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"LEARNED SAFETY" AS A MECHANISM IN LONG-DELAY TASTE-AVERSION LEARNING IN RATS[1]

JAMES W. KALAT[2] AND PAUL ROZIN
University of Pennsylvania

Rats learn taste aversions with unusually long CS–US delays. This has previously been explained as slow decay of a CS trace or as relative lack of interference. We propose, however, that the CS–US delay gradient is a learning curve: During the delay, a rat gradually learns that a taste is "safe." A solution which a rat drinks only once becomes safe and resistant to learned aversions for at least 3 wk., suggesting a learned safety mechanism. If a rat drinks a solution twice (within the effective CS–US interval) before a single poisoning, it learns less aversion than if it received only the second presentation. The learned-safety theory explains this result; a trace-decay or interference model cannot.

Research on taste-aversion learning, beginning with that of Garcia (Garcia, Ervin, & Koelling, 1966; Garcia & Koelling, 1966), has led to findings which suggest a need for major reorientations in our theorizing about learning (see Rozin & Kalat, 1971; Seligman, 1970; Shettleworth, 1972). One of the most striking and controversial aspects of taste-aversion learning is the ability of rats to learn aversions despite delays of several hours between taste and poison, even with only a single trial (Revusky, 1968; Smith & Roll, 1967). This contrasts sharply with the results of many other types of learning experiments in which learning apparently does not occur with delays longer than a few seconds.

Why is long-delay learning possible in the taste–poison situation and not in others? The most conservative answer is what we might call the "aftertaste" theory. This view holds that although the delay between taste and poison is operationally long, it is actually short since some peripheral trace of the taste—such as an aftertaste, taste in the stomach or blood, or regurgitation—mediates the delay. While it is possible that aftertastes may play some secondary role in this type of learning, a number of studies clearly indicate that they play no *necessary* role (for reviews of evidence, see Revusky & Garcia, 1970; Rozin & Kalat, 1971).

An alternative theory proposed by Revusky (1971) involves a reinterpretation of the normal CS–US delay gradient utilizing the principle of "belongingness" or "preparedness" (Seligman, 1970; Thorndike, 1932). According to this principle, certain stimuli are preferentially associated with certain other stimuli—in this case tastes with poisons (Garcia & Koelling, 1966). Revusky posits that the CS–US delay gradient reflects the fact that a US is associated with the most recent potential CS. Ordinarily, a US is readily associable with a wide variety of visual, auditory, proprioceptive, and other cues. Since an animal is constantly bombarded with many cues of this type, any increase in the delay between the would-be CS and the US accidentally introduces other potential CSs so that the US will be associated with these more recent stimuli and not the experimental CS. In taste-aversion learning, however, only tastes (and probably associated smells) are readily associable with poisons, and an animal, either in nature or in the laboratory, experiences very few tastes over a long delay. Thus there is little "concurrent interference" to prevent association of the poison with a taste presented several hours previously.

This clever theory is probably valid in part, but it is not satisfactory as the *sole*

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[2] Requests for reprints should be sent to J. W. Kalat, who is now at the Department of Psychology, Duke University, Durham, N.C. 27706.

explanation for long-delay taste-aversion learning. In particular, it seems to predict that learning should occur with unlimited delays if no taste interference is present. However, increasing delays cause decreasing learning in this situation, even if no tastes are available during the delay (Kalat & Rozin, 1971). It might, of course, be argued that nontaste cues, though poorly associable with poison, manage to generate enough interference to prevent association of poison with the last previous taste. In that case, however, it would be difficult to explain the finding that three novel highly "salient" solutions, which should be highly associable with poison, generate relatively little interference (Kalat & Rozin, 1971).

In short, neither the aftertaste nor Revusky's (1971) interference-plus-belongingness theory is adequate to explain fully the difference between taste-aversion learning and other types of learning.

Let us consider two additional thories: (*a*) the traditional "trace-decay" view, which holds that some central trace of a CS decays gradually during the delay such that after a certain delay it is too weak to be associated with the US; (*b*) the "learned-safety" view, elaborated below, which holds that during the CS–US delay, the rat gradually learns that the taste is "safe." Both the trace-decay and learned-safety models assume that the distinctive features of taste-aversion learning represent an evolutionary specialization of the learning mechanism (see Rozin & Kalat, 1971). Both agree that whatever process underlies the CS–US delay gradient operates more slowly in the case of food-poison combinations so as to make the learning mechanism better adapted to the problem of food selection. The disagreement regards the nature of that process underlying the delay gradient.

