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Memory Formation Processes in Weakly Reinforced Learning

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CROWE, S. F., K. T. NG AND M. E. GIBBS. *Memory formation processes in weakly reinforced learning.* PHARMACOL BIOCHEM BEHAV 33(4) 881–887, 1989.—Day-old chicks trained on a single-trail passive avoidance learning task, with varying concentrations of the aversive stimulus (methyl anthranilate), truncated retention functions for low concentrations. The retention function for a 20% v/v dilution of methyl anthranilate in absolute ethanol yielded high retention levels until approximately 40 to 45 minutes following learning. This retention function appears to consist of only the short-term and intermediate (phase A) memory stages of Gibbs and Ng's three-stage model of memory formation, with the short-term stage susceptible to inhibition by monosodium glutamate, and the intermediate stage by ouabain and dinitrophenol. The results suggest that processing of memory into the relatively permanent long-term stage may depend on the strength of the reinforcer in aversive learning.

Day-old chicks Passive avoidance learning Memory stages Monosodium glutamate Ouabain Dinitrophenol

THERE are good reasons for arguing that it may not be beneficial for an organism to store all of its experience in long-term memory. Nor is it always possible to evaluate the significance of a given experience for subsequent adaptive behaviour until feedback, in terms of reinforcement regarding that experience, becomes available to the organism. These conjectures are the principal assumptions underlying the single trace dual-process model of memory formation proposed by Gold and McGaugh (9). They maintain that the time dependence of memory formation processes may reflect the organism's appraisal of the utility of a given training experience, with a view to selecting biologically significant information for permanent storage. The model proposes that learning produces a single memory trace which develops rapidly and decays rapidly unless decay is arrested by nonmemory processes also instigated by the learning experience and related to the significance of the experience. Under this explanation, what appears to be a short-term memory retention function is a special case in which either the experience produces minimal nonspecific influences or those influences are blocked (9).

McGaugh (14) provides some evidence to support the suggestion of the existence of memory traces which do not display the nonspecific influences. McGaugh gave mice a single training trial on an inhibitory avoidance task (step-through passive avoidance), and found that no significant retention was observed for the five- or 30-second intervals after training, although levels of avoidance of the mice did increase at one and 24 hours. He found that he could improve the levels of retention at the later times by giving massed training trials for the first two minutes. The effect of the number of training trials also varied with the time of administration of electroshock (ECS). When ECS was administered two minutes after the first training trial, retention 24 hours later did not vary significantly with the number of training trials. The number of training trials did affect retention, however, when the ECS was administered one hour after training. The retention of animals given two or more training trials was significantly better than that of animals given a single trial. They observed a similar effect with the administration of a second training trial given one hour after original training. A single additional trial given one hour later significantly increased avoidance in tests made the next day. McGaugh concludes from this data that each training trial, whether given early or later in training, initiates memory storage processes that are time-dependent, and that, due to the observed ability of the animals to display trial-to-trial improvement, that learning cannot be based completely on permanent initial memory storage. Modifications of memory can take place for sometime after the initial exposure to training.

McGaugh's observations have also been noted in passive avoidance learning paradigms in chicks. Cherkin (2), using his single-trial passive avoidance task in the day-old chick, noted that training with a concentrated chemical aversant, methyl anthranilate (MeA), resulted in a latency to peck of greater than 10 seconds at periods of 24 and 48 hours after training. Cherkin construed this latency as indicative of long-term memory (LTM). If a one in 400 dilution of the aversant was used (MeA/400) (this is the maximum dilution of the substance in water), the 24-hour latency was equivalent to the baseline level of pecking. If subjects were trained with MeA and subsequently rendered amnesic by a moderate flurothyl treatment, their latency to peck returned to a baseline level. Cherkin (2) notes that a significantly higher number of birds had high latencies if they had been reminded of the initial

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training with a single presentation of MeA/400 26 hours after initial training, as measured 48 hours after training. This effect was not observed if the subjects had been treated with a strong flurothyl treatment initially and were then reminded.

Cherkin (2) interprets these data as consistent with the possibility that the incomplete amnesia induced by the amnesic agent permits consolidation of a *weak* engram which is subthreshold when tested at 24 hours, but which is raised past the threshold by the reminder treatment. The reminder effect induced under these conditions is deemed to be useful in detecting latent memory encoded in the subthreshold engram. There is some support in the literature for Cherkin's contention that the reminder effect can be attributed to an additional learning trial raising the strength of the subthreshold engram (8), rather than to a failure to retrieve memory existent at the time of initial training (13,17). However, Gold *et al.*'s (8) interpretation needs to be evaluated in the light of the conclusions drawn by Miller, Kasprow and Schatchman (16) following their extensive review of retrieval variability following learning. A relevant issue is whether the subsequent trial with diluted MeA is a reminder trial or an instance of relearning.

