FEATURED ARTICLE

A Comparison of the Effects of State and Non-State Reminder Treatments on Morphine State-Dependency and Cycloheximide-Induced Retrograde Amnesia in Rats

Paula M. Millin* and Emily N. Newman
Department of Psychology, Kenyon College

The modified state dependent retention (MSDR) theory of retrograde amnesia (RA) proposes that the forgetting associated with RA is due to retrieval failure brought about by state dependent memory loss, which is forgetting that occurs when memory recall is attempted in a pharmacological state that differs from the encoding state. In a novel test of the MSDR theory, the current study attempted to determine if RA and state-dependency (SD) share a common mechanism, retrieval failure, by comparing the effects of several reminder cue treatments on RA or SD induced forgetting of a passive avoidance task in rats. The pattern of results was quite similar to morphine SD (Experiment 1) and cycloheximide-induced RA (Experiment 2). Both sources of forgetting were alleviated by reintroduction of the drug state but not by exposure to non-state retrieval cues. Implications for the MSDR theory of RA are discussed.

Key Terms: State-Dependency, Retrograde Amnesia, Memory, Consolidation, Reminder Cue, Morphine, Cycloheximide, Protein Synthesis

The memory consolidation hypothesis contends that newly acquired memories persist in an initially labile state until a complex cascade of neural events leading to memory storage is complete (McGaugh, 2000). This process of slow consolidation is thought to be an advantageous mechanism whereby adrenal stress hormones (such as epinephrine and cortisol) that are released during and after stressful or novel experiences can facilitate memory storage, making emotionally significant events better remembered. The down side of this adaptive characteristic is that newly formed memories are also vulnerable to disruption for a short period following learning (McGaugh, 1990).

Some of the strongest evidence for consolidation theory has come from the study of retrograde amnesia (RA); that is, memory loss for information acquired prior to a physiological insult.

*Corresponding Author:
P.M. Millin, Ph.D.
Department of Psychology
Kenyon College
Gambier, OH 43022
Telephone: (330) 317-0411
Email: millinp@kenyon.edu

In humans, brain trauma from head injury is the most common cause of RA. In the laboratory, RA can be experimentally induced in animals by post-training administration of a number of treatments, including electroconvulsive shock (ECS) (Duncan, 1949), hypothermia (Mactutus, Riccio, & Ferek, 1979), brain lesions (Kaut & Bunsey, 2001; Land, Bunsey, & Riccio, 2000), and protein synthesis inhibition (Nader, Schafe, & LeDoux, 2000). A ubiquitous characteristic of RA is that it displays a temporal gradient, such that recently acquired memories are lost while older memories are spared. In an early study, rats were delivered ECS at various intervals following learning of a passive-avoidance task (Duncan, 1949). ECS delivered within 15 minutes of the task resulted in RA for the original learning, while the same insult delivered at longer intervals after training left the memory intact. Thus, there appeared to be a finite window during which newly acquired memories were vulnerable to the amnesic effects of ECS. According to a traditional consolidation approach, any disruption during this window was thought to preclude memory formation by interrupting the critical neuronal events that lead to memory storage. The temporal gradient of RA,
therefore, provided compelling support for the consolidation hypothesis.

For many decades, the consolidation hypothesis, including its supposition that RA reflects a storage failure, enjoyed widespread popularity despite the fact that questions had been raised about its ability to account for certain features of RA, such as delayed onset (Miller & Springer, 1971) and alleviation via spontaneous or cued recovery (for review, see Millin, Moody, & Riccio, 2001). For example, pre-test exposure to the unconditioned stimulus (US) (Miller & Springer, 1972), the conditioned stimulus (CS) (Brown, Sissman, Kasprów, & Miller, 1985), or a combination of contextual and CS cues (Devitt & Hopfer, 1974) has been shown to alleviate RA. In an especially compelling example, RA was produced for a signaled avoidance Y-maze task via bilateral ablation of the hippocampus in rats (Land et al., 2000). It was found, however, that memory for training recovered if the rats received a non-contingent footshock (US) reminder treatment prior to test. The design ruled out non-memorial explanations for the recovery, such as new learning during the reminder treatment. Moreover, a standard test session has also been used as a reminder treatment. In such cases, animals are tested, and then retested (typically) 24 hours later. Importantly, the test procedure appears to activate the memory of previous learning without promoting any new learning. For instance, Quatermain, McEwen, & Azmitia (1972) reported that amnesia induced by ECS or the protein synthesis inhibitor, cycloheximide, was successfully alleviated for a passive avoidance (PA) task by allowing rats to undergo a reminder test session one day before a second test.