In contrast to the trace-decay theory, the learned-safety theory regards the CS–US delay gradient as representing a learning process, not a forgetting process. With long taste–poison delays, *the animal fails to associate taste with poison not because the animal has forgotten the taste but because it has learned that the taste is safe.* That is, the central representation of the taste has not been lost; it has merely been gradually reclassified from "possibly dangerous, associable with poison" to "probably safe, relatively unassociable with poison." This view assumes, of course, that a rat having no previous experience with poisons regards any new food as potentially dangerous (Barnett, 1958; Barnett & Spencer, 1953; Rozin, 1968, 1969).

One line of evidence favorable to the learned-safety theory is the demonstration that rats, if anesthetized during the interval, can learn taste aversions with taste–poison intervals even longer than those which are usually effective (Rozin & Ree, 1972). This finding could be explained either in terms of the reduction in interference as a result of anesthesia or in terms of the reduction of safety learning. It would be difficult, however, to explain it in terms of the passive decay of a memory trace.

A second line of evidence, more directly supporting the learned-safety interpretation, comes from studies of the effect of novelty of tastes. Rats have a strong tendency to associate poison with novel, rather than familiar, tastes (Maier, Zahorik, & Albin, 1971; McLaurin, Farley, & Scarborough, 1963; Revusky & Bedarf, 1967). Under suitable conditions, rats will learn strong aversions to familiar solutions (Garcia, Kimeldorf, & Koelling, 1955). However, if a rat drinks both a novel and a familiar taste prior to poisoning, it acquires a much stronger aversion to the novel than to the familiar solution, even if the familiar solution was temporally closer to the poison (Kalat, 1971; Revusky & Bedarf, 1967; Wittlin & Brookshire, 1968).

This finding supports the learned-safety theory. We suggest that the taste is associated with the *absence* of certain stimuli, i.e., safety from the negative consequences which might have occurred. (For a discussion of possibly similar mechanisms in other systems, see below.) If at a later time the rat is poisoned after drinking the same solution, its previous learning that the solution is safe will in some manner interfere with its learning that the solution is toxic.

In the experiments cited above, a "familiar" solution was one which rats had drunk

several times. Experiment 1 demonstrates how little previous experience a rat must have with a solution for it to qualify as familiar in this situation. From these results it will be argued that it is implausible that a rat should forget a solution within a few hours after drinking it, as the trace-decay view requires. Experiment 2 offers a more direct test of the learned-safety theory of the CS–US delay gradient.

Experiment 1

Experiment 1 consists of three parts in which various amounts of familiarity with a solution are compared with respect to the resistance to learned aversion that they generate. Experiment 1A compares one and three exposures to sucrose; 1B compares one and seven exposures to casein hydrolysate. Experiment 1C compares one exposure to casein hydrolysate 1 day before the poisoning day and one exposure 3 wk. before the poisoning day.

Method

The rats were kept in individual wire-mesh cages having two openings for insertion of 30-ml. graduated Richter tubes (±.5 ml.). Prior to each experiment, each rat was given 20 min. access to tap water once a day until all rats were consistently drinking from the tube within seconds after its presentation. The rats had ad-lib access to Purina Lab Chow at all times. All solutions were prepared fresh each day in tap water and presented at room temperature.

TABLE 1
Summary of Procedures for Experiment 1

Experiment	Group	Procedure
1A	Three exposure	3 days sucrose,[a] 1 water
	One exposure	2 days water, 1 sucrose, 1 water
	Novel	4 days water
1B	Seven exposure	7 days C.H.,[b] 1 water
	One exposure	6 days water, 1 C.H., 1 water
	Novel	8 days water
1C	3-wk. delay	1 day C.H., 20 water
	1-day delay	20 days water, 1 C.H.
	Novel	21 days water

Note. Group 1A's procedures were followed by 1 poisoning day, 2 days water, 2 of nothing, and 1 test day; Group 1B's were followed by 1 poisoning day, 1 day water, and 1 test day; Group 1C's were followed by 1 poisoning day, 1 day water, and 1 test day.

[a] 10% solution.

[b] C.H. = 5% casein hydrolysate.

There were three subexperiments, 1A–1C. (See Table 1 for experimental design.) The subjects for Experiment 1A were 30 female white rats, experimentally naive, aged 58 days on the final day of the experiment. The same rats were used for 1B, beginning 7 days after the completion of 1A. The subjects for 1C were 28 female white rats, aged 87 days on the final day of the experiment. These rats had previously been in an experiment in which they were poisoned after drinking sucrose and NaCl solutions. In 1B and 1C, experimental groups were reassigned to balance groups for previous experience.

