*COCAINE'S EFFECTS ON FOOD-REINFORCED PECKING IN
PIGEONS DEPEND ON FOOD-DEPRIVATION LEVEL*

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Four pigeons deprived to 80% of their laboratory free-feeding weights pecked keys under a multiple fixed-ratio 30 fixed-interval 5-min schedule of food presentation. Components alternated strictly with 15-s timeouts separating them; each was presented six times. When rates of pecking were stable, 2 pigeons' weights were reduced to 70%, and the other 2 pigeons' weights were increased to 82.5% to 85% of free-feeding levels. Cocaine (1.0, 3.0, 5.6, and 10.0 mg/kg and saline) was administered 5 min prior to sessions. When each dose had been tested twice, pigeons' weights were adjusted to the level that they had not yet experienced, and cocaine was tested again. Cocaine reduced response rates in a dose-dependent manner under the fixed-ratio schedule and under the fixed-interval schedule at high doses, and increased rates under the fixed-interval schedule at low doses. Reductions in pecking rates occurred at lower doses under both schedules in 3 of 4 pigeons when they were less food deprived compared to when they were more food deprived. Low doses of cocaine increased low baseline rates of pecking in the initial portions of the fixed-interval schedules by a greater magnitude when pigeons were more food deprived. Thus, food-deprivation levels altered both the rate-decreasing and rate-increasing effects of cocaine. The implications of these results for the mechanisms by which food deprivation increases cocaine self-administration and for the dependence of cocaine's effects on the baseline strength of operant behavior are discussed.

Key words: cocaine, fixed-interval schedules, fixed-ratio schedules, food deprivation, key peck, pigeon

Changes in levels of food deprivation of nonhuman subjects have been shown to alter several behavioral effects of drugs profoundly. Studies have shown that the rate-suppressive effects of drugs on operant behavior are obtained at lower doses when animals are less food deprived than when they are more severely food deprived (Cole, 1967; Gollub & Mann, 1969; Kelly & Thompson, 1988; Samson, 1986; Schaal & Branch, 1992). Schaal and Branch, for example, tested cocaine's effects on pigeons' key pecking under a fixed-ratio (FR) 30 schedule of food reinforcement when the pigeons were deprived of food to 70%, 80%, and 90% to 100% of their free-feeding weights. Rates of pecking were suppressed following administration of lower doses of cocaine when pigeons were relatively less food deprived.

Another frequently studied behavioral effect of changes in levels of food deprivation is the increase in drug self-administration

that typically accompanies food restriction (see Carroll & Meisch, 1984, for a review). The effect has been demonstrated in rats and monkeys self-administering drugs from several pharmacological classes, including cocaine and *d*-amphetamine (Carroll, France, & Meisch, 1981; Carroll & Stotz, 1983; de la Garza, Bergman, & Hartel, 1981), opiates (Carroll & Meisch, 1979), pentobarbital (Kliner & Meisch, 1982), phencyclidine and ketamine (Carroll & Meisch, 1980; Carroll & Stotz, 1983), ethanol (Meisch & Thompson, 1973; Oei & Singer, 1979), and delta-9-tetrahydrocannabinol (THC) (Takahashi & Singer, 1979, 1980). Schaal and Branch (1992) suggested that alterations of the rate-suppressive effects of drugs by food deprivation may be related to increases in drug self-administration produced by greater levels of food deprivation. Specifically, increases in drug-reinforced behavior may occur, in part, because the rate-suppressive effects of large, cumulative doses of self-administered drug are lessened.

Some findings have suggested, however, that other mechanisms are involved in increases in drug self-administration induced by food deprivation. For example, rates of self-administration are often increased under

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food deprivation *prior to* the ingestion of rate-suppressing doses of drug (Papasava & Singer, 1985). Carroll and Stotz (1983) showed that food deprivation not only increased rhesus monkeys' overall intake of *d*-amphetamine and ketamine but also lowered the dose at which the highest rates of responding were observed. Similar effects were observed for cocaine (de la Garza & Johanson, 1987; Papasava & Singer, 1985), *d*-amphetamine (de la Garza & Johanson, 1987), and delta-9-THC (Takahashi & Singer, 1979). Furthermore, Carroll (1985) showed that food deprivation increased rates of lip contacts of rhesus monkeys on a fluid-yielding spout under a second-order schedule in which lip contacts produced access to phencyclidine only at the end of a 60-min session. Finally, changes in the pattern of self-administration during the session have also been obtained (Carroll, 1987; Carroll, Lac, Walker, Kragh, & Newman, 1986; Carroll & Stotz, 1983, 1984). In these studies, when subjects were satiated, self-administration occurred at a fairly low, constant rate across the session. When subjects were food deprived, however, impressive bursts of intake early in sessions were followed by long periods without responding. These studies suggest that subjects become more sensitive to the reinforcing effects of low doses of drug when they are food deprived. However, because most studies do not present data on patterns of intake, the generality of this effect is unknown.