Especially puzzling is the counterintuitive, yet common finding that re-exposure to the amnesic agent itself prior to test aids in memory retrieval. For example, hypothermia-induced RA has been reversed by recooling rats prior to the memory test (Mactutus, Ferek, George, & Riccio, 1982). Even memory loss for a passive-avoidance task produced by the protein synthesis inhibitor, anisomycin, has been reversed by a second administration of anisomycin given prior to test (Bradley & Galal, 1988). Collectively, these findings demonstrating reversal of RA seem inconsistent with the notion that RA reflects a disruption of the consolidation process; however, there are reported failures to reverse amnesia (Lee, Everitt, & Thomas, 2004; Schulz, Fendt, & Schnitzler, 2002; Xu, & Davis, 1992), making this a hotly debated issue (for a review, see Nader & Wang, 2006).

An alternative theory, which emerged from the finding that RA is reversible by re-instating the amnesic insult, proposes that RA reflects a memory retrieval failure brought about by processes akin to state-dependent memory loss (Hinderliter, Webster, & Riccio, 1975; Mactutus et al., 1982; Riccio, Millin, & Bogart, 2006). According to this theory, because information continues to be processed following the learning event (as proposed by the consolidation hypothesis), memories may become embedded in the specific internal state produced by an amnesic agent. As the amnesic state dissipates, memory retrieval becomes less likely due to a mismatch between the internal states of the organism during encoding and retrieval. This theory is based on the well-documented phenomenon of state dependency (SD), in which memory for a task learned under a drug state is disrupted by a change in pharmacological state at the time of testing. For example, one study reported that when pentobarbital was administered to rats 20 minutes before exposure to a novel flavor, memory for the flavor was impaired two days later when rats were no longer under the influence of the drug as evidenced by a failure to demonstrate attenuation of neophilia (Morilak, Orndoff, Riccio, & Richardson, 1983). However, rats that were re-administered pentobarbital prior to test demonstrated the typical attenuation of neophilia, indicating intact memory for the initial flavor exposure.

This hypothesis is a modified state-dependency interpretation because the amnesic insult (and thus the altered state) occurs after the learning episode, rather than before as with traditional SD (Millin et al., 2001). The modified state dependent retention (MSDR) theory can neatly explain the characteristics of RA that were previously unaccounted for by the consolidation failure hypothesis. First, MSDR theory explains the temporal gradient of RA. As the delay between the learning and amnesic insult increases, a greater proportion of the consolidation process is completed in the unaltered state, and is thus retrievable in the normal state (i.e. the sparing of “old” memories). Moreover, the delayed onset of RA is actually predicted by the MSDR theory, since the memory should be retrievable as long as the amnesic state is present. In other words, amnesia is delayed until the amnesic state wears off, creating a mismatch in encoding and retrieval states. Finally, the MSDR theory explains the paradoxical finding that re-exposure to the amnesic agent prior to testing results in memory retrieval. This occurs because the internal state associated with the original learning is reestablished, providing critical retrieval cues (Ricchio, Moody, & Millin, 2002). Thus, a number of the issues that were problematic for the traditional consolidation model are explained by the MSDR...
theory of RA, which asserts that amnesia occurs because of retrieval, not storage, failure. Although the MSDR theory was first proposed more than three decades ago (Hinderliter et al., 1975) empirical support for the theory has lagged. As a starting point, it would be useful to investigate to what extent forgetting due to SD is similar to forgetting produced by RA. Similarities could be measured along a number of dimensions, including the approach taken in the present study of comparing the effects of retrieval enhancing manipulations on the two sources of forgetting. Since SD is proposed to be the underlying mechanism of RA, the two sources of forgetting should respond similarly to treatments designed to improve recall. Here we compared the effects of both state and non-state reminder treatments on memory loss due to either morphine-induced SD (Experiment 1) or cycloheximide-induced RA (Experiment 2). Rats were administered drugs either prior to (SD) or immediately following (RA) training on a standard PA task. Testing, which occurred either 24 or 48 hours later (depending on group), was preceded by a state cue (morphine or cycloheximide), a non-state cue, or no reminder cue. The non-state reminder cues involved elements of training, and included either exposure to the training context, the CS, or a test session. If the effect of the retrieval cues is similar for SD and RA, it would support the speculation that these sources of forgetting are similar and perhaps share a common mechanism, namely retrieval failure (if cues produce alleviation).