In 1A, the "three-exposure" group was given a 10% (w/v) sucrose solution for 20 min/day for the first 3 days of the experiment and water for 1 hr. on Day 4. The "one-exposure" group was given water for 20 min/day on the first 2 days, sucrose solution for 20 min. on the third, and water for 1 hr. on Day 4. The "novel" group was given water for 20 min. on the first 3 days and water for 1 hr. on Day 4. On Day 5 all rats were offered the sucrose solution for 2½ min. Thirty minutes later[3] they were intubated ig with 6 ml. of .15 M LiCl. On Days 6 and 7 all rats were given water ad lib for 24 hr.; on Days 8 and 9 they were given no liquid at all. On Day 10 they were offered sucrose and water simultaneously for 20 min.

In 1B, the "seven-exposure" group was given 5% (w/v) casein hydrolysate for 20 min/day for 7 days and water for 20 min. on Day 8. The "one-exposure" group was given water for 20 min. on the first 6 days, casein hydrolysate for 20 min. on Day 7, and water again on Day 8. The "novel" group was given water for 20 min. on each of the first 8 days. On Day 9 all rats were given casein hydrolysate for 2½ min. Thirty minutes later they were intubated with 6 ml. of .15 M LiCl. On Day 10 all rats received water for 1 hr. On Day 11 they were offered casein hydrolysate and water simultaneously for 20 min.

In 1C, the "3-wk. delay" group was given 5% (w/v) casein hydrolysate for 20 min. on Day 1. On the next 20 days these rats were given water for 20 min/day. The "one-day delay" group was given water for 20 min/day for 20 days and 5% casein hydrolysate on Day 21. The "novel" group was given water for 20 min. on each of the first 21 days. On Day 22 all rats were given the casein hydrolysate solution for 2½ min. Thirty minutes later they were intubated with 6 ml. of .15 M LiCl. On Day 23 all rats were given water for 1 hr.; on Day 24 all rats were offered the casein hydrolysate

[3] Throughout this article, "x min. later" means x min. after presentation of the solution, not after its removal.

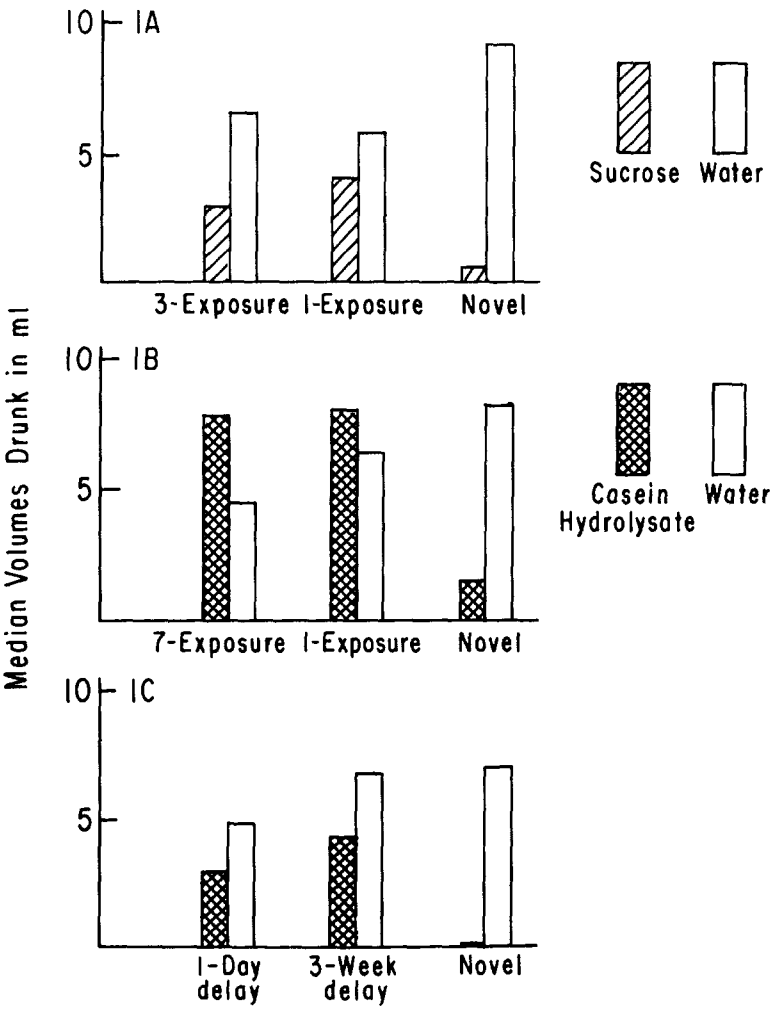

FIG. 1. Results of Experiment 1.

solution and water simultaneously for 20 min. Table 1 summarizes the procedures.

Results

Figure 1 presents the median volumes drunk by each group on the test day of each experiment. All six groups having previous experience with the test solution drank more of it than the novel groups ($p < .01$ for all comparisons except one exposure vs. novel in 1B, for which $p < .04$, and three exposures vs. novel in 1A, for which $p < .06$[4]). In none of the three subexperiments was there a significant difference between the two groups having previous experience with the test solution.

