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# Modification of reactivated memory through "counterconditioning"

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Four experiments using rats were conducted to determine whether a "counterconditioning" procedure would be effective in altering old, but reactivated, memory. The aversiveness of previously established Pavlovian conditioned stimuli was reduced by giving subjects a highly preferred substance (maltose solution) shortly after a brief exposure to the fear cues (Experiments 1 and 2). No evidence of a time-dependent effect was obtained with a 1-hr. delay between reactivation and maltose (Experiment 2). Groups given noncontingent footshocks in lieu of Pavlovian conditioning (whether or not they subsequently received maltose) showed uniformly little aversion to test cues (Experiment 3). This finding suggests that counterconditioning in this paradigm affects associative memory processes. A time-dependent effect of delayed treatment and other evidence that active memory is necessary for counterconditioning were obtained (Experiment 4). These experiments support the notion that in rats as well as in humans, memory is a malleable process susceptible to postacquisition modifications and revealed the potential value of the reactivation paradigm in studying counterconditioning as a model for desensitization.

The nature, or characteristics, of old memory is an issue of relatively recent vintage in research on animal learning. Several studies have focused on whether an established memory, when reactivated or retrieved, becomes susceptible to retrograde amnesia in a manner akin to new learning. One major finding has been that vulnerability of information to retrograde amnesia may be determined more by the state of activity of the memory than by its age (Lewis, 1979). Very generally, these studies have shown that amnesia can be induced after delays far exceeding the usual temporal gradient by exposing subjects to either the conditioned stimuli (DeVietti & Holliday, 1972; Lewis & Bregman, 1973; Mactutus, Riccio, & Ferek, 1979; Misanin, Miller,


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& Lewis, 1968) or the reinforcer (Howard, Glendenning, & Meyer, 1974; Robbins & Meyer, 1970; Schneider & Sherman, 1968) prior to the amnestic treatment; in the absence of such reinstatement the amnestic agent is ineffective. This outcome suggests that reinstatement of certain cognitive or motivational attributes of original training reactivates the earlier memory, and once active, the memory is subject to change (Lewis, 1979). Since there is some evidence that recovery can be induced from this amnesia for old memory (Mactutus et al., 1979), it may be useful to view the phenomenon in terms of altered retrieval, in which the target memory becomes reencoded with respect to the new (amnestic treatment) context (Riccio & Ebner, 1981).

A second major finding is that reactivated memory can interact with drugs or subsequent learning. Examining the hypermnesic effects of strychnine, Gordon and Spear (1973a) demonstrated that the drug facilitated retention of old as well as newly acquired memory, provided that the older memory was reactivated by a cueing exposure. In another series of studies, Gordon and his colleagues (Gordon, 1977; Gordon, Frankl, & Hamburg, 1979; Gordon & Spear, 1973b) have investigated the proactive interference effect produced by reactivated memory. When two competing memories (passive vs. active avoidance) are established with long intervals (24 hr.) between the two tasks, little proactive interference is obtained; however, substantial proactive interference occurs if subjects are exposed to cues (with no primary reinforcement) of the earlier task shortly before acquisition of the criterion task (e.g., Gordon et al., 1979).

These findings suggest that under certain conditions old memories can interact with new events in various ways. This raises the interesting question of whether the content of earlier established learning can be modified or transformed by subsequent information. If ECS or hypothermia impairs later retrieval of memory, can less drastic manipulations modify earlier learning? Once reactivated, can attributes of old memory be altered by providing new information? In short, does memory in nonverbal organisms have a "malleable" quality, as Loftus and her colleagues (Loftus, 1979; Loftus & Zanni, 1975) have demonstrated for humans?

The approach used here was to present a reinforcing stimulus (UCS) that was opposite in hedonic significance to that used in original acquisition. Presentation of this stimulus followed a cueing treatment intended to reactivate memory. Thus, if fear conditioning constitutes the target memory, can retention of the fear be reduced by

administering an appetitive reward after cue exposure? More generally, is the hedonic value or intensity of the original information modifiable? This paradigm might be described as a Pavlovian "counterconditioning" procedure, with the important distinction that the original conditioned stimuli are, in fact, not physically present during presentation of the new UCS.

