Reinstatement-induced recovery of a taste-LiCl association following extinction

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Water-deprived rats were given a single exposure to saccharin and LiCl, either paired or unpaired. Half the subjects then received three saccharin-only exposures (extinction) in the training enclosure, followed by a single LiCl-only presentation (unconditioned stimulus reinstatement) 8 days after conditioning. The remaining subjects received six saccharin-only exposures, followed by LiCl reinstatement 13 days after conditioning. In both cases LiCl reinstatement occurred outside the training/test context. Appreciable recovery from extinction was observed after the partial loss of taste aversion obtained with three extinction sessions and the 8-day conditioning-reinstatement interval, but not after the asymptotic loss of taste aversion obtained with six extinction sessions and the 13-day conditioning-reinstatement interval. Conditioned taste aversions appear to be similar to more traditional associations with respect to both extinction and reinstatement-induced recovery from extinction. The results are discussed with reference to the event-memory, contextual-conditioning, and facilitated-retrieval hypotheses of postextinction reinstatement effects.

Rescorla's event-memory model views conditioning as the formation of representations of the conditioned stimulus (CS), the unconditioned stimulus (US), and the CS-US association (e.g., Rescorla, 1973). The model posits that postconditioning, nonreinforced CS presentations, that is, conventional extinction, will both degrade the US representation and weaken the CS-US association, thereby diminishing the conditioned response. Furthermore, subsequent US-alone exposures are hypothesized to restore the effective US representation with a consequent recovery of performance. Alternative explanations of the reinstatement-induced recovery from extinction include the contextual-conditioning hypothesis of Bouton and Bolles (1979), which posits that, rather than inflating the US representation, US reinstatement is effective because context-US associations formed at the time of reinstatement summate with residual CS-US associations at the time of testing. Additionally, Miller, Kaspro, and Schachtman (in press) view extinction as a retrieval deficit produced by retroactive interference arising from conflicting associations, that is, CS-US and CS-noUS associations. They further suggest that US reinstatement acts as an extremely salient retrieval cue that promotes retrieval and further information processing. This should facilitate subsequent retrieval of the CS-US association, thereby augmenting its effect on test performance.

Reinstatement-induced recovery has repeatedly been observed in conditioned suppression preparations (e.g.,

Bouton & Bolles, 1979; Rescorla & Cunningham, 1978; Rescorla & Heth, 1975). In a recent effort to test the generality of the phenomenon to taste-aversion learning, Bouton (1982) administered LiCl reinstatement treatment to rats following extinction of a saccharin (SAC)-LiCl association. Despite the manipulation of several procedural variables aimed at enhancing sensitivity to detection of the effect, no reinstatement-induced recovery was observed, a result, Bouton suggested, of taste aversions being qualitatively different from more traditional associations.

The event-memory model places great emphasis on the amount of CS-alone exposure administered prior to US reinstatement and on the interval between acquisition and reinstatement. That is, extensive extinction and/or extensive forgetting would likely degrade the CS-US association as well as the US representation, thereby reducing the probability of recovery of the original association due to inflation of the US representation. Bouton (1982) failed to observe a reinstatement effect even when posttraining SAC-alone exposure (extinction) was reduced from seven 20-min exposures and a 16-day conditioning-reinstatement interval (Experiment 1) to five 20-min exposures and an 11-day conditioning-reinstatement interval (Experiment 2). In the framework of the event-memory model, it is possible that both of Bouton's extinction treatments were sufficient to degrade the SAC-LiCl association, thus preventing recovery through reinstatement of the LiCl. Although the final experiment in Bouton's study failed to find a reinstatement effect following incomplete (albeit substantial) manifest extinction resulting from five 20-min SAC-alone exposures, it is possible that the extent of extinction and/or the 11-day conditioning-reinstatement interval degraded the CS-US association sufficiently to deny recovery.

Support for this research was provided by NIMH Grant 33881. We thank Wesley J. Kaspro for discussions that initiated this research, Elizabeth Gordon and Louis Matzel for their critical reading of an earlier version of this manuscript, and Roxanne Vavra for her secretarial assistance. Address reprint requests to Ralph R. Miller, Department of Psychology, SUNY-Binghamton, Binghamton, NY 13901.