Discussion

1. The results of Experiment 1 further document the importance of novelty in taste-aversion learning and are readily interpretable in terms of the rats' learning that a solution is safe.

2. It is clear that very little experience with a solution is necessary for the rat to accept it as familiar and safe. In this experiment, one previous exposure to a solution produced about as much effect as three or seven, and one exposure 21 days before the

[4] Throughout this paper, all statements regarding statistical significance refer to a two-tailed Mann-Whitney U test based on median absolute intakes of the test solution.

poisoning day was about as effective as an exposure 1 day before.

For the present argument it is not important whether the various levels of familiarity actually produced *equal* effects. The point is merely that one exposure to a solution produces a *large* effect and that the rat has a long memory for a single exposure to a solution. After one exposure to a solution, even 21 days previously (Experiment 1C), a rat accepts it as familiar and does not learn as much aversion as it would if the solution were novel.

Let us consider the relevance of this result for the CS–US delay gradient. A rat poisoned 6 hr. after drinking 10% sucrose acquires no significant aversion to it (Kalat & Rozin, 1971). The trace-decay interpretation of this result is that the rat's memory trace of the solution has effectively disappeared by the end of the 6-hr. delay. This interpretation is plainly untenable without modification in the face of evidence that the rat remembers the solution well after 3 wk.

At the end of 6 hr. the rat must still *remember* the sucrose, but that memory is no longer *associable* with poison. We suggest that the decline in associability is due not to a fading of the trace itself but to a reclassification of the trace, dependent on learning.

More evidence is needed to establish that the learned-safety model explains the CS–US delay gradient. It must be established not only that the rat learns that the solution is safe by the expiration of the maximum CS–US delay that would mediate learning but also that with increasing delays, within the limits that would mediate some learning, the rat is gradually learning that the taste is safe. That is, a solution the rat tasted for the first time 3 hr. ago must be safer than one it tasted 30 min. ago.

TABLE 2

SUBJECTS FOR EXPERIMENT 2

Experiment and solution	*n*	Age (in days)	Previous experimental experience
2A—casein hydrolysate	40	67–70	None
2B—casein hydrolysate	48	73–78	Poisoned twice after drinking sucrose, coffee
2C—sucrose	48	83–87	None
2D—NaCl	48	109–112	Poisoned three times after drinking saccharin, vinegar, coffee
		67–70	No poisoning; drank casein hydrolysate, sucrose, coffee

Experiment 2 is an attempt to demonstrate that a rat is gradually learning that a solution is safe during the first few hours after first tasting it.

Consider the situation in which rats are poisoned ½, 4, or 24 hr. after drinking a novel casein hydrolysate solution. As previously demonstrated (Kalat & Rozin, 1971), the ½-hr. group acquires a stronger aversion than the 4-hr. group, but the 4-hr. group acquires some aversion relative to the control group. The trace-decay interpretation is that after 4 hr. the trace has become weak and therefore poorly associable with poison. The learned-safety theory, on the other hand, holds that after 4 hr. the rats have partially learned that the solution is safe[5] although the learned safety has not yet reached asymptote. Experiment 2 includes a group which allows us to decide between these interpretations. Rats were offered the novel casein hydrolysate 4 hr. prior to poisoning and again ½ hr. prior to the same poisoning. The trace-decay theory would predict that these rats should acquire as much aversion as the ½-hr. group, since the trace was reinstated ½ hr. before poisoning. If anything, the 3½–½ hr. group should acquire *more* aversion than the ½-hr. group because the 4-hr. trace alone is associated to some extent with poison and could add to the ½-hr. trace. The learned-safety theory, however, predicts that the 3½–½ hr. group should acquire *less* aversion than the ½-hr. group and perhaps as little as the 4-hr. group, since it has had 4

[5] It may sound strange to say that a rat has learned an aversion to a solution but has also learned that the solution is safe. We are proposing that the rat is in a conflict situation. The solution, after all, has been followed by both a period of safety and a period of illness.

