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Cocaine and Vigilance Task Performance of Rats: Effects of Delay of Reinforcement

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GRILLY, D. M. AND C. NOCJAR. *Cocaine and vigilance task performance of rats: Effects of delay of reinforcement.* PHARMACOL BIOCHEM BEHAV **37**(4) 643–648, 1990.—In two experiments rats were food-reinforced for pressing one of two levers in an operant chamber, with the correct lever being indicated by the position of a briefly illuminated light. In Experiment 1 the levers were always in the chamber, whereas in Experiment 2 the levers were inserted into the chamber immediately after cue light termination and withdrawn immediately after a choice response. The rats were tested under four conditions: after an injection (SC) of saline or 2.5 mg/kg cocaine and with delay of reinforcement (DOR) of either 0 or 8 s. In both experiments, cocaine enhanced accuracy under the 0-s DOR condition. However, in neither experiment was there evidence of facilitation with cocaine under 8-s DOR, which by itself increased choice latencies and decreased accuracy when choice latencies exceeded 0.5 s. These results indicate that cocaine may only enhance performance in vigilance tasks under constrained conditions, e.g., those that require minimal levels of information processing.

Cocaine Performance Vigilance Delay of reinforcement Rats

CONCERNS over the abuse of cocaine have led to a number of studies on its biochemical and behavioral actions that contribute to its value as a reinforcer. In a series of studies focused on the potential performance-enhancing properties of cocaine, low doses of cocaine (2.5 mg/kg) were observed to enhance the accuracy of rats trained in a two-choice vigilance task, i.e., one that required the subjects to maintain a readiness to respond to a simple visual stimulus which occurred periodically over time (10). Subsequently, it was determined that the facilitative effects of cocaine on accuracy in this task were not restricted to conditions under which the organism was performing at suboptimal levels because of fatigue or lack of motivation (11). In contrast, in a similar task requiring selective attention, in which subjects were confronted with two simultaneous stimuli (a constant light and a blinking light) and were required to respond to one while disregarding the other, cocaine (doses between 1.25 and 15.0 mg/kg) did not enhance accuracy (10). Thus, it does appear that low doses of cocaine can enhance performance in some choice tasks under a variety of arousal conditions.

The general purpose of the present research was to determine whether or not cocaine would facilitate accuracy in our vigilance task under delay of reinforcement (DOR) conditions. Disruptions in the temporal contiguity between operants and reinforcement in general have been shown to weaken responding as measured by runway speed, number of errors, response latency, or response rate [see, e.g., (18,20)]. It has also been shown with monkeys that a DOR, either introduced abruptly (17) or gradually (4,5) into a well-learned visual discrimination can cause a deterioration in response accuracy. Therefore, we conducted two experiments to determine whether the effects of a low dose of cocaine interacted with the effects of DOR on vigilance task performance.

METHOD

Animals

Male Sprague-Dawley rats (purchased from Hilltop Lab Animals) were used, 10 in Experiment 1 and 11 in Experiment 2. They were 10 to 21 months of age (mean = 14.1 months) in Experiment 1 and were 14 to 25 months of age (mean = 17.5 months) in Experiment 2 at the beginning of the drug treatment phase of the experiments. At 100 days of age, their individual weights were determined and maintained at these levels (mean = 352 g, range = 316–390 g) through food restriction. Water was available at all times in their home cages. The animals were maintained in a 22°C, 50% humidity facility under a 12-h light-dark cycle (lights on 0800 h). Test sessions were conducted between 1300 and 1900 h. The animals in Experiment 2 had been used in a previous study

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assessing the effect of differential arousal states on vigilance task performance, and had been twice exposed to cocaine (2.5 mg/kg). There was a minimum of 91 days (mean = 143 days) between the animals' last drug exposure and the first test session of Experiment 2.

