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EFFECTS OF COCAINE ON BRIEFLY SIGNALED VERSUS COMPLETELY SIGNALED DELAYS TO REINFORCEMENT

DIANA J. WALKER AND MARC N. BRANCH

UNIVERSITY OF FLORIDA

Key pecking by 4 pigeons was maintained by a multiple schedule consisting of two variable-interval 60-s schedules wherein each food presentation followed a nonresetting 27-s delay that was either briefly signaled at its outset or completely signaled. Brief-signal duration was adjusted so that response rates maintained by the briefly and completely signaled delays of reinforcement were similar. In general, acute administration of small to intermediate doses (0.3 to 3.0 mg/kg) of cocaine produced either small increases in response rates in both components or no change, and larger doses (5.6 to 13.0 mg/kg) decreased response rates. Chronic (i.e., daily) cocaine administration (10.0 mg/kg) resulted in tolerance to the rate-decreasing effects in both components. Cocaine's effects were generally similar whether delays were completely or briefly signaled. Discontinuation of cocaine administration and subsequent removal of the delay signals also had similar effects in both components of the multiple schedule. Taken together, these results are consistent with the view that the two types of delay signals were equally effective in maintaining responding during the variable-interval schedules.

Key words: signaled delay of reinforcement, conditioned reinforcement, cocaine, tolerance, variable-interval schedule, key peck, pigeons

The purpose of the present experiment was to study the effects of acute and chronic (i.e., daily) cocaine administration on responding maintained by briefly and completely signaled delays to reinforcement. Key pecking by 4 pigeons was maintained by a multiple schedule consisting of two variable-interval (VI) 60-s components, wherein each reinforcer followed a 27-s delay. In one component the delay was briefly signaled by a change in key color at its beginning; in the other component the delay was completely signaled. Roughly equal response rates were established in the two components of the multiple schedule via manipulation of the brief-signal duration. Cocaine was administered acutely and then chronically to determine whether drug effects on performance would differ for the two types of signaling arrangements.

Cocaine was chosen for study in the present experiment because it has been suggested that this drug and other psychomotor stimulants may enhance the efficacy of conditioned reinforcers (Beninger, Hanson, & Phillips, 1980; Cohen & Branch, 1991; Files, Branch, & Clody, 1989; Hill, 1970; Robbins, 1975, 1978; Robbins & Koob, 1978). Hill, for example, reinforced lever pressing by rats with sweetened condensed milk according to a VI 2-min schedule. A 6-s hum accompanied operation of the dipper. Then four types of extinction were implemented across groups. One group was injected with placebo, and responses in extinction had no consequences. Another group was injected with placebo, and responses produced the hum that had been paired previously with milk presentation. The other two groups received 10.0 mg/kg pipradrol (a psychomotor stimulant) before extinction sessions; for one group, lever presses produced no consequences, and for the other group, lever presses produced the hum. Responding was enhanced by pipradrol administration when the conditioned reinforcer (i.e., the hum) was presented. Both placebo groups responded similarly, and responding was suppressed by pipradrol when the conditioned reinforcer was not presented in extinction. Hill performed two other experiments that showed that the facilitation of responding observed in Experiment 1 was due to enhanced efficacy of the conditioned reinforcer and not to enhanced efficacy of

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Send correspondence or reprint requests to Diana J. Walker or Marc N. Branch, Department of Psychology, University of Florida, Gainesville, Florida 32611.

sensory reinforcement or to a change in motivation.

Researchers using other procedures have replicated the finding that stimulants can enhance the reinforcing efficacy of stimuli that have been paired with primary reinforcement (e.g., Cohen & Branch, 1991; Files et al., 1989; Robbins, 1975, 1978; Robbins & Koob, 1978). The study by Files et al. has relevance to the present experiments. They found that methylphenidate can enhance key pecking by pigeons in extinction when stimuli are presented that have been paired with food (paired brief stimuli). In their experiment, sessions began with a second-order schedule of paired brief stimuli wherein, after an average of 30 s, a key peck produced food and a stimulus complex or the stimulus complex alone. In the second part of the session, one of two types of extinction occurred. Either key pecks produced the stimulus complex, or they produced no consequences. Methylphenidate was administered occasionally before sessions, and it was found that moderate doses produced higher response rates during extinction when the stimulus complex was presented than when it was not. Interestingly, during sessions that were not preceded by drug administration, no conditioned reinforcement effect was observed. That is, subjects responded similarly in extinction whether the paired brief stimuli were presented or not. Thus, the authors report that conditioned reinforcing functions of the paired brief stimuli during extinction were "revealed" by the drug (cf. Branch, 1984).

In the present study, performance of pigeons was established under two different types of delayed reinforcement. Nonresetting delays of the same length, regardless of the subject's behavior during the delay, were employed. The delays were signaled by a stimulus change that occurred at the beginning of each delay. The signal was present only during the first part of the delay (briefly signaled), or the stimulus remained present throughout the delay (completely signaled).

Delay signals may serve as conditioned reinforcers, and their efficacy may increase as larger proportions of the delays are signaled. Using VI 60-s schedules, Schaal and Branch (1988) and Sizemore and Lattal (1977) found that short (1- to 9-s), unsignaled, nonresetting delays decreased key-pecking rates of pigeons. When such delays were signaled, rates similar to those that occur with immediate VI reinforcement were maintained (Schaal & Branch, 1988). Richards and Richardson (1991) obtained similar results using variable-ratio (VR) 50 and VR 100 schedules of food presentation. Longer (27- to 60-s) delays have also maintained rates similar to those maintained under VI 60-s schedules of immediate reinforcement when the delays were completely signaled (Ferster, 1953; Lattal, 1984; Schaal & Branch, 1990). In addition, Schaal and Branch (1990, Experiment 1) found that relatively long (27-s) delays that were briefly signaled could also maintain rates similar to those maintained by immediate reinforcement (cf. Schaal & Branch, 1988) at some brief-signal durations. That is, in terms of maintenance of responding, brief and complete signals were equally effective.