## EXPERIMENT 1

The aim of the initial experiment was to determine whether the strength of fear could be reduced if subjects received a highly preferred food following a brief reactivation exposure to the conditioned fear stimuli. In order to increase the likelihood of appetitive behavior by a momentarily frightened rat, a strong incentive was provided in the form of maltose solution (Richter & Campbell, 1940) to mildly deprived animals.

The general plan of the experiment was to establish a strong tendency to approach and drink the maltose solution. Subjects subsequently received a Pavlovian fear conditioning session. Following a 1-day retention interval, memory was "reactivated" for some animals by brief exposure to fear cues. For half of these subjects, cue exposure was immediately followed by the presentation of the sugar solution; the other half received only the cue exposure (a condition included in order to assess any extinction effect of the reactivation). Twenty-four hours later, both groups were tested for fear to the conditioned stimuli, as was a third group that had received training but no interpolated treatments.

### METHOD

#### Subjects

In this experiment, 18 naive male albino rats (Holtzman) were used. The animals were approximately 80 - 100 days old at the beginning of the experiment. Each subject was housed in a single wire-mesh cage.

#### Apparatus

A 20 × 20 × 19 cm black Plexiglas box with a grid floor was used as the conditioning chamber. The grid floor consisted of .3-cm metal rods spaced 2 cm apart. The terminals from the floor were connected to a shock scrambler, which delivered a 1-sec, 100-V shock from a matched-impedance source (Campbell & Teghtsoonian, 1958). Technical grade maltose (65% M) purchased from the Fisher Scientific Company was used to make a 10% (w/v)

sugar solution. The solution was presented to subjects in graduated Richter tubes.

### Procedure

All animals were food deprived for 24–48 hr. to reduce them to 90% of their ad lib body weight. For the remainder of the experiment, the rats were on a restricted food diet to maintain this weight level. Fluid consumption was also restricted to a 15-min. period of access to water during these first 2 days and to the maltose solution on subsequent days.

Following the initial period of deprivation, each rat received maltose solution, to be consumed within a daily 15-min. period. Subjects received the solution in their home cages, which were moved into the room housing the experimental apparatus for the 15-min. session. This phase of the experiment lasted 5 days and was intended to establish a strong approach/consummatory response. The mean amount of solution consumed during the 15-min. period increased from 12 ml on the first day to 24 ml on the last day.

Subsequently, subjects were randomly assigned to one of three groups ($N$ = 6). All groups received Pavlovian fear conditioning to the black side of the black-white experimental chamber. Conditioning consisted of delivery of six 1-sec (100-V) shocks on a variable time (VT-30 sec) schedule during a 3 min. placement in the black compartment. Twenty-four hours later, two groups received a "cueing" exposure in which they were returned briefly (5 sec) to the fear cues. Immediately after the cueing, rats in one group (cue/malt) were moved to cages located in the same room and received a 5 min. presentation of maltose solution. The second group (cue/no malt) was also placed in cages in the experimental room for 5 min., but no sugar solution was available. The third group (no cue/no malt) served as a retention control and received neither cueing nor maltose. One day later, a passive avoidance test was employed to measure fear to the black compartment. Each animal was placed on the white (safe) side facing away from the door, and the time until it entered (four paws) into the previously shocked compartment was measured. Subjects not leaving the safe side were assigned the maximum score of 900 sec.

## RESULTS AND DISCUSSION

The median cross-through latencies (in sec) for the three groups were: cue/malt, 75; cue/no malt, 303; and no cue/no malt, 509. A Kruskal-Wallis analysis indicated a significant overall effect of treatment, $H = 8.12$, $p < .02$. Maltose immediately after cue exposure resulted in significantly shorter latencies than did either cue exposure only, $U = 4$, $p = .03$, two-tailed, or no cue exposure, $U = 3$, $p = .016$, two-tailed. The numerical scores suggest that cue exposure produced some extinction of fear, but the comparison was not significant (cue/no malt vs. no cue/no malt, $U = 10.5$, $p > .10$,

two-tailed). While there seems little doubt that a larger sample size would yield an extinction effect, the major finding of interest is the difference between the two experimental groups, which had equal "extinction" treatment but differed in subsequent appetitive experience.

