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Cocaine and automaintained responding in pigeons: Rate-reducing effects and tolerance thereto with different durations of food delivery

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ABSTRACT

Pigeons were exposed to an automaintenance procedure in which 6-s key illuminations in one color (red or white) were immediately followed by 3-s food deliveries and key illuminations in the other color were followed by 9-s food deliveries. Both conditions engendered consistent responding. With both durations of food delivery, acute and chronic cocaine administrations (1.0–17.8 mg/kg) produced dose-dependent decreases in mean percent trials (key illuminations) with a response and mean total response per session. Tolerance developed to the disruptive effects of cocaine on both response measures. Food duration did not significantly affect either response measure or significantly interact with cocaine dose or drug regimen. The orderliness of the present findings, like those of a related study examining whether probability of food delivery modulated the effects of cocaine on automaintained responding [Porritt, M., Arnold, M., Poling, A., Cocaine and automaintained responding in pigeons: rate-reducing effects and tolerance thereto with different CS–US pairing probabilities. Pharmacol Biochem Behav 2007; 87:405–411.], suggests that the automaintenance procedure is a useful assay for examining tolerance to drug effects on classically-conditioned responding. Unlike the results of that study, however, the present findings are inconsistent with a behavioral momentum analysis of drug effects on such responding.

1. Introduction

The concept of behavioral momentum, developed by Nevin (1974, 1988, 1992), proposes (a) that resistance to change is a better measure of the "strength" of responding than response rate and (b) that frequency of reinforcement is the primary determinant of resistance to change. Nevin proposed that behavioral momentum, defined as a particular response's resistance to change, is primarily a function of how frequently a reinforcer, such as food, is delivered in the context of the antecedent stimulus exercising control over the response in question. He also proposed that magnitude of reinforcement directly affects resistance to change (Nevin, 1974). Although the behavioral momentum metaphor has been used primarily to conceptualize schedule-controlled operant behavior (e.g., responding under variable-ratio or variable-interval schedules of food delivery, see Nevin, 2002; Nevin and Grace, 2000), it also could be applied to automaintained responding (Porritt et al., 2007).

Automaintained responding is responding that persists across sessions under an autoshaping procedure. Brown and Jenkins (1968) first described the autoshaping procedure and noted that it involved "the standard arrangement for classical conditioning" (p. 7), in which pairings were arranged between a conditional stimulus (CS), key illumination, and an unconditional stimulus (US), food delivery. As Brown and Jenkins noted, although the experimenter arranges only stimulus–stimulus relations under the autoshaping (or automaintainence) procedure, because food delivery quickly follows any peck that occurs during a trial (key illumination), it is probable that pecks are affected by response-independent (i.e., "adventitious" or "superstitious") operant reinforcement, in addition to classical conditioning mechanisms. The relative contribution of classical conditioning and operant conditioning learning processes to autoshaped (and automaintained) responding has been long debated (e.g., Schwartz and Gamzu, 1977) but never entirely resolved. Indeed, it may be impossible to do so experimentally. Be that as it may, such procedures have proven valuable for delineating drug effects on the acquisition and retention of information (see review by Sparber, 2001).

Research on drug effects on automaintained performance has primarily focused on acute actions of drugs. To our knowledge, only one study (Porritt et al., 2007) has used automaintainance procedures to study tolerance or to examine whether drug effects on such responding are consistent with a behavioral momentum analysis. Porritt et al. investigated the effects of acute and chronic administrations of cocaine (1.0–17.8 mg/kg) on the automaintained responding of pigeons under conditions in which 6-s red, green, and white key illuminations were followed by food with a probability of 0.25, 0.5, and 1.0, respectively. Substantial responding occurred at all probabilities. Acute and chronic cocaine administration reduced percent trials with a response and total responses per session in dose-dependent fashion, with tolerance observed when acute and

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chronic dose–response curves were compared. In general, the disruptive effects of cocaine were larger when the probability of food delivery was 0.25 or 0.5 than when it was 1.0. Porritt et al. suggested that these results are consistent with a behavioral momentum analysis of drug effects. That is, behavior was stronger (more resistant to drug-induced disruption) when the rate of reinforcement was higher, although response rates tended to be lower.

According to a behavioral momentum analysis, if rate of reinforcement is held constant, then magnitude of reinforcement should directly affect resistance to change, with larger magnitudes leading to greater response strength (Nevin, 1974). Research on classical conditioning supports this analysis with respect to resistance to extinction (Savastano and Miller, 2004). Little is known, however, regarding the effects of reinforcer magnitude on resistance to other perturbations, such as drugs, under classical conditioning paradigms, and the extension of the behavioral momentum metaphor to Pavlovian conditional responses, such as automaintained responding, deserves further attention (Grace and Nevin, 2004). Moreover, given the importance of behavioral factors in the occurrence of tolerance (Branch, 1993), further examination of such factors is of interest. Therefore, the present study determined the extent to which reinforcer magnitude (duration of food delivery) influenced the resistance of automaintained responding in pigeons to disruption by cocaine under acute and chronic conditions.

