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The Effect of Extinction on the Delayed Learning of a Conditioned Taste Aversion in the Rat

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THE EFFECT OF EXTINCTION ON THE DELAYED LEARNING
OF A CONDITIONED TASTE AVERSION IN THE RAT

BY

JOHN J. COLBY

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE
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ABSTRACT

This investigation entailed the first systematic analysis of the extinction of a conditioned taste aversion. The study was undertaken because little is known about the elimination of these prepared responses.

Prepared responses appear to be characterized by (1) the formation of associations between specific stimuli; (2) the rapidity with which associations are established and (3) the apparent lack of stimulus contiguity generally considered to be necessary to establish classical conditioning.

Throughout the paper an attempt has been made to contrast the acquisition and extinction phases of the conditioned taste aversion, the prepared response, with the body of evidence that has been derived from decades of research on unprepared responses with the ultimate goal of determining if the laws of unprepared learning apply to prepared learning. Unprepared responses are not characterized by the presence of specific associations; all neutral stimuli being equally conditionable and are acquired when contiguous stim-

uli are repeatedly paired. Unprepared learning is thought to reflect trial and error learning whereas prepared learning phenomena seem to suggest the presence of special memory mechanisms that facilitate the acquisition of adaptative responses to important events in the organisms environment e.g. associating gustatory cues with stomach malaise.

The subjects for this study were 72 male albino rats. Eighteen rats were assigned as control subjects and did not acquire the conditioned taste aversion. The remaining 54 rats were subdivided into groups of 18 and were all made ill after consuming saccharine, a novel food. Each of these four groups of 18 rats were further subdivided into three subgroups and received either 1, 5 or 10 sensitization trials (controls) or extinction trials (experimental subjects). One group of experimental rats ($N=18$) after being subdivided into the three subgroups ($n=6$) received either 1, 5 or 10 forced exposures to the saccharine. A second group of 18 rats after subdivision received 1, 5 or 10 injections of LiCl , the illness inducing agent. The final 18 rats were an illness retention group. The three subgroups of six rats each were merely tested for saccharine aversion after 1, 10 or 20 days.

The effect of the extinction manipulation was measured

over a seven day test period. The results show that forced exposure to the saccharine, the conditioned stimulus, was the superior procedure. However, comparing the amount of saccharine consumed by the groups treated by this procedure with the controls indicated that recovery from the aversion was not complete, even for the subgroup having received 10 forced exposures to saccharine.

The subjects having received no treatment evidenced no recovery from the taste aversion irrespective of the length of the retention interval between the last extinction and the first test day. These results and those observed from the groups that received the forced exposure to saccharine are consistent with the findings from the literature on unprepared learning.

The rats that received the repeated injections of LiCl as an extinction procedure were not recovered on the first test day but recovered very quickly in the test phase. It appears that repeated non-contingent injections of the illness inducing (LiCl) agent and the resulting stomach malaise followed by the opportunity to drink the saccharine in the test phase facilitates recovery during the latter phase. Some investigators have reasoned that a procedure like that

described above might contribute to the formation of cognitions which are inconsistent with those formed during the acquisition phase. That is, the illness that was ascribed to the saccharine in the acquisition phase is, during the test phase no longer so ascribed because of the non-saccharine related illness of the extinction period. This cognition would expedite recovery when the saccharine is later presented with no subsequent illness as is the case in the test phase. This latter result was not expected based on the sensitization data from the unprepared area of learning.

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Arnold Kirshenbaum prepared the figures for this paper. Their quality reflect the careful attention he devoted to his work.

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My wife, Lynne, has been so supportive of my efforts and so giving of herself over the period of my work that nothing I can say can convey my appreciation and love. She and I know, however, that this is another product of our labor.

Finally, many of the intangibles that inspire someone to achieve at this level are probably attributed to one's parents. I recognize and appreciate the things they did long ago that influence my life today.

In conclusion, I have dedicated this work to the memory of my father who taught me by his example that there is no substitute for effort.

TABLE OF CONTENTS

TITLE PAGE	i
APPROVAL PAGE	ii
ABSTRACT	iii
ACKNOWLEDGEMENT	vi
LIST OF TABLES	x
LIST OF ILLUSTRATIONS	xi

CHAPTER

I. INTRODUCTION	1
II. METHOD	34
III. RESULTS	43
IV. DISCUSSION	73
APPENDICES	83
REFERENCES	86

LIST OF TABLES

TABLES	Page
1. Design of the acquisition and extinction phases for the experimental and control groups.....	36
2. Mean amount of saccharine consumed (cubic centimeters) on each of the three acquisition trials.....	45
3. Mean amount of saccharine consumed (cubic centimeters) on each of seven test trials (n=6) by the control groups.....	63
4. Mean amount of saccharine consumed (cubic centimeters) on each of seven test trials (n=6) by the saccharine-only groups.....	64
5. Mean amount of saccharine consumed (cubic centimeters) on each of seven test trials (n=6) by the LiCl-only groups.....	65
6. Mean amount of saccharine consumed (cubic centimeters) on each of seven test trials (n=6) by the retention groups.....	66

LIST OF ILLUSTRATIONS

Figure	Page
1. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for the control rats.....	47
2. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for the rats extinguished by forced exposure to saccharine.....	49
3. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for the rats extinguished with LiCl injections.....	51
4. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for the rats in the retention extinction condition.....	53
5. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for all groups having received one extinction trial.....	55
6. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for all groups having received five extinction trials.....	57
7. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for all groups having received ten extinction trials.....	59

INTRODUCTION

Behaviorists have generally accepted the conclusion that the rat is a passive participant in learning experiments and form associations between any set of contiguous events (Pavlov, 1928). In a recent departure from this tradition some investigators (Garcia, Kimeldorf and Hunt, 1961) have reported results that challenge the view that all stimulus pairs are equally associable. These results suggest that the rat brings to some learning task species specific response tendencies which enhance the likelihood that specific associations, not any association, will be established between stimulus events. These response tendencies are believed to reflect a biological adaptability to the natural environment and can be elicited from every member of the species.

Although species specific response tendencies have only recently been of interest to psychologists, they are not new to all investigators of animal behavior. Specific

associations have been discussed by ethologists for several decades. An approximation to specific response tendencies has been described in Thorndike's Law of Readiness (1932) and has been interpreted by Hilgard and Bower (1966) to mean an "action tendency".

Recently, Seligman and Hager (1972) developed an organizational scheme that defines the necessary conditions for specific associations to be established when all possible associations seem equally likely. Three learning types have resulted from this organization and are defined in terms of the organisms preparedness to form specific relationships between stimuli. Seligman and Hager's efforts to define several learning forms has diminished the conceptual differences in learning held by traditional behaviorists and ethologists.

One form of learning described by Seligman and Hager (1972), unpreparedness learning, has been of primary interest to behaviorists because it seems to reflect the experience associated with trial and error learning.

When relatively permanent changes in behavior occur after numerous acquisition trials the organism is said to be unprepared to learn. The incremental form of the acquisition curve indicates that special associative mechanisms

have not evolved to facilitate the formation of S-S or S-R bonds. Psychologists have inferred from the gradual change in the response topography that unprepared learning studies are untainted by species specific response patterns. Historically, laboratory psychologists have been motivated to create learning paradigms that are free from environmental influences just as the developmental specialist seek culture free tests and Ebbinghaus sought a verbal task independent of previous associations. When laboratory psychologists were unexpectedly confronted with species specific responses they were treated as a nuisance to be attenuated to the extent possible.

The study of unprepared learning has, in fact, established numerous laws of learning (Kimble, 1961). In fact, it has been such a useful paradigm for investigating the gradual formation of associations that psychologists have come to think of it as the model for acquiring new and adaptative modes of behavior. However, a growing body of psychologists believe that unprepared laws are not exhaustive and do not describe or predict the events subsumed under prepared and contraprepared learning categories (Seligman, 1970; Bolles, 1970; Kalat and Rozin, 1971).

When organisms form specific associations between stimulus events, when all possible associations appear equally likely, the organism is said to be displaying prepared learning. That is, specific responses are learned and are evoked by only a subset of all possible conditional stimuli. Garcia et al. (1961) have illustrated this phenomenon by showing that rats associate gastrointestinal illness with novel taste cues only. Extroceptive stimuli, both visual and auditory, and familiar gustatory stimuli did not become associated with the illness even though these stimuli were more temporally contiguous with the stomach malaise. It is interesting to note that Garcia used x-radiation to induce the illness; food items were not poisonous. None-the-less, the rats associated the malaise with the novel food and it only. On subsequent opportunities when the rats were offered a choice of the novel food or a more familiar food, both of which were consumed before the gastrointestinal upset, the rats avoided the novel food but not the more familiar food.