The decreased latency to enter a previously shocked area for subjects receiving maltose shortly after termination of the exposure to the fear cues suggests that a memorial representation of the conditioning episode may have been modified. Although it is not clear how consumption of sugar water in a cage would provide a source of retroactive interference for Pavlovian conditioning administered in a different apparatus, the design of Experiment 1 did not address this possibility. Further, if the outcome obtained depends upon memory reactivation, then a time dependent decrease in the effectiveness of the maltose reinforcement should occur. In Experiment 2, we attempted to replicate the initial finding and examine these additional concerns.

## EXPERIMENT 2

Since a reactivated memory presumably should subside again into an inactive state, introducing a delay between cue exposure and appetitive experience would be expected to lessen the strength of the counterconditioning treatment. Although the effective time intervals involved will likely depend on a variety of task and test parameters, other types of studies have observed time dependent changes following reactivation of old memory (Gordon, 1977; Mactutus et al., 1979). Retroactive interference, on the other hand, is generally considered to be independent of the temporal interval between acquisition and interpolated learning (Newton & Wickens, 1956).

### METHOD

#### Subjects

In this experiment, 32 experimentally naive male albino rats, 80 - 100 days of age and purchased from Holtzman Co., were used. The animals were housed singly in wire-mesh cages.

#### Apparatus and procedure

The apparatus was the same as that employed in Experiment 1. The maltose approach training was also generally the same. In brief, after being food and water deprived to 90% of their ad lib weight, rats received access to

10% maltose solution for 15 min. daily for 5 days. The sugar solution was presented in Richter tubes attached to the home cages. Because of the distance between the fear conditioning room and the colony and the need to provide immediate reward in one of the groups, all subjects received their maltose training while in the room housing the Pavlovian apparatus. Consumption of maltose over the 5 days increased from approximately 11 ml to 22 ml.

Pavlovian fear conditioning was administered 24 hr. following the final maltose training session. In this experiment, five shocks (150-V, 1-sec each) were delivered at irregular intervals during a 5-min. period while the subject was confined to the black compartment.

Subjects were assigned at random to one of four treatment conditions ($N = 8$). Twenty-four hours after conditioning, all groups received a 30-sec exposure without shock ("cueing") to the fear stimuli. This slightly longer cueing exposure was chosen on the assumption that a stronger reactivation might be produced and to determine whether the effect was somehow specific to the particular cueing condition used in Experiment 1. At 0, 5, or 60 min. following the cueing manipulation, three groups were given 5 min. access to maltose. The fourth group served as an extinction control and did not receive the sugar solution. Regardless of their treatment, all subjects remained in the conditioning room (in home cages) for slightly over 1 hr.

Fear of the previously shocked (black) compartment was assessed using a passive avoidance test 24 hr. after the cue exposure. The latency for each subject to cross through into the black compartment was recorded during a 10-min. test.

## RESULTS AND DISCUSSION

Figure 1 presents the median test latencies for the four groups. A Kruskal-Wallis analysis confirmed the presence of a treatment effect, $H = 12.2, p < .01$. As the figure suggests, the pattern of results was quite simple: regardless of the delay interval, each of the cue/malt groups differ significantly from the cue/no malt condition ($U$s = 7 or less; all $p$s $< .01$, two-tailed), but contrary to our expectations, there were no differences between any of the maltose conditions (e.g., immediate vs. 60-min. delay, $U = 25, p > .10$).

