results is limited by the stability of the background source and by noise in the photomultiplier traces from which the absorption coefficients were calculated. From an analysis of dK/K as obtained from Eq. 1 we estimate the accuracy of our results to be better than 15 percent at the major absorption peaks and better than 45 percent at values of K which are less than 500. The position uncertainty of any point on the wavelength scale is $\pm$ 3 Å.

Our work is in excellent qualitative agreement with the results obtained by Price (*4, 10*). As was the case in Price's data, our results show the absorption feature at 1750 Å to be extremely diffuse and relatively less in magnitude than the absorption line at 1560 Å. Our data are in disagreement with those of Harrison (*5*) and her coworkers. Harrison's absorption coefficients (transformed into our units) are shown by the dashed lines in Fig. 1. Because of signal-to-noise considerations, in the present work we did not resolve vibrational features at 1739 and 1722 Å. There is no apparent explanation, however, for the discrepancy in the absolute magnitude of the absorption peaks at 1750 and 1559 Å (*11*).

Our data clearly show a continuum in the H_2CO absorption spectra of considerable intensity at wavelengths shorter than 1570 Å. This continuum will be the major spectral feature contributing to the determination of the H_2CO lifetime in the interstellar radiation field and will yield lifetime values which are several orders of magnitude shorter than would be inferred from previously available data (*12*). As was pointed out in the introduction, the application of these data to a meaningful lifetime calculation must also involve a reevaluation of the generalized interstellar radiation field in the local regions of space where formaldehyde is found (*2*).

Note added in proof: Recent measurements made with 0.25-Å resolution indicate closer agreement with the photographic work of Harrison and her co-workers (*5*) than is evidenced in Fig. 1. These results, which are in good agreement with Fig. 1 for $\lambda < 1700$ Å, nonetheless yield a maximum K of 1360 ± 20 cm^{-1} atm^{-1} at the 1750-Å peak.

E. P. Gentieu, J. E. Mentall
Laboratory for Extraterrestrial Physics, National Aeronautics and Space Administration, Goddard Space Flight Center, Greenbelt, Maryland

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10. Price (*4*) published only the H_2CO spectra and did not attempt to calculate absorption coefficients.
11. Harrison and her co-workers (*5*) did not estimate the absolute accuracy of their measurements. The method by which we collected data, namely, by scanning the background source before and after the H_2CO had flowed through the absorption chamber, precluded the possibility of errors due to the possible polymerization of H_2CO on the photomultiplier window.
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Amnesia Produced by Electroconvulsive Shock or Cycloheximide: Conditions for Recovery

Abstract. *Retrograde amnesia for a passive avoidance response was produced in rats by electroconvulsive shock and in mice by cycloheximide, an inhibitor of protein synthesis. One day after training the memory could be restored if a "reminder" of the original foot shock was given after the retention test on which the amnesia was demonstrated. Memory did not return if the reminder was given without the prior retention test or if the reminder and the test were separated by 23 hours.*

The most commonly accepted explanation of the time-dependent effects of electroconvulsive shock (ECS) and other amnesia-producing agents is that they interfere with the consolidation of the memory trace (*1*). According to this hypothesis, memories are initially in a labile stage, at which time they are vulnerable to destruction by a variety of agents. With the passage of time they become increasingly resistant to disruption, until a time is reached when they are said to be consolidated. This interval is a matter of controversy, with estimates ranging from a few seconds to several days (*2*). It is also commonly assumed that when retrograde amnesia (RA) is produced, it is permanent, since if fixation has been prevented, no memory should be present at any time (*3*). This aspect of the hypothesis has received considerable experimental attention. Although some studies have shown spontaneous recovery of memory after ECS (*4*), most experiments indicate that the amnesia is permanent (*5*). This conclusion has been challenged recently by several experiments which have shown that, by appropriate means, memories apparently lost after ECS can be restored (*6*). We now report a study which describes some of the conditions which determine recovery of memory after amnesia induced by ECS or by cycloheximide, an inhibitor of protein synthesis.

Subjects for our first experiment were male Holtzman rats 90 to 120 days old. The apparatus was a standard two-compartment passive avoidance box. The large compartment was 38 cm square and 44 cm high. The smaller compartment, which served as a start box, was 17 cm long, 15 cm wide, and 33 cm high. The two compartments were separated by a sliding door. The entire apparatus was constructed of ¼-inch (1 inch = 2.54 cm) clear Plexiglas, except for the floors which were made of stainless steel rods 0.24 cm in diameter, spaced 1.25 cm on centers.

