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Simple and Conditional Discriminations in Rats: The Effects of Delays and Scopolamine

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In simple delayed discriminations (DD), the reinforcer depends on information available from the sample stimulus, whereas in delayed conditional discriminations (DCD), the reinforcer depends on information jointly available from the sample and the test stimuli. The present experiments compared performance in rats trained in DD and DCD (a) during acquisition over training sessions, and as a function of increasing (b) delay in the memory interval and (c) dosage with the anticholinergic scopolamine. In this work, clicks and tones served as sample stimuli, and bright and dim overhead lights served as test stimuli for lever pressing for food during the test stimulus. The DD task was acquired more rapidly and was more resistant to the effects of delay between the sample and test stimuli. These results replicate previous work with pigeons. In addition, the dose-performance function of scopolamine differed between DD and DCD. Performance declined as a linear function of drug dose in the DCD group. In contrast, performance declined abruptly as a stepwise function of drug dose in the DD group. These latter results suggest that the cholinergic system may be utilized differently during DD and DCD.

Delayed discriminations are in common use in the study of animal memory. Hunter's (1919, p. 38) description of the essential characteristics of the delayed discrimination task remains definitive: (a) The stimuli must be presented to the animal then withdrawn during the interval of delay. (b) There must be no stimulus left in the apparatus which could determine the animal's subsequent response. (c) The animal's delayed response is used as a measure of memory performance. Today, we recognize two versions of the procedure: simple delayed discrimination and delayed conditional discrimination. In both, a sample stimulus is presented and withdrawn, then the animal must perform some discriminative response

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for reward during the test. In *delayed simple discriminations* (DD), reinforcement depends on the sample stimulus, but the test stimulus is irrelevant. In *delayed conditional discriminations* (DCD), reinforcement depends (is conditional) on both the sample and the test stimulus; delayed matching to sample is a commonly studied example.

Honig and Wasserman (1981) compared DD and DCD in pigeons. Sample stimuli (colors) and test stimuli (white lines) were presented on the response key, separated by a memory interval. Pecking the response key during the test stimulus on positive trials produced food (the reinforcer), whereas negative trials ended with a blackout. In DD, trials beginning with one sample color (e.g., red) were positive and trials beginning with another sample color (e.g., green) were negative; the test stimulus (e.g., vertical or horizontal line) had no bearing on trial outcome. In DCD, two sample–test sequences (e.g., red–horizontal and green–vertical) were positive, and two (e.g., red– vertical and green–horizontal) were negative; here, the color of the sample and the orientation of the test line jointly determined trial outcome. After initial acquisition, memory intervals ranging from 1 to 30 s were introduced. The results were clear: (a) Pigeons acquired DD more rapidly than DCD, and (b) over delays DD was better remembered than DCD.

The laboratory rat has been the most commonly used species in behavioral research and in neuropharmacological extensions of that research. Yet, few studies have used rats in delayed discriminations of the sort studied by Honig and Wasserman. In view of the now well-known differences between species in the expression of behavioral phenomena, one cannot simply assume that mammalian cognition operates the same way as avian cognition in DD and DCD.

In an initial study of DCD in rats, Wallace, Steinert, Scobie, and Spear (1980) examined memory for auditory (white noise) and visual (diffuse chamber illumination) sample stimuli with simultaneous and successive testing procedures. The results showed that (a) rats could acquire accurate symbolic and standard matching to sample performance at a 0-s delay; (b) like pigeons, rats performed increasingly less accurately as the memory interval increased; (c) rats performed more accurately at the longer delays when an auditory, instead of a visual, sample preceded the delay. This work and its extension and replication by Cohen, Escott, and Ricciardi (1984) provide useful parameters for the comparison of DD and DCD in rats.

Memory performance in delayed discriminations is thought to involve interaction between two memory systems. One system, termed "reference memory" by Honig (1978), is concerned with the rules and concepts needed to describe the environment. The other system, termed "working memory" by Honig (1978), is concerned with recently perceived events and currently expected events. In delayed discriminations, information

about the task (e.g., its rules and context) is represented in reference memory. Under stable training conditions, these memories remain constant from trial to trial. In contrast, information specific to an ongoing trial (e.g., memories of the sample and expectancies regarding trial outcome) is represented in working memory. Even under stable training conditions, these working memories must change from trial to trial. Delayed discriminations make intensive use of working memory, but after initial acquisition, the reference memory representation of the task may change little. Thus, delayed discriminations may be useful in the study of working memory in animals.