Apparatus

Two operant chambers [details of which can be found in (9)] were interfaced with Apple IIe 64k microprocessors, which controlled experimental events and collected data. Two levers, which could be mechanically withdrawn or inserted into the chambers, were located at one end of the chambers. Located between the two levers was a food tray, into which single 45 mg food pellets were delivered as reinforcers. A microswitch was activated when the animal's head was inserted into the opening. The cue lights were located directly above each lever, and a house light was centered in the ceiling.

Procedure

Both experiments of this study implemented discrimination training to reach maintenance responding within the criteria outlined below. This was done so that all animals within each experiment began the drug treatment phase at approximately the same level of choice accuracy.

During all training and test sessions, there was no illumination in the room containing the operant chambers. Trials began with the house light in the chamber coming on. Prior to cue light presentation in Experiment 1 (unsignalled delay procedure), the rat had to have its head out of the food tray and had to refrain from pressing either lever for 1.0 s. The cue light above one of the levers was then illuminated (there was a minimum of 1.7 s between house light onset and cue light onset). Cue light duration was individually determined as described below. If the lever beneath the cue light was pressed, food was delivered immediately (or after 8.0-s delay in 8-s DOR test sessions, described below), accompanied by a 40-ms light presentation inside the food tray. After an incorrect response, the house light was turned off immediately; following a correct response, it was turned off 1.0 s after the rat had inserted its head in the food tray following food delivery. This signalled the end of the trial. Intertrial intervals were approximately 7.0 s.

The procedure remained the same in Experiment 2 (signalled delay procedure) with the following exceptions. All trials began with the levers withdrawn from the chamber. Prior to cue light presentation, the rat had to have its head out of the food tray for 1.0 s. The cue light was then illuminated. Immediately following cue light termination, both levers were inserted into the chamber. Following a lever press, the levers were then withdrawn. The food delivery procedure was the same as in Experiment 1.

In both experiments, the position of the cue light was randomly determined, except that there were no more than six successive trials with the light present in the same position. Within a session, the total number of trials with each cue did not differ by more than two.

Cue light duration was individually determined for each animal under 0-s DOR. At the beginning of training in the vigilance task, the cue light duration was set at 1.8 s. If an animal's accuracy exceeded 87% in two successive sessions, the cue light duration was decreased by 0.3 s; if the animal's accuracy dropped below 75% for two successive sessions, the cue light duration was increased by 0.15 s. This titration procedure was employed until the animals' overall percentage of correct responses was maintained between 75 and 87 over four successive 100 trial sessions without a change in cue light duration. Final cue light durations ranged from 0.55 to 1.80 s (mean = 1.21 s) in Experiment 1 and from 0.45 to 1.80 s (mean = 1.17) in Experiment 2. In Experiment 1 the animals were then exposed to eight alternating 0- and 8-s DOR sessions prior to experimental treatment to acclimate them to the 8-s DOR condition. In Experiment 2 the animals were not acclimated to the 8-s DOR condition prior to experimental treatment, because literature suggested to us that DOR effects may be cumulative across test sessions (4, 5, 17). (As noted in the Results section, there was a cumulative effect of 8-s DOR across trials within the test sessions; however, there was none between 8-s DOR sessions, perhaps because we always exposed the rats to a 0-s DOR practice session between 8-s DOR test sessions.)

Four test sessions of 100 trials each were conducted four to seven days apart. Animals were tested after an injection of saline or 2.5 mg/kg cocaine, in either a 0- or 8-s DOR condition. In Experiment 1 the order of treatment was randomly determined for each animal, and a practice session, with 0-s DOR, was conducted between each treatment session. In Experiment 2 the 0-s DOR test sessions were conducted prior to treatment sessions with the 8-s DOR condition. The order of drug treatment was randomly determined within each of the DOR conditions. To make sure that the animals' level of performance remained within the criterion levels, practice sessions with 0-s DOR were conducted between the 8-s DOR treatments. Animals were not run on other days. Each animal was therefore exposed to all treatment conditions with a constant cue light duration. The delay interval was chosen on the basis of pilot work indicating that an 8-s DOR was capable of producing a moderate, but significant, reduction in overall accuracy in this task over a single 100-trial session. The dose was chosen on the basis of its ability to produce optimal performance enhancement in previous work with this task (10); also, this dose has been shown to enhance accuracy in this task under both high and low levels of behavioral arousal (11).