The present experiment was designed to examine the effects of cocaine on responding maintained by stimuli that signal a delay to primary reinforcement. In previous studies of the interaction of stimulants and conditioned reinforcers (mentioned above), the conditioned reinforcer has been a stimulus that was temporally contiguous with food. In the present study, both stimuli signaled a delay to food; the complete signal was contiguous with food, whereas the brief signal was not. As mentioned above, it has been shown that both brief and complete delay signals can function as conditioned reinforcers (i.e., can maintain higher rates of responding than can delayed reinforcement that is not signaled). The question asked in the present experiment was how cocaine would interact with the two types of delay signals to influence key pecking by pigeons when the two types of signals maintained similar rates and patterns of responding under nondrug conditions. It was expected that cocaine might affect responding maintained by the brief and complete signals differently and thereby reveal differences in their conditioned reinforcing efficacy (cf. Branch, 1984; Files et al., 1989).

Cocaine was administered both acutely and chronically in the present study because it has been shown that chronic administration can result in differential effects on performances maintained by different consequences even when the performances are affected similarly

by acute administration of a drug (cf. Branch, 1979).

METHOD

Subjects

Four adult male White Carneau pigeons were maintained at 80% of their free-feeding weights via supplemental feeding of mixed grain and pelleted pigeon food after experimental sessions. The pigeons were housed in individual stainless steel cages in a temperature-controlled colony room under a 16:8 hr light/dark cycle. They had continuous access to water and health grit in their home cages. Three pigeons had been exposed briefly to short (2-s) unsignaled delays of reinforcement, and 1 (Subject 269) had had experience with unsignaled, briefly signaled, and completely signaled delays of reinforcement of various durations (Schaal & Branch, 1988, Experiment 2, 1990, Experiment 1).

Apparatus

Sessions were conducted in an operant conditioning chamber for pigeons that was 30 cm wide, 32 cm long, and 31 cm deep. The walls were painted flat black, and the work panel was brushed aluminum. It contained three response keys (Gerbrands), horizontally aligned and centered 21 cm above the floor. The keys were 2 cm in diameter and could be transilluminated by four 1.1-W 28-Vdc lamps that were covered with red, blue, green, or white translucent caps. Only the center key was used and required a force of approximately 0.15 N to count as a response and produce a feedback click. Two 1.1-W 28-Vdc lamps located behind aluminum shields in either upper corner of the work panel served as houselights. Reinforcement was 3-s access to mixed grain, which was delivered through an aperture (6 cm by 5 cm) below the center key and 7 cm above the floor. The feeder was illuminated by a 1.1-W 28-Vdc lamp during reinforcement, at which time the houselights and keylight were off. White masking noise was present in the room in which the chamber was located, and a ventilation fan was mounted in the ceiling of the chamber. The pigeon could be observed through a peephole located in the door of the chamber.

A custom-built computer that operated under the ECBasic control system (Walter & Palya, 1984) and that was interfaced with an IBM®-compatible computer (Zenith) located in an adjacent room programmed contingencies and collected data. A Gerbrands Model C-3 cumulative response recorder provided continuous recording of responses.

Procedure

Because of the pigeons' previous experience, key-peck shaping was not necessary. After the pigeons were adapted to the chamber, key pecks were maintained by a multiple fixed-ratio (FR) 1 FR 1 schedule of food presentation in the presence of green and red keylights. Following this preliminary training, sessions consisted of a 5-min blackout followed by a multiple schedule of two VI 60-s components in which each reinforcement occurred after a 27-s delay that was either briefly or completely signaled. More specifically, in the first component the key was green during the VI schedule; the first peck after an average of 60 s turned off the green keylight and turned on a white keylight for x s (brief signal), after which the white keylight was extinguished and the green keylight was reinstated for the remainder of the 27-s delay. This component alternated (see below) with a second component in which the key was red during the VI schedule; the first peck after an average of 60 s turned off the red keylight and turned on a blue keylight for 27 s (complete signal). Food was delivered immediately after the 27-s delay in each component regardless of a bird's behavior during the delay. In schedule terminology, this arrangement is a multiple chained VI 60-s fixed-time (FT) x-s FT (27 − x)-s chained VI 60-s FT 27-s schedule. Intervals for the VI schedules came from a list of 20 values determined by Catania and Reynolds' (1968) equation for generating constant-probability VI schedules and ranged from 3.0 to 215.9 s.

Sessions were conducted 7 days per week at about the same time each day and began with a 5-min blackout (all lights in the chamber off) followed by the component with brief signals. Components alternated for three presentations each, separated by 1-min blackouts. Component presentations lasted for 6 min of VI time each, yielding a total of 18 min of VI time in each component. Data were collected during two-component blocks

Table 1

Number of injections received during drug-effect determination.

Subject	Condition	Dose of saline or cocaine (mg/kg)						
		Sal	0.3	1.0	3.0	5.6	10.0	13.0
269	Baseline	2	2	2	3	3	2	3
	Chronic saline	16						
	Chronic cocaine	4	4	2	2	2	162	2
820	Baseline	2	2	3	3	3	3	
	Chronic saline	28						
	Chronic cocaine	3	5	3	5	2	158	
1009	Baseline	2	2	3	3	5	2	
	Chronic saline	26					2	
	Chronic cocaine	3	3	4	3	3	162	
1069	Baseline	2	2	2	3	3	2	
	Chronic saline	22				1		
	Chronic cocaine	3	2	2	3	2	162	

Table 2

Number of sessions in each condition.

Subject (brief-signal duration)	Baseline	Chronic saline	Chronic cocaine	Chronic saline	No injections	No signals
269 (6 s)	145	16	178	20	21	35
820 (12 s)	143	28	176	20	21	35
1009 (12 s)	135	28	178	20	21	12
1069 (6 s)	126	23	174	20	15	35

of the session so that time-course effects of the drug could be examined.