It is possible, however, that subjects also become more sensitive to the *rate-increasing* effects of low doses of drug when they are deprived of food. One of the more reliable generalizations from behavioral pharmacological research is that behavior that occurs at low rates is often increased by drugs, a fact that gave rise to the rate-dependency principle (Dews, 1958). Increases in drug self-administration due to food deprivation could reflect, in part, an enhancement of rate-increasing effects of low doses of drugs, so that food deprivation and the drug act synergistically to increase the rate of drug intake. Rate-dependent increases, typically obtained in the lowest part of the effective dose range, have been obtained with many drugs, including amobarbital, cocaine, *d*-amphetamine (and many other central nervous system stimulants), methadone, morphine, pentobarbital,

chlordiazepoxide, ethanol, chlorpromazine, and imipramine (Barrett, 1976; Byrd, 1979; Dews, 1958, 1964; Howell, Byrd, & Marr, 1986; Kelly & Thompson, 1988; Schama & Branch, 1994; Spealman, Goldberg, Kelleher, Goldberg, & Charlton, 1977; Spealman, Madras, & Bergman, 1989; Wenger, Donald, & Cunney, 1986; Zuccarelli & Barrett, 1980; see Kelleher & Morse, 1968, for a review). Rate-increasing effects of drugs are conveniently studied using fixed-interval (FI) schedules. These schedules typically produce patterns of responding that include pauses immediately after reinforcement followed by transitions to relatively high rates of responding toward the end of the interval. Low doses of many drugs produce increases in rates of responding in the early part of the interval and either reduce or do not affect high rates of responding later in the interval. An FI 5-min schedule was employed in the present study, then, to determine whether the rate-increasing effect of cocaine in pigeons is altered by changes in food-deprivation levels. The FI schedule alternated with an FR 30 schedule, thus producing a multiple FR 30 FI 5-min schedule that permitted the examination of both rate-decreasing and rate-increasing effects.

METHOD

Subjects

Four adult male White Carneau pigeons with no prior experimental experience were used as subjects. They were maintained at a given percentage of their free-feeding weights via postsession feeding. When not in experimental sessions, they were individually housed in a temperature-controlled colony where they were allowed free access to water and digestive grit.

Apparatus

Four custom-made experimental chambers, constructed of wood with aluminum front panels, were used. The internal dimensions of each chamber were 33 cm across the front panel, 31 cm from the front panel to the back wall, and 37.5 cm from floor to ceiling. Three response keys on the front panels were mounted 26 cm from the floor. The center key could be lit from behind with a green or a red light. The side keys were dark and inoperative. The middle key required a force

of approximately 0.19 N to record a response. A 28-V 1.1-W lamp 7 cm above the center key served as a houselight. A rectangular aperture 16 cm below the middle key provided access to a solenoid-operated food hopper. During hopper presentations, the aperture was lit with white light, and the houselight and keylights were extinguished. Extraneous sounds were masked by white noise and ventilation fans. Contingencies were programmed and data were collected by two MS-DOS-based 80386 microcomputers, programmed under Medstate® Notation (MED Associates, Inc. & Tatham, 1991).

Procedure

Experimental sessions were conducted 6 or 7 days a week at approximately the same time each day. Pigeons, deprived to 80% of their free-feeding weights, were trained to eat from grain hoppers; then pecks to the green center key were autoshaped (Brown & Jenkins, 1968). Brief exposure to small FR schedules preceded exposure to the terminal reinforcement schedule. The terminal schedule was a multiple FR 30 (green key) FI 5-min (red key) schedule. Reinforcement consisted of 4-s access to the grain hopper. Sessions began 5 min after pigeons were placed in the chambers with the illumination of the houselight and the center key lit green. After completion of the FR 30 requirement, food was presented followed by a 15-s period in which all lights were extinguished and pecks had no effect (i.e., a blackout). The red key and houselight were then illuminated and the FI 5-min schedule began. A 15-s blackout also separated the FI from the FR schedule. This sequence was repeated six times before the session was over. The time that components could remain in effect in the absence of pecking was limited to 2 min for the FR 30 and 8 min for the FI 5-min schedule. Thus, if no responding occurred, the components and their associated stimuli still alternated.