Experiment 1

Method

Subjects. Fifty-three (> 90 days old) female Sprague-Dawley-derived rats (from the Kenyon College breeding colony) served as subjects. All rats were individually housed in opaque acrylic tubs with corn cob bedding and were maintained on a 12/12h light/dark cycle. All experimental sessions took place during the lighted portion of the photocycle, and at the same time each day. Food and water were not restricted at any time except during experimental sessions. Prior to data collection, approval for this project was granted by Kenyon College's Institutional Animal Care and Use Committee.

Apparatus. A 49x22x21-cm Plexiglas PA chamber (Ugo Basile model 7552) was used for conditioning. The chamber was divided into two equal sized compartments – one black and one white that shared a clear Plexiglas lid. A sliding door (7x7 cm) separated these two compartments. The floor consisted of metal grids spaced 1.2 cm apart, and the grids in the black side of the chamber were connected to a shock source. A 10-W light bulb was suspended 23 cm above the center of the white compartment.

The PA chamber was housed in a dimly lit and heavily scented room. Specifically, the room was illuminated with a single 25 W red bulb, and was scented with an Air Wick “mandarin & green tea” plug-in air freshener set at a constant level of 2. A small fan remained on at all times to provide white noise.

Procedure and Drugs. Rats were handled for a total of 9 minutes (3 minutes on 3 separate days) during the week preceding training. Forty-five minutes prior to training and testing sessions, rats were weighed and then injected sub-cutaneously in the dorsal surface of the neck with either morphine sulfate (dissolved in physiological saline at a concentration of 5mg/ml and injected at a dose of 5 mg/kg; obtained from Sigma Pharmaceuticals) or an equivalent volume of 0.9% physiological saline. The 45-minute delay, which was spent in the colony room, was included to allow time for drug onset prior to the experimental sessions.

Group S-S (n = 10), which received saline prior to both training and testing provided a baseline measure of non-drugged performance on the PA task. Group M-S (n = 10), which was injected with morphine before training and saline prior to test, provided a measure of the effect of a switch in drug state between training and testing on PA performance (i.e. state dependency). Group M-M (n = 10), which received morphine prior to both training and testing, provided a measure of performance in animals that were similarly drugged during training and test. In other words, this group investigated the effect of re-inducing the drug state on memory retrieval. Similar to Group M-S, several additional groups (M-S-Context, n = 5; M-S-CS, n = 5; M-S-Warmup, n = 5; and M-S-Test, n = 8) received morphine prior to training and saline prior to testing; however, each of these groups was exposed to one of four cueing treatments (to be described shortly) prior to test. The purpose of these groups was to test whether or not non-state cueing treatments were capable of alleviating the memory deficit associated with a change in drug state at the time of test (i.e. of alleviating SD). A group receiving saline prior to training and morphine prior to testing was not included since this group would have no comparable RA group and would thus not provide any additional information.

Training. Rats were transported in their home cages to the experimental room where their cage was placed on a table next to the PA chamber for 30 seconds. The rat was then placed on the experimenter's arm for 30 seconds. It was then gently
placed into the white compartment of the PA chamber and the lid was closed. After 15 seconds, the sliding door separating the two chambers opened automatically, and the latency of the rat to cross into the black compartment (thus causing the tilting floor to connect a circuit) was automatically recorded. Once the animal crossed into the black side of the chamber, the sliding door immediately closed behind it and a 1-second inescapable shock (0.8 mA) was delivered to the animal’s feet. Thus, for all animals, the black side of the PA chamber was a CS paired with a shock US. Fifteen seconds after shock delivery, subjects were removed from the black side of the PA chamber, placed back in their home cage, and returned to the colony room.

Testing. Fear of the black compartment of the PA chamber was measured 24 hours after training by recording the rats’ latency to cross from the white to black compartment in the PA chamber. Testing was identical to training with the exceptions that no shock was delivered upon entry to the black compartment and animals were removed immediately upon crossing. A test ended when the rat either crossed into the black compartment or after 538 seconds.