These data provide further evidence that memory for a fearful episode can be modified by subsequent information in the form of an appetitive event. Although the fear stimuli were not transformed into approach or positive hedonic cues, the marked reduction in passive avoidance latencies implies that the intensity of fear has been attenuated beyond any effect of extinction per se. But the failure to obtain the anticipated temporal gradient leaves open the important question of whether reactivation is a necessary condition for the

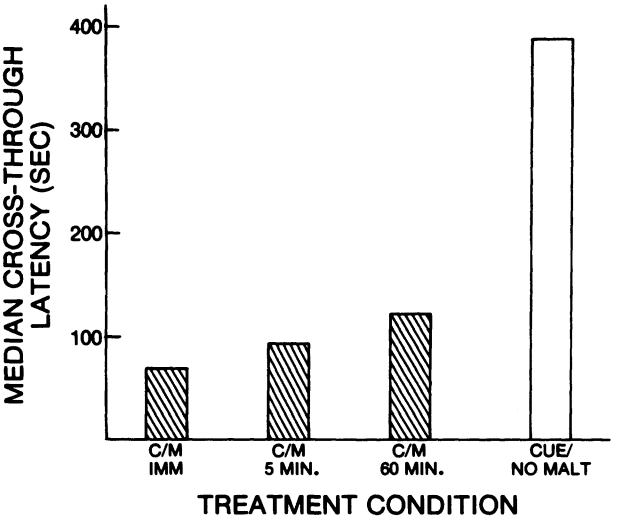


Figure 1. Median cross-through latencies (sec) for animals given a cue exposure followed by maltose at different delay intervals, or only the cue exposure

modification. If not, then the present findings, while representing an interesting type of retroactive interference, may not be pertinent to the original question concerning the properties of retrieved memory.

## EXPERIMENT 3[1]

Before considering further whether cueing is a critical aspect of the paradigm, it seemed advisable to assess the possibility that elevated test latencies in controls were based upon some type of performance artifact rather than learning. More specifically, the notion that memory is modulated by the counterconditioning treatment presupposes that associative processes are involved. However, the employment of Pavlovian procedures in training does not insure that passive avoidance is based upon fear conditioning; perhaps comparable performance would be obtained in rats that have received systemic stress through noncontingent footshocks (NCFS). If this were the case, then the reduced "fear" produced by maltose treatment would not necessarily indicate counterconditioning of a specific memory. Rather, it would suggest that the effect should be viewed in terms of competing hedonic events interacting in some fashion.

Had the delay between reactivation and maltose diminished the counterconditioning effect, the nonassociative interpretation would seem less critical, since a time dependent cueing effect should occur only if memory retrieval is elicited by those cues. But in view of our unexpected success in altering behavior even in the 1-hr. delay condition, it seemed prudent to examine the potential influence of nonassociative factors. Accordingly, in the following experiment, three groups that received NCFS in lieu of training were subsequently exposed to the black "cues" alone or to black cues followed by maltose either immediately or after a 1-hr. delay. These conditions paralleled the design of Experiment 2. A fourth group received Pavlovian conditioning, in which shocks were administered in the black-white apparatus as in Experiment 2, in order to provide an estimate of the level of associative strength, i.e., an acquisition/retention control.

## METHOD

### Subjects

Thirty-nine male Holtzman rats (80–100 days old) were housed in single cages and ear punched for identification.

### Apparatus and procedures

The Pavlovian conditioning apparatus was a 38 cm × 18 cm × 21 cm black-white shuttle box. The floor consisted of 2.5-mm stainless steel grids spaced 1 cm apart. Only the grids on the black side of the apparatus could be electrified. A guillotine doorway, 8 cm × 8 cm, separated the two chambers. A 15-W bulb was suspended 30 cm above the white side. The noncontingent footshock (NCFS) box was a 19 cm × 15.5 cm × 20 cm unpainted pinewood box. The floor consisted of 5-mm stainless steel grids spaced 1 cm apart. Shocks in both the Pavlovian and NCFS situations were delivered from the same shock source.