Several weeks after overall rates of pecking became stable (as judged visually), the pigeons were divided randomly into 2 pairs. One pair (78 and 53) were fed more food until their weights reached 85% of free-feeding weights, and the other pair (40 and 10) were reduced in weight over several days until they reached 70% of free-feeding weights. Pigeons 78 and 53 occasionally failed to peck

Table 1

Feeding conditions, order of feeding conditions, number of training sessions preceding drug tests, and pigeons' actual weights.

Subject	Feeding condition (%FFW)		Training sessions		Actual weights (g)	
	1 ^a	2 ^a	1	2	1	2
78	82.5	70	270	47	532	451
53	82.5	70	263	70	424	359
40	70	85	276	48	380	448
10	70	85	277	46	385	454

^a Numbers depict order of event.

during some components at their 85% weights. It was decided that a minimally acceptable baseline level of responding during the low-deprivation condition would include the collection of all available reinforcers. Therefore, these pigeons' weights were reduced to 82.5% of free-feeding weights, at which they consistently earned each reinforcer during baseline sessions. Cocaine and vehicle were administered to all pigeons twice at each body weight in the following mixed order: 3.0 mg/kg, 5.6 mg/kg, saline, 1.0 mg/kg, and 10.0 mg/kg. At least four baseline sessions, which were not preceded by cocaine or vehicle administration, separated each drug or vehicle test. Upon completion of two tests of each dose at the initial weights, pigeons' weights were increased or decreased to the level they had not yet experienced, and when response rates were stable again, cocaine and vehicle were tested as before. Thus, the order in which pigeons were exposed to the deprivation conditions (listed in Table 1) was counterbalanced across pairs.

Overall rates of pecking and the temporal location within the session of each peck and food presentation were collected daily. The latter data were used to examine rates during the 10 30-s segments of each 5-min interval, so that rate increases could be assessed.

Drug Administration

Cocaine HCl (NIDA) was dissolved in sterile 0.9% saline to be administered in a volume of 1.0 ml/kg body weight. Cocaine and vehicle were injected into the breast muscle, after which pigeons were placed in the chambers for the 5-min interval prior to the start of the session. For Pigeons 10 and 40, which

were initially maintained at 70% of free-feeding weights, cocaine doses were administered in a volume that was proportional to their 70% weights. This procedure reduced the absolute amount of drug the pigeons received compared to what they would receive at their 85% weights. It was decided that the injection volume for each bird during each deprivation condition should be fixed at the volume proportional to their 82.5% to 85% weights. Cocaine and vehicle were tested two more times, at the adjusted volumes, for Pigeons 10 and 40 at the 70% weights prior to increasing their weights to 85% of free-feeding weights.

RESULTS

The number of sessions of training prior to the first test of cocaine under both conditions of food deprivation, the order of the conditions, and each pigeon's actual weight are listed in Table 1. For all pigeons, rates and patterns of performance typical of the component schedules were obtained. Figure 1 illustrates representative baseline performance in the form of cumulative records for Pigeon 53, selected from sessions not preceded by injections under both deprivation conditions. A brief pause was followed by high, steady rates of pecking during the FR component. During the FI component, near-zero rates at the beginning of the intervals were followed by transitions to high terminal rates.

Figure 2 shows the effects of cocaine on overall rates of pecking under both conditions of food deprivation for both schedules of reinforcement. If cumulative records were not available for examination of patterns of responding within fixed intervals for a certain session (shown below), the next available preceding session was included in the computation of means of control rates. The administration of saline did not affect baseline rates of pecking. Overall rates of pecking maintained by the FR 30 schedule decreased as a function of dose under both levels of food deprivation for all 4 pigeons. Under the FI schedule, rates decreased at a lower dose for 3 of 4 pigeons under the 82.5% to 85% of free-feeding weight condition compared to the 70% of free-feeding weight condition. For Pigeons 40 and 10 under the 70% free-feeding weight condition, rates of pecking maintained by the FI schedule increased at the 3.0

mg/kg dose relative to the rate following saline. Rates after saline administration were lower under the 82.5% to 85% condition for 3 of 4 subjects.