This type of avoidance behavior, like other forms of preparedness learning such as imprinting (Lorenz, 1950), specific hunger (Rozin and Rodgers, 1967) and species speci-

fic defensive reactions (Bolles, 1970), are thought to have had a long and specialized evolutionary history paralleling the development of specialized sensory and receptor organs. This parallel development has been succinctly illustrated by Lettvin, Maturana, Pitts and McCulloch (1959) who have described specialized cells in the retina of the frog (*Rana Pipiens*). These receptor cells are uniquely designed to detect convex edges of various insects only. Seligman (1970) reasons that specific response tendencies, like specialized sensory mechanisms, tend to enhance the survival of the species in the natural environment.

Contraprepared learning refers to Seligman's third discrete category and is characterized by a very lengthy acquisition phase with learning occurring only after an inordinate number of trials if at all. Conceptually, contrapreparedness results when the experimenter attempts to teach an organism to make a response antagonistic to a prepared behavior in situations where natural selection influences the response probability.

Breland and Breland (1961), reporting on the "misbehavior" of organisms, described their inability to train a raccoon to deposit two tokens in a container for food

reinforcement (illustrating for the youthful observer fiscal responsibility). Prior to their reported failure to teach this response, the Brelands had no difficulty conditioning the subject to deposit a single token, thus they returned to this earlier phase to further establish the simpler response. They reported, however, that overlearning the first response did not facilitate the acquisition of the second two-token phase. The raccoon persisted in his misbehavior seemingly oblivious to the concept of positive transfer. The Brelands reasoned that the pattern of perservative behaviors exhibited by the raccoon, consisting of preconsummatory responses specific to its species was so prepared that in the presence of the considerable stimulus excitation produced by the two food-related tokens, the prepared response was unavoidably emitted.

In an analogous fashion, phobic humans treated by implosion therapy with a full array of imaginal cues report relief (Seligman, 1972). The appearance of diminished fear of the imaginal phobic objects(s) may occur because cognitions are not sufficiently salient to elicit the more prepared avoidance response. The fact that clients appear improved when the fear is measured by standard clinical in-

inventories MMPI (Lewis & Penner, 1967): Fear Thermometers and Fear Survey Schedules (Lang & Lazovik, 1963) may be misleading. The client's verbal report is discrepant with the results based on these inventories (Lang & Lazovik, 1963; Lang, 1968; Miller & Nawas, 1969). Verbal reports reflect the client's real experiences with the phobic-related conditioned stimuli. The discrepancy suggests that when the cues are imaginal the client is able to make an unprepared or even a contraprepared response and remain in the presence of the fear inducing cognitions. However, when the phobic subject is confronted with the real phobic object a prepared response appears inevitable. The critical factor may be the extent to which the client is stimulated by the different fear inducing cues.

Recent analyses of prepared learning underscore the conclusion arrived at by the Brelands "...that the behavior of any species cannot be adequately understood, predicted or controlled without knowledge of its instinctive pattern, evolutionary and ecological niche." (Breland & Breland, 1961, p. 608). It is no longer appropriate to conclude that behavioral repertoires are identical except for differences in the manner in which organisms contact and act upon

their environment. This latter contention espoused by Skinner, one of the foremost spokesmen of the behaviorists' tradition, [Skinner in Koch (ed.), 1959] and others who share his view of ethological influences on learning grant only "...that hormones, genetic determinants...and the like are ...determinants of parameter values only." [Estes in Koch (ed.), 1959] .

The remainder of this introduction will more fully develop the recent body of evidence reflecting on the significance of prepared learning, the factors that have been hypothesized to account for this phenomenon and will examine the ethological-learning and general process learning position to determine if those learning forms vary in significant ways or merely parametrically.

Place Versus Taste Cues

Since the original paper by Garcia et al. (1961), there has been an increasing interest in the learning-ethological model. Garcia and Koelling (1967) investigating the acquisition of taste aversion found that gustatory stimuli (1 gm/liter saccharine in water) paired with x-radiation or Lithium Chloride (LiCl) produced a strong aversion to the taste of the saccharine, a novel food for the rats. No aversion

to a unique set of place cues was established in this study. Garcia and Koelling described the distinctive place cues as "bright-noisy" water. When, upon drinking, the rat activated an electric circuit, an incandescent lamp (five watts) flashed and a clicking relay was repeatedly closed. These potential CS's did not become aversive in spite of the fact that x-radiation and LiCl are powerful illness inducing agents. Rats shocked while drinking "bright-noisy" water-saccharine solutions subsequently avoided "bright-noisy" drinkometers but not the gustatory stimulus, the saccharine, delivered in the absence of the audiovisual cues.

Subsequently, Revusky & Garcia (1970) reported data that appears to control for backward conditioning. Employing x-radiation as the UCS, saccharine was presented to rats four to six hours after irradiation with no resulting aversion to the saccharine. An aversion was established when the rats were permitted to drink saccharine for the first time within two hours of the irradiation.

More recent designs investigating the extent to which exteroceptive stimuli get established as aversive conditioned stimuli have produced results that are inconsistent with those reported by Garcia and Koelling (1967). Honggeler

(1974) placed rats in a distinctive black box before injecting the rats with LiCl. On trials not reinforced with LiCl injections the rats were placed in a translucent plastic box. During the test phase the subjects showed an increased latency to enter the black box and a decreased latency to leave the translucent, safe box. These results were consistent over several trials. Braveman (1974) using guinea pigs found that subjects acquired an aversion to the color of solutions if the original colored solutions were followed by LiCl. However, the recovery from the conditioned aversion is faster if color is the only cue associated with the LiCl. When taste is the aversive conditioned stimulus, recovery is more gradual indicating that taste is a more salient cue. Braveman suggests that it is not unusual to find that exteroceptive-visual cues become effective aversive cues for guinea pigs. These organisms are more dependent upon visual cues to secure their food than are rats. To control for the possibility that the food coloring was adding subtle gustatory cues to the solutions, Braveman replicated the study using various colored tubes to deliver the food solution. The results of this control study were consistent with the original findings. In perhaps the most

sensitive test of exteroceptive conditioning to LiCl; Morrison and Collyer (1974) taught rats to lick for fluid delivered on a VI3 schedule. During the conditioned aversion acquisition phase the rats licked a distinctive tasting solution paired with the onset of the house light. This was the only time the house light was on. Each of three acquisition trials was reinforced with an injection of LiCl. When tested in the presence of the house light a significant suppression of the licking response on the VI3 was evident. The investigation suggests that the ability to establish an exteroceptive cue as an aversive conditioned stimulus may be due to the fact that there were three acquisition trials. Theoretically, the light was paired with the LiCl on the second and third trial by the mediation of the distinctive tasting solution. In the Garcia and Koelling (1967) study only one acquisition trial was employed thus the exteroceptive stimulus, the audio-visual cue, was never paired with the LiCl after illness but before another acquisition trial. Perhaps after one illness trial the rat would have been able to associate the audiovisual cue with the LiCl via the injected solution, a paradigm suggestive of higher order conditioning. In support of this explanation, Morrison and

12

Collyer found no suppression of the licking response without a taste mediator. Also, there was no suppression of licking to the light alone when the light-taste relationship was initially established in preconditioning trials followed by LiCl reinforcement of the taste stimulus. Thus, there was no evidence of sensory preconditioning.

Although some evidence for exteroceptive aversion conditioning is evident in the last three studies described here, it seems that more input, measured in trials is necessary to form this type of aversion. An aversion to taste stimuli, however, is apparent after a single acquisition trial suggesting that taste aversion is a form of prepared learning while conditioned aversion to exteroceptive, audio-visual cues, constitutes a form of unprepared learning. In this regard, Lorden, Kenfield and Braun (1970) have reported significant suppression of responding on a VI 30 for water reinforcement in the presence of isopropanol vapor when rats had previously smelled, or smelled and tasted isopropanol followed by illness. Lorden et al. reported that on the first day of testing of a sequence of five test trials the rats that had smelled and tasted isopropanol followed by LiCl evidenced a greater suppression of the licking response in the presence of the

vapor than rats having previously smelled the isopropanol only. There were no differences between the smell only and taste-smell groups on the second through fifth test days. Further, Smith and Balagura (1969) have reported intragastric loading of LiCl (.12M) does not establish an aversion to LiCl or NaCl both of which are indistinguishable salts. In the same investigation an aversion to LiCl and NaCl was established after the rats drank a .12M solution of LiCl. Thus it appears that gustatory cues are necessary to establish an aversion to novel solutions and exteroceptive stimuli and are always more salient than exteroceptive stimuli. However, not all taste stimuli are equally salient. Kalat and Rozin (1970) have shown that rats poisoned following the injection of two novel solutions associate poisoning more with one solution than the other. Casin hydrolysate was always more salient (i.e. became more aversive) than sucrose, NaCl or vanilla when casin hydrolysate was paired with one of the other solutions prior to the LiCl injection. The aversion was more equally distributed between the two solutions, however, when casin hydrolysate was the second of the two solutions consumed by the rats. Parenthetically, Kalat and Rozin report there is no direct re-

relationship between palatability and salience. The most salient solution, casein hydrolysate, is the least preferred solution by control rats but sucrose, the second most salient solution is preferred to all other solutions by controls.