Cholinergic neurons may play a critical role in the expression of cognition in working memory (e.g., Bartus & Johnson, 1976; Wirsching, Beninger, Jhamandas, Boegman, & El-Defrawy, 1984). For example, Bartus and Johnson (1976) studied delayed discrimination in monkeys under two doses of the anticholinergic scopolamine. The greatest impairments occurred at the longest memory interval delays, and the highest dose. Bartus and Johnson concluded that acetylcholine may be intimately involved in the expression of primate working memory.

The purpose of the present experiments was twofold: (a) to replicate and extend the comparison of DD and DCD using rats, and (b) to compare the effects of cholinergic blockade by scopolamine on the two discriminations.

EXPERIMENT 1

This study provides a systematic replication in rats of Honig and Wasserman's (1981) comparison of the acquisition of DD and DCD. Differences in salience between auditory and visual stimuli as samples for rats in DCD (Wallace *et al.*, 1980; Cohen *et al.*, 1984) present methodological problems in comparisons between DD and DCD. For example, counterbalancing sample stimuli could introduce complex interactions with effects resulting from differences between the two delayed discrimination tasks. Accordingly, two auditory stimuli of approximately equal discriminability served as samples in the present experiments.

Method

Subjects

Twenty-four experimentally naive male Wistar rats, obtained from Charles River Canada were about 90 days old at the beginning of the experiment. They were housed individually with free access to water. During preliminary training, the rats were maintained at about 80 percent of their free-feeding weight on a diet of Purina Rat Chow. During Experiments 1–3, the rats were fed to 85% of their free-feeding weight, adjusted weekly according to growth charts available from the breeder.

Apparatus

Six identical operant conditioning chambers (29 × 23 × 19 cm high) were housed within separate sound-attenuating chests. Two side walls and the ceiling of each chamber were Plexiglas. The rear wall and the intelligence panels were 3-mm sheet aluminum. The dipper, located in a recessed area at the center of the intelligence panel presented 0.01 mL of Mazola brand corn oil for 2.5 s as the reinforcer. A stainless steel retractable lever (Coulbourn Instruments E23-05), requiring about 15 N force for operation, was mounted 6 cm above a grid floor about 7 cm to the right of the dipper opening. The two auditory stimuli were a 1000-Hz tone and repeated clicks. The tone was produced by a Grundig generator (model TG-4) and presented via a 12-cm speaker located behind a perforated area in the center of the ceiling. Repeated clicks, six beats per second controlled by a potentiometer, were produced by a unijunction transistor and presented via a 5-cm speaker located at grid level opposite the dipper. Sound levels at the lever, as measured by a sound level meter (Radio Shack 33-1028) were 78 dB for the tone and 79 dB for the click against a background level of 68 dB provided by white masking noise and ventilation fans. A dim house light and a bright overhead light were used as test stimuli. The house light was a 0.5-W General Electric 1819 lamp mounted on the back of the intelligence panel. The bright light was a frosted General Electric 40-W lamp mounted 10 cm above the Plexiglas ceiling and 2 cm in front of the intelligence panel. The operant conditioning chambers were interfaced to a Digital Equipment Corporation PDP 8/e minicomputer located in another room. Procedures and data collection were programmed with the SKED system (State Systems Inc., Kalamazoo, Michigan).

Procedure

Preliminary training. The lever was inserted for 30-s trials and withdrawn during 20-s intertrial intervals. A 20-s intertrial interval remained in effect throughout the experiment. On each trial the dipper was presented for 5 s immediately after the lever was retracted. Once the rats were pressing the lever reliably, they were trained with fixed ratios of 1–20 presses for 5 days, followed by variable intervals of 15 s for 5–7 days. Finally, 15-s variable interval trials were correlated with a bright overhead light, tone, or clicks, presented equally often and at random for 5 days.

Discrimination training. Each trial progressed through the following steps: intertrial interval, sample presentation, memory interval, test stimulus presentation, and trial outcome.