Cocaine HCl (obtained from Sigma Chemical Co.) was diluted with 0.9% saline, and solutions were prepared so that all injections were given in a volume of 1.0 ml/kg. Dose is expressed as the salt. Both saline and cocaine doses were administered subcutaneously 15 min prior to testing.

RESULTS

The following behavioral measures were derived for each animal under each condition: 1) percent correct lever choices (accuracy); and 2) median choice latency or latency to respond (time between cue light offset and lever-press). Because the probability of a correct choice (accuracy) is inversely related to the choice latency in this task (9,10), accuracy scores were derived for each of three choice latency categories: 1) 0.0–0.5 s; 2) 0.5–1.5 s; and 3) 1.5–3.5 s (these categories were chosen because of the highly positively skewed distribution of the latencies, e.g., see Fig. 2). Treatments × blocks ANOVAs, with drug treatment, delay condition, and blocks (choice latency category) as factors, were used for initial statistical analyses. Significant F values were then followed up with planned comparisons (two-tailed *t*-tests).

Percent correct lever choices as a function of drug treatment, delay condition, and choice latency category are shown for both experiments in Fig. 1. The results in the two experiments were very similar. In both experiments accuracy was a joint function of all three factors. In Experiment 1 (unsignalled delay procedure) there was a significant main effect of drug treatment, $F(1,9) = 5.13$, $p<0.05$, and significant interactions between drug treatment

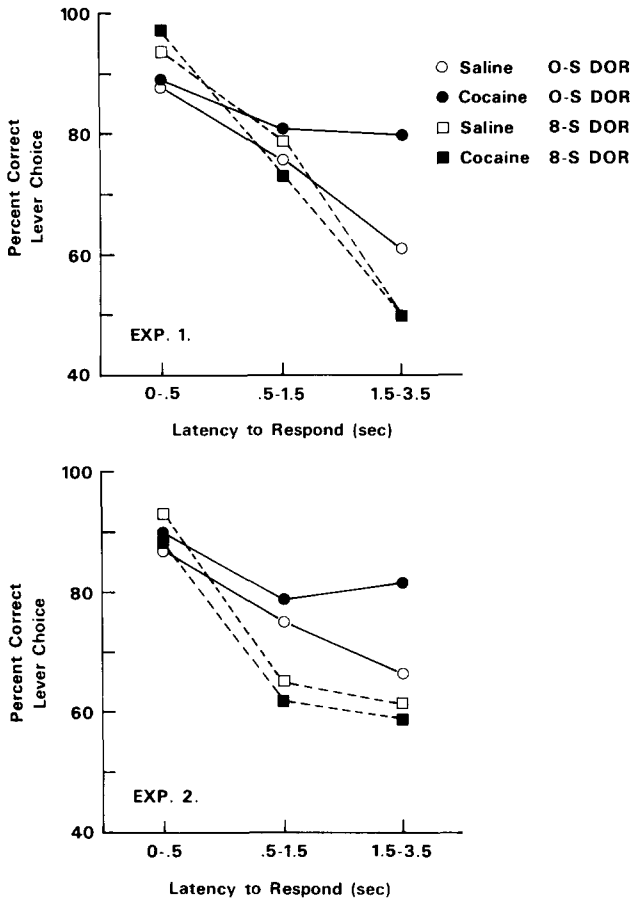

FIG. 1. Accuracy (mean percentage correct lever choices) as a function of latency to respond (choice latency) after saline or 2.5 mg/kg cocaine at either 0-sec DOR or 8-sec DOR. In Experiment 1 (EXP 1, n = 10) response levers were always in the chamber. In Experiment 2 (EXP 2, n = 11) response levers were inserted into the chamber following cue light termination and withdrawn from the chamber following a choice response.

