, was taste-aversion conditioning in the home cage. At this point, three of the four groups of rats were allowed to drink vinegar, a 0.2 percent saccharin solution (weight to volume in tap water), or water for 30 minutes in their home cages. The drinking was followed immediately by an intraperitoneal injection of 0.3*M* lithium chloride (LiCl) (3 percent of body weight). The fourth group was a control that drank vinegar solution but was injected with an equivalent amount of physiological saline. (iii) Phase 3 runway extinction (five trials) was conducted 24 hours after home-cage taste-aversion conditioning. On extinction trials the goal box cup was clean and empty. Thirty minutes after the runway extinction phase, all four groups of rats were allowed to drink vinegar solution in the home cage for 30 minutes as a test of the taste aversion conditioned in phase 2.

Figure 1A summarizes the runway acquisition and extinction data (*6*). All four groups of rats reached asymptotic running speeds within 30 trials. In extinction, the vinegar-LiCl group suppressed running speed on the first extinction trial after taste-aversion conditioning; and the response was extinguished below the operant level across trials. The saline control group was the slowest to extinguish. The other two poisoned groups—saccharin-LiCl and water-LiCl—ran faster than the vinegar-LiCl group, but, perhaps because they were still affected by the illness, these two poisoned groups were slower than the saline control group (*7*). These data (and particularly the first extinction point) are to our knowledge the first demonstration of the suppressive effects of the anticipation of an aversive taste. They also demonstrate a specific relationship between aversive taste conditioning (in the home cage) and the suppression of an instrumental response

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to that taste as reward in the goal box.

During the taste-aversion conditioning trial, before LiCl injection, the two vinegar groups drank about the same amount of vinegar solution, and their intake did not differ from that of the saccharin and water groups (Fig. 1B). After the runway extinction phase all three LiCl groups suppressed drinking in comparison with the saline control group (*8*). This generalization of suppression of drinking corresponds to the generalization of suppression of the running response during extinction.

In experiment 2, we asked whether PRF training with a flavored solution in the goal box would "immunize" rats against the subsequent suppressive effects of anticipating that taste after it had been paired with LiCl. Sixty-four male and female rats were run in four squads of 16 each. The apparatus and all other training procedures were as in experiment 1 except that the reward was a saccharin solution. This was again a three-phase experiment, but it differed from the first in three ways. (i) Runway acquisition (30 trials) included two reinforcement conditions, CRF and 50 percent PRF. (ii) The reward was a 0.2 percent solution of saccharin rather than vinegar. (iii) To reduce the likelihood that our results could depend on direct effects of the illness induced by LiCl, the runway extinction phase (ten trials) was initiated 48 hours after taste-aversion conditioning rather than 24 hours afterward. Because exposure to a taste affects its ability to serve as a conditioned stimulus for taste-aversion conditioning (*9*), the PRF animals, after each training session, were given an additional amount of saccharin solution equal to the difference between the total amount of their rewards and the rewards of the CRF animals (*10*). During the taste-aversion conditioning trial, half of the CRF and PRF subjects were given a LiCl injection immediately after a 30-minute period of access to a saccharin solution in their home cages; the other half of the CRF and PRF animals were injected with saline. On the day between home-cage conditioning and runway testing, all animals were given water for 30 minutes in their home cages.

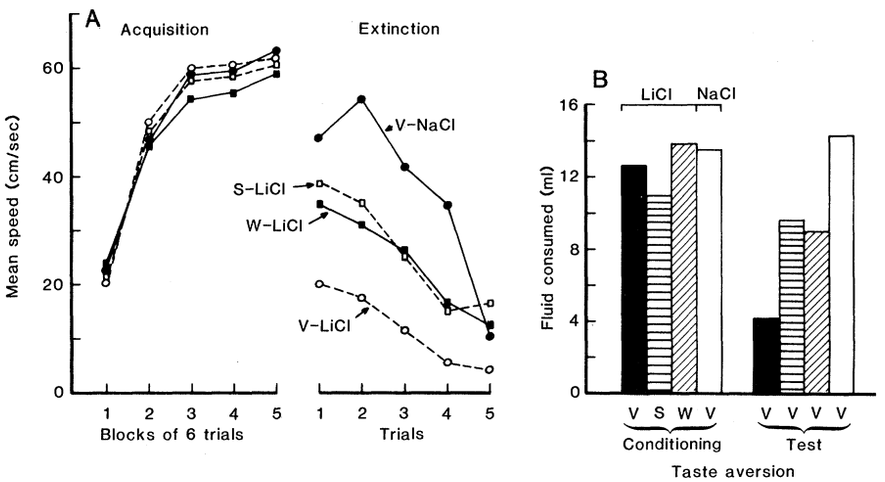

Fig. 1. Experiment 1. (A) Acquisition and extinction speed curves. (B) Fluid intakes during conditioning and testing of taste aversions. Abbreviations: *V*, vinegar; *S*, saccharin; *W*, water.