TABLE 3

PROCEDURES, RESULTS, AND STATISTICAL COMPARISONS FOR EXPERIMENT 2

Group[a]	Experiment 2A		Experiment 2B			Experiment 2C		Experiment 2D		
	n	C.H.	*n*	C.H.	H_2O	*n*	Suc	*n*	NaCl	H_2O
Soln (2½ min.)-30 min.-P	10	1 (.001)	10	.5 (.04)	14.25	12	1 (.05)	13	1 (.03)	12.5
Soln (10 min.)-3½ hr.-Soln (2½ min.)-30 min.-P	10	7.5 (.001, *ns*)	14	3.75 (.10, *ns*)	10	13	3.5 (.02, *ns*)	13	8 (*ns*, *ns*)	13.3
Soln (10 min.)-4 hr.-P	10	5.75 (.002)	14	3 (.06)	11.75	11	11 (*ns*)	8	5 (.07)	11.5
Soln (10 min.)-24 hr.-P	10	17	10	9.25	11.25	12	12.5	14	14.25	8.75

Note. All volumes are *Mdn* ml. drunk in 20 min. All groups were compared statistically with regard to absolute intake of the test solution. The numbers on the lines connecting two groups represent the *p* value as determined by a two-tailed Mann-Whitney *U* test, (*ns* = $p > .10$). Comparisons between the 24-hr. group and the first two groups of each experiment are not shown, but the difference was significant ($p < .03$) in each case. Abbreviations: Soln = solution; P = poison; C.H. = 5% casein hydrolysate; Suc = 10% sucrose.

[a] Delay times given represent those for Experiments 2A, 2B, and 2C. In 2D, delay times for the four groups were, respectively, 15 min., 90 and 15 min., 105 min., and 24 hr.

hr. to learn that the solution is partially safe. That is, the amount of acquired aversion depends largely on the extent to which the rats have learned that the solution is safe, which in turn depends mainly on the time since the rats *first* (not most recently) tasted the solution. The details of the experiment are elaborated below.

EXPERIMENT 2

Method

The subjects were female white rats. The number, age, and previous experience of the subjects are presented in Table 2. In all cases in which the same rats were used for more than one experiment, experimental groups were reassigned for each experiment. The results are pooled for two sets of subjects in the NaCl experiment.

Three solutions were used: 5% (w/v) casein hydrolysate, 10% (w/v) sucrose, and .15 M NaCl. Table 3 outlines both the Day 1 procedures and the results for each group. Each group received the solution for 10 min. and/or 2½ min., with varying delays between the two solutions and between the second solution and poisoning. The number in parentheses after "soln" indicates the period in which the solution was available; the numbers between two solutions or between a solution and poisoning indicate the interval between the presentations of the two solutions or between presentation of the solution and poisoning. The poisoning procedure consisted of intubating the rat with 6 ml. of .15 M LiCl.

Several hours after the 24-hr. group was poisoned, all rats were given water for 1 hr. The following day all were again given water for 1 hr. On the next day all rats were given the test solution and water for 20 min., and the rats' consumption of each was recorded.

In Experiments 2A and 2C, all groups except the 24-hr. group had median intakes of the test solution of less than 1 ml. This floor effect made it difficult to see any differences among the groups. Therefore, on the day following the first test, rats in 2A and 2C were offered casein hydrolysate and sucrose, respectively, with no other solution available (one-bottle test), for 20 min. Table 3 and Figure 2 present the data for only the one-bottle test.

Results

Table 3 and Figure 2 present the median volume drunk in 20 min. by each group; Table 3 also presents certain statistical comparisons.

The experiment is based on the assumption that the ½-hr. groups[6] learn a stronger aversion than the 4-hr. groups and that the latter learn some aversion relative to the 24-hr. groups. In all four experiments, these differences are in the expected direction. The magnitude and significance of these differences is indicated in Table 3.

Given the above results, the critical question is whether the 3½–½ hr. group learns at least as much aversion as the ½-hr.

[6] Throughout the following discussion, each group will be referred to by the time intervals involved in its treatment. For instance, the 3½–½ hr. group drank the test solution of its experiment once, 3½ hr. later drank it again, and another ½ hr. later was poisoned.