A more direct approach to the question of subthreshold engrams has been reported by Gibbs (3), using a variation of the Cherkin task (4). Gibbs and her associates noted that the early posttraining time-course of avoidance of the previously aversive lure for chicks trained with MeA/400 closely resembled that observed with the concentrated aversant (5). Memory was retrievable at 10 minutes following training, a transient retention deficit was observed at 15 minutes after training, then the avoidance returned and remained high until 40 minutes after training. It is at this point that the two levels of the training experience diverged. Avoidance in the concentrated aversant trained birds remained high until 50 minutes, showed another transient retention dip at 55 minutes, and then stayed high until at least 24 hours. Avoidance in birds trained with the dilute aversant solution was high until 40 minutes, but then declined from there to a baseline level. The latter timecourse was similar to that observed by Cherkin (1).

In this paper we report studies aimed at exploring memory processing following training of day-old chicks with a "weak" aversant. In particular, we sought to determine the nature of the retention function obtained with such a learning experience and whether the cellular mechanisms that may be involved in memory processing under these conditions are similar to those postulated to underlie normal (i.e., concentrated aversant) memory processing (4).

METHOD

Animals

Day-old black Australorp White Leghorn chicks were obtained from a local hatchery on the morning of each experiment. Approximately 16 subjects were used for each data point, depending on the number successfully trained from an initial subject pool of 20 birds.

Drugs

All drugs were prepared in 154 mM NaCl. Monosodium glutamate (GLU, 4.0 mM, BDH), ouabain (OUA, 0.034 mM, Sigma), saline (SAL, 154 mM) or 2,4-dinitrophenol (DNP, 0.2 mM, Sigma) was administered to the centre of each forebrain in a 10 μl volume by freehand injection using a Hamilton repeating dispenser syringe. A stop on the needle regulated the injection depth to approximately 3 mm in an area of the brain where previous studies have shown amnesic effects from a range of pharmacological agents, including the present ones (4,6). Drugs were injected blind and the codes were not broken until after the data had been scored.

Procedure

The experimental paradigm is essentially that described in Gibbs and Ng (4). Briefly, chicks were pretrained in pairs to peck at a red and a blue glass bead, dipped in water and presented in succession for 10 seconds each. Following pretraining, a similar red bead to the one used in pretraining was coated with an aversant solution and presented to the chicks for a period of ten seconds. Several different aversant concentrations were used in the study, and these were made up as v/v solutions of methyl anthranilate in absolute ethanol. Chicks pecking at the bead typically show a disgust reaction which includes shaking their heads and wiping their beaks on the floor. The number of pecks per chick in the 10-second test period and the latency to first peck were recorded by an on-line computer via a manual keyboard.

On the retention trials, a dry red and a dry blue bead were presented in succession for 10 seconds each, and the number of pecks and the latency to first peck was recorded for each chick for each bead. The proportion of chicks avoiding the red bead and pecking the blue bead at each learning retention interval and a discrimination ratio for red and blue beads were determined. The discrimination ratio was defined as the number of pecks at the blue bead on the test trial divided by the total number of pecks for each ten second trial at both the red and the blue bead. The discrimination ratio data was used as the primary dependent variable for statistical analysis. All chicks avoiding the blue bead on the test trial were excluded from the final analysis, since the reason for such avoidance is not unequivocally clear in the context of the colour discrimination training paradigm. As there was no training for blue avoidance, such avoidance must occur as a consequence of nonspecific effects of the treatments employed. The number of birds excluded on this basis was generally about 10% for a given training test interval.

RESULTS

Experiment 1: Dose-Response Functions

Independent groups of chicks were trained with various concentrations of methyl anthranilate dissolved in absolute ethanol ranging from 0% v/v (absolute ethanol) to 100% v/v (concentrated methyl anthranilate). Chicks were tested for retention at 180 minutes after the initial training trial. Both the percentage of chicks avoiding the red and pecking the blue bead and the discrimination ratio indicate that retention levels were uniformly low for concentrations of methyl anthranilate up to 25% v/v (see Fig. 1). Concentrations of 33% v/v methyl anthranilate in absolute ethanol and higher produce clear evidence of discriminated memory at 180 minutes.

Simple analysis of variance with unweighted means of the discrimination ratios yielded a significant concentration effect, $F(1,10)=6.59$, $p<0.00$. In fact, the analysis reveals a significant linear trend, $F(1,10)=50.13$, $p<0.00$, and a significant quadratic trend, $F(1,10)=5.9$, $p<0.02$, but no significant cubic trend, $F(1,10)=1.16$, $p=0.28$. The results, therefore, suggest a retention function akin to a negatively accelerated exponential function for the concentration effect.