As other work indicated that mild water deprivation was as effective as food deprivation in inducing vigorous consummatory behavior and as it was more convenient, in this and the following experiment, animals were maintained on a 23.75-hr. water deprivation schedule throughout. On days 1–7, all animals were given 15-min. access to a 10% (w/v) maltose (65% industrial grade M) solution in Richter tubes attached to the home cages. Animals were handled for approximately 3 min. on days 5, 6, and 7. On day 8, shocks were administered. For the Pavlovian conditioned group, training consisted of the rat being placed on the black side of the black-white box for 2 min. During this period, the rat received six footshocks (150 V, 1-sec duration) administered on a variable time schedule. Immediately after this period ended, the rat was placed on the white side of the apparatus for 2 min. No shocks were administered while the rat was on the white side. This dif-

ferential conditioning procedure was then repeated. Thus, the Pavlovian conditioning treatment consisted of four 2-min. sessions (two on the black side and two on the white) in which a total of 12 shocks were administered to the rat (all when the rat was on the black side). The NCFS treatment consisted of administering 12 shocks (150 V, 1-sec duration) during a 6-min. period in the pinewood box. To equate exposure to the black/white chamber with the Pavlovian group, NCFS animals received 8 min. of exposure to the black/white shuttle box 3 to 4 hr. after their series of shocks. The exposures were distributed in 2-min. sessions to each side, as was done for the Pavlovian group. Thus, the NCFS animals received the same aversive treatment as the Pavlovian conditioning group, albeit in a different location, and both groups were matched for duration of exposure to the shuttle box.

On day 9, the animals that had received NCFS and exposure to the shuttle box received a 30-sec exposure to the black side of the Pavlovian conditioning apparatus. Two groups of 10 animals received maltose either immediately or 1 hr. after this exposure, while 9 other animals did not receive any maltose after this exposure. The 10 animals in the Pavlovian conditioned group received no treatments on day 9.

On the 10th day, all animals were tested for their tendency to avoid the black side of the apparatus. Animals were placed on the white side facing away from the door, which was down. The door was removed 10 sec later. Again, passive avoidance was measured by cross-through (all four paws) latency into the black side. To provide a further index of fear, spatial avoidance was also recorded, i.e., the total amount of time spent on the white side (TTW) of the apparatus during the 10-min. test.

## RESULTS

All groups that had received NCFS readily entered into the black side of the apparatus, thus demonstrating little fear. Median latencies were 20, 24, and 30 sec for the immediate, 1-hr. delay, and no maltose conditions, respectively. None of these groups differed statistically, all $U$s $> 37.5$, $p$s $> .10$. However, the Pavlovian conditioning group (median latency = 236 sec) did differ from all of these NCFS groups, all $U$s $< 18$; $p$s $< .05$, two-tailed. An identical pattern was obtained using the total time spent on the safe side (TTW) as the measure. Median scores were 170, 244, and 189 sec for the NCFS groups, compared with 453 sec for the Pavlovian trained group. None of the NCFS groups differed from one another, $U$s $> 34$, $p$s $> .10$, but each differed from the conditioned group, all $U$s $< 12$, $p$s $< .02$.

These findings indicate that the differential treatment effects obtained in Experiments 1 and 2 are not attributable simply to interactions of maltose drinking with stress-related performance artifacts.

Unlike Pavlovian conditioning, NCFS experience, with or without maltose treatment, did not result in avoidance of the black compartment. While NCFS may well produce "pseudoconditioning" (i.e., heightened passive avoidance latencies) in some situations, it did not have that effect here, perhaps because of the opportunity for differential learning afforded by the repeated series of shocks. Also, the potential confounding role of neophobic reactions to a novel test situation was eliminated by providing the NCFS groups with the same amount of exposure to the black/white chamber as the Pavlovian trained subjects. Finally, it should be noted that the retention scores in the conditioned animals were lower than are typically obtained under these conditions by our laboratory. Whatever the reasons for this difference, it should be noted that each experiment in the study was "self-contained"—subjects were from the same shipment and data were collected across all conditions at the same time. Thus, while direct comparison of absolute scores cannot always be made across experiments, interpretation of relationships obtained within an experiment is not affected.