Three Hypotheses to Account for Conditioned Taste Aversion

The body of evidence discussed above identifies two factors that distinguish unprepared learning results from more recent findings based on prepared learning studies. First, investigators of prepared learning phenomena including Garcia and Koelling (1967) and numerous others (Revusky & Bedarf, 1967; Kalat & Rozin, 1970; Seligman, 1970; Revusky & Garcia, 1970) have provided considerable evidence from studies of specific hungers and learned taste aversions to show that all stimuli are not equally associable. Secondly, Smith and Roll (1967) and Revusky (1968) have established delay of reinforcement gradients of six and eight hours respectively and CS-US intervals of 30 min. to 120 min. are typical.

These results are in sharp contrast to those derived from the historically more traditional response measures that characterize unprepared learning (e.g. eyelid, pupillary, heartrate and GSR conditioning). The results from these

learning procedures, taken as a whole, consistently provide evidence that all stimuli are equally associable, assuming temporal contiguity [Pavlov, 1928; Estes in Koch (ed.), 1959]. Furthermore, close temporal contiguity in the order of seconds in thought to be necessary to establish classical conditioning. Indeed maximum associations require that the period separating the CS and US be measured in fractions of a second (Hull, 1952). Gormezano and Moore [In Marx (ed.), 1969] report maximum classical defensive conditioning with ISI of 0.25 to 0.50 sec. and Kimble (1961) summarizing the results from numerous studies of a variety of response systems reports that 0.50 sec. is a frequent ISI value for maximum conditioning. White and Schlosberg (1952) also report 500 msec. as the optimal ISI for classical conditioning of GSR in humans. Although the ISI range varies depending on the response system being conditioned, classical conditioning with an ISI of less than 250 msec. are rare and was found to be too short for White and Schlosberg (1952). Notterman, Schoenfeld and Bersh (1952) were able to establish some heart rate conditioning with a 7000 msec. ISI which seems to approach the upper limit for CS-US separation. Young (1958) was unable to establish a pupillary

dilation response with a 15 sec. ISI.

The obvious difference in ISI range for prepared and unprepared learning suggests that the parameters which were thought to apply to all forms of classical conditioning need to be reevaluated in light of successful prepared learning results employing ISI of one hour or more.

For some investigators a reevaluation of parametric values is not sufficient. Kalat and Rozin [In Seligman & Hager (ed.), 1972] argue that the range of ISI for conditioned taste aversion together with the specificity of CS conditionability warrants a more cognitive interpretation of the conditioning.

Three hypotheses have been suggested to account for the fact that conditioned taste aversion is characterized by specificity of associations and the lack of stimulus contiguity. Two of these hypotheses, aftertaste and interference are not new and have been discussed in other contexts. Both theories are consistent with the view that prepared learning can be described by the general process laws of learning except for the observed parametric values. Learned safety, the third hypothesis, has been more recently formulated as an alternative to the aftertaste and inter-

ference hypotheses and has been cognitively interpreted by some investigators.

Aftertaste Hypothesis

The simplest explanation to account for taste aversion conditioning with long delays of reinforcement is to suggest that some aftertaste of the novel solution is present when the subject becomes ill. If, in fact, this could be demonstrated, further discussion about gradients of delayed reinforcement would be mute and stimulus contiguity would be demonstrated. The data resulting from several critical tests controlling for aftertaste do not support this hypothesis, however. Rozin and Kalat (1971) insist that any aftertaste explanation of delayed learning must involve some unidentified "blood factor" or taste trace in the mouth.

The former mechanism is highly speculative and there is only one study in the literature that is of any relevance to it. Hunt, Carroll and Kimeldorf (1965) joined pairs of rats by vascular anastomosis. These parabiont rats, having a common circulatory system were then differentially treated to establish the extent to which humoral factors influence the acquisition of a conditioned taste aversion for the second of the joined pairs. One rat of a joined pair was permitted

access to saccharine following an exposure to x-radiation. This rat always became ill and acquired a saccharine aversion. The second rat of the pair also drank saccharine but was shielded from the x-radiation. The shielded rats that had consumed the saccharine but had not been irradiated exhibited a strong aversion to the saccharine too. Parabiont pairs consisting of a rat that received saccharine and x-radiation, and a second rat that was shielded and drank unflavored water behaved differently. An aversion to saccharine was obvious for the saccharine-x-radiated rat but the water-shielded rat did not acquire an aversion. These results support the predictions of Hunt et al. that humoral factors influence the acquisition of a conditioned taste aversion. It appears that some metabolic change took place in the x-radiated rat which because of the common circulatory system affected the shielded subject and produced an aversion to saccharine if the shielded subject had also been exposed to saccharine prior to the malaise. These results, although intriguing, do not clarify the significance of peripheral aftertaste because the study is procedurally esoteric and may be unique to those instances where x-radiation is used as the US. Furthermore, as Hunt et al. report, the speci-

fic mechanism to account for this humoral mediation of the aversion remains unidentified.

With the exception of the Hunt et al. investigation, researchers interested in the peripheral aftertaste explanation of delayed learning have concentrated their efforts on controlling for a specific trace of the solution which serves as the CS. Revusky and Bedarf (1967) in a study illustrating the specificity of conditioned taste aversion to the more novel of two gustatory cues have by counterbalancing for cue order also controlled for aftertaste. For some subjects a familiar food (CS₁) was followed by a novel food (CS₂) before the LiCl presentation. The CS order was reversed for other subjects. The primary effect of this manipulation was the establishment of an aversion to the novel substance irrespective of CS order. The fact that the aversion was specific to the novel solution when it preceeded the familiar solution weakens the argument that an aftertaste of the novel solution served to bridge the one hour ISI. If an aftertaste remained, it would be the taste of the familiar solution.

Further results reflecting unsupportably on the aftertaste explanation have been reported by Garcia, Green and

McGowan (1969). In their study an aversion was established to 0.05% HCl using x-radiation as a US. The significance of these results emanates from the fact that the concentration of HCl used by Garcia et al. is negligible compared to the concentration of this acid found in the stomach of the rats. Of equal significance are the results reported by Nachman (1970) indicating that an aversion could be established to 43° C water when it was paired with apomorphine. Water delivered at room temperature and not reinforced did not become aversive. These results were replicated with cold water paired with the US with the same results. It appears that the temperature of water can serve as an effective CS for taste aversion conditioning and rats can discriminate between this CS and room temperature water that is not reinforced. Since the potential CS's were both tap water it is difficult to understand how a differential taste cue associated with the hot (cold) water could serve as a taste-cue mediator. Some temperature-taste interaction must occur, however, since a replication of this experiment using distilled water did not produce differential results for the heated (cold) water versus the room temperature water. Finally, in contradiction to the aftertaste

hypothesis, Rozin (1969) established an aversion to only one of two specific concentrations of a solution and that to the concentration that was followed by LiCl.

Interference Hypothesis. Another mechanism that has strong historical roots in psychology to account for forgetting S-S or S-R events is interference (Kimble, 1961). For the phenomenon of conditioned taste aversion, however, the task is to account for the ability of some organisms, here the rat, to establish and maintain ISI's of up to eight hours (Revusky, 1968). Revusky and Garcia (1970) propose that the maximum CS-US interval depends not on time per se. but on the number of interfering stimuli that are present between the CS-US presentations. Since in taste aversion learning the range of potential taste stimuli that could intervene between the CS and US is restricted and generally non-existent the subjects tend to associate the US or its effect with the only gustatory stimulus available, the novel solution (CS) even though the interstimulus interval is measured in hours. This theory is very attractive because it proposes a general principle, interference, to account for the differences between taste aversion learning and other forms of unprepared learning.

However, as parsimonious as this single explanation may appear, the results of several critical studies indicate that the lack of interfering stimuli cannot alone account for the rats ability to associate stomach malaise with a novel solution. Kalat and Rozin (1971) have reported that the consumption of two or three novel solutions during a 30 minute interstimulus interval between sucrose consumption and poisoning does not eliminate the sucrose aversion. Thus, interference defined as the number of interpolated stimuli between the CS and US does not significantly attenuate the effect of the acquired aversion. Other investigations reflecting on the interference interpretation will be discussed under the next heading.