While the data appear to suggest that there should be a significant cubic trend component rather than a significant quadratic trend component (see Fig. 1), the failure to obtain this may possibly be due to the relatively few data points at widely varying intervals in the latter part of the function. Overall, the results suggest that concentrations of less than 33% v/v methyl anthran-

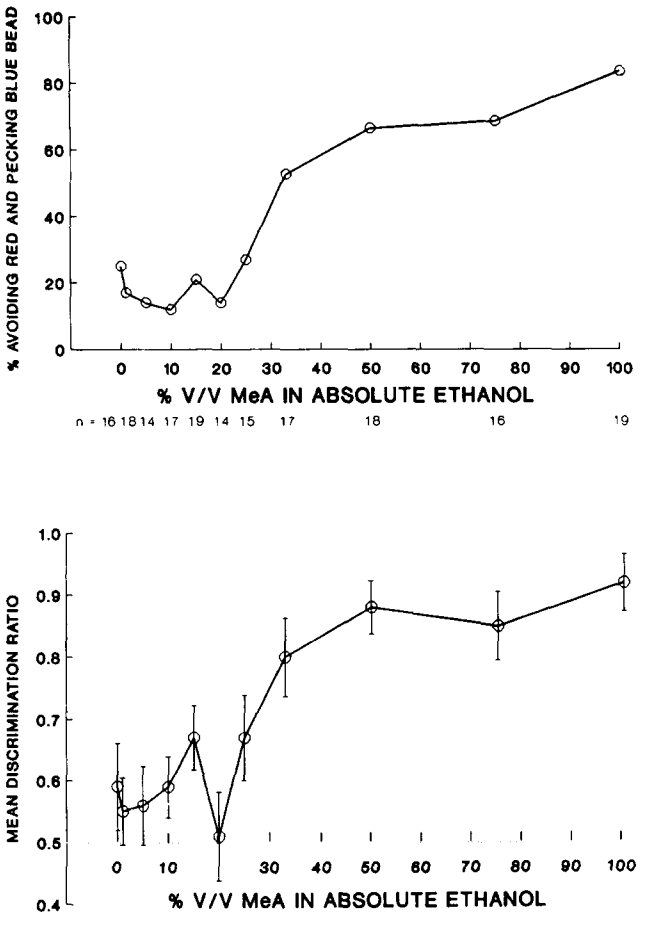

FIG. 1. Percentage of chicks showing discrimination memory (top) and mean discrimination ratios (bottom) measured 180 minutes after training with various dilutions of MeA. Using the technique of planned comparisons of proportions (18), pairwise differences in proportions (P) of chicks showing discrimination memory would be significant at $\alpha = 0.05$, if $(Pi - Pj) \geq 0.33$.

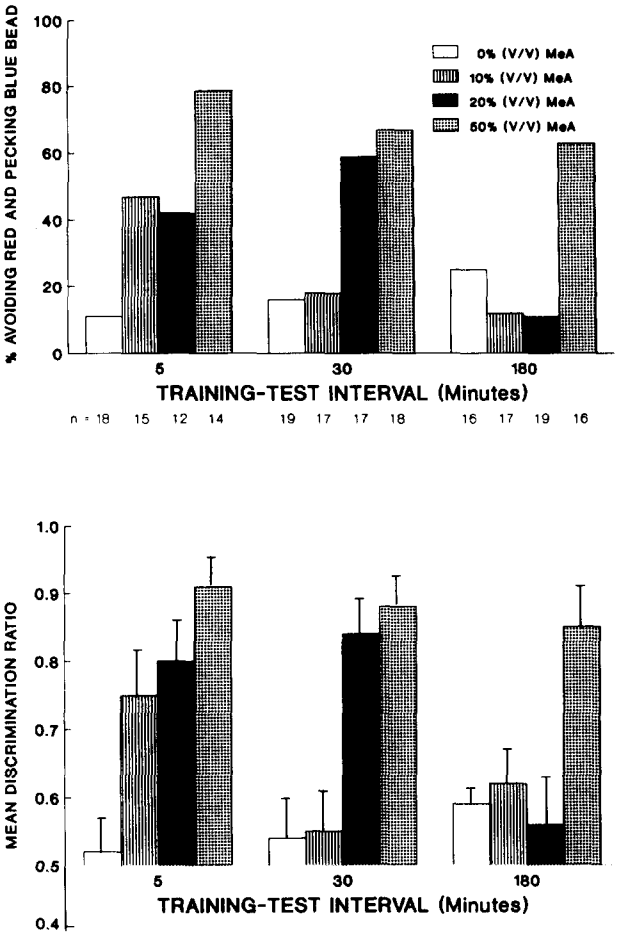

FIG. 2. Retention levels of chicks tested at 5, 30 and 180 minutes following training with various concentrations of MeA. Planned comparisons of proportions would yield significant ($\alpha = 0.05$) pairwise differences in proportions avoiding the red and pecking the blue bead if $(Pi - Pj) \geq 0.33$.

ilate produce no evidence of memory as measured at 180 minutes after initial training.

