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Recovery of Memory Following Forgetting Induced by Depletion of Biogenic Amines[1]

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QUARTERMAIN, D. AND M. E. JUDGE. *Recovery of memory following forgetting induced by depletion of biogenic amines.* PHARMACOL BIOCHEM BEHAV 18(2) 179–184, 1983.—Following depletion of biogenic amines by reserpine, mice were trained to avoid one compartment of a shuttle box by employing the procedures of Pavlovian fear conditioning. Retention was tested one week later using both an active and a passive measure. A robust amnesia was apparent in reserpine-treated animals on both retention measures. Treatment with the mixed dopamine-serotonin agonist lisuride 30 min prior to the test alleviated the memory loss. Since improved retention in the drug treated mice was indexed by increased response latencies in the passive test and decreased latencies in the active test it is unlikely that the improvement in performance was the result of non-specific effects on activity. The results are consistent with the hypothesis that lisuride treatment before testing facilitates retrieval processes.

Retrieval Amnesia Biogenic amines Reserpine Active avoidance Passive avoidance

PHARMACOLOGICAL investigations into the role of biogenic amines in memory processing have revealed that these neurotransmitter systems can influence both the storage and retrieval of information. Evidence that monoamines can modulate storage processes is provided by studies which show that immediate post-training administration of agents which disrupt storage, inhibit synthesis or block receptors result in amnesia while those treatments which stimulate release, block reuptake, prevent transmitter degradation or stimulate receptors facilitate retention [9,24]. Additional support for an effect on storage processes comes from studies which have demonstrated that transient post training decreases in levels of brain norepinephrine correlate with degree of retention of an avoidance response [6,16]. Involvement of the biogenic amines in retrieval processes is suggested by studies which have demonstrated that administration of monoamine stimulants before the retention test can alleviate experimentally-induced amnesias [4, 19, 20, 21], reduce forgetting resulting from a long retention interval [22,23] and improve memory in senescent mice [21].

Recently, attention has been focussed on the relative contribution of central and peripheral amine systems to memory processing. Several experiments have shown that endogenous peripheral catecholamine release in response to the training experience facilitates memory storage. For example, it has been shown that alpha adrenergic antagonists [27] and d-amphetamine [14] can attenuate forgetting if they are administered peripherally, but not centrally, around the time of training. It has also been shown that guanethidine and syrosingopine, two catecholamine (CA) depleting agents which do not readily enter the brain, cause amnesia when administered before training [18,28] and that amnesia induced by CA depletion can be reversed by peripherally administered doses of dopamine and norepineprhine [30]. Additional support for the involvement of peripheral catecholamines comes from studies which show that plasma epinephrine and norepinephrine levels are responsive to levels of footshock used in training [16]. These observations suggest a role for the peripheral catecholamine systems in memory storage processes.

The relative importance of central and peripheral amine systems in retrieval processes has received less attention. The available evidence suggests that central monoamine systems may have a more important function in mediating retrieval than they appear to have in modulating storage. This generalization is suggested by the results of recent studies which have shown that: (a) d-amphetamine administered peripherally before the retention tests enhances retention but the amphetamine analog 4-hydroxyamphetamine (4-OHA) which has difficulty passing the blood brain barrier fails to improve performance [22], (b) both d-amphetamine and the dopamine-serotonin agonist lisuride facilitate retention when administered into the brain before a retention test [1], and (c) the reversal of amnesia induced by pre-test treatment with the monoamine oxidase inhibitor pheniprazine is blocked by reserpine but not by syrosingopine [19]. Taken together, these results suggest a role for central monoaminergic systems in memory retrieval processes.

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It has recently been suggested that amnesias induced by depletion of peripheral and central amines may have different characteristics. Walsh and Palfai [29] have shown that while amnesia induced by the peripheral depleting agent syrosingopine can be alleviated by repeated exposure to the training apparatus, memory loss induced by combined central and peripheral depletion by reserpine cannot. On the basis of this observation Walsh and Palfai [29] suggest that reminder treatments can recover memory following peripheral amine depletion but when central amines are depleted the amnesia is permanent and reflects a loss from storage.