The present study sought to obtain recovery of a SAC-LiCl aversion by varying the amount of posttraining CS extinction and the conditioning-reinstatement interval. Subjects initially either received a SAC-LiCl pairing or were presented with SAC and LiCl explicitly unpaired. One group from each of these treatments was subsequently given three 10-min extinction exposures to SAC followed by one US reinstatement exposure 8 days after conditioning; the additional paired and unpaired groups received six 10-min SAC presentations followed by a US reinstatement exposure 13 days after conditioning. Although initial treatment, extinction, and testing all occurred in the home cages, US reinstatement was given in a highly dissimilar environment in order to minimize the possible influence of reinstated context-US associations on performance at the time of testing. If Bouton's (1982) failure to obtain a reinstatement effect was the result of excessive extinction or too long a conditioning-reinstatement interval, three 10-min extinction exposures over an 8-day interval would be more likely to yield a reinstatement effect than six such exposures over a 13-day interval. The effect of LiCl reinstatement was assessed by within-subject comparisons between SAC consumption on the final extinction exposure and on a subsequent test following reinstatement treatment. The control conditions that received the CS and US explicitly unpaired were used for comparison purposes to determine the degree of extinction in the paired CS-US groups and to detect any nonassociative effects of the LiCl reinstatement treatment. No control group for spontaneous forgetting (i.e., a group given conditioning but no extinction) was included in this study (or Bouton's, 1982), because aversions in general are known to be resistant to forgetting and conditioned taste aversions in particular have been found to be enduring over time (e.g., Gustavson, Garcia, Hankins, & Rusiniak, 1974).

METHOD

Subjects

Twenty-four male and 24 female Sprague-Dawley-derived rats, bred and raised at the SUNY-Binghamton colony, served as subjects. The body weights of the females ranged from 220 to 300 g and the males from 310 to 430 g. Subjects were individually housed in standard wire-mesh cages with ad lib access to Purina Lab Chow in the home cage at all times. Prior to the start of the study, they were exposed to a schedule of increasing water deprivation which culminated in 10 min of access to water each day in the home cage. This schedule of 10 min/day of water was in effect for 5 days prior to Day 1 of the experiment, thereby ensuring that deprivation was asymptotic before the study began. This watering schedule was maintained throughout the study. Experimental treatments were conducted 18-22 h after access to water on the previous day, thereby assuring similar levels of thirst across groups. The vivarium was maintained on a 16 h light/8 h dark cycle and treatments were administered during the light portion of the cycle. All subjects were handled for 30 sec/day for a week prior to the start of the study.

Apparatus

All treatments except the reinstatement exposure occurred in the home cage. The home cage was a standard hanging stainless-steel

cage with sheet-metal rear and side walls and wire-mesh front wall and floor. The cages measured 24 × 17.8 × 18.2 cm (l × w × h). The LiCl reinstatement treatment was administered in 12 truncated V-shaped enclosures that measured 18.5 cm in length, 17 cm in height, and 20.5 cm wide at the top tapering to 5.5 cm wide at the floor. These reinstatement chambers had sloping stainless-steel-plate side walls. The roofs were constructed of clear Plexiglas and the end walls of white Plexiglas. The floors were two 2-cm-wide parallel stainless steel plates separated lengthwise by a 1.5-cm gap. The room housing the reinstatement enclosures was illuminated by fluorescent bulbs and had an ambient noise level of 56 dB(C). Unpublished conditioned-lick suppression data from our laboratory have shown that there is very little generalization between this context and the home cage. A 10-cc syringe with a 25-ga, 1.59-cm hypodermic needle was used to inject 1.3% body weight of 0.3 M LiCl [in contrast to Bouton's (1982) 0.5% body weight of 0.6 M LiCl]. A 0.1% (w/v) saccharin solution was provided via a graduated plastic tube with a metal lick tube attached.