## EXPERIMENT 4

Experiment 2 indicated that test performance was altered comparably in subjects receiving maltose either immediately or 1 hr. after reactivation. In retrospect, our failure to obtain a temporally graded reactivation effect may have been related to the presence of background contextual cues throughout the delay interval. Because of time constraints in running squads of animals, subjects in the delay conditions were not returned to the colony following reactivation exposure, but were held in the same room that housed the fear conditioning apparatus. Given the contribution of contextual cues to memory retrieval in other types of studies (Spear, 1978; Gordon, 1981), it is possible that the room itself served as a source of stimuli that either reactivated memory or helped maintain the reactivated memory throughout the "delay" interval. Another possibility, of course, is that the intervals employed were not long enough. Accordingly, the aim of Experiment 4 was to examine further the effects of varying the time between reactivation of memory and presentation of appetitive reinforcement upon the modulation of fear. An ancillary aim was to determine directly whether memory could be reactivated, as measured by the counterconditioning phenomenon, by exposure to room cues only. If context were to prove ineffective, then it can be

seen that the latter group provides the appropriate condition for determining the effects of maltose without cueing, i.e., a maltose only control.

## METHOD

### Subjects

Fifty-one Holtzman rats (80–100 days old) were housed in single cages and ear punched for identification. As in Experiment 3, food was available ad lib in the home cages.

### Apparatus and procedures

The apparatus was identical to that of Experiment 3. Water deprivation regime, maltose training, and Pavlovian conditioning were the same as described for the preceding experiment. Twenty-four hours after fear conditioning (day 9), all rats were randomly assigned to one of five different groups. Three groups received a 30-sec black cue exposure (no shock) and maltose outside the experimental room. These three groups differed in the delay between cue exposure and maltose delivery with the intervals being 0, 1, or 4 hr. A fourth group (cues inside) received 30-sec cue exposure and immediate presentation (0 delay) of maltose, but, for these subjects, the bottles containing sugar solution were attached to cages inside the experimental room. This group served as a replication of a condition in Experiment 2. The final group (room/outside) received a 30-sec exposure to the cues of the room housing the experiment chamber (but not to the apparatus cues constituting the CS), followed by maltose outside the room immediately after the reexposure. This group tested for the possibility that the room cues alone were sufficient to "reactivate" the target memory.

On day 11, all animals were tested for fear of the black side of the apparatus. Animals were placed on the white side facing away from the door, which was down. The door was removed 10 sec later. Both the time for initial cross-through (all four paws) into the black side (latency) and total amount of time spent on the white side (TTW) of the apparatus during the 10-min. test were recorded as indices of fear.

## RESULTS

Figure 2 presents the median test scores, both latency (panel A) and TTW (panel B), for all five groups. The solid line curves represent the groups that received maltose after cueing. A Kruskal-Wallis analysis revealed a significant treatment effect with either dependent variable, both $H$s $> 15$, $p$s $< .01$. While no differences were found among any of the cue groups receiving maltose immediately or after a 1-hr. delay (for either measure, $p$s $> .10$), each of these three condi-