A foot shock (FS) of 1.6 ma for a duration of 2 seconds was delivered to the grid floor of the large compartment from a scrambled constant-current shock source. Electroconvulsive shock (100 ma for 300 msec) was delivered through small padded alligator clips which were attached to the rat's ears before training. Intensities of FS and ECS were monitored on milliammeters and checked regularly on an oscilloscope.

On the training day, rats were put in the starting compartment with alligator clips attached. After 15 seconds the door was opened; when the rat entered the large compartment, the door was

closed and FS was automatically delivered. Electroconvulsive shock was administered 1.0 second after the termination of the FS. On the next day, we attempted to restore the memory by giving the rats a reminder of the foot shock they had previously received. The reminder shock (1.6 ma for 2.0 seconds) was given in a small (10 cm wide, 20 cm long, 12 cm high) black Plexiglas box and did not itself produce any avoidance of the test apparatus. This procedure is essentially a relearning test of retention.

The basic phenomenon of ECS-induced RA is shown in Table 1 which gives median latencies to step-out (stepping into the large compartment) on the training day and on two retests, the first (T_1) 24 hours after foot shock and the second (T_2) 24 hours after T_1. Rats which were given FS without the ECS showed good memory, as indicated by latencies of 180 seconds on both tests (group 1). Administration of ECS 1.0 second after foot shock produced amnesia (group 2). Test 1 and test 2 latencies of the group given both FS and ECS are significantly faster than those of the group given FS and sham ECS ($P < .01$; *7*). These latencies are indistinguishable from those of a group given ECS without prior FS (group 3).

Table 1 also shows the results of attempts to restore memory by giving a reminder shock (RS) in a different place. In our initial attempts rats were given the reminder shock 24 hours after training and tested for recovery 24 hours later. This procedure does not result in any return of memory (group 4). A T_2 latency of 31.5 seconds is not significantly different from latencies of the group receiving no reminder shock (group 2). We repeated testing of this group on a number of occasions and were never able to demonstrate recovery of memory. On studying the procedures of two published reports of the reminder-shock phenomenon (*6*), we felt that an initial test before reminder shock was administered might be a crucial factor in producing recovery of memory, although the importance of this was not indicated in the reports. We therefore introduced an initial test for amnesia (T_1), which was given 24 hours after foot shock. One hour after this we gave the reminder shock and 23 hours after that we gave T_2. This procedure results in a T_2 latency of 171.5 seconds (group 5). This is a significant increase over T_1 latency ($P < .01$) and is comparable to latencies of animals which did not get an ECS. That this increase in T_2 latencies is not simply due to association of the reminder shock with the training apparatus is shown by the fact that rats given neither FS nor ECS but given reminder shock 1 hour after T_1 have short T_2 latencies (group 6).

To describe more fully the dependence on T_1 of memory recovery, after a reminder shock, we carried out a further series of experiments. We first determined whether the handling involved in T_1 was by itself sufficient to cause reminder shock to restore the memory. A group of rats was given foot shock and ECS as usual, but 24 hours later, instead of being given T_1, they were put in a brown cardboard box, which had the same dimensions as the training apparatus, and left there for a time equal to that spent by rats in completing T_1 in the apparatus. One hour later, RS was administered. This did not bring back the memory (Table 1, group 7). Test 2 latencies of this group were not significantly different from those of the group which was not given T_1 (group 4).

It began to appear that the importance of T_1 for the reminder-shock phenomenon might be that it provided the opportunity for the animal to recognize the training situation and so reactivate a partially suppressed memory of the situational cues. If this were the case, we reasoned that the reactivated memory trace might fade with the passage of time, in which event RS would be progressively less effective. This is indeed the case. Reminder shock given 4 hours after T_1 still produced a significant amount of memory recovery when T_1 and T_2 latencies are compared ($P < .01$, group 8); but when RS was delayed for 23 hours (group 10), it had no significant effect in reversing the amnesia. For both of these intervals we were able to show that, in the absence of the original punishment shock, the reminder shock did not change T_2 latencies significantly (groups 9 and 11). As a final control we gave a group of rats FS followed 1.0 second later by ECS in a small white compartment which had two aluminum plates as floors. Test 1 was given as usual in the regular training apparatus followed by RS 4 hours later. The T_2 latencies of this group (group 12) are significantly shorter ($P < .01$) than those of the group which was given FS and ECS in the training apparatus (group 8). This indicates that the FS has to be given in the training apparatus in order for the RS to produce long T_2 latencies and thus tends to minimize the importance of nonspecific effects of combining FS with ECS.