2. Method

2.1. Subjects

Five experimentally-naïve adult female pigeons, obtained from Palmetto Pigeon Plant (Sumter, SC), served as subjects. They were individually housed with unlimited access to water and grit in a temperature-controlled colony room (20–22 °C), maintained under a 12-hr light/12-hr dark schedule. Throughout the experiment, access to food was restricted to maintain each bird at approximately 80% of its free-feeding weight. This study was approved by the Institutional Animal Care and Use Committee and conducted in accordance with the *Guide for the Care and Use of Laboratory Animals* promulgated by the National Research Council (1996).

2.2. Apparatus

Four commercially available test chambers (MED Associates, St. Albans, VT) housed in sound attenuating shells were used. The front wall of each chamber contained three response keys, symmetrically located 24 cm above the floor, and a horizontally centered opening through which mixed grain could be reached when the food hopper was raised. Each response key could be illuminated in red, white, or green. A white bulb centered at the top of the back wall provided ambient chamber illumination and an exhaust fan provided continuous ventilation and masking noise. A personal computer equipped with MED PC® software was used to collect data and to arrange experimental events.

2.3. Behavioral procedure

The behavioral procedure was a discrete-trials procedure similar to that used by Porritt et al. (2007). In the present study, 6-s key illuminations were immediately followed by either 3- or 9-s access to food, regardless of the bird's behavior. Half of the key illuminations, selected at random with the provision that each color appeared equally often each session, were red. The other key illuminations were white. The key that was illuminated on a given trial (left, center, and right) also was selected at random, with the provision that each key was lighted equally often each session. For three birds, red key illuminations were always followed by 3-s food deliveries and white key illuminations were always followed by 9-s food deliveries. For the other two birds, red key illuminations were always followed by 9-s food deliveries and white key illuminations were always followed by 3-s food deliveries. Trials were separated by a variable inter-trial interval (ITI) with a mean length of 45 s and a range of 15 to 120 s. All keys were darkened during the ITI. Daily sessions comprised 24 trials and were conducted at about the same time each day (during the light portion of the light/dark cycle), seven days a week.

2.4. Pharmacological procedure

The automaintenance procedure was in effect for 25 sessions prior to pharmacological testing. By the end of this period, all birds regularly pecked during red and white key illuminations, and the percentage of trials with one or more responses (percent trials with a response) showed no obvious trend across 5 consecutive sessions. The same is true of the total number of responses per session.

Acute drug testing involved the administration of 1.0, 3.2, 5.6, and 10 mg/kg of cocaine to each bird. No higher dose was given because 10 mg/kg greatly reduced responding. Each bird received every dose twice, in an irregular order. Cocaine was given every third day; the session immediately preceding drug testing was a vehicle-control (0 mg/kg) session and the day preceding vehicle injection was a baseline session with no injection given. Throughout the study, cocaine hydrochloride (Sigma, St. Louis) was dissolved in isotonic saline solution and injected into the breast muscles at a volume of 1 ml/kg 5 min before behavioral testing. Vehicle (isotonic saline solution) was administered in comparable fashion.

During chronic administration, each bird received a daily dose of 5.6 mg/kg cocaine 5 min before the onset of behavioral testing. After 20 consecutive sessions of exposure to this dose, percent trials with a response and total responses per session showed no obvious trend across 5 consecutive sessions and post-chronic testing began. During chronic testing, doses of 0 (vehicle), 1.0, 3.2, 10, and 17.8 mg/kg, as well as the usual chronic dose of 5.6 mg/kg, were evaluated. Each bird received every dose other than 5.6 mg/kg twice, in an irregular sequence. Tests of doses other than 5.6 mg/kg were separated by 2 consecutive sessions during which 5.6 mg/kg was administered. When 0, 1.0, or 3.2 mg/kg doses were administered prior to the session, sufficient cocaine to equal the 5.6 mg/kg daily dose was administered immediately after behavioral testing.

2.5. Dependent variables and data analysis

We measured two dependent variables: (a) percent of trials with a response, and (b) total number of responses across all trials. For data analyses, data points for each bird are the means of the two administrations. We used three-factor repeated-measures analysis of variance (RM ANOVA) to analyze these data. Because pigeons did not receive 17.8 mg/kg in the acute phase, the RM ANOVA results exclude this dose. The RM ANOVA included the following within-subject factors: regimen (acute vs. chronic), duration of food delivery (3- vs. 9-s), and dose of cocaine (1.0, 3.2, 5.6, and 10.0 mg/kg). All data were transformed to percent vehicle control prior to testing. When significant interactions were present, simple effect tests were conducted using paired-samples *t* tests, with the Bonferroni correction applied to adjust the Type I error rate. These tests were only conducted when the graphed means did not overlap.