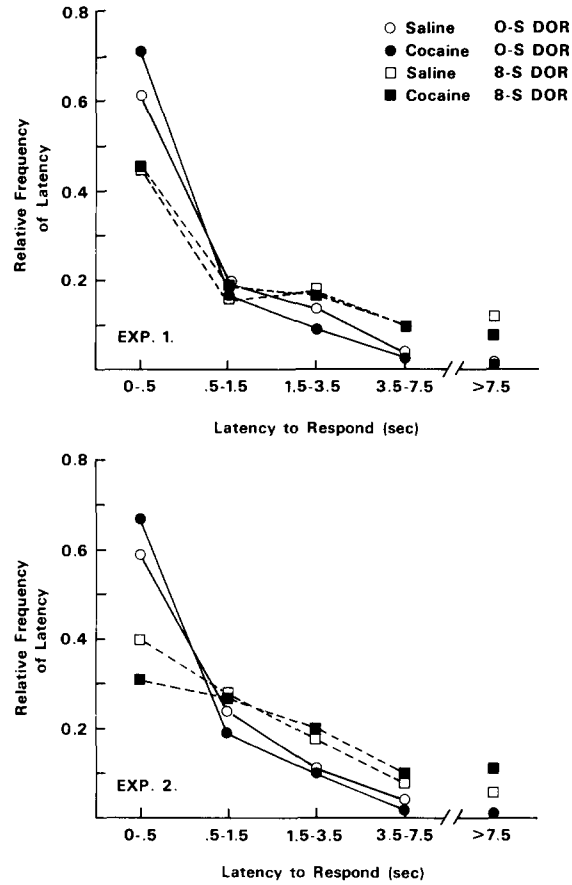

FIG. 2. Relative frequency distributions of latencies to respond (choice latencies) in Experiments 1 and 2 (EXP 1 and 2) after saline or 2.5 mg/kg cocaine at either 0-sec DOR or 8-sec DOR.

and delay condition, $F(1,9) = 9.10$, $p<0.05$, and between delay condition and choice latency category, $F(2,18) = 13.99$, $p<0.01$. In Experiment 2 (signalled delay procedure) there was a significant main effect of delay condition, $F(1,10) = 14.43$, $p<0.01$, and significant interactions between drug treatment and delay condition, $F(1,10) = 5.64$, $p<0.05$, and between delay condition and choice latency category, $F(2,20) = 5.07$, $p<0.05$.

In Experiment 1 animals treated with cocaine exhibited significantly higher accuracy levels than saline-treated animals in the 0-s DOR condition, $t(9) = 3.53$, $p<0.01$, but there was no significant difference between drug treatments under the 8-s DOR condition. In comparisons between 0-s DOR and 8-s DOR conditions, the 8-s DOR condition resulted in significantly higher accuracy in the 0.0–0.5-s choice latency category, $t(9) = 3.38$, $p<0.01$, and significantly lower accuracy in the 1.5–3.5-s choice category, $t(9) = 3.32$, $p<0.01$.

In Experiment 2 in the 0-s DOR condition, cocaine-treated animals exhibited significantly higher accuracy levels than saline-treated animals, $t(10) = 3.86$, $p<0.01$, but there was no significant difference in accuracy between cocaine-treated and saline-treated animals in the 8-s DOR condition. In comparisons between 0-s DOR and 8-s DOR conditions, accuracy was significantly lower in the 8-s DOR condition in both the 0.5–1.5 and 1.5–3.5-s choice latency categories, $t(10)$s $= 5.21$ and 2.88, ps <0.01 and 0.05.