During the 20-s intertrial interval and 7.5-s sample stimulus, the lever was retracted and the houselight continuously illuminated. On half the

trials the tone was presented and on the remaining trials the click was presented as the sample.

The memory interval followed offset of the sample; delay values of 0.01 (nominally 0 s), 1, 2.5, 5, and 10 s were randomly scheduled, each on one-fifth of the trials. The houselight remained on and the lever retracted.

The test stimulus was either the house light or the bright light. The lever was inserted for 7.5 s, during which responses were recorded. On negative trials, the lever was retracted after 7.5 s and either lamp turned off. On positive trials, the first press after 7.5 s retracted the lever, turned off either lamp, and presented the dipper for 2.5 s. If a further 5 s elapsed without a lever press (a limited hold), then the lever was retracted without presenting the reinforcer.

Discrimination groups. There were four types of training trials: Tone-bright light, tone-house light, click-bright light, and click-house light. These were presented equally often during each session. Assignment of 12 rats each to the DD and DCD groups was random. In the DD group, for 6 rats all trials beginning with tone were positive and all trials beginning with click were negative, without regard to the test stimulus; for the other rats these contingencies were reversed. In the DCD group, half of the trials beginning with each auditory stimulus were positive and half were negative, depending on the test stimulus. For 6 rats, the click-house light and tone-bright light sequences were positive and the click-bright light and tone-house light sequences were negative; for the other rats, the opposite contingencies were in effect.

The four trial types were presented once at each of the five delays, in random order, in blocks of 20 trials, for a total of 100 trials per session. Discrimination training continued, Monday to Friday, for 56 sessions.

Analysis of discrimination performance. Discrimination ratios were calculated to assess performance during the test stimulus. An example for six rats in the DCD group follows: (a) Responses during the bright test light following the clicker sample (positive trials) were divided by responses during the bright test light following both the clicker and tone samples (postive and negative trials). (b) Responses during the house light following the tone sample (positive trials) were divided by responses during the house light following both the clicker and tone samples. (c) The two ratios were averaged to provide an overall ratio. The ratio normally ranged from 0.50, indicating no discrimination, to 1.00, indicating perfect discrimination.

Results

Higher discrimination ratios, that is, improved discrimination of negative sample–test sequences, can result from either decreased rates of responding on negative trials or increased rates of responding on positive trials.

Analysis of variance of the response rates on positive trials found no reliable effects of groups, delay intervals, blocks of sessions, or their interactions, Fs(13, 286) < 1.54, ps > .05. Thus, higher discrimination ratios result mainly from decreased responding on negative trials. Mean response rates per minute were 40.1 and 46.6 for the DD and DCD groups, respectively.

Figure 1 presents mean discrimination ratios for 14 four-session blocks of training for the DD and the DCD groups. Analysis of variance found reliable effects for Discrimination Group, $F(1, 22) = 178.64$, $p < .01$; Delay Interval, $F(4, 88) = 26.07$, $p < .01$; Blocks, $F(13, 286) = 62.75$, $p < .01$; Delay Interval × Blocks, $F(52, 1144) = 1.35$, $p < .05$; and Discrimination Group × Blocks, $F(13, 286) = 20.20$, $p < .01$. These analyses and inspection of Fig. 1 suggest that (a) the DD group acquired the discrimination more rapidly and to a higher performance level, and (b) increasing delay reduced performance in both discrimination groups. Also, both groups responded less on negative trials than on positive trials (i.e., with discrimination ratios above 0.50), during Block 14 of training (ts > 6.74, $p < .01$), indicating reliable acquisition of delayed discriminative performance.

EXPERIMENT 2

Honig and Wasserman (1981) found better discriminative performance after long delays in DD than in DCD. One purpose of the present study was to investigate the effect of increasing the memory interval between the sample and the test stimuli on DD and DCD in rats. In pilot work, 1-, 10-, and 20-s memory interval delays yielded intermediate performance that declined as a function of increasing delay in the DCD group, but yielded excellent performance unaffected by delay in the DD group. In contrast, 5-, 20-, and 40-s delays yielded poor performance unaffected by delay in the DCD group, but yielded high intermediate performance that declined as a function of delay in the DD group. Accordingly, a different but overlapping series of delays was chosen for each group, namely, 1, 10, and 20 s for the DCD and 5, 20, and 40 s for the DD group.