The principal purpose of the present experiment was to test this hypothesis by determining whether forgetting induced by pre-training reserpine can be alleviated by a pharmacological treatment administered prior to the retention test. Reserpine has been widely employed as an amnestic treatment. This agent depletes neuronal stores of all the monoamines both centrally and peripherally [26] and is therefore useful for investigating the durability of amnesias induced by depletion of biogenic amines. It has been shown that the amnestic effects of reserpine are not due to acute toxicity at time of testing, altered footshock thresholds or state dependent learning [13,17]. We have previously shown [21] that lisuride can restore memory following forgetting induced by a protein synthesis inhibitor or by a long retention interval. Lisuride is a potent agonist for both dopamine and serotonin receptors and in addition has weak noradrenergic blocking activity at high doses [11]. Our intention in the present study was to determine if lisuride administered before the retention test would alleviate forgetting induced by depletion of central and peripheral monoamines by reserpine.

An additional purpose of the study was to evaluate the utility of a testing procedure which permits memory loss and memory recovery to be indexed by both an active and a passive avoidance response. The use of such a procedure would provide more convincing evidence that pharmacological treatments are influencing memory processes rather than producing non-specific behavioral reactions.

METHOD

Animals

Male Swiss Webster mice (West Jersey Biological Supply) approximately 12 weeks old and 40 grams body weight were the subjects for this experiment. Subjects were housed 4/cage with ad lib food and water for the duration of the experiment.

Apparatus

Pavlovian fear conditioning was carried out in one side of a two compartment mouse shuttle box (LVE #MSC-022). The shock side, which was 9 cm wide, 11 cm high and 23 cm long had black Plexiglas walls and a black lid. The floor consisted of stainless steel rods (0.3 cm diameter, 7 cm between rods) through which a scrambled shock (0.2 mA) could be delivered from a Coulbourn constant current shocker. The conditioned stimulus (CS) was a 24 VDC light flashing on for 0.5 sec at a frequency of 1 Hz. The CS was positioned on the lid above the shock compartment. The non-shocked (safe) side of the compartment measured 9 × 11 × 23 cm and had white Plexiglas walls, a white cardboard floor and was covered with a white Plexiglas lid. The two sides were separated by a partition which contained a guillotine door 4 cm wide × 4 cm high. Sham training was carried out in an adjacent room in a chamber of the same dimensions as the training shock chamber but having a V-shaped aluminum floor with a separation of 1 cm between the two sides of the trough. Each side was connected to one pole of a constant current shock source set to the same intensity (0.2 mA) as the UCS used in fear conditioning. The chamber was yoked to the training apparatus so that the sham trained mice received shocks at the same time as their classically conditioned counterparts.

Procedure

Pavlovian fear conditioning (C). Mice were first placed in the white ("safe") side of the conditioning chamber where they remained for 2 minutes. No shock was administered. The door was then opened and the animal was gently moved into the black side of the apparatus where 4 fear conditioning trials were administered. Each conditioning trial consisted of a 13 sec presentation of the CS accompanied for the final 3 sec by the UCS. CS and UCS terminated together. The 4 trials were given on a variable interval 30 sec schedule. Following the fourth shock the door was raised and the mice were gently pushed into the white side where they remained for 2 minutes. They were then returned to the shock side where they received another 4 conditioning trials. The conditioning procedure thus consisted of a total of 8 CS-UCS pairings and exposure for an approximately equal period to the stimuli of the safe (non-shocked) compartment.

Sham Conditioning (S). Animals scheduled for sham conditioning were yoked to a mouse receiving classical conditioning. Thus number and intensity of shocks was identical for the two groups but no formal CS-UCS pairing occurred for the yoked controls.

Retention testing. Retention was tested 7 days after conditioning. Animals were divided into two groups one of which was tested with a passive and the other with an active avoidance procedure. No shock was administered during testing.