Just prior to testing, Group M-S-Context subjects received a context cueing treatment that involved a 180-second exposure to the salient context room, followed by a 5-minute delay spent in an unfamiliar darkened room. During context exposure, rats were simply placed in their home cages on the table in the experimental (red light, scented) room in plain view of the PA chamber. Group M-S-CS received a cueing treatment in which subjects were placed in the black side of the PA chamber (i.e., were exposed to the CS from training) for 25 seconds, then were moved to the unfamiliar darkened room for 10 minutes. Rats were given time in the darkened room between exposure to the reminder procedure and testing because of evidence showing that it is beneficial to allow some “processing” time between the reminder cue and test (Gordon, & Mowrer, 1980; Land et al., 2000). The chosen dark room delays (5 and 10 minutes) were based on prior work that effectively used similar delays (Litvin & Anokhin, 2000; Land et al., 2000). Group M-S-Warm-up received a cueing treatment that included a 5-minute context exposure immediately before testing. Finally, Group M-S-Test received two test sessions spaced 24 hours apart. The first test session took place 24 hours after training just as in all other groups. This “reminder” test was identical to the standard test session (no shock upon crossing); with the exception that the rat was retained in the black side of the chamber for 10 seconds after crossing over to allow continued processing of the chamber cues. A standard test trial was then conducted 24 hours after the reminder test session for this group. Groups were run in squads of five. Sample sizes are smaller in the cued groups because it became clear after examining the results from the first squads that none of these groups showed even the slightest trend toward memory recovery. Moreover, there was so little variability in the data to suggest that running more animals might be beneficial that it seemed like a misuse of our limited resources to complete the replications.

Results

A one-way analysis of variance (ANOVA) compared mean training latencies across groups. This test was non-significant at an alpha level of .05, F(6, 46) = 2.08, p > .05, indicating that all groups (S-S, M-S, M-M, M-S-Context, M-S-CS, M-S-Warm-up, M-S-Test) had similar latencies at training (23.8, 48.5, 61.5, 108.6, 23.6, 85.7, 48.8, respectively). A one-way ANOVA compared mean test latencies across groups. This test was significant at an alpha level of .05, F(6, 46) = 3.80, p < .01. Because we were only interested in a small number of pair-wise comparisons, a priori contrasts were used to determine important between-groups differences. Homogeneity of variances was violated; therefore, all reported values are based on adjusted values that do not assume equal variances. As is evident from Figure 1, the mean test latency of Group S-S (M = 224.7, SEM = 69.9) was significantly longer than that of Group M-S (M = 34.2, SEM = 9.9; t(9.36) = -2.7, p < .05), reflecting state dependent memory loss in the latter group. Group M-M (M = 211.6, SEM = 72.4) performed significantly better than Group M-S at test (t(9.33) = .037, p < .05), suggesting that, as predicted by a SD view, re-inducing the morphine state prior to test alleviated the memory decrement. Groups M-M and S-S were not significantly different from one another, t(17.98) = -.130, p > .05, indicating that memory for the task was good regardless of state, so long as the state was consistent between training and testing. Moreover, the fact that Group M-M performed well rules out the possibility that the poor performance in the M-S groups was due to poor learning in groups trained under the morphine state. Similarly, because training cross latencies were comparable for groups trained under morphine and saline states, there is no reason to suspect that the long test latencies in Group M-M reflect motor or motivational effects of morphine. Finally, none of the M-S cued groups (M-S-Context, M-S-CS, M-S-Warm-up, or M-S-Test) had mean test latencies (M = 36.9, SEM = 9.4; M = 22.1, SEM = 8.5; M = 20.3,
Figure 1: Mean cross-through test latencies for groups in Experiment 1. Error bars represent +/- 1 standard error of the mean.

SEM = 7.2; M = 18.3, SEM = 3.7, respectively) significantly longer than Group M-S, t(11.64) = -.92, p > .05, indicating that none of the non-state cueing treatments was effective in alleviating the memory loss due to state-dependency.

Experiment 2

Method

Subjects. Sixty-four (> 90 days old) female Sprague-Dawley-derived rats (from the Kenyon College breeding colony) served as subjects. All housing and other care conditions were the same as for rats in Experiment 1.

Apparatus. The apparatus used in Experiment 1 was also used in this experiment.

Procedure and Drugs. The handling and training and testing protocols were identical to that described for Experiment 1, with the exception that there was a 10-second delay between the animals' entry (cross) into the black compartment and the onset of shock during training. This change was implemented in an attempt to increase the overall cross latencies at test (i.e. improve avoidance learning). Since there are no direct statistical comparisons made between groups from the two experiments, this minor adjustment should not create interpretive difficulties.