Relative frequency distributions of choice latencies for the two drug treatments and two delay conditions are shown in Fig. 2. As expected, 8-s DOR significantly increased choice latencies in both experiments, $F(1,9) = 25.72$ and $F(1,10) = 33.31$, ps <0.01. As noted in previous studies, in the no delay condition choice latencies were slightly, but reliably, shorter with cocaine than with saline, $t(9) = 2.82$ and $t(10) = 3.59$, ps <0.05 and 0.01. However, under 8-s DOR conditions, there was no reliable difference between cocaine and saline-treated animals in Experiment 1, and in Experiment 2 choice latencies were actually significantly longer with cocaine (mean = 1.19 s) than with saline (mean = 0.85 s), $t(10) = 2.28$, $p<0.05$.

The above results are for 100 trial sessions. To see whether performance measures changed over trials, we broke the sessions into thirds and reanalyzed the data. Mean performance levels for accuracy and choice latency are shown in Table 1. Performance levels under the 0-s DOR condition were fairly constant across test sessions. However, under the 8-s DOR condition, as one might expect, choice latency distributions gradually shifted across trials for both drug treatments in both experiments. Thus, as can be seen in Table 1, as the relative frequency of longer choice la-

TABLE 1

GROUP MEANS (SEM INDICATED IN PARENTHESES) FOR PERCENTAGE CORRECT LEVER CHOICE (%) AND CHOICE LATENCY SCORE (CL) IN S AS A FUNCTION OF DRUG TREATMENT, DELAY OF REINFORCEMENT CONDITION, AND TRIALS WITHIN TEST SESSIONS IN EXPERIMENTS 1 AND 2

Treatment		Trials 1–33	34–67	68–100	All Trials
Experiment 1					
Saline, 0-s delay	%	80.8 (2.2)	80.9 (3.0)	77.0 (3.7)	79.6 (2.4)
	CL	0.40 (0.03)	0.43 (0.07)	0.40 (0.06)	0.41 (0.05)
Saline, 8-s delay	%	82.7 (2.0)	70.3 (2.5)	73.3 (2.6)	75.4 (1.5)
	CL	0.44 (0.06)	1.11 (0.17)	1.35 (0.55)	0.85 (0.15)
Cocaine, 0-s delay	%	82.7 (2.4)	87.1 (2.6)	85.5 (3.5)	85.1 (2.1)
	CL	0.37 (0.09)	0.34 (0.01)	0.36 (0.05)	0.36 (0.02)
Cocaine, 8-s delay	%	78.7 (1.8)	77.9 (2.6)	71.7 (3.4)	76.1 (2.3)
	CL	0.47 (0.07)	0.72 (0.14)	1.21 (0.23)	0.71 (0.11)
Experiment 2					
Saline, 0-s delay	%	79.6 (2.1)	82.4 (2.9)	82.9 (1.9)	81.6 (1.1)
	CL	0.49 (0.09)	0.40 (0.06)	0.38 (0.06)	0.42 (0.04)
Saline, 8-s delay	%	82.6 (1.6)	73.2 (3.8)	66.1 (4.2)	74.0 (2.1)
	CL	0.56 (0.14)	0.81 (0.16)	1.14 (0.21)	0.85 (0.13)
Cocaine, 0-s delay	%	86.8 (2.3)	85.3 (2.5)	86.0 (1.6)	86.0 (1.1)
	CL	0.39 (0.06)	0.35 (0.06)	0.37 (0.03)	0.37 (0.03)
Cocaine, 8-s delay	%	78.2 (2.1)	63.1 (3.2)	60.9 (2.5)	67.4 (1.8)
	CL	0.72 (0.16)	1.32 (0.56)	1.89 (0.62)	1.19 (0.15)

tencies increased across trials, overall accuracy declined across trials.

DISCUSSION

The results of these experiments replicate previous findings (10,11) that a low dose of cocaine (2.5 mg/kg) can significantly enhance accuracy in a choice task heavily dependent on vigilance. In these earlier studies, the response levers were always accessible during test trials. The present study demonstrates that these earlier results were not totally dependent on this aspect of the task, because in Experiment 2 the levers were only accessible after the cue light was terminated and were immediately retracted following the animal's response. However, the results of both of the present experiments further indicate that cocaine-induced facilitation only occurred when there was no delay between the choice response and reinforcement. When reinforcement was delayed 8 s, the results were very different.