To characterize the development of tolerance, ED_{50} values and associated 95% confidence intervals (95% CIs) were computed for the acute and chronic data (cf., Tallarida, 2000, pp. 26–31). All doses were transformed to $\log_{10}$ (dose) for these calculations. The ED_{50} values for $\log_{10}$ (dose) were obtained by fitting regression lines to data points between 80% and 20% (or nearest values) of vehicle-control values on the descending limb of the dose–response curve. All tests were conducted with familywise $\alpha = .05$. The data for both dependent

measures met the RM ANOVA sphericity assumption. SPSS for Mac OSX (v. 16; SPSS, Inc., Chicago, IL) was used to analyze data; GraphPad Prism for Mac OSX (v. 5.0; GraphPad Software, Inc., San Diego, CA) was used to analyze data and create graphs.

3. Results

3.1. Percent of trials with a response

Fig. 1 (Panel A) depicts vehicle-control data for percent trials with a response across all experimental conditions. Paired-samples *t* tests revealed no significant differences (both $p>.10$) between the two food durations under acute (95% CI of the difference = −11.81, 4.61) or chronic (95% CI of the difference = −27.21, 5.61) conditions. Likewise there were no significant differences (both $p>.30$) between the acute and chronic regimen at either 3-s (95% CI on the difference = −15.69, 35.09) or 9-s (95% on the difference = −15.92, 20.92) food duration. Thus, the vehicle-control data appeared comparable under all conditions.

Fig. 2 (Panel A) depicts the mean percent of trials with a response (±1 SE) for all pigeons under all experimental conditions. These data are expressed in terms of percent vehicle control. The food duration by regimen by dose interaction was not significant, $F(3, 12)=0.24$, $p=.87$, MSE = 143.68. That is, the significant regimen by dose interaction [$F(3,12)=5.59$, $p=.01$, MSE = 682.54] did not significantly differ as a function of the duration of food delivery. In other words, across both durations of food delivery, the form of the regimen by dose interaction was similar. This can be seen in Fig. 2, in which the dose–response curves for the two food duration conditions show a similar form within each regimen condition, whereas the regimen curves show separation across the intermediate doses. The significant regimen by dose interaction indicates that tolerance occurred to the effects of cocaine, with the drug producing larger reductions in the percent of trials with a response under acute than under chronic conditions.

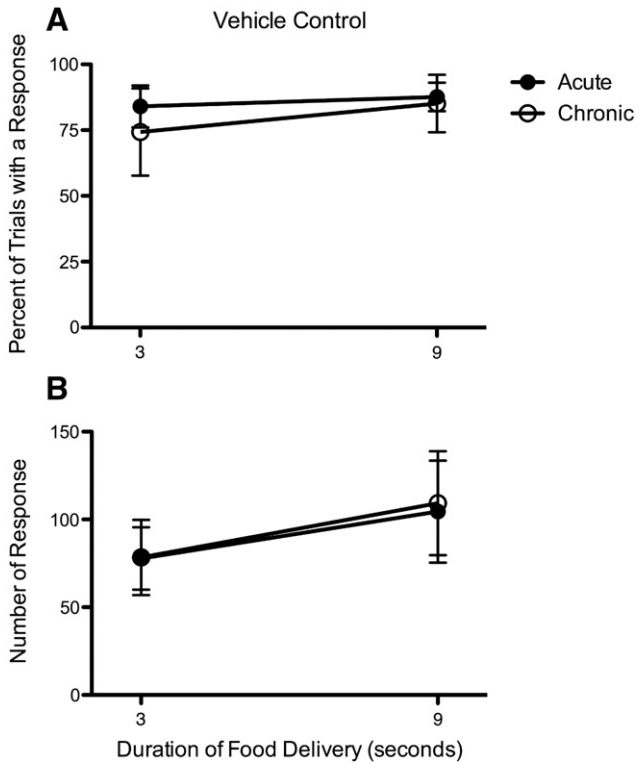

Fig. 1. Vehicle-control data for the food duration and drug regimen conditions. Panel A depicts the mean (±1 SE) percent of trials with a response and Panel B depicts the mean (±1 SE) number of responses. Closed symbols represent acute data and open symbols represent chronic data. Note that the two *Y*-axes have different scales.