Scopolamine is a muscarinic cholinergic receptor blocker with both central and peripheral actions (Gilman, Goodman, & Gilman, 1980). Numerous previous studies (e.g., Eckerman, Gordon, Edwards, MacPhail, & Gage, 1980, Meyers, 1965; Meyers & Domino, 1964) have shown that the memorial effects of scopolamine are attributable to its central action. A second purpose of the present study was to investigate the effects of scopolamine on performance in DD and DCD.

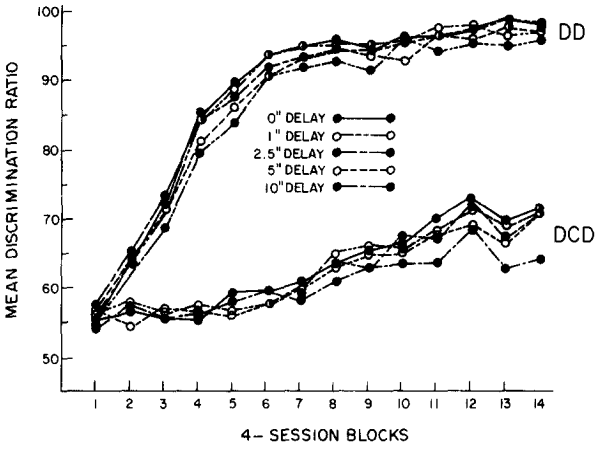

FIG. 1. Discrimination ratios for the acquisition of delayed simple (DD) and conditional (DCD) discriminations in Experiment 1.

Methods

Subjects and Apparatus

The rats, now between 190 and 240 days old, and the apparatus were those used in Experiment 1.

Drugs

Scopolamine hydrobromide (Sigma Laboratories) was mixed fresh each day in distilled water at doses of 0.1, 0.5, and 1.0 mg/kg. In the control condition, the dose was 1 mL/kg of 0.9% saline. All doses were injected intraperitoneally in a volume of 1 mL/kg delivered 30 min before testing.

Procedure

Preliminary training. Training with the same sequence of within trial events as in Experiment 1 continued for 12 sessions. However, the memory intervals in the DCD group were 1, 10, and 20 s, and the memory intervals in the DD group were 5, 20, and 40 s. Within each session, the four trial types were presented once at each of the three delays, in random order, in eight blocks of trials for a total of 96 trials.

The DD and DCD groups continued in the above procedure without change except for the addition of drug testing. The rats were tested in an experimental design composed of four 3-session drug test blocks separated by nondrug sessions devoted to the recovery of baseline. Nondrug sessions continued for a minimum of 3 consecutive days or until performance did not differ significantly from predrug baseline.

During the first block of test sessions the rats had saline as a control for nonspecific effects of the injection procedure. During each of the

remaining three blocks, one dose level of scopolamine (0.1, 0.5, or 1.0 mg/kg) was injected. Rats were randomly assigned to one of six subgroups; each was administered the three doses in a different order; thus, two rats in each discrimination group received each order.

Results and Discussion

Mean rates of test-stimulus responses per minute on positive trials for the DD and DCD groups were 48.1 and 47.9, respectively. Analysis of variance found no reliable effects for groups, delays, or drug doses, or their interactions, Fs < 1. As in Experiment 1, variation in the discrimination ratio appears to result from changes in the rate of responding on negative trials.

Figure 2 presents discrimination ratios as a function of scopolamine dose and delay interval for the DD (left panel) and the DCD (right panel) groups. The results were averaged for 3-day test blocks. An analysis of variance conducted on the results at the common 20-s delay found reliable effects for Discrimination Group, $F(1, 22) = 74.35$, $p < .01$; Drug Dose, $F(3, 66) = 29.86$, $p < .01$; and Discrimination Group × Drug Dose, $F = 7.72$, $p < .01$. Trend analysis of the dose-response function at the 20-s delay in the two groups showed that performance declined as a linear, $F(1, 11) = 106.65$, $p < .01$, quadratic, $F(1, 11) = 8.28$, $p < .02$, and cubic function, $F(1, 11) = 11.39$, $p < .01$, of dose in the DD group, and as a linear function, $F(1, 11) = 10.73$, $p < .01$, of drug dose in the