First, as expected, 8-s DOR was detrimental to accuracy performance, but only when choice latencies were longer than 0.5 s. In Experiment 1 accuracy was actually higher with 8-s DOR under both drug treatments when choice latencies were less than 0.5 s. In Experiment 2, this phenomenon did not occur. Because there were several differences between the two experiments in terms of the subjects' ages and the procedures used, there could be a number of reasons for this minor difference in the results. However, the differences are most likely due to the animals' having access to the levers in Experiment 1 and being able to respond immediately following, or even prior to, cue light termination. In Experiment 2 these types of responses were prohibited because there was approximately ⅓ s between cue light termination and lever accessibility. In either case, in the early portion of the test sessions, because most choice latencies were short, the effect of the 8-s DOR on overall accuracy levels was minimal. However, as the relative frequency of longer choice latencies increased across trials under 8-s DOR conditions, overall accuracy levels declined considerably under both drug treatments.

Second, with 8-s DOR there was no reliable difference between cocaine- and saline-treated animals in terms of overall accuracy levels. The only evidence for a cocaine facilitation effect occurred in Experiment 1 when choice latencies were short. There was no evidence for a cocaine facilitation effect in Experiment 2 for any choice latency category or portion of the test sessions.

These results raise a number of interesting questions regarding the effects of DOR on choice behavior and how these interact with the effect of cocaine. Unfortunately, we have very little information which we can use to answer these questions. While numerous studies have shown that disruptions in temporal contiguity between operant responses and reinforcement weaken responding in a variety of ways, these studies have most frequently been conducted in the context of response acquisition and extinction, rather than in the context of operant response maintenance (14). The literature on the effect of DOR on the maintenance of discrete trial choice behavior is even more sparse and sometimes contradictory. Lawrence and Hommel (15), using a Grice apparatus, reported that rats which had mastered a black-white discrimination were able to maintain their accuracy when subsequently given 50 trials of training at delay intervals of 20, 30, and 60 s. However, decrements in accuracy may not have been observed in that study because of an insufficient number of trials; in three studies with monkeys in which more trials were assessed, discrimination task accuracy in highly trained subjects has been shown to be disrupted when DOR was introduced. Mishkin and Weiskrantz (17) reported a significant reduction in the accuracy of visual (successive) discrimination performance in rhesus monkeys when a DOR of only 8 s was introduced abruptly. In two other studies (4,5), the accuracy of Cebus monkeys that were highly trained on a two-choice simultaneous visual discrimination task was severely disrupted even when DOR was gradually increased from 2 to 32 s. Thus, it appears that in highly trained animals it may take several exposures to the DOR in order for

accuracy deficits to occur.

Unfortunately, none of these studies appear to offer any clues as to why 8-s DOR caused the accuracy deficits in our animals nor any clues as to why cocaine was not found to facilitate accuracy in our task when 8-s DOR was introduced. There are several hypotheses regarding DOR-induced accuracy deficits. One is that when a discriminative response is separated from its consequences by a DOR and no differential cues exist to mediate the delay interval, the association between the two events becomes weakened (4). A second hypothesis is that the performance decrement with DOR is due to a loss of incentive value, either because of a reduction in reward value or because the delay period becomes aversive (4,5). A third possibility is that during the DOR the animals begin to engage in irrelevant behaviors that are spuriously reinforced when reinforcers are delivered and that these behaviors begin to compete with the previously acquired response (6,12).

Of these three hypotheses, our results appear most compatible with the last two. From our interpretation of the association deficit hypothesis, we would have predicted that accuracy should have declined with DOR regardless of the animals' choice latency. It is clear that this did not happen in either experiment. In fact, in Experiment 1 accuracy was actually higher under 8-s DOR when the animals' choice latencies were very short. Conversely, we interpret the incentive loss hypothesis to predict that choice latencies should increase across trials under the 8-s DOR condition, which would then lead to lower overall accuracy levels. Our results were consistent with this prediction.