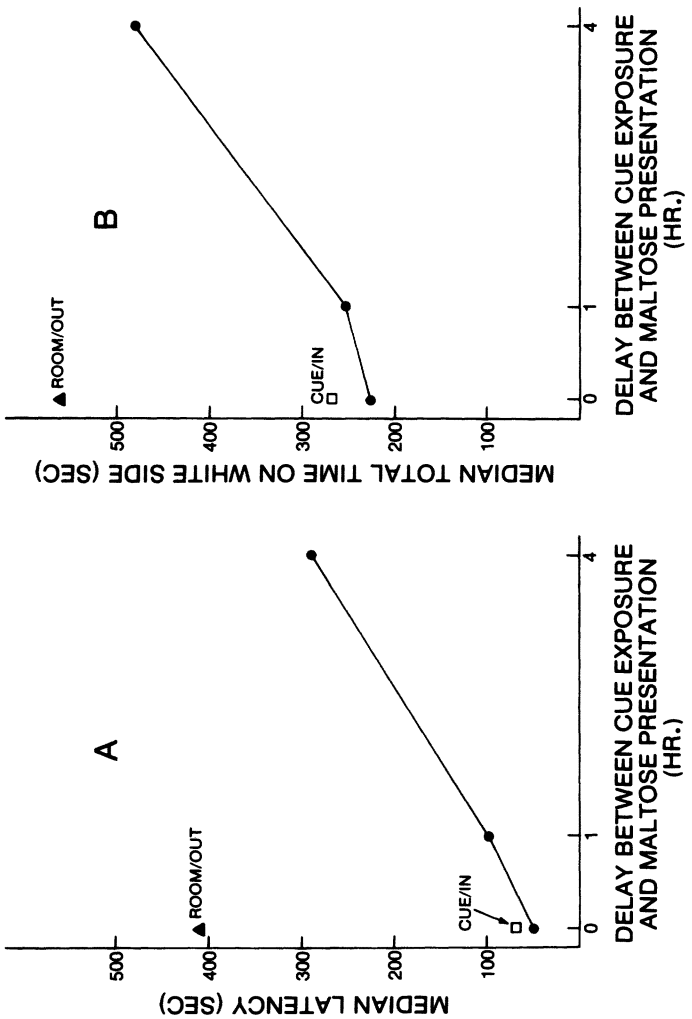


Figure 2. Median cross-through latencies and total time on white measures (both in sec) for Experiment 4

tions differed significantly from the cue/4-hr. delay and the room/out conditions (for latency, all $U$s $< 20$, all $p$s $< .05$; for TTW, all $U$s $< 22$, all $p$s $< .05$). The lack of effect with the 4-hr. delay is also reflected in the fact that this group did not differ from the room/outside condition, for either measure, $p$s $> .10$.

## DISCUSSION

These data replicate Experiments 1 and 2 in demonstrating that presentation of maltose reward following brief exposure to an aversive CS reduces the strength of conditioned fear. Unlike traditional counterconditioning, however, the conditioned fear cues were not present during the period of appetitive reinforcement. Thus, it appears that "counterconditioning" occurs with respect to some memorial representation of the earlier aversive episode. Experiment 4 provides evidence that memory reactivation in conjunction with maltose consumption, rather than simply consumption of maltose during the retention interval, is critical. This conclusion is supported by two lines of evidence: first, the counterconditioning effect was significantly attenuated when the maltose treatment was delayed by 4 hr. after cue exposure; second, rats that received maltose but had not been exposed to the specific fear cues of training (group room/outside) continued to show high levels of fear. Since context alone proved ineffective, this latter group provides a no cue/maltose control condition. Apparently, a representation of the target episode needs to be in an active state in order for a change to be induced.

As in Experiment 2, the 1-hr. delay failed to degrade the phenomenon, which may suggest that even weak levels of memory activation are sufficient in this situation. While the temporal gradient seems long in comparison with retrograde amnesia studies, it is not out of line with the length of the delays that are effective in other paradigms, such as those involving conditioned taste aversion (Garcia, Ervin, & Koelling, 1966; Revusky & Garcia, 1970).

The present data also demonstrate that the location in which subjects received the maltose is not critical to obtaining the modification of memory. When exposure to conditioned fear cues was followed immediately by maltose presentation, either in the same or in a different room, the effects were comparable. Indeed, presentation of the maltose in a different context even 1 hr. after cue exposure was still effective in counterconditioning. But apparently the contextual cues of the room alone are not sufficient to reactivate memory, as this group (room/outside) showed significantly more fear than the comparably

treated group (cue/outside) that received exposure to the conditioned fear cues as well as the context of the room.