Regardless of the schedule of reinforcement, rate decreases occurred at lower doses for 3 of 4 pigeons when they were maintained at 82.5% to 85% of free-feeding weights compared to when they were maintained at 70% of free-feeding weights (see Figure 2). Differences in cocaine's effects were most apparent following the 5.6 mg/kg dose. For Pigeons 53 and 78, rates following this dose were completely suppressed when weights were maintained at 82.5% of free-feeding weights, but were only moderately suppressed under the FR schedule when weights were maintained at 70% of free-feeding weights. Pigeon 10's rate of pecking on the FI schedule was half the control rate at the 5.6 mg/kg dose and was completely suppressed at the 10.0 mg/kg dose when maintained at 85% of free-feeding weight. When maintained at 70% of free-feeding weight, Pigeon 10's rate of pecking on the FI schedule was not completely suppressed until the 10.0 mg/kg dose. For Pigeon 40, rates of pecking on the FR and FI schedules were completely suppressed following 5.6 mg/kg under the 70% of free-feeding weight condition, whereas rates were either unaffected (FI) or suppressed (FR) under the 85% of free-feeding weight condition at this dose.

Figures 3 through 6 show, for Pigeons 53, 78, 10, and 40, respectively, response rates during successive 10ths of the FI. In general, cocaine increased response rates early in the interval. Response rates in latter parts of the interval were unchanged or suppressed. Rate increases in the first half of the interval were most evident following the administration of 3.0 mg/kg of cocaine for Pigeons 78, 40, and 10 and following 1.0 or 3.0 mg/kg for Pigeon 53. The rate-increasing effects of cocaine were more pronounced at 70% of free-feeding weights than at higher body weights for every subject. In general, both determinations of 1.0, 3.0, and 5.6 mg/kg produced greater increases in responding in the first half of the interval at 70% of free-feeding weights than at higher body weights. For Pigeon 40 (Figure 6), the second determination of 1.0 mg/kg at 70% of free-feeding weight produced greater increases in the first

Fig. 1. Representative cumulative record of pecking, obtained from Pigeon 53 at 70% of free-feeding weight (top panel) and 82.5% of free-feeding weight (lower panel), under a multiple FR 30 FI 5-min schedule of food reinforcement. Diagonal deflections of the pen indicate food presentations. The pen reset to the baseline after each food presentation.

half of the interval than both determinations at 85% of free-feeding weight. The first determination of this dose at this weight, however, did not increase response rates. The second administration of 5.6 mg/kg cocaine

increased this pigeon's rates when it was maintained at 85% of free-feeding weight but suppressed rates completely when it was tested at 70% of free-feeding weight.

Although within-interval rates for two ses-

53

Fig. 3. Effects of cocaine on rates of pecking in successive 30-s segments (i.e., 10ths) of the FI 5-min schedule, expressed as means of rates obtained during each interval, for Pigeon 53. The pecks that produced reinforcement and the time between that peck and the presentation of the reinforcer were not included in the calculation of these points. The left panels show performance at 70% of free-feeding weight; the right panels show performance at 82.5% of free-feeding weight. Closed circles represent the mean rates from control sessions that were not preceded by the administration of cocaine or saline (there were 10 such sessions for each body weight). Control sessions immediately preceded the session prior to cocaine or saline administration. If records were not available for this session, the next available preceding session was analyzed. Broken lines indicate one standard deviation above and below control points. Upright open triangles represent the effects of first determinations, and inverted open triangles represent the effects of second determinations. The effects of a dose are not depicted if rates were reduced to zero or very near zero by that dose. Records from the first determination of 3.0 mg/kg at 70% of free-feeding weight were unavailable.

sions are unavailable due to a computer error, inspection of the range bars in Figure 2 reveals that the overall response rates obtained during these sessions very nearly

matched those obtained in the sessions represented in Figure 3 (Pigeon 53) and Figure 4 (Pigeon 78), suggesting that the within-interval rates during those sessions were not

Fig. 4. Effects of cocaine on rates of pecking in successive 10ths of the FI for Pigeon 78. Records were unavailable for the first determination of 3.0 mg/kg at 82.5% of free-feeding weight. Other details are as in Figure 3.

substantially different from those represented in Figures 3 and 4.