In order to determine whether concentrations of methyl anthranilate which do not produce evidence of memory at 180 minutes after learning yield evidence of memory at earlier times, independent groups of chicks were trained with 0, 10, 20 and 50% v/v methyl anthranilate in absolute ethanol and tested at five and 30 minutes after training. The results (see Fig. 2) indicate that all concentrations of methyl anthranilate other than 0% v/v yielded evidence of memory five minutes after learning, although levels appear highest for 50% methyl anthranilate in ethanol. At 30 minutes after learning, the same effects are noted but 10% v/v yields a much lower level of retention than that observed with the 20% and 50% concentrations. These contrast with the 180-minute retention time, when as indicated earlier, only a concentration of at least 50% methyl anthranilate yielded evidence of memory.

A two-way ANOVA with unweighted means yielded a significant concentration effect, $F(3,205) = 18.29$, $p < 0.00$, and a significant interval by concentration interaction, $F(6,205) = 2.87$, $p < 0.01$, but no significant intervals main effect, $F(2,205) = 1.83$, $p = 0.16$. Post hoc Newman-Keuls tests confirm that at five minutes after learning, 20 and 50% concentrations of methyl anthranilate yielded significantly higher levels of retention than 10% or 0% v/v methyl anthranilate in absolute ethanol. A similar result was obtained at 30 minutes after learning, while at 180 minutes after learning the retention levels of chicks trained with 50% methyl anthranilate were significantly different from that of chicks trained with any of the other concentrations.

Overall the findings from Experiment I suggest that, while training with at least 50% methyl anthranilate produces long-term memory, training with 20% methyl anthranilate produces evidence of memory only for a short period after learning. The findings are equivocal with lower concentrations of methyl anthranilate; indeed absolute ethanol does not appear to be effective at any time after training.

Experiment II: Retention Functions for Varying Concentrations of Aversant

Since 20% methyl anthranilate appeared to yield memory for a short period after training, it was of interest to determine the retention function with this concentration. Different groups of chicks were trained with either 100% or 20% v/v methyl anthranilate in absolute ethanol and retention tested at various times

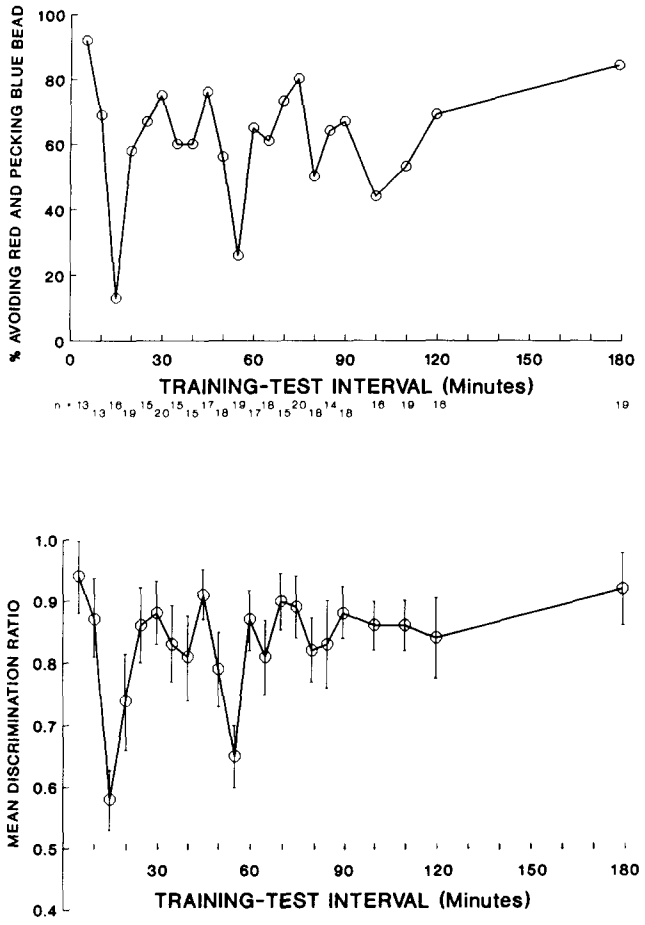

FIG. 3. Retention levels measured by percentage discrimination memory (top) and discrimination ratio (bottom) at various times after training with 100% concentration of MeA. Pairwise differences (Pi − Pj) in proportion of chicks showing discrimination memory would be significant at $\alpha = 0.05$, if they are greater than or equal to 0.33.