Passive avoidance. Animals tested with this procedure were placed in the safe side facing away from the door. After 5 sec the guillotine door was raised allowing access to the shock chamber in which the CS was continuously present. Initial latency to cross into the shock compartment, the total number of crosses and the total time spent in the shock compartment during the 5 minute test period was recorded for all animals. Mice failing to cross within 5 min were given the maximum latency score.

Active avoidance. Mice were placed in the shock compartment and after 5 sec the door was raised and the CS initiated. When the mouse crossed into the safe compartment the CS terminated and the response latency was automatically recorded. Mice remained for 60 sec in the safe side between trials. Animals failing to respond within 60 sec were lifted into the safe side by the experimenter and given the maximum latency as a test score. All animals received 5 active avoidance trials.

Drug administration. Peripheral and central biogenic amines were depleted by injecting mice with 2.5 mg/kg reserpine (Serpasil,® Ciba) 2 hours prior to conditioning or sham training. This dose and time value was chosen in order to duplicate the conditions employed by Walsh and Palfai [29]. Control animals were injected with the drug vehicle which consisted of 2.5 mg of citric acid, and 0.1 ml of propylene glycol per ml of water. Thirty minutes prior to

TABLE 1
EXPERIMENTAL DESIGN

Drug	Training Condition	Drug	Testing Condition
SAL	S	SAL	PA
SAL	C	SAL	or
RES	C	SAL	
RES	C	LIS	AA

SAL=Saline, RES=Reserpine, LIS=Lisuride, C=Conditioning, S=Sham Conditioning, PA=Passive Avoidance, AA=Active Avoidance.

testing mice were injected with either saline or 0.5 mg/kg lisuride hydrogen maleate (Schering AG, Berlin). This dose has previously been shown to induce robust facilitation of retention following forgetting induced by a variety of sources [21]. All drugs were injected subcutaneously at 0.01 ml per gram of body weight.

Experimental design. On the training day mice were given either fear conditioning or sham conditioning using the procedures described above. At testing 7 days later, some animals were tested with a passive avoidance test and some with an active avoidance procedure. Thirty minutes before the test mice were injected with either saline or lisuride. This design is outlined in Table 1 and group numbers are presented in the results section. Latencies were transformed to logarithms before statistical analysis.

RESULTS

Passive Avoidance

Passive avoidance test scores are shown in Fig. 1. Strong fear conditioning is evident when Saline injected mice are compared with the sham conditioned control group. Relative to the sham group, conditioned mice showed a significant increase in latency to enter the shocked side, $t(16)=6.39$, $p<0.001$ (all t-tests, 2-tailed), a significant decrease in total number of entries, $t(16)=11.91$, $p<0.001$, and a significant decrease in percentage time spent in the shock side, $t(16)=6.96$, $p<0.001$. Reserpine produced a robust amnesia for this conditioning. A comparison of the conditioned Saline group with the conditioned Reserpine group revealed that reserpine-treated mice initially entered the shock compartment significantly faster, $t(22)=4.56$, $p<0.001$, made significantly more entries, $t(22)=8.70$, $p<0.001$, and spent a significantly longer percentage of the test period in the shock compartment, $t(22)=4.84$, $p<0.001$, than conditioned mice treated with Saline. Pre-test treatment with lisuride restored the conditioned response to the level of the Saline treated controls. When the conditioned reserpine-Saline and conditioned, reserpine-lisuride groups are compared it is apparent that lisuride treatment results in a significant increase in initial latency, $t(21)=5.85$, $p<0.01$, a significant decrease in number of entries, $t(21)=8.23$, $p<0.001$, and a significant decrease in the percentage of time spent in the previously shocked compartment, $t(21)=3.69$, $p<0.02$. Retention performance following lisuride treatment is not different from that observed in mice with unimpaired memory. These findings indicate that lisuride results in an almost total restoration of the fear conditioning.