DISCUSSION

Cocaine reduced overall response rates of 3 of 4 pigeons maintained by FR and FI schedules at lower doses when pigeons were relatively less food deprived compared to when they were more severely deprived. Cocaine was more likely to increase response rates during fixed intervals when pigeons

were more food deprived. Finally, increases in response rates were larger in magnitude when pigeons were more food deprived. Thus, food deprivation altered both rate-decreasing effects of high doses and rate-increasing effects of low doses of cocaine. Specific aspects of these data will be considered with respect to their implications for the mechanisms by which food deprivation alters drug-reinforced behavior and whether these effects may be considered to be indications that cocaine's effects are altered by a behav-

Fig. 5. Effects of cocaine on rates of pecking in successive 10ths of the FI for Pigeon 10. Other details are as in Figure 3.

ior's strength as indicated by its resistance to drug-induced disruption.

Cocaine's rate-reducing effects were obtained at lower doses in 3 of 4 pigeons when they were relatively less food deprived (82.5% to 85% of free-feeding weights). The same alteration of cocaine's rate-suppressive effects by alterations in body weight was also observed in pigeons pecking under a single FR 30 schedule of food presentation (Schaal & Branch, 1992). In that study, however, differences in cocaine's effects were most reliably

observed when 90% to 100% of free-feeding weight was compared to 70% of free-feeding weight. In several cases, dose-effect curves obtained at 80% free-feeding weight did not fall neatly between those obtained at the extreme body weights. In the current study, increases in body weight above 82.5% or 85% resulted in occasional failure to respond on a schedule within the allotted time limit; thus, available reinforcers were missed. Weights were adjusted to levels that assured that pigeons would earn all the available reinforcers

Fig. 6. Effects of cocaine on rates of pecking in successive 10ths of the FI for Pigeon 40. Other details are as in Figure 3.

during sessions without drug; thus, the low-food-deprivation condition was arranged by maintaining pigeons at 82.5% to 85% of free-feeding weights. Although that difference in body weights was sufficient to alter cocaine's rate-suppressive effects in 3 of 4 pigeons, it may not have been sufficient to alter Pigeon 40's sensitivity to the extent observed in the other pigeons, as indicated, perhaps, by the effects of 5.6 mg/kg cocaine in this pigeon. It should be noted, however, that the effect of 3.0 mg/kg cocaine under the FR schedule

at the two deprivation levels matched that obtained for the other pigeons (i.e., rates were suppressed by this dose when Pigeon 40 was maintained at the heavier weight but not at the lighter weight). In addition, four complete determinations of cocaine's effects (two at the volume proportional to 70% of free-feeding weight) had been tested in this pigeon (and in Pigeon 10) prior to determining cocaine's effects under the 85% of free-feeding weight condition. Although it cannot be determined from the present ex-

perimental design, the possibility exists that the smaller decreases in rates following administration of 5.6 mg/kg cocaine when Pigeon 40 was maintained at 85% of free-feeding weight may reflect the development of tolerance to the drug's rate-decreasing effects with repeated acute administrations. This suggestion is weakened, however, by the absence of a similar effect in Pigeon 10, which had experienced the same number of cocaine tests. It is not known for certain, therefore, why cocaine's effects differed for Pigeon 40.

For each subject, low doses of cocaine (1.0 and 3.0 mg/kg) increased response rates in the first half of fixed intervals to a greater degree when pigeons were relatively more food deprived than when relatively satiated (Figures 3 through 6). Even for Pigeon 40, whose rates were completely suppressed by 5.6 mg/kg when relatively more food deprived, rate *increases* following 3.0 mg/kg were greater than those obtained when this pigeon was maintained at 85% of free-feeding weight. Evidence is provided, therefore, that more severe food deprivation both enhances the rate-increasing effects of cocaine and attenuates cocaine's rate-decreasing effects.