Duration of the brief signal was 6 s at the start of the experiment for each pigeon. For the 3 pigeons with no signaled-delay experience (all but Subject 269), the duration of the signal was varied across sessions until the shortest brief signal that maintained response rates similar to those maintained by the complete signal was found. These brief signals were 6 s for Subjects 269 and 1069 and 12 s for Subjects 820 and 1009. Brief-signal durations were determined during a phase in which component presentations lasted for 4, 6, or 10 min of VI time each, yielding sessions that lasted 45 min. Because effects of the drug observed during acute administration appeared to diminish near the end of sessions, the procedure was changed so that component presentations lasted for 6 min each, as mentioned above. This phase was designated the baseline phase, during which acute drug effects were examined. When response rates in both components were judged to be stable under the new baseline conditions (through visual inspection of daily plots), acute drug effects were determined by immediately preceding occasional sessions with intramuscular injections of saline (the vehicle) and different doses of cocaine hydrochloride into the breast muscle. (Drug administration, therefore, occurred 5 min before onset of the multiple schedule.) Injection volume was 1.0 ml per kilogram of body weight, and doses ranged from 0.3 to 10.0 mg/kg for 3 pigeons and from 0.3 to 13.0 mg/kg for 1 pigeon (Subject 269). All drug injections were separated by at least a week, and each dose, as well as saline, was administered at least twice (see Table 1). Drug doses were determined as the salt, and cocaine was provided by the National Institute on Drug Abuse.

Following acute dose–response determinations, all sessions were preceded by saline injections (chronic saline) for at least 16 days. Then all sessions were preceded by injections of cocaine (10.0 mg/kg). When predelay response rates became stable in this phase (after 56, 69, 69, and 72 sessions for Subjects 269, 820, 1009, and 1069, respectively), a cocaine dose–response curve was determined in the context of chronic cocaine by substituting other (probe) doses for the chronic dose in a manner similar to that during the acute drug phase: All probe-dose injections were separated by at least a week, and each dose, as well as saline, was administered at least twice. The numbers of injections of each dose in each condition are shown in Table 1.

Another chronic saline phase followed the chronic drug phase. Next, no injections preceded sessions. For the final phase of the experiment the signals were removed, and the schedule was a multiple tandem VI 60-s FT 27-s tandem VI 60-s FT 27-s schedule. The sequence and number of sessions of all conditions are summarized in Table 2.

RESULTS

Because the focus of the research was on the relative efficacy of the two delay-signaling procedures, analysis concentrated on responding during the periods preceding delays (i.e., during the VI periods). (Information re-

lating to behavior during delays will be presented below.)

Figure 1 shows overall response rates from the first and last five sessions of the final baseline condition and all subsequent conditions for each subject. Acute dose–response data were obtained during the baseline condition (before chronic saline) and will be presented in the remaining figures. For all subjects response rates in the two components were similar under baseline conditions, with Subject 820's rates being the most dissimilar. Chronic saline administration resulted in little change in response rates. However, for Subject 269, response rates were higher in the brief-signal component than in the complete-signal component at the end of chronic saline administration.

Chronic cocaine administration (10.0 mg/kg) resulted in an initial decrease in response rate (except in the complete-signal component for Subject 269) and then in recovery from the rate-decreasing effects in both components for all subjects. Overall, performance in the two components was similar during daily administration. Performance recovered in both components, with little evidence of differential control by the two signaling arrangements (except for Subject 269 at the beginning of chronic dosing).

Discontinuation of the chronic administration of cocaine had similar effects in the two components. For Subject 269 response rates in both components declined below baseline levels and stayed relatively low for the remainder of the study. For the remaining 3 subjects, response rates returned to values close to those seen under baseline conditions.

Removing the signals resulted in lower response rates for all 4 birds. Response rates for Subject 269 decreased gradually to below-baseline levels. Subject 1069's response rates decreased immediately, then increased to baseline levels after two sessions of no signals, and then stabilized at a very low rate by the end of the experiment. Response rates for Subjects 820 and 1009 decreased quickly and stabilized rapidly. In all cases, removal of the signals resulted in similar declines in both components. In summary, the figure reveals that effects across the study usually were similar in the two components.

Similarities and differences in performances in the two components were analyzed further by examining effects of different doses of cocaine during the baseline (acute) and chronic drug phases. Figure 2 shows predelay response rates during the VI segments of the two components, averaged over the entire session, plotted as a function of dose of cocaine during the acute and chronic drug phases for all subjects. Control response rates maintained by the briefly and completely signaled delays of reinforcement were similar for 3 of the 4 subjects (Subjects 269, 1009, and 1069). Subject 820's average response rate was higher in the complete-signal component, although the ranges overlapped considerably. Given acutely, cocaine generally resulted in dose-related decreases in response rate maintained by the VI schedule. Data for Subject 269 in the complete-signal component are an exception. Dose–response functions for behavior controlled by the two types of delay contingencies during acute cocaine administration were similar for Subject 820, with small to intermediate doses resulting in no effect on overall response rates in the two components and the largest dose of cocaine (10.0 mg/kg) suppressing rates greatly in both components. For the other 3 subjects, response rates controlled by the complete signals were slightly more resistant to the rate-decreasing effects than were rates in the brief-signal component, and in two cases (Subjects 269 and 1009) some doses of cocaine increased rates in the complete-signal component.

The right panels of Figure 2 show response rates during the VI segments averaged over the entire session, plotted as a function of the substituted dose of cocaine during the chronic drug phase for all subjects. Compared to the acute-dosing functions, rate-decreasing effects were less evident in all subjects. Dose–response functions for the two components were similar for Subjects 820, 1009, and 1069, with ranges of effects overlapping at every dose. For Subject 269 during chronic administration, rates in the two components differed following saline injections, and effects in the two components also differed in that at doses smaller than 10.0 mg/kg, rates were higher in the brief-signal component.