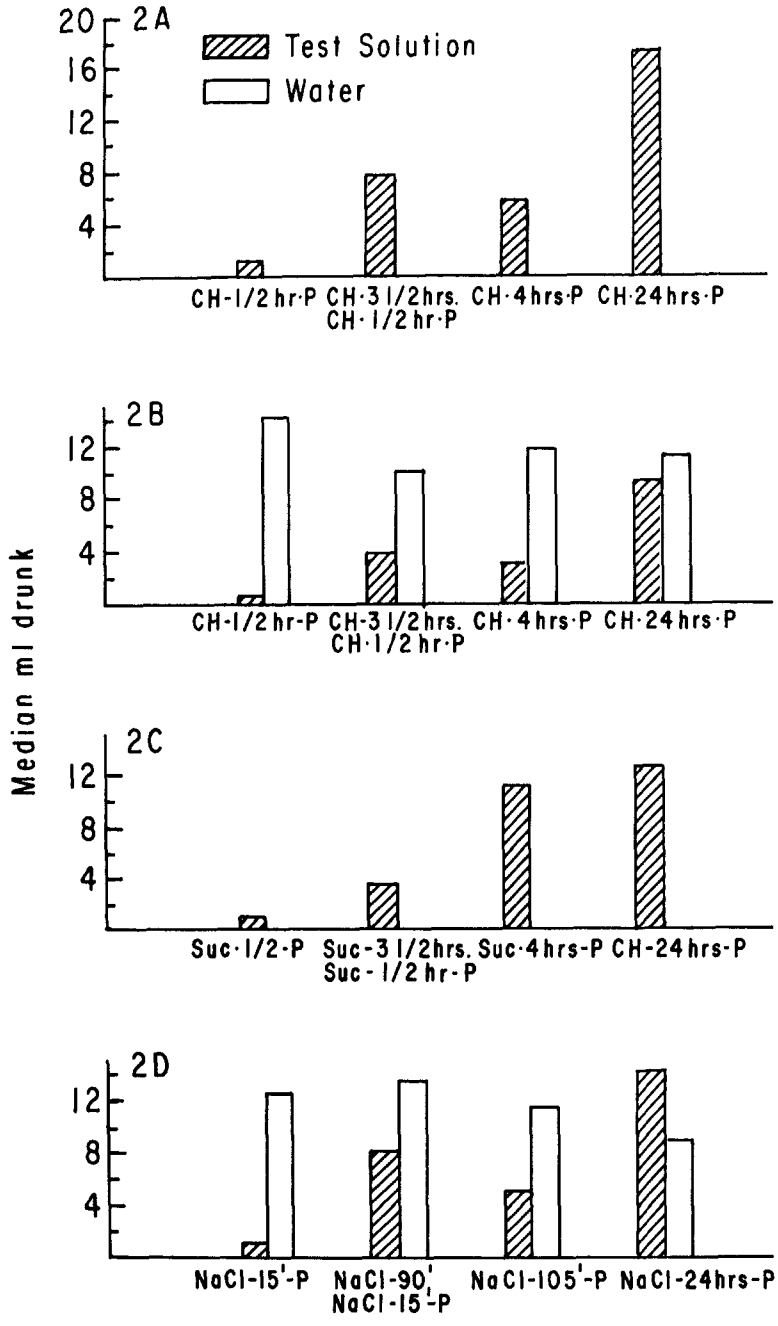

FIG. 2. Results of Experiment 2. (Data are presented for only the one-bottle tests in Parts 2A and 2C.)

group, as predicted by the trace-decay theory, or less aversion, as predicted by the learned-safety theory—perhaps (but not necessarily) as little aversion as the 4-hr. group. In accord with the latter theory, the 3½–½ hr. group acquired a significantly weaker aversion than the ½-hr. group in all four experiments (Table 3). It did not differ significantly from the 4-hr. group in any of the four experiments; in fact, it showed slightly less aversion than the 4-hr. group in three of the four experiments.

Discussion

The results of Experiment 2 are in agreement with the predictions of the learned-safety theory, are incompatible with the trace-decay theory, and are certainly not predicted by interference theory. The argument is no longer tenable that the casein hydrolysate 4-hr. group acquires less aversion to casein than the ½-hr. group because the trace has decayed during the 4 hr., for rats acquire about an equal aversion if the trace is reinstated ½ hr. prior to poisoning. It appears that the rats poisoned 4 hr. after drinking the casein hydrolysate acquire little aversion to it because they have learned to some degree that the solution is safe. A similar conclusion holds for the sucrose and NaCl experiments.

General Discussion

Experiments 1 and 2 both support the theory that the CS–US delay gradient represents a learning process rather than a forgetting process. Learning is diminished with long delays because, at least in the case of tastes, the rats are learning during the delay that the taste CS is safe. We propose that rats learn taste aversions with unusually long CS–US intervals because they learn very slowly that tastes (and perhaps certain other stimuli) are safe. After a rat first tastes a solution, the learned safety of the solution rises gradually toward asymptote at a rate presumably varying with the salience of the solution and probably with various factors in the animal's previous experience (previous safe tastes, previous poisonings, etc.).