Immediately after training and again twenty minutes before testing, rats were injected subcutaneously with either 1.0 mg/kg of cycloheximide (pre-mixed at a concentration of .1%, obtained from Oxoid Limited) or an equivalent volume of 0.9% physiological saline. Cycloheximide is a protein-synthesis inhibitor commonly used to induce RA in laboratory animals (Berman, Kesner, & Partlow, 1978; Litvin & Anokhin, 2000). It has been reported that subcutaneous injection of a dose half of that being used in the current study (.50mg/kg) decreases cerebral protein synthesis in rats by 75% within 20 minutes (Pavlik & Teisinger, 1980). The long-term
amnesic effects of this antifungal antibiotic are not due to a conditioned aversion to aspects of the training situation (Schmaltz & Clement-Forestier, 1977).

Group S-S (n = 8) received saline immediately after training and prior to test. This group provided a baseline measure of memory in normal, untreated rats. Group C-S (n = 8) received cycloheximide immediately after training and saline 20 minutes prior to testing. This group provided a baseline measure of RA due to cycloheximide. For Group C-C (n = 8), cycloheximide was administered immediately after training and 20 minutes before test. This group was included to test for the ability of the cycloheximide state itself to act as a retrieval cue for an amnesic memory. Group Pseudo C-C (n = 8) received non-contingent PA training, in which no shock was delivered during the training procedure. A non-contingent footshock identical to the one given to other groups during training was administered an hour later in a different apparatus. In other words, this group received exposure to all elements of the training procedure, but in a non-contingent manner. This group received an injection of cycloheximide following pseudo-training, and again 20 minutes before test. The purpose of this group was to provide a measure of any non-specific effects of cycloheximide on behavior at the time of testing.

Four groups of rats (all n = 8) received cycloheximide following training and saline prior to testing. In addition, these groups received various reminder treatments prior to testing. The cueing treatments for these groups (C-S-Context, C-S-CS, C-S-Warm-up, and C-S-Test) were identical to those described for the comparable groups in Experiment 1 (M-S-Context, M-S-CS, M-S-Warm-up, and M-S-Test, respectively). For all groups, testing commenced 24 hours after training (and a second time 24 hours following the first test in Group C-S-Test) and was conducted according to the procedures outlined for SD groups.

**Results**

A one-way ANOVA compared mean training latencies across groups. This test was non-significant at an alpha level of .05, F(7, 58) = .998, p > .05, indicating that all groups (S-S, C-S, C-C, C-S-Context, C-S-CS, C-S-Warm-up, and C-S-Test, Pseudo-C-C) had similar training latencies (30.6, 15.4, 19.2, 21.7, 21.7, 14.7, 21.6, 20.9, respectively). A one-way ANOVA compared mean test latencies

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**Figure 2**: Mean cross-through test latencies for groups in Experiment 2. Error bars represent +/- 1 standard error of the mean.
across groups. This test was significant at an alpha level of .05, \(F(7, 58) = 5.05, p < .001\). Because we were only interested in a small number of pair-wise comparisons, a priori contrasts were used to determine important between-groups differences. Homogeneity of variances was violated; therefore, all reported values are based on adjusted values that do not assume equal variances. As can be seen in Figure 2, the mean test latency of Group S-S (\(M = 300.2, SEM = 52.4\)) was significantly longer than that of Group C-S (\(M = 51.8, SEM = 11.3; t(7.7) = -4.63, p < .01\)) reflecting strong RA in the latter group. Group C-C (\(M = 310.3, SEM = 79.0\)) performed significantly better than Group C-S at test, \(t(8.33) = 3.24, p < .05\), suggesting that, as predicted by a MSDR view, reinstating the cycloheximide state prior to test alleviated RA. Mean test latencies for Groups S-S and C-C were not significantly different from one another, \(t(13.58) = 106, p > .05\), indicating that again, as predicted by a SD view, memory for the task was good regardless of state, so long as the state was consistent between training and testing. None of the C-S cued groups (C-S-Context, C-S-CS, C-S-Warm-up, or C-S-Test) had mean test latencies (\(M = 137.1, SEM = 59.9; M = 44.4, SEM = 18.3; M = 88.3, SEM = 46.6; M = 154.4, SEM = 61.2\), respectively) significantly longer than Group C-S, \(t(28.8) = 1.99, p > .05\). These findings are quite similar to those from Experiment 1, showing that none of the non-state cueing treatments was effective in alleviating the memory loss due to RA. Finally, cycloheximide administration prior to test did not increase test latencies in the absence of contingent PA training. To this point, Group Pseudo-C-C had significantly shorter latencies (\(M = 18.58, SEM = 3.15\)) than Group C-C, indicating that the relatively long mean test latency in Group C-C was not due to a non-specific effect of cycloheximide that was unrelated to memory recovery.