Learned Safety Hypothesis of Acquired Taste Aversion. Nachman and Jones (1974) and others have proposed a learned safety hypothesis to account for the conditioned taste aversion phenomenon. They suggest that when a rat ingests a nontoxic novel substance it gradually learns in the ensuing hours that the substance is safe. This explanation would account for the limited amount of novel solutions consumed on the first exposure trial (neophobia), the increased amount con-

sumed on the second and subsequent trials when the subjects are not poisoned and the aversion when the novel food is followed by poison.

In series of experiments testing the efficacy of the learned safety hypothesis, Nachman and Jones (1974) first made rats mildly ill by injecting them with LiCl four hours after drinking saccharine. The long delay of reinforcement attenuated the degree of the illness. Two days later the subjects were given a two minute exposure to saccharine and at the end of one of four intervals; 4, 30, 120 or 480 minutes, a 10-minute reexposure to the saccharine. The amount of saccharine consumed in the ten minute test increased as the period between the 2-minute and 10-minute reexposure increased suggesting that the rats were learning that the saccharine was now safe. Four control groups were run using the same time parameters to control for water deprivation as an alternative explanation for these results. No significant difference in the amount consumed was found for the 4-30- 120- and 480-minute groups when water replaced saccharine.

In a second experiment, four groups of rats were given two exposures to saccharine. The interval between the first

and second session was again 4, 30, 120 and 480 minutes for the four separate groups. On the original exposure trial the rats exhibited the typical neophobic avoidance of the novel solution and drank only small quantities of the saccharine. The amount of saccharine consumed on the second exposure was, however, different for the four groups. As in the original experiment in this series, the longer the interval between the original exposure and the reexposure to saccharine the more fluid ingested. A third experiment in this series was essentially a replication of Experiment II except that six preexposures were administered. No direct effect of the various delays was noted in this instance assumedly because some asymptote of learned safety had been achieved by the sixth exposure. These rats were subsequently injected with LiCl at 4, 15, 30, 60, 120 or 480 minutes after the sixth saccharine exposure and for the 120- 240 and 480-minute groups no aversion was noted. The degree of aversion for the subjects in the 4- 15- 30 and 60-minute groups was greatly reduced compared to Nachman's data for subjects not having received repeated nonreinforced pre-exposures to saccharine suggesting that learned safety attenuated the subsequent effect of the poisoning. However,

the fact that there is some effect for the groups having CS₆-US interstimulus intervals of less than 120 minutes compared to the 120- 240 and 480-minute groups indicates a trace decay effect. One might conclude that two mechanisms, trace decay and learned safety, are necessary to account for the acquired taste aversion phenomenon established with delayed reinforcement.

In a direct test of learned safety versus passive trace decay, Kalat and Rozin (1973) reported that one preexposure to a novel substance attenuated the subsequent conditioned taste aversion as much as three or seven preexposures. Also, one preexposure 21 days before poisoning was about as effective as a single preexposure the day before a subsequent reinforced trial. These results do not appear to enhance the passive trace decay explanation. However, they are not consistent with the results reported by Nachman and Jones (1974).

In another experiment in this series, a group of rats were permitted access to the novel solution (CS) at 3.5 and 0.5 hours before poisoning. Another group of rats received only a single exposure to the novel solution 0.5 hours before poisoning. The rats in the former group devel-

oped a substantially ~~lesser~~ aversion to the CS. The trace decay theory predicts an equal aversion for both groups while learned safety proponents predict, as the results indicate, a lesser aversion for the 1.5-0.5-hour group because the latter group has four hours to learn that the novel food substance is safe.

Finally, in a study directed to the same end, Domjan (1972) infused 8 ml of 0.2% saccharine into the mouth of rats via oral cannulae. Three groups were infused at different flow rates; 0.6, 1.2 or 2.9 ml/minute resulting in a total saccharine contact time of 13.3, 6.7 or 2.8 minutes respectively before being poisoned. The two groups having received the two slower infusion rates and thus more exposure time to the CS exhibited a weaker aversion to the saccharine. These results corroborate those of Kalat and Rozin (1973) and support the contention that some unidentified mechanism actively monitors the subsequent gastric effect resulting from the ingestion of novel foods. This mechanism appears to function as long as a trace, probably neural, remains active. This hypothesized neural trace appears to remain active for something in the order of 8 hours (Revusky, 1968) but can be extended if the subject is anesthe-

sized following the presentation of the CS (Rozin & Ree, 1972). The trace can ~~be eliminated~~ by cortical spread of depression procedures (Davis & Bures, 1972) which is consistent with the hypothesis that the CS trace is neurological and located in the central as opposed to the peripheral nervous system. Whether this mechanism is cognitive or more "primitive" [Kalat & Rozin in Seligman & Hager, 1972] remains unanswered.

Statement of the Problem

Thus far investigators have shown that the laws of general process (unprepared) learning do not adequately describe the acquisition phase of prepared learning. The results of numerous investigations, many of which have been detailed here, consistently indicate that prepared responses are acquired in a single trial and are specific to a subset of all potentially associable stimuli. Somewhat surprisingly, little is known about the extinction phase to prepared learning. What little information is available seems to suggest that the extinction of an acquired taste aversion, in contrast to acquisition, corresponds more closely to the extinction of unprepared responses (Garcia, Erwin & Koelling, 1966). With the exception of the Garcia et al. (1966) data and a report by Grote and Brown (1973) indicating that deprivation expedites the extinction of a conditioned taste aversion, no systematic investigation of extinction procedures appears in the literature.

To further determine the variables that affect prepared learning this study sought to describe the extent to which learned taste aversions were subject to three ex-

tion procedures. These three procedures were (1) the repeated presentation of the CS without reinforcement (US), (2) a no treatment condition which served as a retention control, and (3) the repeated presentation of the US alone.

The predictions of the effectiveness of these procedures were based on the conclusions, viewed collectively, from the unprepared learning literature. Extinction of classically conditioned unprepared responses occurs when the CS is repeatedly presented without the US. This fact is true for all species of organisms that have been conditioned irrespective of the response system involved (Razran, 1939). Although little is known about the elimination of prepared behaviors established by classical conditioning, the available evidence does suggest that repeated CS presentations will extinguish the prepared response (Garcia et. al., 1966; Grote & Brown, 1973). These investigations were not studies of extinction per se. but did provide some evidence that reflect on this process. Since no further information was available and because these studies are consistent with the unprepared learning data relative to extinction a conservative prediction based on the systematic elimination of a prepared avoidance response

was appropriate. Thus, the first hypothesis was; the same variables that influence the extinction of an unprepared avoidance response would apply specifically to the extinction of a prepared avoidance response. The conclusions resulting from this hypothesis were (1) Extinction involving the repeated presentation of the CS in the absence of the US will be superior to the other extinction technique used in this study. (2) The more trials of this variety administered, the more extinction that will occur. Stated in terms of the dependent measure this means that the rats administered, 10 forced exposures to the CS will consume more saccharine than those rats administered five extinction trials to the CS. Those rats given one forced CS trial will evidence the least recovery of the subjects treated in this manner when tested after extinction.

Kalat and Rozin (1971) have concluded that learned taste aversions are adaptive responses having significant survival value. Inferences from this conclusion can be outlined in a series of statements; (1) the consummatory behavior of the rat is primarily influenced by gustatory cues. (2) stomach malaise probably results from the consumption of poisoned "food", since the eating of many foods has occurred before,

without poisoning, (3) future avoidance of this "food" is essential and dependent on the rat's ability to recognize this substance by its gustatory quality.

It was logical to conclude, applying these statements of adaptability, that aversions to gustatory cues associated with illness are acute rather than chronic. This conclusion has merit from a natural selection standpoint i.e. it limits the size of the category of poison "food" and would result in future sampling of previously poisoning "food". If this speculation has merit, one would expect to see a reduction in the avoidance of the CS simply as a function of time. Thus, the second hypothesis was; not only do the variables of unprepared extinction apply to the elimination of a conditioned taste aversion but adaptive mechanisms based on natural selection augment the likelihood of extinction in the absence of any treatment. The predictions based on this logic were; (1) "treatment" involving neither the presentation of the CS nor the US will eventually diminish the conditioned taste aversion, (2) the longer the retention interval between the last poisoning and the test presentation of the CS, the weaker the aversion to the CS. Thus, a retention interval of 20 days will result in more ex-

tion of the avoidance response than one of 10 days. A test of the aversion after a single day following poisoning will not be an effective treatment procedure.