At this point we recognize six phenomena which any complete theory of long-delay taste-aversion learning must explain: (*a*) the CS–US delay gradient (Kalat & Rozin, 1971); (*b*) the smaller aversion produced by the 3½–½ hr. procedure than by the ½-hr. procedure (Experiment 2 above); (*c*) the increased resistance to aversion of familiar solutions (McLaurin, Farley, & Scarborough, 1963; Revusky & Bedarf, 1967; Experiment 1 above); (*d*) the fact that in spite of Phenomenon *c*, rats can learn some aversion to a familiar solution (Garcia, Kimeldorf, & Koelling, 1955); (*e*) the variation in the intensity of learned aversions as a function of stimulus properties (Kalat & Rozin, 1970) and strength of the poison (Revusky, 1968; Wright, Foschee, & McCleary, 1971); (*f*) the reduction of aversions as a result of explicitly introduced taste interference (Kalat & Rozin, 1971; Revusky, 1971). As we have argued above, Phenomena *b* and *c* require a learned-safety mechanism of some type, and we have proposed a form of this mechanism which would adequately account for Phenomenon *a*. Phenomena *d*, *e*, and *f* require additional or modified assumptions, regarding which we hesitate to commit ourselves at the present. Learned aversion is clearly something other than the absence of learned safety, and the fundamental remaining question is exactly how learned safety interacts with other aspects of the animal's experience to produce learned aversion and to what extent learned aversions are independent of learned safety.

Implications for Other Types of Learning

The learned-safety interpretation of the CS–US delay gradient is based on evidence drawn entirely from taste-aversion learning. There is reason to believe that a similar mechanism applies in other situations involving associational learning.

Several researchers in the field of shock-avoidance learning have described a phenomenon whereby a stimulus becomes a "safety signal," i.e., a signal associated with the absence of shock (LoLordo, 1967; Miller & Weiss, 1969; Moscovitch & LoLordo, 1968; Rescorla & LoLordo, 1965). Such a signal becomes inhibitory to shock-avoidance behavior. However, it seems to be a necessary condition for the establishment of a safety signal that the stimulus be presented in a situation in which the animal previously experienced shock. If a stimulus is presented alone before the animal has experienced shock or in a previously shock-free situation, there is no evidence that it acquires fear-reducing properties (Rescorla, 1971).

Nevertheless, such a stimulus does become resistant to later association with

shock. This phenomenon, known as "latent inhibition" (Carlton & Vogel, 1967; Lubow, 1965; Lubow & Moore, 1959; Siegel, 1969, 1970), is certainly analogous to the learned-safety process discussed in this paper. Rescorla's (1971) recent study of latent inhibition suggests that it is misnamed. The stimulus does not really become inhibitory; it merely becomes less salient. Rats are slower to condition to this stimulus as *either* an excitatory *or* an inhibitory stimulus. Thus, it does not really become a signal for safety; the rat does not learn "This stimulus means no shock." Rather, it is as if the rat learns "This stimulus predicts nothing; I need not pay attention to it."

It is possible that there is a fundamental difference between tastes and other stimuli in this regard. Perhaps a rat, even without previous relevant experiences, is more fearful of new tastes than of novel stimuli in other modalities. Consequently rats which experience a novel taste without distinct consequences would learn that it is safe while under analogous conditions they would learn that another type of stimulus is "meaningless." On the other hand, it is of course possible that the learned safety of tastes is actually "learned meaninglessness," as it is for other stimuli. The tests Rescorla (1971) employed in this regard would be difficult to apply to taste-aversion learning, since it has been difficult to demonstrate positive learned taste preferences (Rozin & Kalat, 1971). It does not, however, appear that rats regard familiar tastes as meaningless or something not attractive of attention.

Regardless of whether rats learn that stimuli are safe or "predictive of nothing" we propose that it is a learning process, not trace decay, that serves as the mechanism of the CS–US delay gradient, and we regard it as at least highly plausible that the long CS–US delay gradients characteristic of taste-aversion learning reflect the slowness with which this learning process operates in the case of tastes.

REFERENCES

Barnett, S. A. Experiments on "neophobia" in wild and laboratory rats. *British Journal of Psychology*, 1958, **49**, 195–201.

Barnett, S. A., & Spencer, M. M. Experiments on the food preferences of wild rats. *Journal of Hygiene*, 1953, **51**, 16–34.

Carlton, P. L., & Vogel, J. R. Habituation and conditioning. *Journal of Comparative and Physiological Psychology*, 1967, **63**, 348–351.

Garcia, J., Ervin, F. R., & Koelling, R. A. Learning with prolonged delay of reinforcement. *Psychonomic Science*, 1966, **5**, 121–122.

Garcia, J., Kimeldorf, D. J., & Koelling, R. A. Conditioned aversion to saccharin resulting from exposure to gamma radiation. *Science*, 1955, **122**, 157–158.

Garcia, J., & Koelling, R. A. Relation of cue to consequence in avoidance learning. *Psychonomic Science*, 1966, **4**, 123–124.

Kalat, J. W. The CS–US delay gradient as a learning curve. Unpublished doctoral dissertation, University of Pennsylvania, 1971.

Kalat, J. W., & Rozin, P. "Salience": A factor which can override temporal contiguity in taste-aversion learning. *Journal of Comparative and Physiological Psychology*, 1970, **71**, 192–197.