Our results under 8-s DOR conditions may also have been due to the specific requirements of the task in combination with the fact that when reinforcement was delayed a number of irrelevant responses could occur between the measured response and the reinforcer and, thus, could be spuriously reinforced. These responses could then compete with the nominal response for which reinforcement was intended. Therefore, as the sessions with 8-s DOR wore on, the subject may have become less able to discriminate the reinforcement contingency. On some trials, the animal may have engaged in the behavior that was most successful in obtaining reward in the past (i.e., quickly pressing the lever underneath the cue light). On other trials, the animal may have attended to stimuli other than the cue light or engaged in other behaviors (or from a cognitive view employed other strategies) before pressing one of the levers. On such trials, longer choice latencies should occur and accuracy should be close to chance levels.

This possibility, that presses to the incorrect lever during the delay interval could be spuriously reinforced, was the primary reason for conducting Experiment 2 in which we employed lever removal immediately following a choice response. Since the levers were always available in Experiment 1, we speculated that cocaine may not have facilitated task performance in the 8-s DOR condition because it could lead to an increase in incorrect lever presses during the delay that were coincidently followed by reinforcement. This hypothesis was not supported because the cocaine-treated animals in Experiment 2 actually performed more poorly in the 8-s DOR condition than in Experiment 1 (see Table 1). However, because it is virtually impossible to eliminate all irrelevant responses that could be spuriously reinforced under a DOR condition, this hypothesis may still be valid.

Although we only used one dose of cocaine in the present studies, it is unlikely that larger doses would have facilitated accuracy in the 8-s DOR condition, because even the low dose used in these studies appeared to exert disruptive effects on performance, particularly in Experiment 2 (see Table 1). We did not use smaller doses of cocaine because previous work (10) had indicated that doses lower than 2.5 mg/kg produced qualitatively the same effects as 2.5 mg/kg, but the effects were less reliable. Also, we are not aware of any other studies indicating that cocaine in doses below 2.5 mg/kg (SC or IP) produces any reliable effects on choice behavior in rodents. However, we cannot rule out the possibility that other doses of cocaine might have enhanced accuracy under an 8-s DOR.

Until recently, it was not clear whether cocaine, like amphetamine, could bring choice performance deteriorated by boredom or fatigue back to baseline levels, or for that matter, whether cocaine had any positive effect at all on choice performance (1). We now know that cocaine's behavioral effects are quite similar to those of amphetamine [e.g., (3, 8, 10, 13, 19)]. It is also clear that cocaine can enhance choice performance even in well-rested, highly motivated rats (11). However, cocaine's ability to enhance performance is highly dependent on the dose. In rats (10) and mice (3) only doses of cocaine of 10 mg/kg or less reliably facilitate choice performance; doses higher than 10 mg/kg may not facilitate performance and may actually disrupt it. Cocaine-induced choice performance enhancement also appears to be highly task specific. Enhancement may not occur if baseline performance is close to ceiling levels (2,7). Also, it may only occur when the task requires predominantly sustained attention, but does not appear to facilitate performance if the task is heavily dependent on selective attention (10). Finally, the present studies indicate that the conditions under which cocaine enhances choice performance are even more limited than these previous studies had indicated. That is, when a delay between the choice and reinforcement is introduced, even low doses of cocaine may not facilitate performance. These results do suggest that when the task is changed from one that requires predominantly vigilance to a task that requires a more complex level of information processing, cocaine may no longer facilitate performance.

This interpretation is consistent with earlier attempts to explain the types of effects on choice behavior that can occur with psychostimulants (2, 3, 10, 16). That is, low doses of psychostimulants may only enhance choice behavior in moderately difficult tasks that require the animal to be ready to process the relevant information and respond as quickly as possible after its occurrence. If more complicated test paradigms are used, particularly if enhanced motor output is nonadaptive for the task contingencies, then even low doses of psychostimulants may not facilitate performance or may induce suboptimal performance.

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