Kalat and Rozin in Seligman and Hager (1972) have suggested that conditioned taste aversions may be a form of cognitive learning. They reason that the rat actively ascribes it's illness to the novel CS. This interpretation is in contrast to a more "primitive" (non-cognitive) form of learning and has provoked considerable discussion among researchers who subscribe to the learned safety interpretation of conditioned taste aversion. Although Kalat and Rozin have not provided data that supports a cognitive explanation for the taste aversion phenomenon, a test of it's explanatory efficacy was included in this study. This test involved the repeated injection of poison (LiCl) after conditioning. If the rat has cognitions and actively attributes it's malaise to the novel saccharine solution then repeated non-contingent illness should elicit other cognitions inconsistent with the original ones. These cognitions should facilitate extinction when the CS is subsequently presented without the US. Relative to the predictions of outcome it was inconsistent with the original hypothesis to

conclude that repeated LiCl injections would facilitate extinction. The original hypothesis stated that the principles of unprepared learning apply to the situation described in this study. In the unprepared literature one finds that repeated US presentations subsequent to conditioning sensitize the subjects i.e. maintain the aversion when the CS is subsequently presented (Kimble, Mann & Dufort, 1955). Therefore, the predictions relative to this were; (1) LiCl injections following saccharine-LiCl pairings will maintain the conditioned taste aversion, and (2) the sensitization will be most evident for those subjects receiving 10 non-contingent injections. Sensitization will also be evident for the five-injection and single-injection groups..

METHOD

Subjects

The subjects were 72 experimentally naive male rats about 65-days old obtained from the Charles River Breeding Laboratory. Subjects were randomly assigned to control or experimental groups.

Apparatus

The experiment was conducted in standard 20 cm. square wire mesh housing units. The solutions were presented in 100 cc.-drinking tubes (Kimble #44875) constructed to prevent drippage and accurate to 0.25 cc. Interperitoneal injections were administered with a 5 cc.-glass syringe.

Procedure

Twelve subjects were run in each of six replications. Each group of 12 rats was subdivided into four groups of three subjects each and run in one of the experimental conditions. Placement within each of these groups was random as was the choice of experimental groups for each replica-

tion. The description of the procedure is a general one that does not describe each of these replications but does describe the differential treatment of each of the twelve subgroups (See Table 1).

Training trials. For five days prior to training subjects were handled and adapted to a 22-hour-water-deprivation schedule. On day six and seven subjects were placed in experimental cages and permitted one hour access to water, made available in two drinking tubes mounted on either corner of the rear wall of the cage. This procedure familiarized subjects with the process of drinking from the tubes. Following the two tube training sessions subjects were presented a choice of water or a 0.1%-saccharine solution in each of the drinking tubes. Subjects had access to the solution for one half hour and were then removed to their home cages. No food or water was available in the home cages at this time.

Acquisition trials. Two hours after having been returned to their home cages three groups of 18 subjects were injected with LiCl (2% of body weight of .15M) ip. The fourth group of 18 rats was injected with an equal dose of NaCl. This concluded the first acquisition trial for the

TABLE 1

Design of the Acquisition and Extinction Phases for the Experimental and Control Groups

Acquisition Conditions			
Control rats n=18	Experimental rats n=54		
Extinction Condition			
Cs (LiCl) n=18	CS s (saccharine) n=18	US s (LiCl) n=18	R s (retention) n=18

n=6 for all cells

Number of
Extinction trials

four groups.

After the injections all subjects were given access to water for two hours in their home cages. Food was also available in the form of standard lab blocks. The food remained available until the next tube-drinking session. The removal of the water after two hours resulted in a 19.5 hour deprivation schedule for the next and all subsequent sessions. The next session, on the following day, constituted a check for prolonged illness. All subjects were placed in the experimental cages and were allowed to drink from the tubes for one half hour. Both tubes were filled with water only. Forty-eight hours after the first session a second acquisition trial was run with the same deprivation level and delay of reinforcement (LiCl or NaCl) maintained. The water tube and saccharine tube positions were reversed, however. A final acquisition trial occurred 48 hours after the second with an illness check session (water in both tubes) on the interpolated day between session two and three. This concluded the acquired aversion phase of the investigation. The dependent measure during this phase was the amount of saccharine consumed on each of the three acquisition days. The amount of water consumed on the in-

terpolated days was also recorded.

Extinction trials. The 54 subjects which received saccharine followed by LiCl were assigned to one of three extinction conditions. Eighteen subjects were subdivided into three subgroups of six subjects each that received either 1, 5 or 10 forced exposures to saccharine only. Forced exposure was accomplished by filling both drinking tubes with saccharine. The extinction phase began for the three subgroups on the day following the last acquisition trial. For those subjects receiving more than one forced exposure to the saccharine, extinction sessions occurred every other day. On interpolated days subjects were given water in both tubes. The specific extinction procedure was as follows; after 19.5 hours of water deprivation the three groups of rats to be administered the forced exposure to saccharine were placed in the experimental cages for one half hour. Since only saccharine was available this procedure exposed subjects to the conditioned stimulus (CS) in the absence of the unconditioned stimulus (US), the LiCl. The three saccharine forced exposure subgroups are hereafter referred to as CS₁, CS₅ and CS₁₀. At the conclusion of the half hour session the CSs were given access to food and

water in their home cages. Four hours after terminating the extinction session the home-cage water bottles were removed. The food remained available ad lib. until the beginning of the next session. This process continued for the CS₅ and CS₁₀ groups for 10 and 20 days respectively. On the interpolated days, CS subjects were given water in both tubes in the experimental cages. The experimental and home cage procedure, however, remained invariant.

A second group of 18 rats was subdivided into three subgroups of six rats each and received 1, 5 or 10 non-contingent injections of LiCl. Since LiCl served as the unconditioned stimulus, these three groups are hereafter referred to as US₁, US₅ and US₁₀. The injections, 2% of body weight of .15M LiCl ip, were administered every other day to the US₅ and US₁₀ subgroups for 10 and 20 days respectively. The procedure on the interpolated days was the same procedure administered to the CS subjects.

The remaining 18 rats of the 54 that received saccharine and LiCl in acquisition were not given any treatment during the extinction phase of the study. Since no treatment followed by a test of the aversion strength to saccharine constitutes a test of the retention of the saccharine-

LiCl association these latter 18 rats are hereafter referred to as Rs. Three subgroups of Rs were formed; R_1 which was tested one day after acquisition and R_5 and R_{10} which were tested after 10 and 20 days respectively. While the rats in the CS and US conditions were receiving their extinction sessions the Rs were merely handled for several minutes. On interpolated, non-extinction, days the R_5 and R_{10} groups were treated in the same manner as the CS and US groups.

The 18 subjects that had been injected with NaCl on each of the acquisition trials served as dual-purpose controls. First, all 18 served as conditioning-acquisition controls. Since NaCl does not produce gastric upset in rats no aversion to saccharine (conditioning) should result. Thus, the amount of saccharine consumed should increase from trial to trial during acquisition. Prior to extinction the 18 control subjects, hereafter referred to as Cs were subdivided in three equal subgroups; C_1 , C_5 and C_{10} . C_1 received one non-contingent injection of LiCl while the latter two subgroups received 5 and 10 injections respectively over 10 and 20 days respectively. On the interpolated non-extinction days the control rats

were given access to water in both drinking tubes in the experimental cages as described for the groups above. Thus in the extinction phase the C's served a second purpose as sensitization controls for the effect of illness per. se.

Testing trials. The final phase of this study constituted a test of the effectiveness of the three extinction procedures. At the conclusion of their respective extinction phase each of the twelve subgroups (See Table 1) was returned to the experimental apparatus and reexposed to the saccharine-water choice procedure. Water was always available in one tube while the other tube contained the 0.1% saccharine solution. Counterbalancing for tube position was continued throughout the test phase. There were seven test sessions of one half hour duration on seven successive days. Subjects were never administered LiCl during the test phase. In all cases subjects were 19.5 hours water deprived at the beginning of a test session and were given access to food and water in their home cages two hours after the conclusion of each test session. The water remained available for two hours only while the food remained accessible ad lib. until the next test trial.

Two dependent measures were of interest in the test of

extinction effectiveness; (1) the amount of saccharine consumed on the first test day and, (2) the recovery sequence as measured by saccharine consumption over the entire test phase.

Despite some heterogeneity of variance in some of the 12 subgroups the major analyses were done using analyses of variance. The ANOVA was used because heterogeneity of variance is not a serious problem when other conditions are met. Boneau (1960) describes these conditions as does the Lindquist (1953) presentation of the Norton study (1951). In brief, if the sampling distributions have different variances or deviate from normality but are approximately symmetrical in shape, there will be little effect of heterogeneity on the F test. This conclusion is predicated on equal cell size. A perusal of the group distributions in this study indicated very little skewedness or departure from normality and all cell frequencies were equal. Relative to the multiple comparison test employed in this study, the Scheffe test, (1963) reports that it too is insensitive to departures from normality.