In summary, these data show that, in order for a reminder shock to produce recovery of the original memory, it must be preceded by exposure to the apparatus where the original training took place. Furthermore, the memory of this experience appears to decay with the passage of time, so that giving a reminder shock 23 hours after T_1 is equivalent to not giving a reminder shock at all. That the RS can be effective as long as 4 hours after T_1 is surprising and suggests that this is not a typical gradient of association but rather some decay in the ability of the RS to rearouse the memory of the training cues. Lewis *et al.* (*6*) also report that a delay of 4 hours between T_1 and RS will still produce substantial memory recovery (*8*).

In our second experiment, we attempted to determine whether memory could be recovered after amnesia had been induced by an inhibitor of protein synthesis, cycloheximide. Using a one-trial passive avoidance task, we have shown (*9*) that, when mice are injected with cycloheximide 30 minutes before a training trial on which they are punished with a high shock (1.6 ma for 2 seconds), they do not show amnesia when tested 1 minute, 5 minutes, or 6 hours after training, but they do show amnesia when they are tested 24 hours after training. However, at 48 hours the memory returns spontaneously. No such effects were shown by mice punished by a low foot shock (0.16 ma). These animals showed amnesia at all testing times, and no spontaneous return of memory was observed. It was of interest, therefore, to determine whether this memory can be restored by giving a reminder shock.

Subjects were male C_{57}/BL.6J mice (25 g). The apparatus was a two-compartment passive avoidance box. The smaller compartment was made of clear Plexiglas, and was 10 cm long by 7 cm wide. A circular hole 5 cm in diameter served as an entrance to the large compartment, which had black Plexiglas sides and was 10 cm wide and 20 cm long. The floor was made of stainless steel rods ⅛ inch in diameter and set 0.5 inch apart. A black Plexiglas guillotine door separated the two compartments. A flashing light, which operated when the door was raised, was set into the end wall of the large compartment. The entire apparatus was 12 cm high, and both compartments were covered with separate hinged lids.

Mice were injected subcutaneously in the back of the neck with 3.0 mg of cycloheximide (Actidione; Upjohn Co., Kalamazoo, Michigan) dissolved in 0.3 ml of 0.85 percent saline. All mice were injected 30 minutes before the training trial to permit inhibition of protein synthesis to reach its maximum (*10*). Mice were placed in the small compartment and, after 15 seconds, the door was raised. This operated a timer and started a flashing light which continued for 20 seconds. When the mouse entered the large compartment, the door was closed and the timer automatically stopped. The light continued to flash and, for the last 2 seconds of the 20-second period, FS (0.16 ma for 2.0 seconds) was automatically delivered through the bars of the large compartment. The light and the FS terminated together. Reminder shock was given in a large, clear Plexiglas compartment 38 cm square and 40 cm high.

Our design, outlined in Table 2, was similar to that used in our first experiment except that T_2 was given 4 hours after T_1 (*11*). The amnesic effect produced by injecting cycloheximide 30 minutes before training is presented in Table 2. Compared to controls injected with saline (group 1), which show good retention, mice injected with cycloheximide (group 2) have a well-developed amnesia ($P < .001$). When given to untrained mice, cycloheximide does not significantly increase test latencies (group 3). Also shown are the effects of giving a reminder shock 1 hour after T_1 (group 4). These mice show a significant increase from T_1 to T_2 latency ($P < .01$) and also have significantly longer T_2 latencies ($P < .001$) than mice given no RS (group 2). When we omitted T_1 and left the mice in their home cages (group 5) reminder shock failed to bring back the memory. We ruled out the effect of a nonspecific interaction of FS with the cycloheximide treatment by giving mice an FS in a small white box instead of the training apparatus. They were given T_1, RS, and T_2 as usual. This procedure (group 6) did not produce T_2 latencies significantly longer than those for the group given no RS (group 2). Finally, we ruled out the direct effect of the RS alone by giving cycloheximide to a group of mice which were given an initial trial in the apparatus but not given FS. As usual, T_1 was followed 1 hour later by the RS. This group did not show a significant increase in T_2 latency (group 7).

Table 1. Median step-out latencies on the training trial and on the two retention-test trials for the groups in experiment 1. Key: FS, foot shock; RS, reminder shock; T_1, first retention test; T_2, second retention test; Handle, place in cardboard box (see text); ECS, electroconvulsive shock; NFS, no foot shock; NECS, no electroconvulsive shock. Time refers to the time after T_1 that RS was administered.