Procedure

Prior to Day 1, subjects were randomly assigned to one of four groups ($n=12$), with the constraint that groups were counterbalanced for sex, litter, and body weight. On Day 1, subjects in Groups UE (unpaired, extensive extinction) and Ue (unpaired, limited extinction) were exposed to SAC for 10 min and the amounts consumed were recorded. Groups PE (paired, extensive extinction) and Pe (paired, limited extinction) received the same 10 min of exposure to SAC, but on Day 2 rather than Day 1. Approximately 10 sec after the removal of the SAC solution on Day 2, all animals in Groups PE and Pe were removed, given an ip injection of LiCl, and then returned to the home cage. Groups UE and Ue were not exposed to SAC on Day 2, but were given a LiCl injection identical to that given to Groups PE and Pe. Ten minutes after injection, all subjects were observed and were found to exhibit behavior indicative of LiCl-induced illness, that is, concave back, nonlocomotion, etc. Subjects that consumed less than 3.5 ml SAC on Day 1 (Groups UE and Ue) or Day 2 (Groups PE and Pe) were eliminated from the study due to insufficient CS exposure. In practice, this criterion eliminated one animal from each group. In order to allow subjects to fully recover from illness, no treatments were administered on Days 3 and 4.

Extinction was conducted on Days 5-7. On each of these days, all subjects were exposed to SAC for 10 min and the amount of SAC consumed daily was recorded. On Day 8, US reinstatement treatment was administered to Groups Pe and Ue. These subjects were placed into the reinstatement chamber for 10 min to allow acclimation to the context, were given a LiCl injection identical to that received on Day 2, and then were returned to the reinstatement chamber for an additional 60 min. Overt symptoms of illness (i.e., inactivity, defecation, and concave back) ceased within 30 min of injection, suggesting that much of the induced malaise was over before the animal was returned to its home cage. Groups PE and UE remained undisturbed in the home cage on Day 8 and all subjects remained in the home cage without treatment on Day 9, to allow subjects given LiCl on Day 8 to recover from illness. On Day 10, Groups Ue and Pe were tested for SAC consumption. The response measure was the amount of SAC consumed from the lick tube in 10 min, that is, the test was equivalent to another extinction trial. On Days 10-12, Groups PE and UE were given additional extinction trials identical to those given on Days 5-7. Thus, subjects in Groups PE and UE received six extinction trials, rather than three (the number given to Groups Pe and Ue). On Day 13, Groups PE and UE were given LiCl reinstatement treatment exactly like that received by Groups Pe and Ue on Day 8. No treatment was given on Day 14, to allow for recovery from illness. On Day 15, subjects in Groups PE and UE were tested for SAC consumption in the same manner as Groups Ue and Pe on Day 10, that is, the equivalent of another extinction trial. Notably, testing on Days 10 and 15 followed a 48-h recovery interval (in contrast to

Bouton's, 1982, 72-h recovery interval). However, as will be seen, with similar degree of extinction (Group PE), our data do not differ from Bouton's.

RESULTS AND DISCUSSION

The criterion for rejection of the null hypothesis was set at $p < 0.05$ for all statistical analyses. Mean volumes of SAC consumption during Day 1 (Groups Ue and UE), Day 2 (Groups Pe and PE), extinction, and testing are illustrated in Figure 1. The common treatments of Groups Pe and PE and of Groups Ue and UE along with the high similarity of their means on Days 1-7 prompted us to pool their data in Figure 1. Typical of these similarities were the means for consumption on Day 7 (for Group PE, 9.4 ml; for Group Pe, 10.1 ml; for Group UE, 16.8 ml; for Group Ue, 16.8 ml). No between-group differences were evident for initial SAC consumption on Day 1 or 2 between Groups Ue and Pe [$t(20) = 0.40$] or Groups UE and PE [$t(20) = 1.58$] (both $ps > 1.0$). Statistical assessment of the effects of the SAC-LiCl pairing, as indexed by consumption on Day 5, yielded significantly less SAC consumed by Group PE than by Group UE [$t(20) = 7.45$] and by Group Pe than by Group Ue [$t(20) = 13.27$] (both $ps < .001$). The increase in consumption by Groups Ue and UE (pooled) between Days 1 and 5 [$F(1,21) = 70.34$, $p < .001$] presumably reflects a loss of neophobia over successive exposures to the saccharin.