RESULTS

Acquisition of the Conditioned Taste Aversion

Prior to analyzing the acquisition data the 54 experimental subjects were assigned to one of the three extinction conditions; CS, US or R. These 18 subjects per condition were further subdivided so that six subjects from each condition would ultimately receive 1, 5 or 10 extinction trials. This grouping resulted in the formation of nine subgroups. The 18 control rats were also subdivided into three groups of six subjects each (See Table 1).

Throughout the acquisition phase the nine experimental groups were treated identically. They were injected with LiCl after being exposed to saccharine. The three control subgroups were administered NaCl after their saccharine exposure. The subgroups were analyzed, after assignment to their respective extinction conditions, to illustrate that prior to the extinction manipulation all experimental groups (and all control groups) were equated in their de-

gree of saccharine aversion.

The acquisition procedure entailed the differential treatment of the nine experimental groups relative to the three control groups. All 72 subjects were given three half hour daily exposures to saccharine every other day. The experimental subjects were injected with LiCl two hours after the termination of each exposure trial. The control subjects, on the other hand, were injected with NaCl.

The prediction was that the experimental subjects would acquire an aversion to the novel tasting substance, the saccharine, because of the LiCl induced illness. The controls, however, would not become ill and no saccharine aversion would result.

Table 2 illustrates the mean amount of saccharine consumed on each of the three acquisition trials for the control and experimental subjects. The acquisition phase of Figures 1-7 visually illustrate these data. The figures clearly indicate that the control subjects increase their intake of saccharine on each of the three acquisition trials. The experimental groups markedly decrease their consumption of saccharine on the second acquisition trial with a near floor effect after the first LiCl in-

TABLE 2

Mean Amount of Saccharine Consumed (Cubic Centimeters) on
Each of the Three Acquisition Trials

Groups	N	Day 1		Day 2		Day 3	
		Mean	SD	Mean	SD	Mean	SD
Controls	18	6.22	4.03	11.21	3.25	12.53	4.43
Conditioned Stimulus	18	5.29	2.80	1.11	0.86	0.72	0.77
Unconditioned Stimulus	18	5.74	3.67	1.07	0.85	0.56	0.36
Retention	18	5.35	3.47	1.39	1.03	0.72	0.41

Fig. 1. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for the control rats.

Fig. 2. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for the rats extinguished by the forced exposure to saccharine.

MEAN SACCHARINE CONSUMED (cc)

Fig. 3. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for the rats extinguished with LiCl injections.

MEAN SACCHARINE CONSUMED
(cc)

Fig. 4. Mean amount of saccharine consumed on each of the three acquisition trials and on each of the seven test trials for the rats in the retention extinction condition.

Fig. 5. Mean amount of saccharine consumed on the three acquisition trials and the seven test trials for all groups having received one extinction trial.

Fig. 6. Mean amount of saccharine consumed on the three acquisition trials and the seven test trials for all groups having received five extinction trials.

Fig. 7. Mean amount of saccharine consumed on the three acquisition trials and the seven test trials for all groups having received ten extinction trials.

jection illustrated by the amount of saccharine consumed on the second acquisition trial relative to the third trial.

A three factor mixed design ANOVA, extinction condition by number of extinction trials by sessions (acquisition days), was used to determine the significant main effects and interactions. The source table for this analysis appears as Appendix A. This three way ANOVA with one repeated factor (sessions) indicated a significant extinction condition effect ($F = 96.41$, $df = 3/60$, $P < .001$). A Scheffe Multiple Comparison Test (Winer, 1971) was employed to determine the group differences. The Scheffe test indicated that the Cs drank significantly more saccharine than the CSs, USs or Rs ($F = 18.51$, $df = 3/60$, $P < .001$). It further indicated no differences between the CSs, USs or Rs. The ANOVA also indicated a significant sessions (acquisition days) effect and the sessions by extinction condition interaction also was significant ($F = 36.28$, $df = 2/120$, $P < .001$ and $F = 32.58$, $df = 6/120$, $P < .001$ respectively).

The Scheffe multiple comparison of the interaction data indicated that the controls and all the experimental groups were equated on the amount of saccharine consumed on the first acquisition day ($F = 67.87$, $df = 11/120$, $P < .001$).

Furthermore, the controls drank the same amount of saccharine on acquisition days two and three but more than they drank on the first exposure to saccharine. The controls drank significantly more saccharine than the CSs, USs and Rs on days two and three also. There was significantly more saccharine consumed by the CSs, USs and Rs on the original acquisition trial compared to trials two and three but no difference between any of these groups on the second and third acquisition trial. Apparently a single saccharine-LiCl pairing is sufficient to produce a near maximum aversion to the saccharine. The means shown in Table 2 clearly illustrate these results.

The Amount of Saccharine Consumed on the First Test Day

The effect of the extinction procedure was determined by recording the amount of saccharine consumed on the first of the seven test trials. The predictions were that the CSs would evidence the most recovery and no recovery would be indicated for the USs. Some effect of recovery was expected for the R_5 and R_{10} groups. The extinction condition by number of extinction trials interaction showed that the CSs having received 5 or 10 extinction trials

drank as much saccharine as C_1 and C_5 on the first test day and significantly more than the USs and Rs which were equated on saccharine consumed. This indicates that CS_5 and CS_{10} are nearly fully recovered as a result of forced exposure to the saccharine. CS_1 is not different from the USs or Rs providing evidence that although the forced exposure to the saccharine is an effective extinction procedure, a single treatment in this manner is not sufficient. The data for the first test day on Tables 3-6 and Figures 1-7 graphically display these results. A two way ANOVA, factorial design, was employed to determine the effect of the extinction procedures and the number of extinction trials as well as the extinction condition by trials interaction. The source table for this analysis appears as Appendix B.

This two way ANOVA showed a significant extinction condition effect ($F= 141.40$, $df= 3/60$, $P<.001$). The Scheffe test indicated that the Cs drank significantly more saccharine than the CSs, USs and Rs on the first test session ($F= 8.28$, $df= 3/60$, $P<.001$).

The number of extinction trials was also a significant factor in therecovery from the acquired aversion to saccharine ($F= 11.60$, $df= 2/60$, $P<.001$). The Scheffe Test

TABLE 3

Mean Amount of Saccharine Consumed (Cubic Centimeters) on
Each of Seven Test Trials (n=6) by the Control Groups

Groups		1	2	3	4	5	6	7
C ₁	Mean	16.83	15.96	17.08	19.88	17.86	19.21	17.25
	SD	6.34	6.01	4.43	4.34	5.10	5.55	5.04
C ₅	Mean	15.25	18.79	18.04	20.08	18.50	19.00	16.86
	SD	6.13	5.24	4.84	3.01	2.15	2.65	3.20
C ₁₀	Mean	22.04	21.96	21.88	21.29	22.58	22.58	23.13
	SD	7.85	4.90	7.58	6.61	7.43	9.34	6.40

TABLE 4

Mean Amount of Saccharine Consumed (Cubic Centimeters) on Each of
Seven Test Trials (n=6) by the Saccharine-Only Groups

Groups		1	2	3	4	5	6	7
CS ₁	Mean	0.92	1.96	1.17	1.71	2.54	6.83	8.86
	SD	0.63	1.75	1.11	1.15	2.38	4.72	7.77
CS ₅	Mean	11.63	14.25	11.33	13.86	13.96	13.79	14.29
	SD	4.05	6.46	5.08	4.85	3.77	2.14	4.44
CS ₁₀	Mean	12.54	13.96	13.50	14.25	14.75	14.86	12.42
	SD	5.86	3.70	4.72	7.44	5.59	6.43	7.14

TABLE 5

Mean Amount of Saccharine Consumed (Cubic Centimeters) on
Each of Seven Test Trials (n=6) by the LiCl-Only Groups

Groups		1	2	3	4	5	6	7
US ₁	Mean	0.54	0.38	0.46	0.58	1.25	4.04	1.42
	SD	0.37	0.21	0.68	0.30	1.86	2.56	1.48
US ₅	Mean	1.29	2.96	3.71	4.25	6.29	6.17	11.38
	SD	0.62	2.53	3.64	3.74	3.43	5.42	7.42
US ₁₀	Mean	0.50	1.50	2.75	4.54	7.50	8.54	11.96
	SD	0.45	0.91	2.19	4.29	6.97	8.85	9.36

TABLE 6

Mean Amount of Saccharine Consumed (Cubic Centimeters) on
Each of Seven Test Trials (n=6) by the Retention Groups

Groups		1	2	3	4	5	6	7
R ₁	Mean	0.54	0.45	0.58	0.96	1.25	3.50	3.08
	SD	0.25	0.33	0.56	0.29	0.39	2.74	3.76
R ₅	Mean	0.36	0.79	0.96	1.86	1.96	5.13	7.46
	SD	0.44	0.71	0.70	2.27	1.60	5.13	8.99
R ₁₀	Mean	0.42	0.38	1.00	0.88	0.71	1.33	2.33
	SD	0.30	0.49	0.42	0.41	0.46	1.03	3.18

indicated that 10 extinction trials are superior to five extinction trials which are more effective than one extinction trial ($F = 15.52$, $df = 2/60$, $P < .001$). The significant interaction effect ($F = 9.00$, $df = 6/60$, $P < .001$) added clarity to these results. The Scheffe Multiple Comparison Test of the interaction effect showed that the Cs having received 10 extinction (sensitization) trials drank more saccharine than any of the CSs regardless of the number of extinction trials. However, the CSs having received five or 10 extinction trials drank as much saccharine as the controls having received one or five sensitization trials. Finally, all of the Cs and the CSs having received five or 10 extinction trials drank significantly more than the USs or Rs irrespective of the number of extinction trials administered to the latter two groups ($F = 36.75$, $df = 11/60$, $P < .001$).