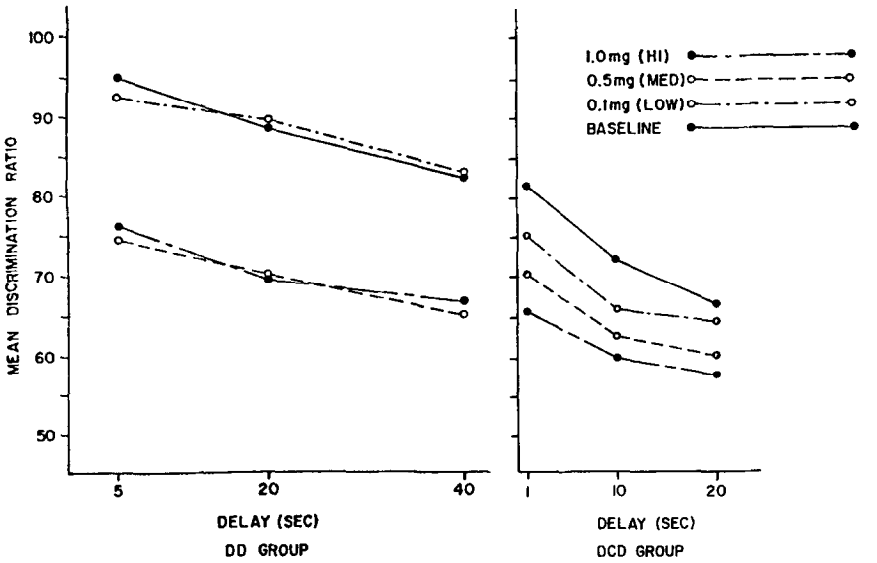

FIG. 2. Discrimination ratios as function of delay and scopolamine dose for delayed simple (DD) and conditional (DCD) discriminations in Experiment 2.

DCD group. (These and subsequent trend analyses used orthogonal weights corrected for unequal intervals.) Also, performance differed between the DD and DCD groups in the baseline (control) condition, $F(1, 22) = 12.65$, $p < .01$. These analyses show that at the 20-s delay, the scopolamine dose-response functions of the DD and DCD groups differed reliably.

Separate analyses were conducted for the DD and DCD groups using the results for all delays. In the DD group, analysis of variance found reliable effects for Drug Dose, $F(3, 33) = 33.57$, $p < .01$, and Delay Interval, $F(2, 22) = 53.31$, $p < .01$. Inspection of the left panel of Fig. 2 suggests (a) gradual decline in performance with increasing delay and (b) an abrupt, stepwise decline with increasing drug dose at each delay. Trend analyses support these impressions. Analysis of the delay effect found a linear trend, $F(1, 11) = 74.26$, $p < .01$. Analysis of the drug dose effect found linear, $F(1, 11) = 81.16$, $p < .01$, quadratic, $F(1, 11) = 12.48$, $p < .01$, and cubic trends, $F(1, 11) = 10.19$, $p < .01$.

In the DCD group, analysis of variance found reliable effects for Drug Dose, $F(3, 33) = 10.16$, $p < .01$, and Delay Interval $F(2, 22) = 33.44$, $p < .01$. Inspection of the right panel of Fig. 2 suggests (a) a steep, continuous decline in performance with increasing delay and (b) a continuous decline with increasing drug dose at each delay. Trend analyses support these impressions. Analysis of the delay effect found linear, $F(1, 11) = 55.45$, $p < .01$, and quadratic, $F(1, 11) = 8.85$, $p < .01$, trends. Analysis of the drug dose effect found a linear trend, $F(1, 11) = 31.53$, $p < .01$.

In summary, the results showed that both delay and scopolamine differentially impaired performance in the two groups. Performance declined (a) more rapidly with increasing delay in the DCD group and (b) more abruptly with increasing drug dose in the DD group.