## GENERAL DISCUSSION

Taken together, these findings are consistent with the general proposition that memory, during or shortly following retrieval, is susceptible to changes or modification from contemporary environmental conditions. The modification of memory depends upon a memory-cueing exposure, as the presentation of maltose without direct cueing was ineffective (Experiment 4). Moreover, like amnesia, the degree of change reflects a time-dependent process following reactivation. Data from the control study (Experiment 3) indicate that the performance undergoing alteration is based upon associative processes, since very poor passive avoidance was obtained in groups receiving noncontingent footshocks in lieu of Pavlovian conditioning. This conclusion is further strengthened by the fact that a cue exposure (Experiment 4) proved to be a necessary condition for the phenomenon. If systemic stress were the basis for passive avoidance responding, then, except for the associative influence of stimulus generalization, one would not expect that changes in responding would be linked specifically to the manipulation of particular cues. While the aversiveness of NCFS may well be altered by maltose consumption, such an outcome seems not to account for the present findings. Thus, the counterconditioning effect appears to represent the assimilation or integration of new information into the old, reactivated memory (cf. Gordon, 1981).

It will now be of interest to compare old and new memories in terms of their sensitivity to countercontitioning. Will similar effects be obtained for newly acquired learning, or is the phenomenon restricted to old memories? While we have just begun to explore this issue, preliminary data indicate that counterconditioning can be obtained for new and old memories, although their temporal gradients appear to differ.

A question of some importance is whether these findings can be considered as an instance of retroactive interference. Since the conflicting responses were not trained in the same stimulus situation and the presentation of maltose without cueing was ineffective, one might conclude that retroactive interference is not involved. On the other hand, approach vs. avoidance tendencies were involved, if one assumes that the cue exposure activated a memory that was then "paired" with new information. Rescorla (1974) has advocated the

thesis that a CS evokes a representation of the UCS and has presented empirical evidence supporting this view. A particularly intriguing application of this approach is provided by Holland's (1981) demonstration that taste aversion can be established when poisoning follows exteroceptive cues—if those cues have previously been associated with the particular flavor. (It is interesting to note that a very similar experiment independently initiated in our laboratory prior to publication of Holland's article was unsuccessful, probably because of the opposing influence of latent inhibition.) Thus, one set of cues appears to activate a representation of the previous constellation of events. Viewed from this perspective, the counterconditioning phenomenon may represent an illuminating form of interference. Lewis (1979) has pointed out that the very nature of an A-B, A-C paradigm involves the conditions necessary for activating the original memory at time of interpolated training. Our data support speculation by Lewis (1979) that disruption (interference) may also occur under different stimulus situations if the memory of the original episode is made active. If this interpretation is correct, it suggests a process by which retroactive interference may provide a source of retention loss across a wide range of divergent conditions.

The malleability of human memory has been demonstrated in a number of ingenious studies by Loftus and her colleagues (cf. Loftus, 1979). In their work, the presentation of information during the retention interval can have a distorting effect on later recall of the target memory. Evidence of reconstructive memory has also been reported in the rat. Using an overshadowing paradigm, Bolles and Kaufman (Note 1) showed that if the salient component of the compound CS in a CER task undergoes extinction, then the "overshadowed" element comes to elicit suppression. One interpretation is that subjects reconstructed, or reattributed, their source of discomfort to the weaker stimulus. In the current study, presenting additional "information" in the form of a "pleasant" event in conjunction with memory of an unpleasant episode attenuated the strength of fear in a subsequent retention test. It appears that the notion of malleability may be useful in studying memory processes in animals as well as in humans.

Finally, although our major interest has been on modifications of memory, the present paradigm may also prove useful as a model for examining processes presumably underlying psychotherapies such as desensitization (Davison, 1968; cf. Murray & Jacobson, 1978). Interpretations of previous studies using counterconditioning in animals as an analog to desensitization have been complicated by several

potential artifacts (Wilson & Davison, 1971). For example, the fear eliciting CS may disrupt contact with a positive UCS such as food, rendering the counterconditioning "treatment" relatively meaningless. Conversely, it is difficult to specify the functional amount of reexposure to the CS received by subjects, as the new UCS can redirect behavior away from the CS. Or the new UCS may elicit motor responses that later distort the assessment of fear. These problems are largely alleviated when the appetitive UCS is introduced after the cueing episode. And it is tempting to consider that the use of memory reactivation is reminiscent of the role of "imagery" when desensitization therapy is conducted in vitro.

## Notes

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1. Experiment 3 was carried out several months following completion of the other experiments in this study.

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