A Treatment (PE versus UE) $\times$ Days (5-7) analysis of variance (ANOVA) indicated an effect of paired versus

unpaired treatment [$F(1,20) = 98.82$], an increase of consumption across days [$F(2,40) = 30.38$], and an interaction indicating that extinction across days was dependent on treatment [$F(2,40) = 13.11$] (all $ps < .001$). A similar analysis comparing Groups Pe and Ue on Days 5-7 also found an effect of treatment [$F(1,20) = 118.41$], an increase in consumption across days [$F(2,40) = 24.71$], and an interaction of extinction with treatment [$F(2,40) = 12.54$] (all $ps < .001$). Within-subject analyses of SAC consumption on Day 5 versus Day 7 indicated significant extinction for Group Pe [$F(1,10) = 44.22$] and Group PE [$F(1,10) = 84.22$] (both $ps < .001$). A similar analysis of Day 7 versus Day 10 consumption found a significant decrease in consumption following the Day 8 reinstatement exposure by Group Pe [$F(1,10) = 11.60$, $p < .01$], but not by Group Ue ($F < 1$). This enhancement of Group Pe's aversion to SAC was probably not the result of spontaneous recovery, as such an effect would also have been manifested in decrease consumption by Group PE on Day 10 relative to Day 7. In fact, subjects in Group PE consumed more SAC on Day 10 than on Day 7 [$F(1,10) = 12.75$, $p < .01$], and differed from Group Pe on Day 10 [$F(1,20) = 48.16$, $p < .001$] but not on Day 7 ($F < 1$). Thus, any spontaneous recovery that might have occurred in Group Pe between Days 7 and 10 was more than compensated for by the effects of extinction on Day 7 that would not be evident until Day 10, the next day saccharin was presented. Moreover, the absence of a decrease in consumption by Group Ue between Days 7 and 10 suggests that the decrease seen

Figure 1. Mean daily intake of saccharin during a 10-min exposure as a function of treatment. On Days 1-7 Groups PE and Pe and Groups UE and Ue have been pooled because they received identical treatment through Day 7. The leftmost two data points represent initial intake prior to initial poisoning, with this measure being taken on Day 1 for Groups UE and Ue and on Day 2 for Groups PE and Pe. All animals were poisoned on Day 2. Groups Pe and Ue were poisoned a second time on Day 8 and Groups PE and UE were poisoned a second time on Day 13.

in Group Pe was not an unconditioned consequence of the LiCl treatment on Day 8. A within-subjects comparison of Group PE's drinking on Day 7 and Day 12 found that additional SAC-alone exposures given on Days 10–12 produced continued behavioral extinction [$F(1,10) = 16.14$, $p < .01$]. A within-subjects analysis of consumption by Group PE on Days 12 and 15 yielded no reinstatement-induced recovery of the aversion ($F < 1$). Moreover, although on Day 15 Group PE differed from Group UE [$F(1,20) = 7.90$, $p < .02$], this difference carried over largely from the nonsignificant difference between these two groups on Day 12. When within-subjects difference scores between Day 12 and Day 15 were compared, Groups UE and PE did not differ [$F(1,20) = 3.06$, $p > .05$].

These results confirm findings of prior studies in which reinstatement-induced recovery was unobtainable following extensive extinction and a long conditioning-reinstatement interval (Bouton, 1982). However, with fewer posttraining CS-alone exposures and a shorter conditioning-reinstatement interval, significant recovery from extinction was observed following US reinstatement. Although the present research does not differentiate between degree of extinction and interval between conditioning and reinstatement as the critical factor that permitted a reinstatement effect in Group Pe but not in Group PE, the critical observation is that, as with more traditional learning tasks, postextinction reinstatement effects following taste-aversion learning can be obtained, given appropriate treatment parameters. Admittedly, with tasks such as lick or barpress suppression, reinstatement effects have been obtained even after extensive extinction (e.g., Bouton & Bolles, 1979; Rescorla & Cunningham, 1978; Rescorla & Heth, 1975); but now, at worst, we are faced only with quantitative, as opposed to qualitative, distinctions between conditioned and other kinds of learning.