Acute and chronic effects of cocaine were also analyzed separately across the three blocks of the session (data not shown). (Ses-

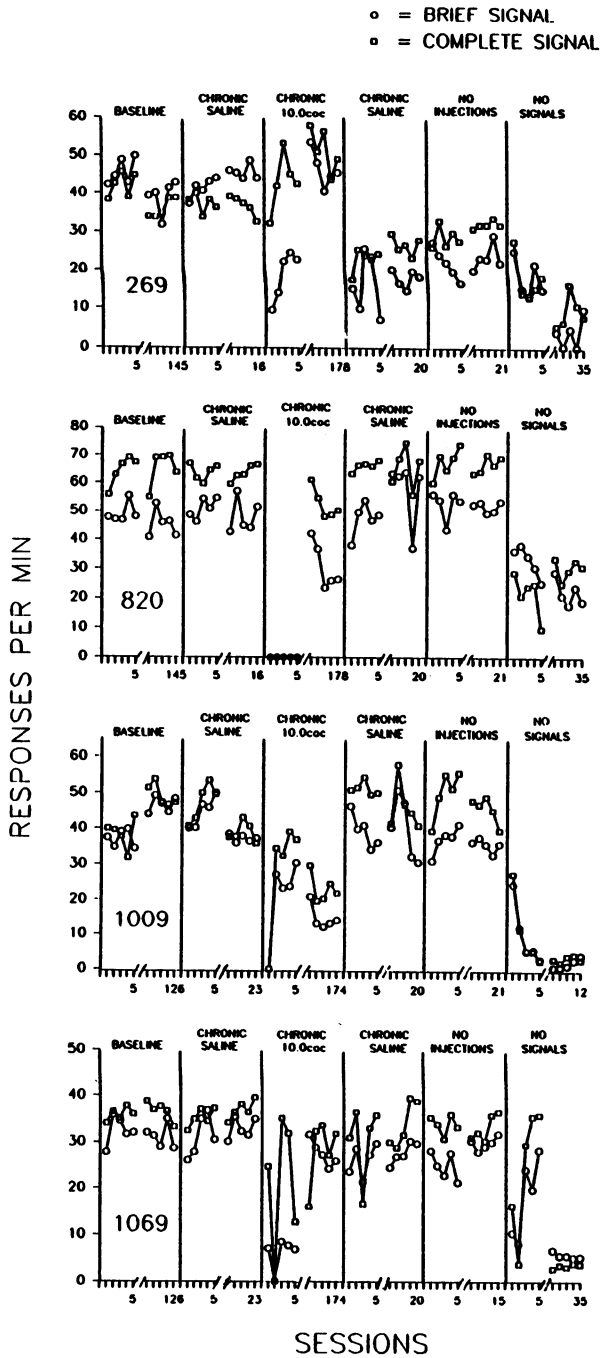

Fig. 1. Average response rates during the first and last five sessions of the last baseline condition and all subsequent conditions of the experiment for all subjects.