Group	Treatment	Time (hours)	Animals (No.)	Median training latency (sec)	Median test 1 latency (sec)	Median test 2 latency (sec)
			No reminder shock			
1	FS-Sham ECS		12	7.50	180.00	180.00
2	FS-ECS		12	7.10	32.50	23.50
3	NFS-ECS		12	9.65	30.50	25.50
			Reminder shock			
4	FS-ECS-RS		12	5.60	None	31.50
5	FS-ECS-T_1-RS	1	12	6.45	33.75	171.50
6	NFS-NECS-T_1-RS	1	12	6.80	8.60	24.50
7	FS-ECS-Handle-RS	1	12	9.50	None	44.50
8	FS-ECS-T_1-RS	4	12	9.80	14.00	141.25
9	NFS-ECS-T_1-RS	4	12	6.80	13.00	25.20
10	FS-ECS-T_1-RS	23	12	7.25	32.85	40.50
11	NFS-ECS-T_1-RS	23	12	10.00	15.00	32.60
12	FS-ECS (white box)-T_1-RS	4	12	None	10.50	29.50

This experiment duplicates the essential features of our ECS experiment and indicates that a reminder shock can also produce recovery of memory after amnesia induced by administration of cycloheximide. These data also indicate that the conditions necessary for recovery are similar after amnesia caused by two quite different agents.

We have also found that (i) a second cycloheximide injection will prevent the recovery of the memory if it is given both before T_1 and before the RS, and (ii) the memory which is restored by the RS procedure is neither extinguished nor forgotten any more rapidly than normal memory of the foot shock.

Clearly, fixation of the memory trace is not completely prevented by ECS or cycloheximide; at least some part of the trace must remain intact after these treatments since the memory can be recovered. The performance deficit might therefore be more appropriately described in terms of a failure of retrieval rather than a disruption of a consolidation process. The nature of the retrieval failure cannot be determined from these experiments. It is possible that ECS and cycloheximide suppress or inhibit the expression of a fully formed and intact memory trace (*12*). It seems to us, however, that it is more likely that the amnesic treatments weaken some part of the memory but that enough of the trace remains to be restored by the reminder shock and testing. For example, amnesic agents may weaken the association between fear and the situational cues to which it is attached. Animals may still be fearful but be unable to associate their fear with the situational cues in the presence of which foot shock was originally administered (*13*). This could provide an explanation for the fact that the animals have to be reexposed to the training apparatus before the reminder shock will restore memory. Whatever the mechanism involved, our data sug-

Table 2. Median step-out latencies for the training trial and the two retention-test trials for the groups in experiment 2. Key: Sal, saline; Cyc, cycloheximide; FS, foot shock; NFS, no foot shock; T_1, first retention test; T_2, second retention test; RS, reminder shock.

Group	Treatment	Animals (No.)	Median training latency (sec)	Median test 1 latency (sec)	Median test 2 latency (sec)
		No reminder shock			
1	Sal-FS-T_1-T_2	15	6.60	180.00	180.00
2	Cyc-FS-T_1-T_2	15	5.30	8.60	20.00
3	Cyc-NFS-T_1-T_2	15	6.00	9.00	7.90
		Reminder shock			
4	Cyc-FS-T_1-T_2	15	3.80	9.30	180.00
5	Cyc-FS-NT_1-T_2	15	7.80	None	17.00
6	Cyc-FS (white box)-T_1-T_2	15	None	9.00	10.40
7	Cyc-NFS-T_1-T_2	15	6.30	7.90	16.70

gest that more attention should be given to experimentally induced amnesias from the point of view of failures of retrieval.

DAVID QUARTERMAIN
BRUCE S. MCEWEN
EFRAIN C. AZMITIA, JR.
The Rockefeller University,
New York 10021

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7. Mann-Whitney U tests were used for all statistical comparisons.
8. A reviewer has raised the possibility that had we not had an arbitrary cut-off point of 180 seconds and had we included a group receiving FS and no ECS, reminder shock might have increased these latencies also. This might have happened, but it could only be accounted for by an enhancement of an already established memory and would therefore in no way change the interpretation that the increase in latency from T_1 to T_2 in group 5 is due to a recovery of the original memory.
9. D. Quartermain and B. McEwen, *Nature*, in press.
10. Cycloheximide (Actidione, Upjohn) at a dose of 3 mg blocked incorporation of [^{3}H]leucine into total cerebral proteins by 94 percent at 30 minutes after subcutaneous administration in saline. Incorporation returned to normal within the ensuing 7 hours. With this dosage less than 5 percent of the mice showed any sickness at the time of testing. Those which did were easily recognized and discarded from the experiment. These results and a more detailed description of the experimental procedure can be found elsewhere (*9*).
11. We have also shown that memory can be recovered after ECS when RS is given 1 hour after T_1, and T_2 follows 4 hours rather than 24 hours later.
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13. We often observe that when rats are placed in the start compartment for T_1, they show a brief freezing response often accompanied by defecation and urination. After a few seconds they relax, explore the start box, and then tentatively walk out into the large compartment.
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Fitness of an Escherichia coli Mutator Gene