The observation of recovery when US reinstatement occurred in a context distinctly different from that used for testing is not compatible with the hypothesis that US-induced recovery from extinction is due uniquely to context-US associations formed during US reinstatement directly influencing test performance. According to this position, US reinstatement results in a strengthening of context-US associations, which summate with the residual CS-US association at test to elevate conditioned responding. We do not deny that this process may contribute to recovery effects when reinstatement occurs in the test context (Bouton & Bolles, 1979), but it is not a compelling explanation of the present findings. Of course it is possible that conditioning to the home cage occurred when the animals were returned 60 min after reinstatement treatment, but presumably the 48-h recovery interval that preceded testing would have permitted appreciable extinction of any such home-cage-LiCl associations.

The current results, viewed in isolation, are compatible with the event-memory model. Extensive extinction may be regarded as weakening the CS-US association as

well as degrading the US representation. In this view, extensive extinction trials would militate against reinstatement-induced recovery regardless of a reestablished US representation. Alternatively, the reinstatement effect evident in Group Pe but not in Group PE might have been a function of the degrading of the CS-US association by the longer conditioning-reinstatement interval employed with the latter group.

The present findings are also consistent with the retrieval hypothesis of Miller et al. (in press). The retrieval hypothesis posits that US reinstatement promotes current retrieval of the CS-US association that allows for further information processing, thereby augmenting its effect on subsequent behavior. In essence, a postextinction US presentation acts as a "reminder" of conditions that prevailed during acquisition, that is, a highly potent retrieval cue. Once retrieved, the target memory is subject to further rehearsal and, consequently, easier retrieval on a subsequent test trial. The failure of US reinstatement-induced recovery to follow extensive extinction, but not moderate extinction, presumably is the result of superior learning of the extinction contingency in the former case. Reinstatement of the US presumably facilitates retrieval of initial training, provided that the competing memory of extinction is not so strong that "reminder treatments" will be ineffective. Reinstatement treatment may be expected to be somewhat more effective in the conditioning context than outside the conditioning context because in the former case the context could augment the US in inducing retrieval of the target memory. However, in the present case, reinstatement in Group Pe was obviously obtained without benefit of the conditioning context, either as a retrieval cue for the CS-US association at the time of reinstatement or as a source of direct association to the US at the time of testing (as has been demonstrated in other situations by Bouton & Bolles, 1979).

Evidence favoring the retrieval hypothesis over the event-memory model comes from a number of sources. First, the retrieval hypothesis has been successful in explaining the occurrence of and recovery from a large number of other performance deficits besides extinction, that is, blocking (Balaz, Gutsin, Cacheiro, & Miller, 1982), experimentally induced amnesia (Deweert, Sara, & Hars, 1980), and latent inhibition (Kaspro, Catterson, Schachtman, & Miller, 1984). It is difficult to postulate how these demonstrations of recovery might be explained from an event-memory viewpoint. Second, these recovery effects can be elicited by posttraining exposure to constituents of the training episode other than the US, that is, the CS or apparatus cues (Balaz et al., 1982; Deweert et al., 1980; Gordon & Mowrer, 1980). Finally, there is direct evidence against the view that extinction appreciably degrades the representation of the US. Kaspro, Schachtman, Cacheiro, and Miller (1984) and Richards and Sargent (1983) found that when two cues were separately trained with the same US and then one cue was extinguished, a reliable decrease in conditioned responding to

the other cue was not observed, despite the event-memory model stating that identical US presentations ought to be encoded as a single US representation (Rescorla & Heth, 1975). In toto, reinstatement treatments that reverse performance deficits appear to be most readily explained in terms of retrieval processes rather than in terms of restoration of degraded US memories.

Bouton (1982) suggested that his failure to obtain a US reinstatement effect following extinction of a conditioned taste aversion may have reflected the fact that dissimilar processes mediate conditioned taste aversion and more traditional learning tasks. Although this was a reasonable suggestion based on his results, the present observation of a US reinstatement effect following conditioned taste aversion renders this position untenable. Rather, it appears that the US reinstatement effect joins a large number of other phenomena in demonstrating that conditioned taste aversions and traditional learning tasks frequently obey at least the same qualitative rules (Domjan, 1983), although there are some phenomena that may yet be found to reflect fundamentally different learning processes between conditioned taste aversion and more traditional learning tasks (e.g., Holman, 1976; Jacobs, Zellner, LoLordo, & Riley, 1981; Rusiniak, Palmerino, Rice, Forthman, & Garcia, 1982).