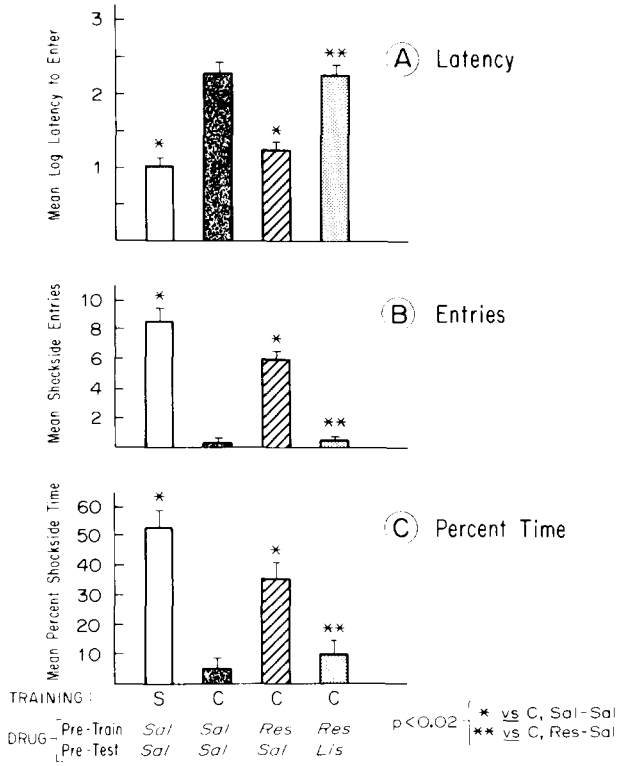

FIG. 1. Passive avoidance retention test scores. Panel A, latency to enter the shock chamber, Panel B, total number of entries, and Panel C, percent time spent in the shock chamber. The test period was five minutes in duration. S=sham conditioning; C=fear conditioning; Sal=Saline; Res=Reserpine (2.5 mg/kg); Lis=lisuride (0.5 mg/kg).

Active Avoidance

Data from the active avoidance test are displayed in Fig. 2 and the results of various statistical comparisons are shown in Table 2. Good retention of fear conditioning is indicated by a decrease in latencies to leave the previously shocked compartment over the 5 test trials. This progessive decline in latencies is characteristic of performance in this task [15] and reflects rapid learning of an escape response from stimuli previously associated with punishment shock. A linear trend analysis [12] carried out on the scores from the conditioned, Saline injected (C,Sal-Sal) group indicates a significant negative slope. Sham conditioned mice (S,Sal-Sal group) exhibit a significant positive slope; their latencies increasing over trials so that at the end of the test session they are effectively distributing their time to about 50% in each compartment (the ITI is spent in the "safe" side). An analysis of variance comparing the scores of these two groups reveals significant groups and interaction effects (Table 2).

Pre-training treatment with reserpine resulted in a pattern of test scores similar to that seen in sham conditioned controls. The C,Res-Sal group exhibited a significant positive linear trend indicating increasing latencies over the 5 test trials. An ANOVA comparing this group to mice with intact memory of conditioning (C,Res-Sal vs. C,Sal-Sal) revealed significant groups and interaction effects (Table 2). Figure 2 shows that reserpine and saline treated mice perform comparably on the first test trial. The relatively long escape latencies in the C,Sal-Sal group occurs because animals tend