DISCUSSION

The results of the present experiments provide further evidence that DD and DCD generate different discriminative performance. (a) The DD group acquired the discrimination more rapidly over sessions and maintained a higher final level of performance throughout Experiments 1 and 2. (b) The DCD group suffered a greater decline in performance with increasing delay, even though it was exposed to shorter memory intervals than the DD group in Experiment 2. Furthermore, extensive pilot work (conducted between Experiments 1 and 2) found near chance performance in the DCD group with the longer delays (i.e., 5, 20, and 40s) used in the DD group in Experiment 2. (c) The effect of scopolamine on discrimminative performance differed dramatically between the two tasks in Experiment 2. In the DCD group, the dose-response function was continuous, with proportional decrements in performance due to 0.1, 0.5, and 1.0 mg/kg doses of scopolamine. In the DD group, on the other

hand, the dose-response function was much less continuous; performance declined abruptly between the 0.1 and 0.5 mg/kg doses, and was very similar at 0 and 0.1 mg/kg and at 0.5 and 1.0 mg/kg. In general, these results obtained with rats are in good agreement with those obtained with pigeons (Honig & Wasserman, 1981). Differences in performance between DD and DCD might arise from a number of sources (e.g., differences in mastery, in delay context, in motivation, or in cognitive requirements) considered below.

Incomplete Mastery

Differences in the final level of discriminative performance attained by the two groups could reflect more complete mastery by the DD group. According to this hypothesis, subsequent differences in the effects of increasing delay and of scopolamine might result mainly from unequal original learning of the two tasks. This implies that further practice should make the performance of the two groups more similar. In support of this implication, as a result of over 30 intervening sessions of pilot work (with increasing delays), discriminative performance improved from the final sessions of Experiment 1 to the pretraining sessions of Experiment 2. Against the hypothesis, performance remained stable over the pretraining sessions and the baseline (nondrug) sessions of Experiment 2. This later finding suggests that performance of the rats in the DCD group was asymptotic prior to the baseline and drug sessions of Experiment 2. Furthermore, studies conducted with pigeons have equated final performance in simple and conditional discriminations and still found greater sensitivity to delay in conditional discriminations (see Honig & Dodd, 1986). Hence, it seems unlikely that incomplete learning accounts for the differential effects of delay and scopolamine on DCD performance.

The Context of Delays

Presenting the 20-s delay in the context of shorter memory intervals in the DCD group and in the context of a longer (40-s) interval in the DD group had the advantage of generating more comparable delay functions in the two groups without changing the nature of the discrimination tasks. Changing the delay context between groups also had a disadvantage. Delay context is known to affect performance at any specific delay (Honig & Wasserman, 1981; Honig & Dodd, 1986). Specifically, performance at the 20-s delay should be poorer in the context of longer delays and better in the context of shorter delays. In Experiment 2, the "context effect" likely minimized differences between the groups, since performance at the 20-s delay was poorer in DCD (where the context included only shorter delays) than in DD (where the context included a longer delay). For this reason, the present work would seem to provide a minimum estimate of the differential effect of delay on the two tasks.

Perceptual, Motivational, or Motor Processes

Scopolamine appears to disrupt several processes important to discriminative performance. (a) Bohdanecky, Jarvik, and Carley (1967) suggested that scopolamine reduced performance in their delayed conditional discrimination task by disrupting perceptual processes, because monkeys performed poorly when the sample and test stimuli were present simultaneously, at near zero delay, and at longer delays. Also, Evans (1975) found that scopolamine reduced performance of a nondelayed visual discrimination. (b) In other work (e.g., Poulos & Hinson, 1984; Stein, 1963), scopolamine affected brain systems that regulate water intake in the rat. (c) It is also possible that scopolamine might affect the motor systems responsible for emission of the lever press response. (d) Bartus and Johnson (1976) reasoned that scopolamine affected their delayed conditional discrimination task by disrupting cognitive (memorial) processes, on the basis of evidence that monkeys performed similarly with and without the drug at zero delay, but much more poorly at long delays with the drug.

Given that scopolamine has so many effects, it is difficult to be certain concerning its locus in the present work. Nonetheless, Experiment 2 does not provide evidence that simple drug effects on perception, motivation, or motor processes account for the differential effect of scopolamine on DD and DCD. Although the rate of lever pressing (on positive trials) is a commonly used measure of food motivation and motor performance, response rate did not vary with drug dose in Experiment 2. Moreover, given the fairly constant rate of responding on positive trials, decreased discriminability (as measured by the discrimination ratio) must result from increased responding on negative trials. Thus, to account for the observed effects, scopolamine would have to increase food motivation or increase motor performance on negative trials. Neither have been reported in the scopolamine literature. A simple scopolamine-induced perceptual deficiency also would appear to be ruled out; the DD and DCD groups were presented the same stimuli, yet their scopolamine dose-performance functions were different. A final possibility is that differences in the cognitive requirements of DD and DCD interact with perceptual processes to produce the observed differential effect of scopolamine. This hypothesis is discussed after contrasting the cognitive requirements of DD and DCD.