REFERENCES

- BALAZ, M. A., GUTSIN, P., CACHEIRO, H., & MILLER, R. R. (1982). Blocking as a retrieval failure: Reactivation of associations to a blocked stimulus. *Quarterly Journal of Experimental Psychology*, **34B**, 99-113.
- BOUTON, M. E. (1982). Lack of reinstatement of an extinguished taste aversion. *Animal Learning & Behavior*, **10**, 233-241.
- BOUTON, M. E., & BOLLES, R. C. (1979). Role of conditioned contextual stimuli in reinstatement of extinguished fear. *Journal of Experimental Psychology: Animal Behavior Processes*, **5**, 363-378.
- DEWEER, B., SARA, S. J., & HARS, B. (1980). Contextual cues and memory retrieval in rats: Alleviation of forgetting by pretest exposure to background stimuli. *Animal Learning & Behavior*, **8**, 265-272.
- DOMJAN, M. (1983). Biological constraints on instrumental and classical conditioning: Implications for general process theory. In G. H. Bower (Ed.), *The psychology of learning and motivation* (Vol. 17). New York: Academic Press.
- GORDON, W. C., & MOWRER, R. R. (1980). An extinction trial as a reminder treatment following electroconvulsive shock. *Animal Learning & Behavior*, **8**, 363-367.
- GUSTAVSON, C. R., GARCIA, J., HANKINS, W. G., & RUSINIAK, K. W. (1974). Coyote predation control by aversive conditioning. *Science*, **184**, 581-583.
- HOLMAN, E. W. (1976). The effect of drug habituation before and after taste aversion learning in rats. *Animal Learning & Behavior*, **4**, 329-332.
- JACOBS, W. J., ZELLNER, D. A., LOLORDO, V. M., & RILEY, A. L. (1981). The effect of post-conditioning exposure to morphine on the retention of a morphine-induced conditioned taste aversion. *Pharmacology, Biochemistry, & Behavior*, **14**, 779-785.
- KASPROW, W. J., CATTERSON, D., SCHACHTMAN, T. R., & MILLER, R. R. (1984). Attenuation of latent inhibition by postacquisition reminder. *Quarterly Journal of Experimental Psychology*, **36B**, 53-63.
- KASPROW, W. J., SCHACHTMAN, T. R., CACHEIRO, H., & MILLER, R. R. (1984). Extinction does not depend upon degradation of event memories. *Bulletin of the Psychonomic Society*, **22**, 95-98.
- MILLER, R. R., KASPROW, W. J., & SCHACHTMAN, T. R. (in press). Retrieval variability: Sources and consequences. *American Journal of Psychology*.
- RESCORLA, R. A. (1973). Effect of US habituation following conditioning. *Journal of Comparative & Physiological Psychology*, **82**, 137-143.
- RESCORLA, R. A., & CUNNINGHAM, C. L. (1978). Recovery of the US representation over time during extinction. *Learning & Motivation*, **9**, 373-391.
- RESCORLA, R. A., & HETH, C. D. (1975). Reinstatement of fear to an extinguished conditioned stimulus. *Journal of Experimental Psychology: Animal Behavior Processes*, **1**, 88-96.
- RICHARDS, R. W., & SARGENT, D. M. (1983). The order of presentation of conditioned stimuli during extinction. *Animal Learning & Behavior*, **11**, 229-236.
- RUSINIAK, K. W., PALMERINO, C. C., RICE, A. G., FORTHMAN, D. L., & GARCIA, J. (1982). Flavor-illness aversions: Potentiation of odor by taste with toxin but not shock in rats. *Journal of Comparative & Physiological Psychology*, **96**, 527-539.

(Manuscript received May 20, 1985;
revision accepted for publication September 9, 1985.)