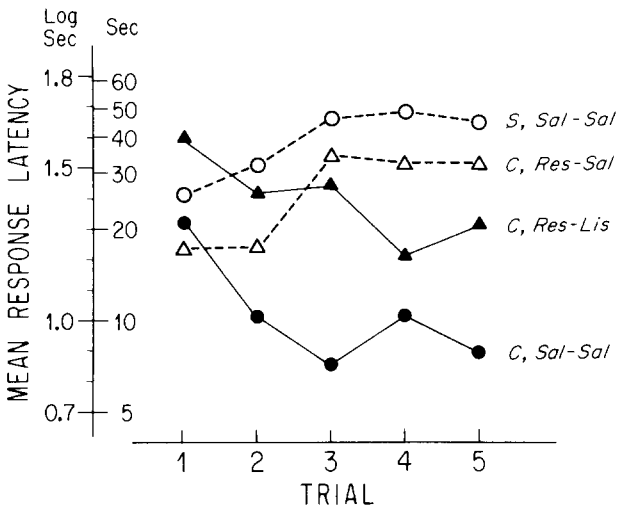

FIG. 2. Active avoidance scores expressed as mean latency to cross from the shock to the safe side under extinction conditions. S, Sal-Sal=Saline before sham conditioning, Saline before testing; C, Sal-Sal=Saline before fear conditioning, Saline before testing; C, Res-Sal=Reserpine before fear conditioning, Saline before testing; C, Res-Lis=Reserpine before fear conditioning, Lisuride before testing.

to freeze when first introduced to the shock chamber. Since mice had no opportunity to escape during training they have to learn this response during the test session. Evidence for retention of fear in this paradigm is provided by the significant linear decline in escape latencies in the C,Sal-Sal group. Mice treated with reserpine do not show this decline; their latencies more closely parallel those of the sham conditioned group, who exhibit a progressive increase in latency over the 5 test trials.

Lisuride treatment before testing (C,Res-Lis) effectively reversed the reserpine amnesia shifting the slope of the latency curve back to a significant negative value. An analysis of variance comparing the C,Res-Lis scores with those of the C,Res-Sal group reveals a significant interaction effect (Table 2); lisuride treated mice run faster while saline animals run more slowly over the 5 test trials. These results are confirmed by trend analyses which indicate that the lisuride latencies exhibit a significant negative trend while saline scores are significantly positive (Table 2). It should be emphasized that relative to the amnesic group (C,Res-Lis), lisuride treatment produces different patterns of responding depending on the test used to index retention. When a passive test is employed mice treated with lisuride respond more slowly than mice treated with saline; when an active response is required however, lisuride results in a pattern of responding in which the mice eventually run faster than the saline controls.

Unlike the passive avoidance measure where reserpine mice treated with lisuride were indistinguishable from animals with intact memory, in the active avoidance test lisuride treated animals, though exhibiting a similar increase in running speed over trials did not respond as quickly as saline treated controls. The most probable explanation for this difference is that the active measure is more sensitive to the side effects of the lisuride treatment. High doses of lisuride, especially when combined with the after effects of reserpine, probably depress motor functioning so that while memory of the conditioning is restored (as indicated by the significant linear decline in latency) mice are unable to respond as rapidly as saline controls. We have previously observed that response speed is frequently reduced following 0.5 mg/kg lisuride.

DISCUSSION

The principal finding of this study is that pre-test treat-

TABLE 2

ACTIVE AVOIDANCE TEST: STATISTICAL EVALUATIONS

Training	C	C	C	S
PreTraining Drug	SAL	RES	RES	SAL
PreTest Drug	SAL	SAL	LIS	SAL
N	12	10	13	8
Trend Analysis				
Coefficient	−0.87	+0.85	−0.76	+0.64
F	6.14	5.21	6.69	8.39
df	1,11	1,9	1,12	1,7
p	<0.85	<0.05	<0.05	<0.05
ANOVA		vs. C,S-S	vs. C,R-S	vs. C,S-S
Groups				
F		11.08	0.01	25.23
df		1,20	1,21	1,18
p		<0.01	>0.1	<0.01
Trials				
F		0.80	0.63	0.52
df		4,80	4,84	4,72
p		>0.1	>0.1	>0.1
Interaction				
F		5.69	3.37	4.30
df		4,80	4,84	4,72
p		<0.01	<0.05	<0.01

ment with lisuride can alleviate memory loss induced by reserpine. The demonstration that lisuride increases test latencies when retention is indexed by a passive measure but decreases latencies when an active test is employed strongly suggests that the effects of the drug cannot be attributed to activity changes or other non-specific effects of the treatment. The change in the direction of the latency measure depending on the response requirements of the retention test indicates that lisuride is probably influencing performance by its actions on memory processes.