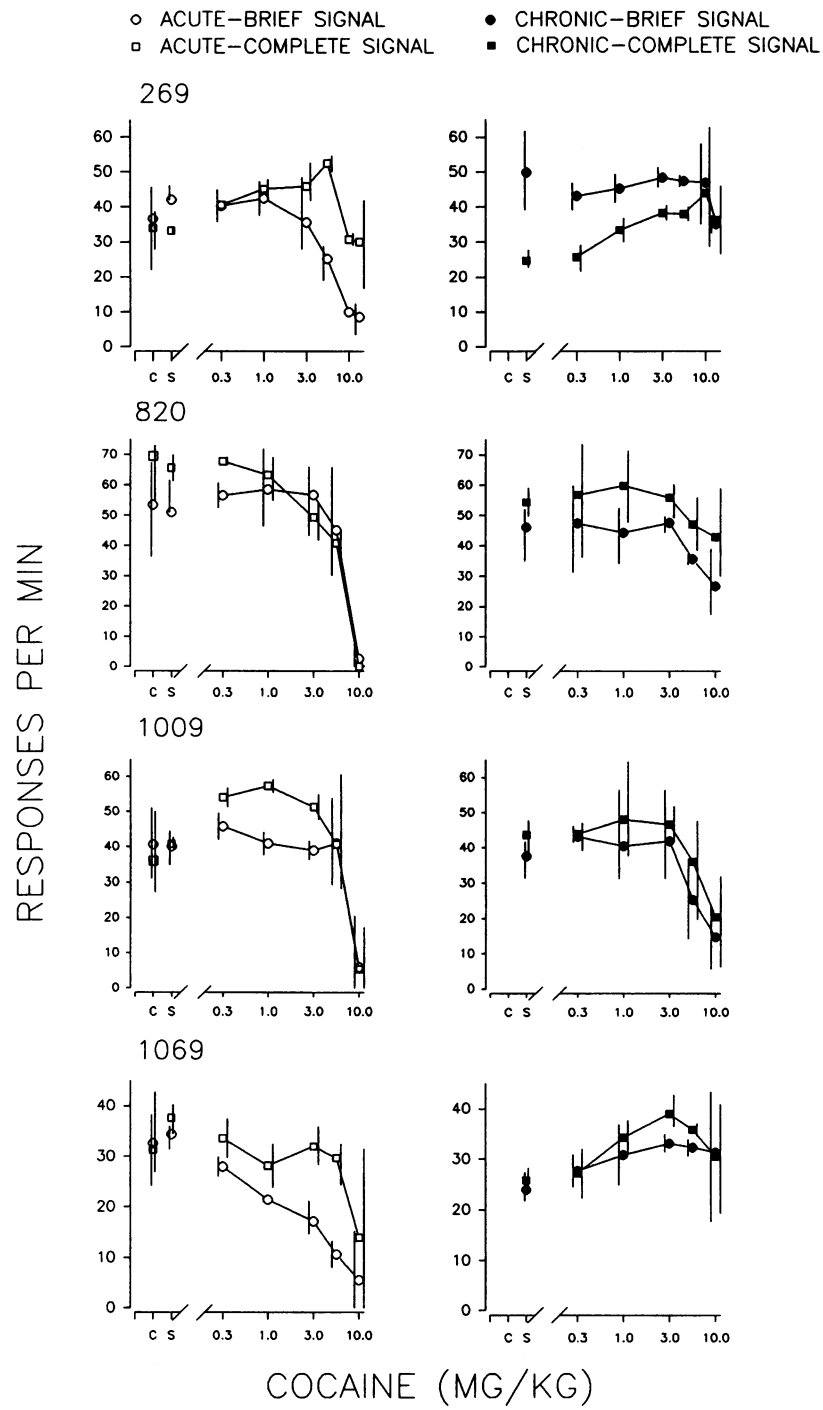

Fig. 2. Average overall response rates during VI segments from both components plotted as a function of dose of cocaine for all subjects during the acute drug phase (left panels) and the chronic drug phase (right panels). Average response rates under drug and saline conditions are means of rates from all sessions that were preceded by the indicated dose. Control rates are means of response rates for all sessions that immediately preceded sessions before which cocaine or saline was administered. Bars to the left of points represent ranges for the brief-signal component. Bars to the right of points represent ranges for the complete-signal component.

sions consisted of three blocks, each block consisting of a presentation of the brief-signal component followed by the complete-signal component.) Under conditions of acute administration, dose–response functions generally were similar from block to block in the two components of the multiple schedule. An exception was evident in the data of Subject 269, whose overall (whole-session) response rate in the complete-signal component did not decrease following acute administration of cocaine (see Figure 2). Response rates in this component during the first block of the session were decreased following acute administration of 10.0 mg/kg cocaine, but not during subsequent blocks. Consistent with results from the other pigeons, rate-decreasing effects of 10.0 mg/kg were not observed in the dose–response functions determined in the chronic drug phase. Rate decreases in the complete-signal component are not evident in Figure 2 because of increases in response rate in this component during the last two blocks of the session following acute drug administration.

To assess tolerance to the effects of cocaine, Figure 3 shows dose–response functions as a proportion of response rates under saline-administration conditions. Tolerance may be defined as a shift to the right of the dose–response curve following chronic drug administration. Alternatively, tolerance may be said to have occurred if dose effects are diminished following chronic drug administration. Clear shifts to the right of the dose–response curve were not observed in this experiment. However, response rate-decreasing effects of cocaine were diminished following chronic cocaine administration relative to acute administration for all subjects at some dose. For Subject 1009, in addition, response rate-increasing effects of cocaine during acute administration in the complete-signal component were absent following chronic cocaine administration. For Subject 269 in the complete-signal component and for Subject 1069 in both components, whose response rates following saline administration during the chronic drug phase were lower than during acute administration (see Figure 2), response rates during chronic cocaine administration increased relative to rates following saline administration.

Figure 4 shows rates of reinforcement, averaged over the entire session, plotted as a function of dose of cocaine during the acute and chronic drug phases for all subjects. Acute dose–reinforcer rate curves were similar in the two components. Performance by all subjects changed during chronic administration such that reinforcer-rate decreases due to cocaine's behavioral effects no longer occurred. Again, dose–reinforcer rate functions were similar in the two components during chronic administration as well.

Response rates during the delays themselves varied from subject to subject and are shown in Figure 5. Response rates during delay periods were very low for 3 subjects (all but Subject 820) under nondrug conditions. There was some tendency for lower rates to be increased by cocaine and higher rates to be decreased during both acute and chronic administration. Overall, however, response rates varied unsystematically across different delay periods and subjects.

DISCUSSION

The present results suggest that, in general, the two signaling procedures were equally effective in maintaining key pecking by pigeons, and the comparability in response rates under the two conditions across the phases of the experiment (see Figure 1) provides support for the notion that the brief and complete signals were equally effective as conditioned reinforcers. Response rates were similar in both components under nondrug conditions, acute drug effects were similar, daily cocaine administration usually resulted in tolerance to cocaine's rate-decreasing effects, and reinforcement-rate decreases observed during acute administration recovered during chronic administration to a similar extent in both components. Discontinuation of daily drug administration resulted in similar effects in the two components. Finally, removal of the signals resulted in similar declines in rate of responding in the two components. These similar patterns of decline validate the efficacy of the signals as conditioned reinforcers and provide evidence that is consistent with a view that the brief and complete signals in the present study were equally effective as conditioned reinforcers. They are also consistent with a view that emphasizes the importance of nondrug rate of