Kalat, J. W., & Rozin, P. Role of interference in taste-aversion learning. *Journal of Comparative and Physiological Psychology*, 1971, **77**, 53–58.

LoLordo, V. M. Similarity of conditioned fear responses based on different aversive events. *Journal of Comparative and Physiological Psychology*, 1967, **64**, 154–157.

Lubow, R. E. Latent inhibition: Effects of frequency of nonreinforced preexposure of the CS. *Journal of Comparative and Physiological Psychology*, 1965, **60**, 454–457.

Lubow, R. E., & Moore, A. V. Latent inhibition: The effect of nonreinforced preexposure to the conditioned stimulus. *Journal of Comparative and Physiological Psychology*, 1959, **52**, 415–419.

Maier, S. F., Zahorik, D. M., & Albin, R. W. Relative novelty of solid and liquid diet during thiamine deficiency determines development of thiamine-specific hunger. *Journal of Comparative and Physiological Psychology*, 1971, **74**, 254–262.

McLaurin, W. A., Farley, J. A., & Scarborough, B. B. Inhibitory effect of preirradiation saccharine habituation on conditioned avoidance behavior. *Radiation Research*, 1963, **18**, 473–478.

Miller, N. E., & Weiss, J. M. Effects of the somatic or visceral responses to punishment. In B. A. Campbell & R. M. Church (Eds.), *Punishment and aversive behavior*. New York: Appleton-Century-Crofts, 1969.

Moscovitch, A., & LoLordo, V. M. Role of safety in the Pavlovian backward fear conditioning procedure. *Journal of Comparative and Physiological Psychology*, 1968, **66**, 673–678.

Rescorla, R. A. Summation and retardation tests of latent inhibition. *Journal of Comparative and Physiological Psychology*, 1971, **75**, 77–81.

Rescorla, R. A., & LoLordo, V. M. Inhibition of avoidance behavior. *Journal of Comparative*

and Physiological Psychology, 1965, **59,** 406–412.

REVUSKY, S. H. Aversion to sucrose produced by contingent X irradiation: Temporal and dosage parameters. *Journal of Comparative and Physiological Psychology*, 1968, **65,** 17–22.

REVUSKY, S. H. The role of interference in association over a delay. In W. Honig & H. James (Eds.), *Animal memory*. New York: Academic Press, 1971.

REVUSKY, S. H., & BEDARF, E. W. Association of illness with prior ingestion of novel foods. *Science*, 1967, **155,** 219–220.

REVUSKY, S. H., & GARCIA, J. Learned associations over long delays. In G. H. Bower (Ed.), *The psychology of learning and motivation: Advances in research and theory*. Vol. 4. New York: Academic Press, 1970.

ROZIN, P. Specific aversions and neophobia as a consequence of vitamin deficiency and/or poisoning in half-wild and domestic rats. *Journal of Comparative and Physiological Psychology*, 1968, **66,** 82–88.

ROZIN, P. Adaptive food-sampling patterns in vitamin deficient rats. *Journal of Comparative and Physiological Psychology*, 1969, **69,** 126–132.

ROZIN, P., & KALAT, J. W. Specific hungers and poison avoidance as adaptive specializations of learning. *Psychological Review*, 1971, **78,** 459–486.

ROZIN, P., & REE, P. Long extension of effective CS–US interval by anesthesia between CS and US. *Journal of Comparative and Physiological Psychology*, 1972, **80,** 43–48.

SELIGMAN, M. E. P. On the generality of the laws of learning. *Psychological Review*, 1970, **77,** 406–418.

SHETTLEWORTH, S. Constraints on learning. In D. S. Lehrman, R. A. Hinde, & E. Shaw (Eds.), *Advances in the study of behavior*. Vol. 4. New York: Academic Press, 1972.

SIEGEL, S. Effect of CS habituation on eyelid conditioning. *Journal of Comparative and Physiological Psychology*, 1969, **68,** 245–248.

SIEGEL, S. Retention of latent inhibition. *Psychonomic Science*, 1970, **20,** 161–162.

SMITH, J. C., & ROLL, D. L. Trace conditioning with X-rays as an aversive stimulus. *Psychonomic Science*, 1967, **9,** 11–12.

THORNDIKE, E. L. *Fundamentals of learning*. New York: Bureau of Publications, Teachers College of Columbia University, 1932.

WITTLIN, W. A., & BROOKSHIRE, K. H. Apomorphine-induced conditioned aversion to a novel food. *Psychonomic Science*, 1968, **12,** 217–218.

WRIGHT, W. E., FOSHEE, D. P., & MCCLEARY, G. E. Comparison of taste aversion with various delays and cyclophosphamide dose levels. *Psychonomic Science*, 1971, **22,** 55–56.

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