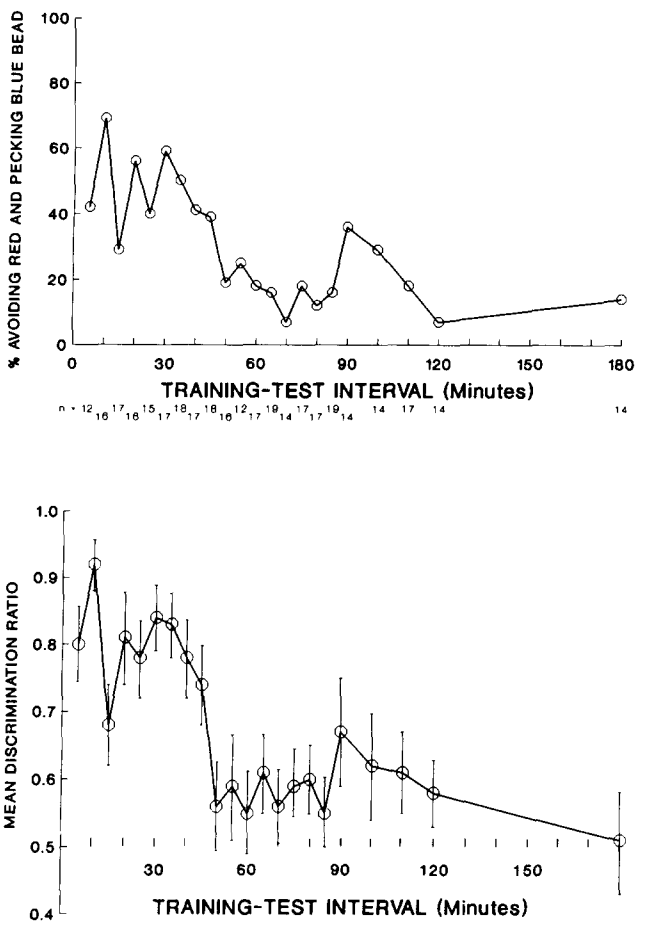

FIG. 4. Retention levels measured by percentage discrimination memory (top) and discrimination ratio (bottom) at various times after training with 20% dilution of MeA in absolute ethanol. Pairwise differences (Pi − Pj) in proportion of chicks showing discrimination memory would be significant at $\alpha = 0.05$, if they are greater than or equal to 0.32.

between five and 180 minutes after learning.

The retention function for 100% methyl anthranilate based on percentage of chicks avoiding the red but pecking the blue bead is comparable to that observed in previous studies [Fig. 3, top; cf. (5)], with transient retention deficits again observed at 15 and 55 minutes after learning. The retention function using discrimination ratios is similar (Fig. 3, bottom). With 20% methyl anthranilate, however, retention levels are relatively high between five and 35 minutes after learning and decline progressively thereafter. After 50 minutes following learning, retention levels are uniformly low (Fig. 4). It may also be noted that, between 5 and 35 minutes after training, retention levels for 20% methyl anthranilate are somewhat lower than those observed with 100% methyl anthranilate. Again, a transient retention deficit is observed at 15 minutes after training, although this is not particularly evident when measured by the discrimination ratio.

A two-way ANOVA [concentration (2) by interval (22)] yielded significant main effects for concentration, $F(1,675) = 83.94$, $p<0.00$, and interval, $F(21,675) = 3.41$, $p<0.00$, and a significant concentration by interval effect, $F(21,675) = 3.10$, $p<0.00$. A simple main effects analysis between concentrations within each training-test interval showed significant differences at all training-test intervals after 40 minutes following initial training, except for the 55-minute training-test interval when no significant differences were observed for the two concentrations of methyl anthranilate.

Within the framework of the Gibbs and Ng three-stage model (5), it would appear that training with a 20% concentration of methyl anthranilate yielded evidence of a short- (STM) and an intermediate- (ITM) term memory stage, but not a long-term memory (LTM) stage.

Experiment III: Effects of Monosodium Glutamate and Ouabain on Memory for the Weak Aversant Task

To the extent that Gibbs and Ng (4) have suggested specific cellular processes involved in the early stages of memory formation, it is important to determine whether the memory observed with 20% methyl anthranilate shortly after training involves similar cellular processes as those observed with concentrated aversant.