The Amount of Saccharine Consumed Over the Entire Test Sequence

The effect of the extinction procedures were also examined over the entire seven day test sequence. The predictions were the same as those discussed under the previous heading. This procedure was employed for a specific

purpose. It was reasoned that the effect of the extinction procedure may not be evident from a single observation of the saccharine intake on one test day. The extinction procedure effect, it was thought, might interact with the saccharine presentation over the entire choice test phase. This result might occur for the USs which were made repeatedly and non-contingently ill during extinction.

The effect of the extinction manipulation when measured over the entire test sequence shows that the CSs never fully recover and do not consume as much saccharine as the Cs. Clearly, however, forced exposure to the saccharine is the most effective extinction procedure for rats having acquired a conditioned taste aversion. Five forced exposures are as effective as 10 but a single forced exposure is ineffective. Although at the outset of the test phase the USs manifested as strong an aversion to the saccharine as the Rs. The recovery exhibited by US₅ and US₁₀ suggests that the repeated non-contingent illness has a delayed effect which is potentiated by the choice procedure of the test phase. US₅ and US₁₀ consume much more saccharine than US₁ and all of the Rs by the end of the test

sequence. Tables 3-6 and the test data of Figures 1-7 graphically display these results.

A three factor mixed design ANOVA, extinction procedure by number of extinction trials by sessions (test days), was employed to determine the effect of the extinction manipulations. The Source Table for this analysis appears as Appendix C. This three way ANOVA indicated a significant extinction effect ($F = 122.67$, $df = 3/6$, $P < .001$). A comparison of the extinction conditions by the Scheffe test showed that the Cs drank more saccharine than the CSs, USs or Rs over the consecutive seven day period. The CSs consumed significantly more than the USs or Rs ($F = 18.51$, $df = 3/60$, $P < .001$). There was also a significant F statistic for the number of extinction trials effect ($F = 18.06$, $df = 2/60$, $P < .001$). The Scheffe comparison of these three parameters indicated that overall the subjects that had 10 extinction trials consumed more saccharine than the subjects that were administered five extinction trials. Both of these groups drank more than the subjects having received a single extinction trial ($F = 12.34$, $df = 2/60$, $P < .001$). The interaction of extinction conditions by trials was also significant and of interest ($F = 5.70$, $df = 6/60$, $P < .001$).

The Scheffe method of comparing these groups showed that C_1 and C_5 were equated on the amount of saccharine consumed over the entire test phase. However, C_{10} drank a greater amount than either C_1 or C_5 . Since none of these groups were made contingently ill during the acquisition phase i.e. saccharine and LiCl were never paired, the increased C_{10} intake may simply be a function of age and weight. The Cs irrespective of number of extinction trials consumed more saccharine than any of the Cs. These results indicate that the non-contingent LiCl injections administered to the Cs as a sensitization control for illness pers. se. confirm the necessity for saccharine-LiCl pairing in order to establish an aversion to the saccharine. These results, furthermore, would seem to suggest that full recovery from conditioned aversion does not occur at least with the parameters employed in this investigation. Consistent with those findings described for the single test of recovery, CS_{10} consume no more saccharine than CS_5 but significantly more than all other groups with the exception of the Cs noted above. Of particular interest is the fact that US_5 consumes as much saccharine as US_{10} , and both consume significantly more saccharine than US_1 , CS_1 and all Rs. These

results would tend to confirm an extinction procedure-test phase interaction. Repeated non-contingent injections of LiCl followed by saccharine in a water-saccharine choice situation has a beneficial effect on recovery from the aversion. Finally, there are no differences between R_1 , R_5 and R_{10} nor between these groups and CS_1 or US_1 .

The ANOVA indicated a significant sessions (test days) effect ($F = 25.00$, $df = 6/360$, $P < .001$) which after further analysis using the Scheffe test showed an increase in saccharine consumption over the seven day test phase ($F = 37.02$, $df = 6/360$, $P < .001$). The sessions by extinction condition interaction was also significant ($F = 3.75$, $df = 18/360$, $P < .001$) but is not of particular interest because the minimal amount consumed by CS_1 , US_1 and R_1 depresses the other CS's, US's and R's to such an extent as to make a comparison of the experimental groups meaningless. The sessions by trials interaction was not significant ($F = 1.25$, $df = 12/360$, n.s.) but is of no interest to this investigation in any case. Finally there was a significant three way interaction: sessions by extinction condition by number of extinction trials ($F = 2.25$, $df = 36/360$, $P < .01$). The interpretation of this interaction can be gleaned from Figures 1-7. It is ob-

vious that C_{10} drank an equal amount of saccharine on each of the seven test days. Also they drank more than either C_1 or C_5 probably as a function of being at least 10 days older and thus heavier. Of greater interest is the fact that CS_5 and CS_{10} are significantly recovered from their aversion to saccharine at the outset of the test phase.

This is not the case for the US s and R s that had received five or 10 extinction trials. CS_1 recovers during the test phase but at the same rate as US_1 . However, CS_1 never recovers to the level of CS_5 or CS_{10} . These figures also indicate, as noted above, that the rate of recovery for US_5 and US_{10} appears to be potentiated by the non-contingent $LiCl$ injections relative to the R s which received no extinction treatment.

DISCUSSION

Interpretation of Acquisition Results

The results depicting the amount of saccharine consumed by the three control groups on each of the three acquisition sessions indicates an initial neophobia for the saccharine. On the second and third acquisition trials, the C's significantly increased their ingestion of saccharine (refer to the acquisition data of Table 2). These results are consistent with those reported by Nachman and Jones (1974) who interpret the initial avoidance as supportive of the learned safety hypothesis. That is, the rats restrict their intake of saccharine until the consequences of ingestion become obvious. When no illness occurs the rats increase their consumption of the novel food.

In sharp contrast to the ingestion pattern exhibited by the C's the experimental subjects significantly decrease their saccharine consumption on the second and third acquisition trials (They do evidence a neophobia for the novel solution on the first trial, however).

The decreased consumption of saccharine by groups CS, US and R on sessions two and three is consistent with the results from the classical conditioning literature on unprepared learning and was expected because they were injected with LiCl after they drank saccharine.

In the literature relating to the acquisition of a classically conditioned response two points are consistently confirmed. First, before a CS can elicit a CR the CS must be repeatedly paired with the US. Second, not only must there be repeated pairings of the CS and US; they must be presented contiguously.

Although the data of this study does not provide evidence that is relevant to the equal associability of stimuli, the results are important to a discussion of the delay of reinforcement gradient. Conclusions based on classical conditioning of unprepared learning lead to the prediction that conditioning would not occur where LiCl followed the saccharine because the ISI was two hours long. Hull (1952), White and Schlosberg (1952), and Gormezano and Moore In Marx (ed.), 1969 have determined that the optimal ISI for most response systems is 500 msec. Noble and Adams (1963) and Notterman, Schoenfeld and Bersh (1952) have described

long ISI as 4,000 msec. and 7,000 msec. respectively. Young (1958) was unable to establish a classical conditioned pupillary response with a 15,000 msec. ISI.

The long delay of reinforcement (two hours) used in this study clearly indicates that at the very least the parameters that apply to the acquisition of unprepared responses do not apply to prepared learning. This fact together with data reported elsewhere indicating that specific associations are established in prepared learning (Garcia & Koelling, 1967 and Kalat & Rozin, 1971) provide efficient evidence to question more than the range of ISI.