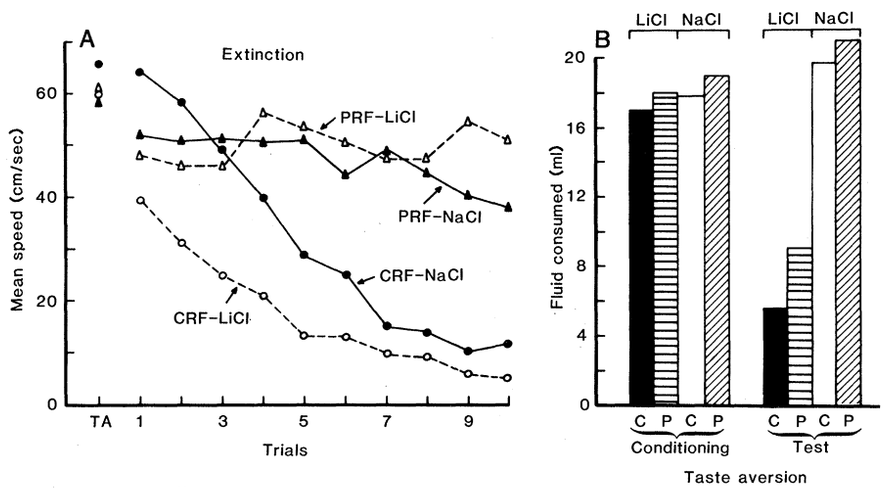

Fig. 2. Experiment 2. (A) Speed during terminal acquisition (*TA*) and extinction. (B) Fluid intakes during conditioning and testing of taste aversions. *C*, Continuous reinforcement; *P*, partial reinforcement.

The CRF-LiCl group extinguished more rapidly than the CRF-NaCl group (Fig. 2A). Again, saccharin was avoided on the first extinction trial. The finding confirms the result of experiment 1 that rats can anticipate and avoid a taste without ever having contacted it after it has been made aversive by conditioning. The confirmation is generalized to a new flavor (saccharin) and a longer interval after the illness (48 hours).

The new finding was that the PRF-LiCl group was as resistant to extinction as the PRF-NaCl group, both groups extinguishing more slowly than the CRF groups (*11*). This indicates that the initial PRF training can successfully immunize against the subsequent suppressive effect of the anticipation of an aversive taste. One explanation is that the rat learns, on a PRF schedule in phase 1, to persist in approaching a flavored solution in the face of frustration, and this persistence transfers in phase 3 to approaching and drinking the solution in the face of the aversiveness of the taste. We cannot, however, rule out the possibility that the PRF training blocks the conditioning of the aversiveness in phase 2, and that the attenuation of avoidance in phase 3 is a secondary effect. [The studies of attenuation of aversions by chemical treatment (*4*, *5*) are also subject to alternative interpretations in terms of original learning as opposed to expression of the flavor aversion.]

As expected, there were no differences in fluid intake among the four groups of rats during the taste-aversion conditioning (Fig. 2B). Compared with the two saline controls, however, the poisoned groups, both CRF and PRF, suppressed saccharin consumption after extinction. The PRF-LiCl group drank more saccharin solution than the CRF-LiCl group (*12*). This result indicates that learned persistence based on the PRF runway training may generalize to the consummatory behavior of drinking an aversive taste solution, reducing the disruptive-suppressive effect on intake of the aversive taste. However, our earlier caveat that preliminary PRF training may have a direct effect on the taste-aversion conditioning applies here as well.

Both experiments demonstrate that a

taste paired with LiCl can be anticipated and avoided—that the anticipation of a taste, rather than the taste itself, can be aversive. The usual taste-aversion experiment demonstrates only escape from an aversive taste. As a historical sidelight, this is a particularly clear demonstration in the rat of what Tolman called an "insight" or "foresight" mechanism (a sign-gestalt-expectation). More than 40 years ago, Miller showed that such a mechanism could be deduced from Pavlovian conditioning principles in the form of Hull's fractional anticipatory goal response (*13*).

Experiment 2 adds the finding that PRF training can reduce (immunize the rat against) the suppressive effects of the anticipation of the conditioned aversive taste and that such training attenuates the suppression of drinking of such a taste solution. If rats are reinforced intermittently and inconsistently with a particular flavored solution, they will avoid that flavor less and drink more of it when it is subsequently paired with gastrointestinal illness. Such a finding has potential practical as well as theoretical implications. One practical application might be to therapeutic situations in which taste aversions and anorexia frequently result from drug or radiation treatments or chemotherapy (*14*). The theoretical implications are for broadening the range of generalization and transfer of persistence in responding across motivational-reward systems (*15*).