Independent groups of chicks were trained with 20% v/v methyl anthranilate in absolute ethanol. Chicks were administered

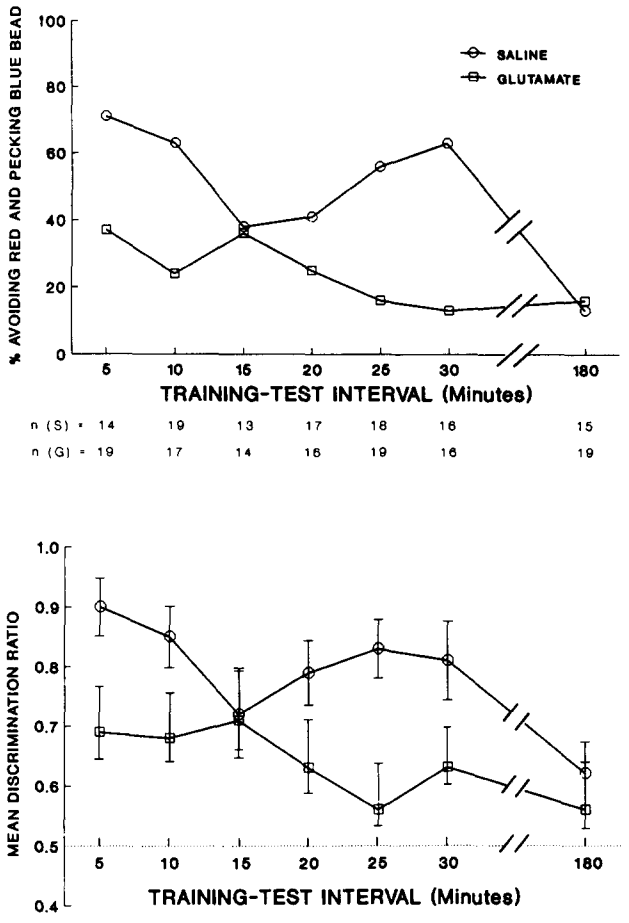

FIG. 5. Chicks trained with 20% MeA were given intracranial monosodium glutamate or saline 5 minutes before training and tested for retention at various times after training. Pairwise differences (Pi − Pj) in proportion of chicks showing discrimination memory (top) would be significant at $\alpha = 0.05$, if they are greater than or equal to 0.33.

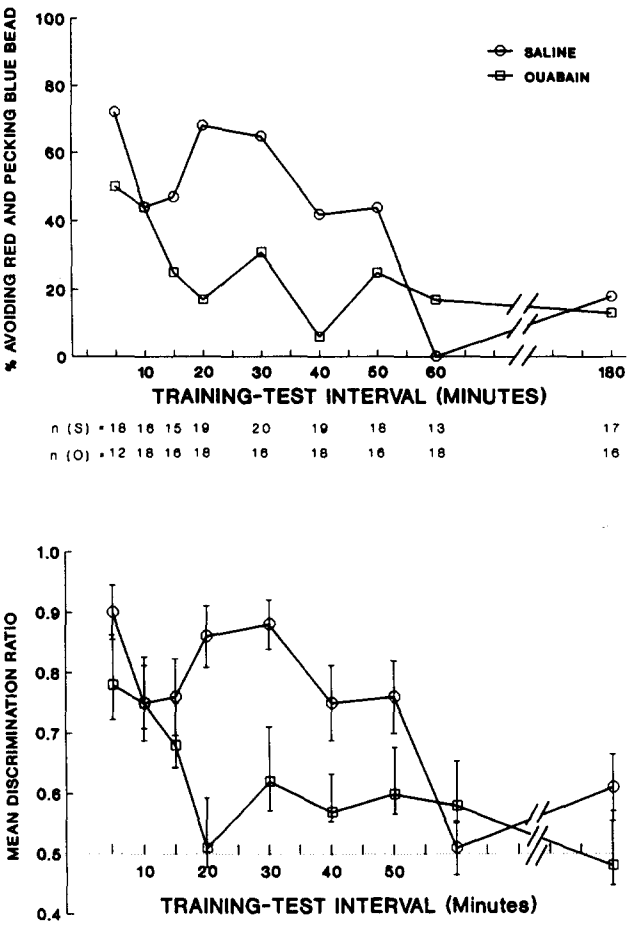

FIG. 6. Effect of ouabain, administered intracranially 5 minutes before training with 20% MeA, on retention levels at various times after training. Pairwise comparisons of differences in proportion of chicks showing discrimination memory (top figure) would be significant ($\alpha = 0.05$), if (Pi − Pj) ≥ 0.32.

intracranially monosodium glutamate, ouabain or saline five minutes before training and were tested at various times after training.