Kalat and Rozin (1971) propose to treat prepared learning as a biological characteristic of some species, which is subject to natural selection and therefore adaptative. Prepared learning indicates the presence of special neural mechanisms which have developed because of important environmental problems (e.g. obtaining adequate foods). The same mechanisms involved in the acquisition of unprepared responses are probably involved in taste aversion learning, too. But, some stimuli are far more salient than others. This may mean that specific neural circuits and pathways associated with these stimuli and their potential consequence have

a lower threshold of excitability and a tendency, acquired through natural selection, to remain active for a long period of time when activated by relevant afferents. A complete discussion of the CS, US and R extinction results is of particular importance for an analysis of conditioned taste aversion relative to prepared and unprepared learning.

The results from classical conditioning unprepared studies predicts that the CS procedure will be the only effective extinction technique. Every significant investigation involving prepared or unprepared learning indicates the superiority of this method. This is true of all response systems across all species (Razran, 1939; Garcia, Erwin & Koelling, 1966; Grote & Brown, 1973). When the response is an appetitive one the extinction is usually complete. However, when an avoidance response is classically conditioned unprepared extinction is often only partial. Solomon and Wynne (1954) have reasoned that incomplete extinction of avoidance responses may be due to response conservation and partial irreversibility. Response conservation refers to the inferred state of anxiety that is assumed to result from the presentation of the aversive CS. As the aversive conditioning progresses, the subjects latency to emit the conditioned

avoidance response decreases. The latency to respond is also short on most extinction trials suggesting that the fear component may not be available to extinguish. When the latency to respond following the CS presentation is long, as it is on some trials, fear is assumed to be present from the behavior of the subject and is reduced by avoiding. This inferred process is offered by Solomon and Wynne to account for the persistent responding in the absence of the US. Blocking which forces the subject to experience both the external CS and the inferred fear does result in extinction (Schiff, Smith & Prochaska, 1972) and supports the analysis of response conservation. Bolles (1970) has concluded that all avoidance learning is of the prepared variety thus, Solomon and Wynne's (1954) analysis has some relevance to the results reported here.

The classical conditioning unprepared literature is also consistent relative to the decay of the association established in avoidance learning. Marx (1969) reports that the evidence always supports the superiority of repeated CS presentations over forgetting (trace decay as a function of disuse). Furthermore, Skinner (1960) has dramatically shown the stability of associations over long

periods. In a widely read paper, Skinner showed that a specific pecking response required to guide a missile remained available to pigeons after a period of four years with no maintenance sessions following the acquisition of this response. Thus, from the unprepared learning perspective the effect of the "retention" manipulation used in this study should have been minimal. This prediction contrasts with the analysis of adaptive behavior described by Kalat and Rozin (1971) and is contrary to the prediction that decay of the CS-US association would occur in this study.

The results described earlier show that the CSs do not fully recover to the level of the CS. Recovery is substantial, however, and both of these facts are consistent with what is known about avoidance studies reported under the unprepared (Solomon & Wynne, 1954) and prepared categories (Garcia, Erwin & Koelling, 1966; Grote & Brown, 1973). An analysis of the parametric effects of the 1, 5 and 10 extinction trials is also consistent with previously reported results in that a single CS presentation is ineffective. Five or 10 trials were equally effective which is somewhat surprising given the extent of the aversion to the saccharine (The test phase of Figures 5-7 illustrate these re-

sults).

The effectiveness of the Retention procedure proved to be totally unsupportive of the hypothesis that conditioned taste aversions are acute rather than chronic. The complete absence of recovery for all subjects treated in the retention manner shows that the association between the novel taste cue and the subsequent stomach malaise does not deteriorate as a function of disuse; at least this is not the case 20 days after the last acquisition trial. These results are fully supportive of the predictions based on the unprepared classical conditioning evidence (Marx, 1969; Skinner, 1960). A test of the aversion after a retention interval considerably longer than 20 days might provide different results but this is mere speculation at this time. Finally, the partial recovery evidenced at the end of the seven day test phase is not a good measure of an extended disuse period because decay was confounded with the CS extinction procedure since all rats drank at least some saccharine during the test phase.

The extinction procedure that utilizes the repeated presentation of the US alone (LiCl) provides some interesting results that deserve space here. The predictions about

the effectiveness of this procedure were drawn from results reported in the unprepared learning area. Reynolds (1968) reports that the repeated presentation of the US alone after conditioning sensitizes the organism to the CS when it is presented again. A specific test of this prediction (Kimble, Mann and Dufort, 1955) supports Reynolds' generalization but data inconsistent with the Kimble et al. results have been reported (McAllister & McAllister, 1960). The prediction, relative to this study, was based on the sensitization data. It was expected that rats having been repeatedly and non-contingently injected with LiCl would, on the subsequent test of the CS aversion show a persistent avoidance of the saccharine. In contrast Kalat and Rozin

In Seligman & Hager, 1972 have reasoned that a procedure that indicates to the rat that the saccharine did not produce the stomach malaise will enhance recovery by producing cognitions inconsistent with the saccharine-illness attribution. Since repeated illness resulting from the non-contingent US injections provides information indicating that the initial novel stimulus-illness relationship was non-causal this procedure served as a test of Kalat and Rozin's hypothesis that conditioned taste aversion is a cognitive

form of learning. A single study by Kalat and Rozin reported in Seligman and Hager (1972) provides the only other data that reflects on this point. These results were unsupportive of a cognitive interpretation of taste avoidance learning.

Although CS₅ and CS₁₀ were more effective extinction procedures than US extinction, a check of Figures 3 and 4 provides some evidence that the US procedure was at least partially effective. US₅ and US₁₀ did consume significantly more saccharine than CS₁, US₁ and all R's. Since it did result in significantly more saccharine consumption relative to R₅ and R₁₀ one might conclude that repeated stomach malaise not associated with the CS after conditioning tends to facilitate recovery from the conditioned avoidance when the CS is later presented. As an alternative to the possibility that US presentations enhance CR decrement (and extinction) as the McAllisters' suggest, Kalat and Rozin's cognitive learning hypothesis can presently incorporate the results of this study. Data that support Kalat and Rozin's thesis are not in agreement with unprepared learning analysis. Unprepared learning theorists do not make reference to cognitions but rely on more "primitive"

learning mechanisms consistent with unprepared learning apply. But, when the rats are made repeatedly ill in the absence of saccharine after saccharine had been previously paired with LiCl an opportunity may exist for the rat to conclude that it could have been the case that the saccharine did not cause the illness. The first test of this cognition occurs when the rat is presented the saccharine solution on the first test day. Since the rat does not become ill subsequent to this test trial the cognition is confirmed and recovery (extinction) is expedited.

The results based on the CS and R procedures fully support the conclusion that the extinction of a conditioned taste aversion parallels the elimination of an unprepared response. However, the US data do not appear to support the general finding that US presentations after conditioning enhances the aversion to the CS when it is subsequently reintroduced (Kimble et. al., 1955). There is some evidence for CR decrement following this procedure, however (McAllister & McAllister, 1960) and should further investigations corroborate the latter findings the US results of this study might also be incorporated into the unprepared learning category.

APPENDIX A

Analysis of Variance of the Amount of Saccharine Consumed
on the Three Acquisition Trials for All Groups of Rats.

Source	Sums of Squares	df	Mean Squares	F
Total	3.513	215		
Between Ss	2.069	71		
Extinction Cond.	1.678	3	0.559	96.414*
Trials	0.008	2	0.004	<1
EC X Trials	0.034	6	0.006	<1
Error _b	0.349	60	0.006	
Within Ss	1.444	144		
Sessions	0.261	2	0.131	36.278*
Sessions X EC	0.704	6	0.117	32.583*
Sessions X Trials	0.003	4	0.001	<1
Sessions X EC X Trials	0.035	12	0.003	<1
Error _w	0.439	120	0.004	

*P<.001

APPENDIX B

Analysis of Variance of the Amount of Saccharine Consumed
on the Seven Test Trials for All Groups of Rats.

Source	Sums of Squares	df	Mean Squares	F
Total	19.141	503		
Between Ss	16.457	71		
Extinction Cond.	12.143	3	4.048	122.667*
Trials	1.191	2	0.596	18.061*
EC X Trials	1.126	6	0.188	5.697*
Error _b	1.997	60	0.033	
Within Ss		432		
Sessions	0.604	6	0.100	25.000*
Sessions X EC	0.263	18	0.015	3.750*
Sessions X Trials	0.063	12	0.005	1.250
Sessions X EC X Trials	0.316	36	0.009	2.250**
Error _w	1.438	360	0.004	

* P<.001

** P<.01

APPENDIX C

Analysis of Variance of the Amount of Saccharine Consumed
on the First Test Trial for All Groups of Rats.

Source	Sums of Squares	df	Mean Squares	F
Total		71		
Extinction Cond.	2.120	3	0.707	141.400
Trials	0.116	2	0.058	11.600
EC X Trials	0.269	6	0.045	9.000
Error	0.293	60	0.005	

All P's < .001

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