The enhancement by food deprivation of cocaine's rate-increasing effects may have implications for the increase in cocaine and other drug self-administration typically observed when experimental subjects are food deprived. In three studies, when rhesus monkeys were relatively more food deprived, prolonged, high-rate bursts of responding maintained by access to cocaine (Carroll et al., 1986), phencyclidine (Carroll, 1985; Carroll & Stotz, 1984), *d*-amphetamine, and ketamine (Carroll & Stotz, 1983) were observed. These findings suggest that the combination of food deprivation and self-administered drug acted synergistically to produce large increases in response rates. Thus, part of the reason animals self-administer more drug when they are food deprived may be that food deprivation enhances the rate-increasing effects of self-administered drug. It should be noted again, however, that such a mechanism should only be considered a contributing factor in accounting for the effects of food deprivation on drug self-administration. Other factors, including increases in sensitivity to the reinforcing effects of low doses of drug (de la Garza & Johanson, 1987;

Papasava & Singer, 1985; Takahashi & Singer, 1979) and decreases in susceptibility to the rate-suppressive effects of high doses of self-administered drug (Schaal & Branch, 1992), must also be considered.

Finally, food deprivation's effects in this and other studies (Schaal & Branch, 1992) suggest that rate-suppressive effects of drugs on operant behavior are determined in part by the behavior's strength, as indicated by its resistance to suppression by the drug. This conception of response strength has been proven useful in dozens of circumstances by the work of Nevin and colleagues (Nevin, 1974, 1992). Despite evidence to the contrary (e.g., Cohen, 1986), several studies suggest that its utility extends as well to understanding how variables alter the behavioral effects of drugs. The strategy is to determine whether the rate suppression produced by drugs is modified by variables that modify the effects of various behavioral operations, such as extinction, prefeeding, and the presentation of response-independent reinforcers (Nevin, Mandell, & Atak, 1983; Nevin, Mandell, & Yarensky, 1981). For example, rate suppression in response to these operations is inversely related to the baseline frequency of reinforcement (Nevin et al., 1983). Likewise, the effects of the opiates methadone and buprenorphine have been shown to be inversely proportional to the baseline frequency of reinforcement associated with five alternating variable-interval (VI) schedules (Egli, Schaal, Thompson, & Cleary, 1992).

A response-strength interpretation of cocaine's disruptive effects would be bolstered by demonstrations that variables that alter response strength also alter cocaine's effects. In the current experiment (and in Schaal & Branch, 1992), food deprivation increased the resistance of behavior to cocaine-induced suppression. Likewise, cocaine reduces response rates less under small FR schedules than under larger ones (Hoffman, Branch, & Sizemore, 1987; Hughes & Branch, 1991). Cocaine has also been shown to reduce response rates of pigeons maintained under a VI 60-s schedule of food presentation delayed by 27 s in a manner that depended on the proportion of the delay that was signaled; rates were more resistant to suppression when the entire delay was signaled than when a small portion of the delay was signaled

(Hughes, Branch, & Schaal, 1989). Psychomotor stimulants (and other drugs) typically decrease accuracy on repeated-acquisition-of-behavioral-chains tasks to a greater extent during the "learning" portion of the session, when animals are learning an unfamiliar chain, than during the "performance" portion, when animals are performing a well-learned chain (Schrot, Boren, Moerschbacher, & Simoes Fontes, 1978; Thompson, 1976). Cocaine reduced accuracy of pigeons on a delayed matching-to-sample task to a greater extent at longer delays (i.e., 4 s) than at shorter ones (i.e., 0.5 s; Branch & Dearing, 1982). In each of these studies, cocaine and other stimulants had larger rate- or accuracy-reducing effects on behavior that could be conceived of as "weaker."

Despite the cogency of such an interpretation, other results are not consistent with a response-strength interpretation of cocaine's effects. For example, although parameter-dependent rate suppression has frequently been observed under ratio schedules, the effects obtained with comparable interval schedules are often not parameter dependent (Branch, 1990; Schama & Branch, 1989). Cohen's (1986) tests of *d*-amphetamine in rats lever pressing on chained and multiple FI or random-interval schedules produced little evidence of schedule or link dependence, and hence also fail to support the response-strength interpretation. Finally, although cocaine disrupted performance of behavioral chains of varying length in squirrel monkeys, the disruption was not clearly altered by the chain length (Branch & Sizemore, 1988).

These consistent and inconsistent results neither fully endorse nor condemn a response-strength analysis of cocaine's effects at this time. They may, in fact, set the agenda for a program of research in which the values of behavioral variables (e.g., rate, magnitude, and quality of reinforcement, delay to reinforcement, response characteristics, etc.) are varied parametrically to assess their contribution to differences in cocaine's behavioral effects. A response-strength analysis may provide a means with which to determine whether and to what extent cocaine's behavioral effects are dependent on behavioral variables.

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