Abstract. *Competition experiments between* Escherichia coli mutT1 *and* mut+ *populations show that the mutator gene confers selective advantage on the strain that carries it. The observed increase in fitness varies, with an average increase in mutator growth rate of 1.4 percent when mutator and wild-type strains are grown together in chemostats.*

Mutator genes are genes that increase the mutation rate of other genes. They have been studied in *Drosophila*, maize, several strains of bacteria, and bacteriophage (*1*). One such gene, the Treffers mutator gene of *Escherichia coli*, increases the mutation rate at least 100-fold at most, if not all, chromosomal loci (*2*, *3*). Because this mutator gene (the *mutT1* allele) preferentially increases transversions of adenine-thymine (AT) to cytosine-guanine (CG) (*2*), the DNA isolated from a *mutT1* population exhibits a growth-dependent increase in the buoyant density of its DNA, and a decrease in the frequency of thymidylate sequences within that DNA (*3*). Both changes are interpreted to mean that the GC content of the *mutT1* DNA has increased, a consequence of a unique mutational pressure (*3*).

We have examined the fitness of a population carrying the *mutT1* allele and report here that *mutT1* populations consistently outgrow coisogenic *mut*+ populations when the two are grown together in a chemostat.

Cultures were grown in phosphate minimal medium (*4*) with limiting glucose as the sole carbon source in chemostats similar in design to those described by Monod (*5*, *6*). Samples were removed from the chemostat at appropriate intervals and assayed for *mut*+ and *mutT1* cells. So that large populations could be screened rapidly for *mutT1* cells, a *mutT1*-induced clone unable to ferment lactose (Lac−) was isolated and purified. Changes in the *mutT1*/*mut*+ ratio in a population were then followed by plating samples on tetrazolium-lactose plates (*7*). If we are to infer the selective advantage of *mutT1* from this experiment, we must establish that the Lac− mutant so used is selectively neutral. In order to show that the Lac− allele was neutral, we grew *mutT1* Lac+ cells with *mutT1* Lac− siblings under our standard conditions (Fig. 1 and Table 1). For a variety of growth conditions in which cell density and generation time were varied, the Lac− mutant chosen [mutated in the permease gene (*6*)] was at a slight disadvantage in the chemostat. It is therefore unlikely that the results that we will attribute to the *mutT1* allele are in fact attributable to the Lac− mutation.

When a *mutT1* Lac− population competes with a *mut*+ Lac+ population in a glucose-limited chemostat the mutator strain consistently outgrows the strain without the mutator (Fig. 2). The increase in the *mutT1* population is characteristic and reproducible and occurs under a variety of cell densities, generation times, and *mutT1*/*mut*+ ratios (Table 1). Similar results have been obtained with strains that were not coisogenic, and under conditions where the culture vessel contained a linearly increasing concentration of casein hydrolysate (chemostat 5, Table 1).

Do these results have a trivial explanation? We have already shown that the Lac− allele used to monitor the mutator level puts the mutator population at a slight selective disadvantage, and therefore the increased fitness of these strains is not a consequence of the Lac− mutation. In fact, it is likely that our estimates of the fitness of the mutator gene are for this reason conservative. In any case, when *mut*+ Lac+ and *mutT1* Lac+ populations are followed in the chemostat and assayed directly for mutator activity (*8*) the conclusion is the same (chemostat 19 of Fig. 2 and Table 1). Further checks on each chemostat showed that the cultures remained free of bacteriophage and colicins, and the *p*H of the culture medium remained constant. Each population was also assayed at the middle and end of each experiment to guarantee that all Lac− colonies were mutator active and all Lac+ colonies were mutator inactive.

In two chemostats we did find mutator active Lac+ colonies at the termination of the experiment. In both cases however, the Lac+ mutator activity was cotransducible with leucine at the same frequency as the *mutT1* allele, showing that the mutator gene present was evidently *mutT1* (*6*, *8*). We therefore assume that in these two cases the Lac− allele had reverted to Lac+ in the *mutT1* background and was selected for, an assumption that is consistent with the data of Fig. 1.

Included in Table 1 is a measure of the fitness of the *mutT1* population estimated from the rate of increase in the *mutT1*/*mut*+ ratio. This value, *K*, is related in a rather complex way to the growth and death rates of the two strains, competition between them for food and space, and cross effects such