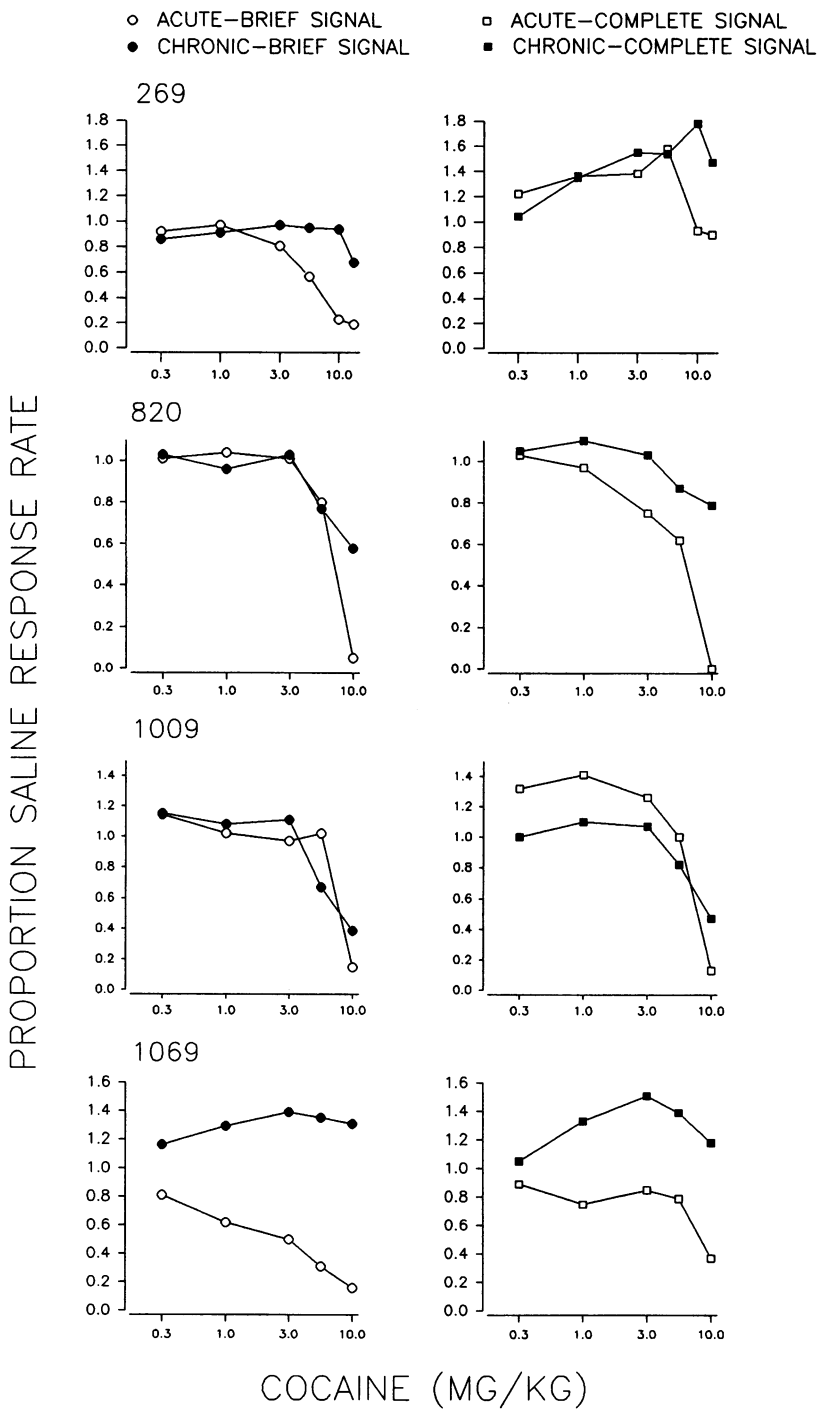

Fig. 3. Dose–response functions from both components plotted as proportion of saline response rates for all subjects during acute and chronic cocaine administration. Data from the brief-signal component are shown in the left panels, and data from the complete-signal component are shown in the right panels. Proportions are calculated by dividing response rates obtained following drug administration by response rates obtained under saline conditions. (Actual response rates are shown in Figures 1 and 2.)