**Discussion**

Problems with the storage failure account of RA led to an alternative MSDR theory, which proposes that the forgetting associated with RA is due to a retrieval failure brought about by state dependent memory loss (Hinderliter et al., 1975; Millin et al., 2001). The current study attempted to evaluate this theory by comparing the effects of several reminder cues on forgetting due to SD or RA. Theoretically, if SD underlies RA, then the cues ought to affect them similarly. Both sources of forgetting were completely alleviated by re-induction of the processing state, while none of the non-state cues significantly improved performance. Although null effects can be difficult to interpret, that the pattern of results for cycloheximide-induced RA and morphine SD was very similar is consistent with the hypothesis that the two sources of forgetting share a common mechanism. That both sources of memory loss were reversible suggests that the shared mechanism is retrieval failure. That the most (and in fact, only) effective retrieval cue was re-induction of the physiological state is in harmony with the MSDR theory of RA.

Moreover, that re-induction of the cycloheximide state alleviated RA contradicts the widely held view that new protein synthesis is a critical molecular link in the chain of events that defines consolidation (Nader et al., 2000). However, because we did not independently measure the degree of protein synthesis inhibition (PSI), one could argue that we may not have disrupted the process completely enough to preclude memory storage, and thus failed to induce true RA. Of course, earlier reports that interpreted PSI-induced RA as a storage failure did not measure the amount of inhibition either, so that argument seems strained and unenlightening. It would be instructive for future investigations to include a broader range of cycloheximide doses so that a dissociation, if one exists, between the amnesic and state-dependent effects of the drug might be elucidated. It is possible that the dose of cycloheximide used in the current experiment produced state-dependency without significantly inhibiting cerebral protein synthesis; however, this seems unlikely given that a dose half as large as the one used in this experiment has been reported to result in PSI of 75% twenty minutes following subcutaneous injection. Nonetheless, it is possible that the state-dependent properties of cycloheximide might be unrelated to its disruptive effect on protein synthesis. Studies correlating the degree of PSI with the degree of RA and SD would be informative as to whether the SD and amnesic effects of cycloheximide are interdependent.

It was somewhat surprising that none of the non-state cues was effective in alleviating RA since they were modeled after cues that had been shown to be effective in past reports (Brown et al., 1985; Devietti & Hopfer, 1974). Although it is not clear why these manipulations did not reverse amnesia in the present study, it does imply that the amnesia produced by both agents was profound and that internal state cues may be especially crucial for memory retrieval under such circumstances. Although not significant, the effects of the context, warm-up, and test trial cues did appear to be greater on RA than SD. This may reflect the fact that in the SD design, in which the state was induced prior to the learning episode, a larger proportion of the memory
was encoded in the altered state, making it especially difficult to retrieve in the normal state.

It was also somewhat disappointing that none of the non-state reminders alleviated morphine SD, since, in our view, the MSDR theory would be strengthened by such a demonstration. One potential criticism of the MSDR theory is that it is not clear from a state dependency standpoint why reminder treatments involving non-state cues would, as has been shown in earlier studies, alleviate RA since the processing state would still be absent during recall. We are aware of a single relevant study in which pentobarbital-induced SD for a PA task was alleviated by a pre-test exposure to a compound stimulus consisting of the US from training (non-contingent foot shocks) and the conditioning context (Meehan, Gordon, & Riccio, 1994). Interestingly, these authors were able to alleviate hypothermia-induced RA by exposing rats to the US cue alone, supporting the idea that traditional SD may create a more profound retrieval failure than RA on account of the greater amount of processing completed in the altered state. While Meehan’s findings are encouraging for the MSDR theory, an alternative explanation for their results is that the reminder shock induced a generalized “stress” state similar to that induced by the initial training episode and subsequent pentobarbital injection, thus reinstating the critical internal state cues associated with the memory. We had hoped to demonstrate that, like other forgetting that arises from retrieval failure, SD could be alleviated by retrieval cues unconfounded by the effects of stress.

The current study represents a novel approach for testing the notion that SD and RA share a common mechanism, an important first step in empirically validating the MSDR theory of RA. Our results indicated that under our experimental conditions, forgetting due to SD and RA were similarly affected (or not) by several types of reminder cues. Most notably, reintroducing the processing state at the time of retrieval led to recovery in both cases, suggesting that at a minimum, these two types of forgetting both result from a failure to retrieve the memory. More research is clearly needed to clarify the relationship between these two types of forgetting, which may provide support for an often discussed but seldom tested theory.

References