If the short-term memory stage observed with the 20% methyl anthranilate involves the same processes as that postulated for concentrated methyl anthranilate (4), it would be expected that glutamate would produce a significant reduction in level of retention compared with saline at each training-test interval except at 15 and 180 minutes after learning. The results appear to confirm these expectations (see Fig. 5). Planned contrasts on mean differences at each training-test interval yielded significant differences at all time intervals tested except 15, 20 and 180 minutes after training, for a type one error rate of $\alpha = 0.05$. At 20 minutes after training, the F(1,218) value was in fact 3.63, with a p of 0.058.

In the case of ouabain, it would be expected that no differences in retention level between glutamate- and saline-treated chicks should be observed before 15 minutes after learning, if the same cellular processes underly formation of ITM as has been suggested for chicks trained with concentrated methyl anthranilate (4). The results (Fig. 6) show relatively high retention levels in ouabain-treated chicks before 10 minutes following learning with a sharp decline thereafter. In contrast, saline-treated chicks did not show a substantial drop in retention levels until after 50 minutes following learning, although some decline was observed after 30 minutes.

Planned contrasts on difference between means of the discrimination ratios at each training-test interval ($\alpha = 0.05$) yielded significant differences between ouabain- and saline-treated chicks at 5, 20, 30, and 40 minutes postlearning but not at other times. The significant difference observed at five minutes after training is surprising, $F(1,286) = 4.25$, $p = 0.04$. However, the retention level at this time for ouabain-treated chicks is clearly considerably higher than those observed at 20 minutes (Fig. 6 bottom). It may also be noted that at 50 minutes after training the F(1,286) value is 3.80, $p = 0.052$, while at 60 minutes after learning, $F(1,286) = 0.05$, $p = 0.82$.

Overall, the results of this experiment are consistent with the view that the memory observed within the first 40 minutes or so after training with diluted methyl anthranilate may reflect the same stages of memory observed with concentrated methyl anthranilate and is subject to inhibition by the same drugs (4).

Experiment IV: Effects of 2,4-Dinitrophenol

It has been suggested that the ITM stage isolated by Gibbs and

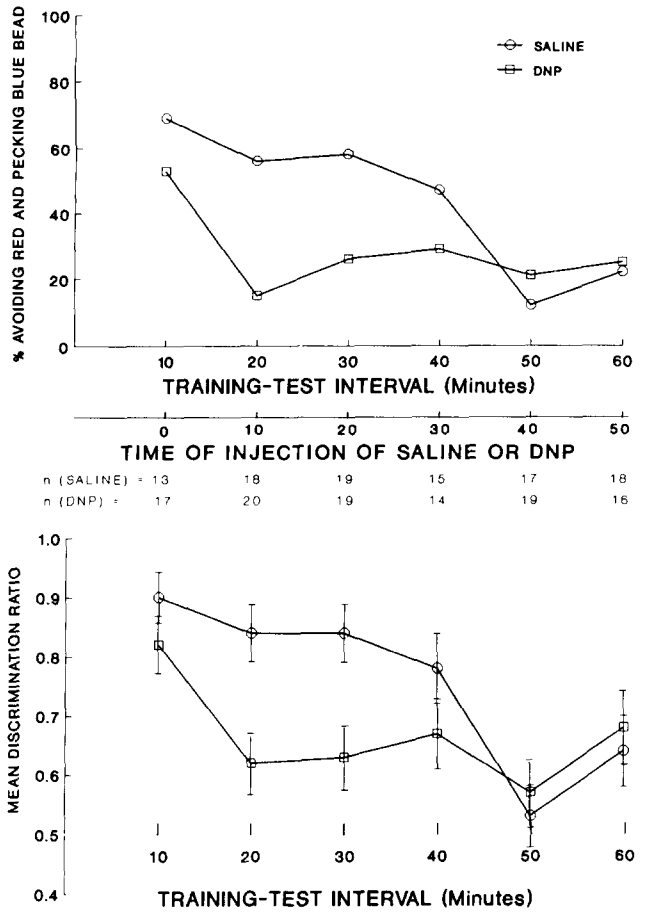

FIG. 7. Effect of DNP, administered intracranially at various times after training with 20% MeA, on retention levels measured 10 minutes after DNP injections. Pairwise differences in the top figure would be significant at $\alpha = 0.05$, if the differences are equal to or greater than 0.32

Ng (6) using concentrated methyl anthranilate may consist of two phases: a phase A susceptible to inhibition by the metabolic inhibitor, 2,4-dinitrophenol (DNP), and lasting between 20 and 30 minutes after training, followed by a phase B, not susceptible to inhibition by DNP and lasting between 35 and 55 minutes after training. In order to establish whether the memory observed between 20 and 40 minutes after training with diluted methyl anthranilate consists of the same phases as postulated for training with concentrated methyl anthranilate, DNP or saline was administered intracranially between 0 and 50 minutes after training and retention was tested 10 minutes after drug administration.