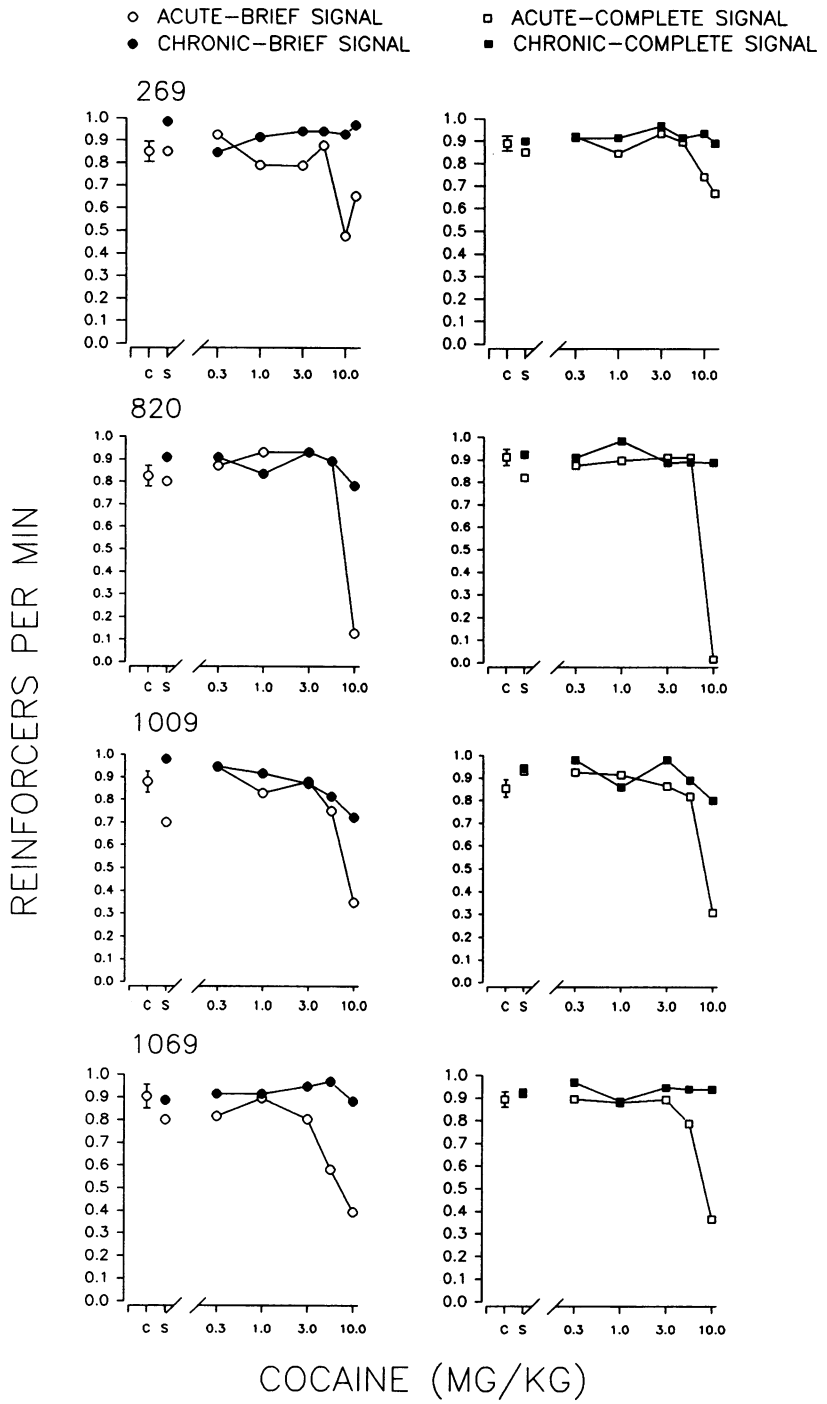

Fig. 4. Average overall reinforcement rates from both components plotted as a function of dose of cocaine for all subjects during acute and chronic drug phases. Bars around control points represent 95% confidence intervals.

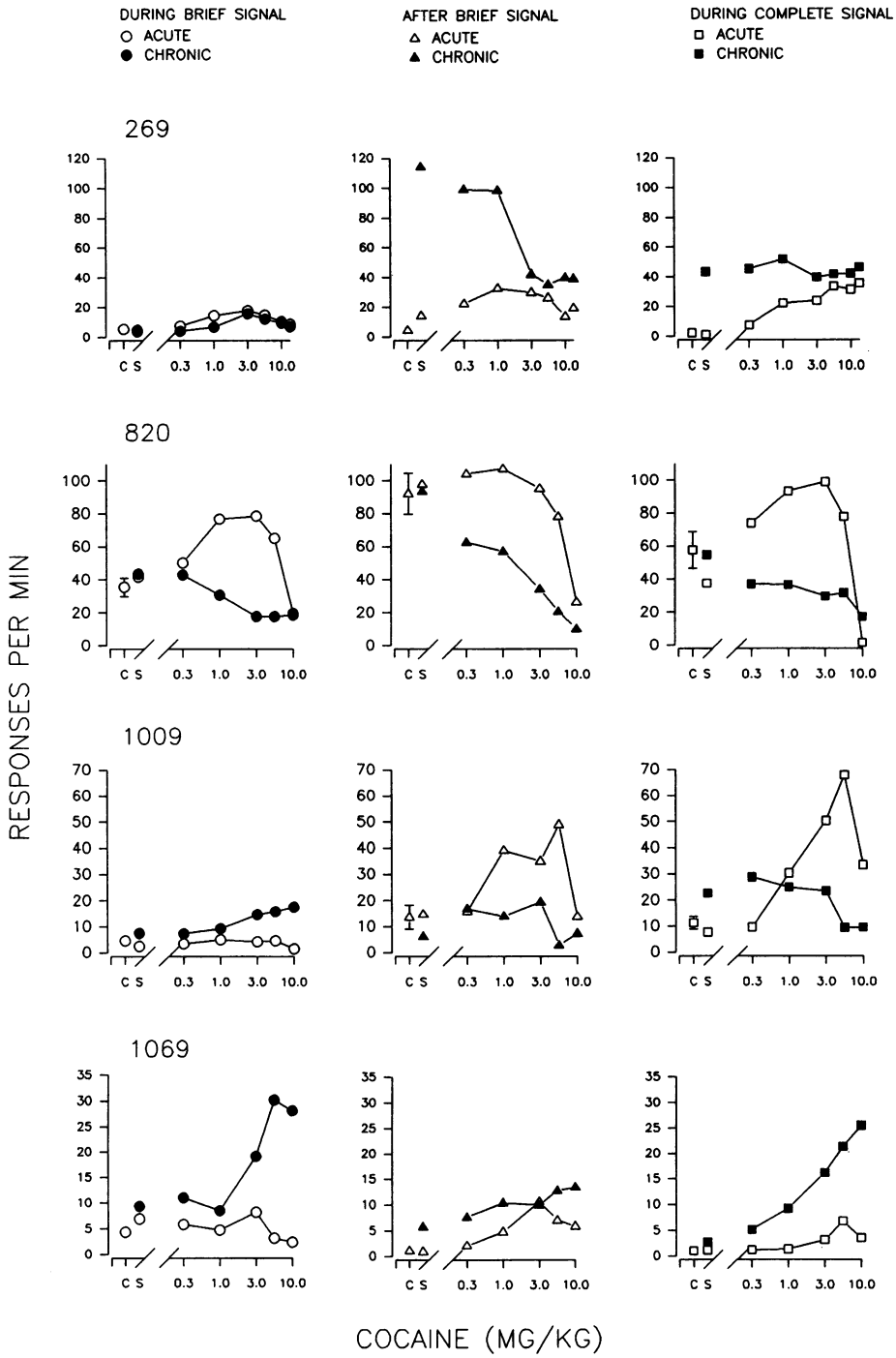

Fig. 5. Average overall response rates during delay periods plotted as a function of dose of cocaine for all subjects during acute and chronic drug phases. 95% confidence intervals are within control symbols. (Note scale differences.)

responding as a determinant of effects of acute and chronic cocaine exposure. Roughly equal response rates were established and roughly equivalent effects were observed in the two components of the multiple schedule (cf. Kelleher & Morse, 1968).