The mechanisms by which memory is restored following lisuride treatment cannot be specified from this experiment. Two explanations of pharmacologically-induced retention enhancement following forgetting can be advanced. The first is based on the view that forgetting is the result of retrieval failure and holds that pharmacological agents improve retention performance by increasing the accessibility or retrievability of stored memories. According to this formulation amnesia treatments do not diminish the strength of the trace but rather they disrupt processes by which internal and external stimuli become associated with the target memory during training. The stimulus associations which normally function as retrieval cues are thus relatively weak and memory failure occurs because of the resultant lack of perceived congruence between conditions which existed at encoding and those present at the time of testing. Pharmacological agents are believed to facilitate retention because they enhance the similarity between conditions of testing and original training [22].

An alternative explanation is based on the premise that performance following amnesia treatments reflects a loss of information from storage. The memory trace is either obliterated or weakened by the treatment [3,5]. According to this position, pharmacological agents enhance retention because they provide additional opportunities for new learning to take place. This new learning when added to the strength of the residual training memory results in an above threshold response. This hypothesis was originally proposed as an explanation for the retention enhancement which occurred following behavioral reactivation treatments such as presentation of CS or UCS [5]. The new learning hypothesis has not provided a satisfactory account of these phenomena [7]. Behavioral reactivating agents do not induce learning in untrained (non-contingently or sham trained) animals and many of the procedures are operationally equivalent to extinction [8].

Data relevant to the evaluation of these interpretations are beginning to appear. While it appears unlikely that pharmacological treatments by themselves could induce new learning, it is possible that they might produce motivational alterations which could strengthen a weakened response. For example, in shock avoidance paradigms agents such as d-amphetamine might induce non-specific anxiety or fear which could summate with the residual training strength to reinstate the avoidance response. In a test of this possibility [22] mice which had been made amnesic for a lick of suppression response were treated before testing with either d-amphetamine or pentylenetetrazol (PTZ), an agent which has been shown to possess anxiogenic properties [25]. Retention was enhanced following amphetamine injection but PTZ was without effect. In the same study, a group of mice was given several strong footshocks prior to the retention test. This procedure failed to facilitate retrieval of the lick avoidance response indicating that a behavioral treatment which induces generalized fear and stress was also ineffective. In another experiment [10] several doses of epinephrine did not improve retention of a step-through avoidance response under conditions in which vasopressin produced strong avoidance. These findings make it unlikely that generalized fear and stress by themselves are sufficient to induce memory retrieval. These findings are more consistent with the hypothesis that retention enhancement induced by pre-test treatment with pharmacological agents is the result of increased accessibility of the stored memory. The mechanisms by which drugs enhance retrieval processes is unknown. Current evidence [23] indicates that agents which increase activity of catecholamine (CA) neurotransmission are particularly effective in facilitating retrieval. This suggests that CA neuronal systems may be an important component of a general memory retrieval mechanism. However, the possible contribution of the serotonergic system must also be considered in view of the potent effect of drugs such as reserpine and lisuride.

Finally, the results of this study confirm previous findings [2, 4, 19] indicating that depletion of biogenic amines before training does not prevent information from being stored in memory. Under normal circumstances this information is inaccessible either because the trace has been reduced in strength or because the retrieval cues necessary for gaining access to an intact memory are unavailable or weakly encoded. When CA stimulants are administered before the test, accessibility to the trace is facilitated. This may result because the agents reinstate certain internal cues associated with training thereby making the retrieval environment more similar to the conditions under which the response was encoded. Alternatively the drugs may facilitate processes associated with selective attention thus making the relevant training stimuli more discriminable in the retrieval environment. The evaluation of these and other possible explanations is one of the goals of our current work.

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