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7. Analysis of variance on extinction data revealed a significant groups effect [$F(3, 36) = 5.41$, $P < .005$] and a groups by trials interaction [$F(12, 144) = 2.45$, $P < .01$]. Subsequent Newman-Keuls tests of the overall means for the four groups indicated that the saline control group ran faster than the vinegar-LiCl ($P < .01$), the saccharin-LiCl ($P < .05$), and the water-LiCl ($P < .05$) groups, and that the saccharin-LiCl and water-LiCl groups ran faster than the vinegar-LiCl groups (P's $< .05$).
8. The group effect [$F(3, 36) = 12.23$, $P < .001$] indicated a significant difference in vinegar intake among the four groups after runway extinction. Newman-Keuls tests showed that the saline control animals drank more vinegar solution than the vinegar-LiCl ($P < .01$), the saccharin-LiCl ($P < .05$), and the water-LiCl ($P < .05$) groups, and that the latter two groups, in turn drank more than the vinegar-LiCl group (P's $< .05$).
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10. This additional precaution was probably unnecessary in view of recent research showing that (i) after a single preliminary exposure to sucrose, neither additional exposures nor length of delay between successive exposures has any additional attenuating effect on taste-aversion conditioning [J. W. Kalat and P. Rozin, *J. Comp. Physiol. Psychol.* **83**, 198 (1973)], and (ii) under conditions of severe water deprivation, preliminary exposure to saccharin does not greatly attenuate the taste-aversion conditioning [M. Domjan, *Learn. Motiv.* **3**, 389 (1972)].
11. Analysis of variance yielded a significant effect of reward [$F(1, 48) = 45.93$, $P < .001$] and a significant interaction of reward and trials, [$F(9, 432) = 18.99$, $P < .001$], indicating a clear PRF extinction effect. The interaction of poison (LiCl versus NaCl) and trials was significant [$F(9, 432) = 4.37$, $P < .001$], indicating that the poisoned groups (CRF-LiCl and PRF-LiCl) extinguished faster than the saline controls (CRF-NaCl and PRF-NaCl). Perhaps more important is the significant interaction of reward and poison [$F(1, 48) = 5.95$, $P < .025$]. Subsequent Newman-Keuls tests showed that the PRF-LiCl group extinguished at about the same rate as PRF-NaCl.
12. After extinction, the poisoned groups (CRF-LiCl and PRF-LiCl) consumed less saccharin solution than the saline controls [$F(1, 48) = 346.09$, $P < .001$]. The PRF animals (PRF-LiCl and PRF-NaCl) drank more solution than their CRF counterparts [$F(1, 48) = 12.38$, $P < .001$]. Newman-Keuls tests among the means of the four groups showed that the PRF-LiCl group drank more saccharin solution than the CRF-LiCl group ($P < .01$); the PRF-NaCl and CRF-NaCl groups did not differ from each other.
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16. Supported by NIMH grant R01-MH-30778. We are indebted to M. Domjan for a very helpful critical reading of the manuscript and for bringing several references to our attention.
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Asymmetry in Facial Expression

The conclusion of Sackeim *et al.* (*1*) that there is "greater right-hemispheric involvement in the production of emotional expression" is unwarranted. They found that observers judge double-left composite faces as showing more intense emotion than double-right composite faces. However, they failed to consider the possibility that peripheral neural and anatomical differences rather than differences in the activity of the right and left cerebral hemispheres could explain such results. Facial surgeons note (*2*) that the two sides of the face differ in the size of the muscles, in fatty deposits (*3*), and in the neural supply from the facial nerve nucleus to the facial muscles. Without controls for such differences, the findings of Sackeim *et al.* cannot be interpreted as being due solely to differences in the impulses sent from the two cerebral hemispheres to the facial nuclei.

There is also reason to question whether Sackeim *et al.* were justified in talking about lateralization in emotional expressions, since they studied a different type of facial movement. Neurologists distinguish between voluntary facial movements (by which they usually mean the ability to perform requested actions) and spontaneous emotional expressions. The evidence is clear that these two types of facial activity depend upon different neural pathways (*4*). The potential independence of these two types of facial actions is dramatically shown in clinical cases in which lesions in the pyramidal system (for example, the precentral gyrus) impair requested facial movements but leave emotional facial movements intact, whereas lesions in nonpyramidal systems produce the reverse pattern. This evidence emphasizes the need for caution in generalizing from studies of requested facial movements to emotional expression and vice versa. Thus, it is crucial to know whether the facial movements studied by Sackeim *et al.* were requested or more spontaneous emotional expressions.

Sackeim *et al.* did not accurately describe the photographs they used, which W. V. Friesen and I supplied to them. They wrote that the pictures showed "posed" emotions, or "subjects deliberately attempting to convey particular emotions." Posing may involve either deliberate performance or some attempt to reexperience an emotion to produce the expression. If our photographs had been posed it would be unclear which kind of facial movements Sackeim *et al.* had studied. With few exceptions, however, the photographs they used were not even poses, but the most deliberate

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