More consistent and substantial differences in drug effects between the two components might have been expected, given that the pairing operations of the two types of signals with food differed. According to Fantino (1977), the pairing hypothesis of conditioned reinforcement states that "the simple pairing of a stimulus with a primary reinforcer imparts conditioned reinforcing strength to that stimulus" (p. 313). In addition, the strength of that stimulus as a conditioned reinforcer is a function of the degree of contiguity between the stimulus and the primary reinforcer (where degree of contiguity is measured as the interval between stimulus offset and the presentation of the primary reinforcer). By this measure, the complete signal in the present experiment should have been maximally effective as a conditioned reinforcer because there was a 0-s interval between complete-signal offset and food delivery. The brief signal, on the other hand, should have been less effective than the complete signal because 15 or 21 s elapsed between offset of the signal and onset of food delivery.

The lack of evidence for differential efficacy of the two types of signals in the present study may have been a function of specific parameters of the two conditioning regimens. In this experiment, the brief-signal durations were manipulated until the shortest brief-signal duration that maintained response rates similar to those maintained by the complete signal was found. Schaal and Branch (1990) found that some brief-signal durations can maintain response rates similar to rates maintained by complete signals, whereas other, shorter brief-signal durations do not. Perhaps there is a threshold duration of brief signals that is as effective in maintaining behavior as are complete signals, and lengthening the brief signal further results in no further changes in performance or in effects of cocaine.

Fantino's (1977) delay-reduction hypothesis of conditioned reinforcement states that "the strength of a stimulus as a conditioned reinforcer is a function of the reduction in time to reinforcement correlated with the onset of that stimulus" (p. 313). According to this simple version of the hypothesis, the brief and complete signals should be equally effective as conditioned reinforcers because onset of both stimuli is correlated with a 27-s delay to food. The present results are consistent with this view.

The view that delay signals gain their function via pairing operations or by signaling a relative reduction in delay to primary reinforcement can be compared to that of Richards (1981), who has hypothesized that delay signals serve as discriminative stimuli for behavior that is adventitiously reinforced during the delay. According to this view, longer brief signals result in delay periods that are more discriminable, with the result that behavior during the delay interferes less with key pecking before the delay. Key-pecking rates are the only measures of behavior that we collected during delay periods (Figure 5). Response rates were indeed higher during the VI periods than during the delays. Because measures of other behavior were limited, however, the role of discriminatively controlled or elicited other behavior remains to be determined. The present results, nevertheless, support the view that brief and complete delay signals that have onsets equally distant in time from food maintained comparable response rates and were of equal efficacy by that measure.

One difference between Subject 269 and the other 3 subjects may account for the minor differences in performance observed between this pigeon and the others. This subject had an extensive history with delays and delay signals of various durations (Schaal & Branch, 1988, Experiment 2, 1990, Experiment 1). In fact, this subject's history included conditions in which the brief-signal stimulus (white keylight) had served as a complete delay signal. Despite the minor anomalies presented by the data of Subject 269, the overall picture is one that indicates considerable similarity in cocaine's effects, both acute and chronic, on response rates and reinforcement rates in the two components of the multiple schedule.

Tolerance developed to effects of doses of cocaine that, when administered acutely, affected behavior such that reinforcement frequency was decreased. This result is consis-

tent with Schuster, Dockens, and Woods' (1966) interpretation that tolerance is likely to develop to drug effects that initially result in reinforcement loss. A comparison of Figures 2 and 4 shows that Subject 269, whose overall response rates during the complete-signal component did not decrease under acute administration, nevertheless developed tolerance to the behavioral effects of cocaine that resulted in decreases in reinforcer rate during chronic administration. (The decreases in reinforcer rate were related to the decreases in responding in the first block of the session, as described in the Results.) These figures and Figure 3 also show, however, that tolerance developed to cocaine's rate-decreasing effects even at doses that resulted in little change in rate of reinforcement (e.g., Subject 269 during the brief-signal component at 5.6 mg/kg and Subject 1069 during the brief-signal component at 1.0 and 3.0 mg/kg cocaine; Figure 4). It is possible that the tolerance that develops to doses that do decrease reinforcement rate generalizes to other behaviorally active doses.

In the few cases in which cocaine-induced response-rate increases were observed, tolerance was also observed, especially for Subject 1009 (Figures 2 and 3) during the complete-signal component. Branch (1979) obtained similar results when *d*-amphetamine was administered to squirrel monkeys, and he related his findings to the possibility that tolerance is more likely to develop when the initial effect of the drug results in some cost to the subject. In the present experiment, the cost associated with the increases in response rates observed when cocaine was administered would be defined as an increased number of pecks per reinforcement. Whether cost, defined this way, has relevance as a behavioral factor influencing tolerance is a topic that merits further experimentation (Schama & Branch, 1994).

The present experiment also provided evidence concerning drug dependence. Behavioral dependence is often illustrated by a disruption of performance when chronic drug exposure ceases (Schuster & Thompson, 1969). Three subjects, despite more than 5 months of receiving the drug daily, showed performance that returned immediately to baseline levels when daily cocaine injections were replaced by daily saline injections (cf. Branch & Dearing, 1982). Subject 269 (Figure 1) appeared to exhibit a withdrawal effect. Interestingly, however, response rates for Subject 269 stayed lower than original baseline rates for all 41 sessions that followed cessation of drug administration, casting doubt on whether the effect seen immediately upon cessation of drug administration should be considered evidence of withdrawal.

The present study illustrates that behaviorally active drugs can serve to illuminate behavioral processes (cf. Branch, 1984; Thompson & Schuster, 1968). In the present study, similar performances were established under two sets of contingencies (i.e., completely signaled and briefly signaled delays). Challenged by cocaine injection, performance in the two components generally was affected similarly. These outcomes suggest that under the conditions of the current experiment, the brief and complete signals were equally effective in maintaining behavior. That cocaine did not interact differentially with the two types of signals in controlling behavior indicates that the two stimuli were equally effective conditioned reinforcers.

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