Cognitive Requirements

We suggest that the differences observed in the present experiments may be best understood with reference to the cognitive requirements of DD and DCD (Honig & Dodd, 1986; Wasserman, 1986). Thus, it is appropriate to review those requirements here. First, compare the decision rules. The DD decision rule specifies the relationship between the samples

and the availability of the reinforcer; for example, if the sample stimulus was tone, then respond for food during the test. By contrast, the DCD decision rule specifies the relationship between the samples, the test stimuli, and the availability of the reinforcer; for example, if the sample stimulus was tone and the test stimulus bright light (or if the sample stimulus was click and the test stimulus dim light), then respond for food during the test. Second, compare the memory products of the sample. In DD, over the memory interval the animal need only retain a simple instruction or outcome prediction to discriminate positive from negative trials. In DCD, over the memory interval the animal may (according to current theorizing) either (a) retain a retrospective perceptual memory of the sample, then decide to peck on the basis of that memory and subsequent test information, or (b) anticipate the remainder of the trial by retaining prospective memory that includes both perceptual information about the expected test stimulus and outcome information about the availability of the reinforcer. Whether DCD requires retrospective or prospective memories (or both) remains quite undecided (see Honig & Dodd, 1986; Wasserman, 1986). Even so, it is clear that DCD has more complex rules (reference memories), and requires retention of a perceptual working memory over the delay.

It remains to explain how these differences in cognitive requirement translate into the differences in discriminative performance observed in the present experiments. First, the slower acquisition, and lower asymptote of DCD performance may be attributed to its increased cognitive complexity. In general, discriminations with more complex rules are acquired more slowly than those with simpler rules. Second, the greater sensitivity of DCD to increasing delay may be attributed to differences between the representations required over the memory interval. The representation in DCD has a perceptual component (whether retrospective or prospective), whereas the representation in DD does not. Most investigators agree in suggesting that perceptual memories of stimuli are more adversely affected by delay than instructional memories of trial outcomes; for example, "Current research, some of it reviewed here, has led to the conclusion that response instructions are better remembered than stimuli" (Honig & Dodd, 1986). Third, one can attribute differential scopolamine dose-response functions in DD and DCD to the blocking of central cholinergic receptors mediating cognition in the two tasks. Stated another way, the differential effect of scopolamine suggests that the cholinergic system does not function identically in DD and DCD. For example, the DCD task may make greater demands on the brain's cholinergic neurons.

A difficulty arises in deciding precisely how reduced cholinergic function affected cognition in the present work. Bartus and Johnson (1976) suggested that an increasing effect of scopolamine at longer delays (i.e., an interaction between drug dose and memory delay) such as they obtained, demonstrated

a specific effect of the drug on working memory. Important to their demonstration was the inclusion of a simultaneous sample–test condition, because performance with and without the drug was nearly identical in the simultaneous condition, but often different at even near zero seconds (nonoverlapping) delay. In preliminary experiments, the inclusion of a simultaneous condition made acquisition by rats problematic. For this reason, the present experiments lacked the simultaneous condition, and no interaction between drug dose and delay was obtained. Thus, unequivocal evidence of an effect of scopolamine on working memory in rats is not available. Perhaps the successful inclusion of a simultaneous condition would result in an interaction between drug dose and delay. Alternatively, scopolamine may have affected different processes in the two tasks (e.g., perceptual encoding in DCD and recognition of the positive sample in DD), and no interaction would be obtained even with the inclusion of a simultaneous condition. According to a quite different hypothesis, scopolamine may have affected the retrieval of discrimination rules from reference memory. According to this view, reference memory retrieval of the more complex DCD rules may have been affected more at lower doses than retrieval of simpler DD rules. In summary, although the present experiments appear to implicate a differential effect of scopolamine on cognition in DD and DCD, they do not decide the issue of which cognitive mechanism was affected.

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