An unweighted means ANOVA [drug (2) by interval (6)] yielded significant drugs, $F(1,93) = 8.36$, $p<0.00$, and interval, $F(5,193) = 6.67$, $p<0.00$, main effect as well as a significant drug by interval interaction, $F(5,93) = 2.32$, $p<0.05$. Simple main effects analysis comparing drug effects within each interval yielded significant differences between drug and saline groups when tested at 20 and 30 minutes following training. Thus, DNP significantly depressed retention levels at 20 and 30 minutes following learning (see Fig. 7). Although the difference at 40 minutes is not significant, $F(1,193) = 1.95$, $p = 0.17$, it is reasonable to note that at this time retention levels for saline-treated chicks were already on the decline. The results suggest, therefore, that all of intermediate memory observed with weakly reinforced training represents Phase A of the ITM stage.

DISCUSSION

The results of the present series of experiments broadly confirm the findings of Cherkin (1,2) regarding the effects of using a diluted aversant in a single trial passive avoidance task in day-old chicks. Varying the concentration of the aversant gives rise not only to differences in the absolute level of retention observed after learning, but also to a failure of some aversant dilutions to yield LTM. In particular, a concentration of 20% methyl anthranilate in absolute ethanol yielded clear evidence of memory for the task for at least up to 40 minutes after learning, albeit possibly at a slightly reduced retention level compared to training with concentrated aversant. What is clear is that beyond this time there is no evidence of memory. The results are similar to those observed by Gibbs and Ng working with a one in 400 dilution of methyl anthranilate in water (3). The results suggest therefore that: 1) the strength of memory established may depend upon the strength of the reinforcement used in the training, a finding that is well known in the learning literature (10), and 2) that at certain low levels of reinforcement, consolidation of memory may not take place, despite evidence of memory for a short period after training.

What is important about the present results is that they show that training with a 20% concentration of methyl anthranilate gives rise to the Gibbs and Ng (4) postulated stages of short- and intermediate-term memory with virtually the same temporal characteristics (3,5). The short-term stage, which lasts for approximately 10 minutes after learning, is susceptible to inhibition by monosodium glutamate, a known depolarizing agent, with an immediate loss of memory following learning. The intermediate stage appears to be present between 20 and at least 40 minutes after learning. The cardiac glycoside, ouabain, a known inhibitor of Na^+/K^+ ATPase activity, produces amnesia after 15 minutes following learning. It would not be unreasonable therefore to conclude that the cellular processes underlying the short and intermediate memory stages observed with the "weak" memory trace [cf. (2)] are the same as those obtained with the concentrated aversant. Of particular interest is the finding that such intermediate memory as is available following the weakly-reinforced training experience appears to be susceptible to inhibition by the metabolic inhibitor DNP. In the terms of the Gibbs and Ng model (4,6), it would appear that the entire intermediate memory stage following weakly-reinforced learning consists of phase A of the ITM arising from a strongly reinforced training task. There appears to be no evidence of Gibbs and Ng's Phase B of ITM. This finding is significant in view of the suggestion that consolidation of a learning experience into LTM may depend on a successful transition from phase A to phase B of ITM (6), and that a "triggering" mechanism for LTM consolidation operates during the time of this transition. The nature of this postulated mechanism is as yet unknown, as is also the case with the nature of the cellular processes underlying the maintenance of phase A and phase B of ITM. Nonetheless, the apparent absence of phase B of ITM following weakly-reinforced learning and the subsequent absence of LTM consolidation is consistent with the above views.

It has been suggested that consolidation of LTM may rely on the "biological significance" of the learning experience (9, 11, 12). Operationally, the concept of "biological significance" may be related to the strength of the aversant in an aversant learning task and may express itself biologically in arousal states associated with the release of hormones (15). Just how hormones modulate this consolidation process is as yet unknown although tentative suggestions have been made within the three-stage model of memory formation which forms the framework for the present study (7). It is possible that hormones act to either maintain phase

A of ITM and/or initiate the transition from phase A to phase B. In the case of the latter, hormones may therefore play a role in initiating the proposed triggering mechanism for LTM formation. Indeed, the reminder effects reported by Cherkin (2) and others [e.g., (8)] may be associated with possible cumulative effects of arousal. We are currently investigating the effects of retraining and of postlearning administration of hormones with a weakly reinforced learning task.

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