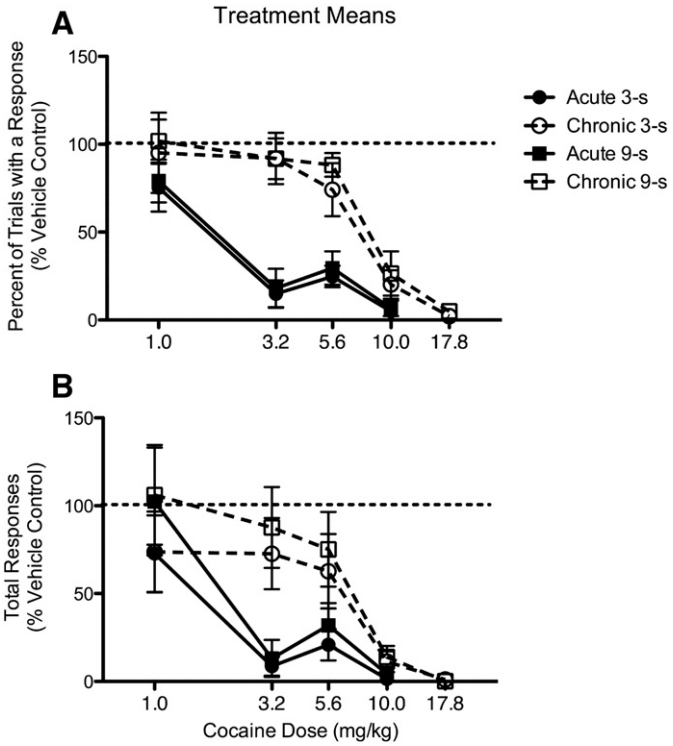

Fig. 2. Dose–effect data for cocaine under acute (closed symbols) and chronic (open symbols) conditions for the 3-s (circles) and 9-s (squares) food durations. Panel A depicts the mean (±1 SE) percent of trials with a response and Panel B depicts the mean (±1 SE) number of responses. All drug data are expressed as a percentage of the mean vehicle-control level. Note that the horizontal dashed line indicates 100% vehicle-control level responding and that the *X*-axis is depicted in logarithmic units.

Because the dose–effect curves for the two food durations overlapped (and there was no significant main effect for food duration — see below), their data were combined for comparisons of acute vs. chronic conditions. The percent of trials with a response was significantly reduced by 3.2 mg/kg of cocaine administered acutely ($M=16.46$, SD = 20.15) vs. chronically ($M=91.85$, SD = 27.84), $t(9)=-7.95$, $p<.0001$, two-tailed, SE = 9.49, mean difference (MD) = 75.39% (95% CI = −96.85, −53.93). Similar results were found at the 5.6 mg/kg dose after acute ($M=27.16$, SD = 16.96) vs. chronic ($M=81.20$, SD = 25.73) administration, $t(9)=-6.61$, $p<.0001$, two-tailed, SE = 8.18, MD = 54.04% (95% CI = −72.55, −35.53). Because the dose–response curves for the 3-s and 9-s food-delivery durations greatly overlapped (and the two conditions did not significantly differ), their data were combined to calculate the ED_{50} values for the acute and chronic conditions. The ED_{50} for the acute phase was 1.95 mg/kg (95% CI = 1.31, 2.60 mg/kg) and the ED_{50} for the chronic phase was 7.67 (95% CI = 6.81, 8.71). These ED_{50} values further confirm that tolerance developed to the effects of cocaine.

The regimen by food duration interaction was not significant, $F(1, 4)=1.21$, $p=.33$, MSE = 44.56. That is, averaging across all doses, the difference between the acute and chronic conditions did not significantly differ across the two food conditions. The dose by food duration interaction also was not significant, $F(3, 12)=0.70$, $p=.57$, MSE = 78.65. Put simply, the effects of cocaine did not appear to differ across the two food durations (when ignoring regimen). Given the presence of the significant regimen by dose interaction, the significant main effects of these two factors should be interpreted with caution. The main effects for regimen and dose were significant, $F(1, 4)=17.37$, $p=.014$, MSE = 2036.10 and $F(3, 12)=12.93$, $p<.001$, MSE = 13.89.97, respectively. In contrast, the main effect of food duration was not

significant, $F(1, 4) = 4.16$, $p = .11$, MSE = 121.69. To summarize these findings with respect to the percent of trials with at least one response: (a) tolerance developed to the effects of cocaine (significant regimen by dose interaction, increased ED_{50} values in the chronic vs. the acute condition); (b) food duration did not modulate the effects of the other independent variables (no significant interactions involving this factor) and responding did not differ across the two durations (no significant main effect of this factor); and (c) cocaine and the drug administration regimen influenced the percent of trials with at least one response (significant main effects of each factor), but these main effects must be qualified due to the presence of the significant interaction involving these two independent variables.

3.2. Number of responses

Fig. 1 (Panel A) depicts vehicle-control data for percent trials with a response across all experimental conditions. Paired-samples t tests revealed no significant differences (both $p > .10$) between the two food durations under acute (95% CI of the difference = −64.85, 11.45) or chronic (95% CI of the difference = −74.66, 12.86) conditions. Likewise there were no significant differences (both $p > .30$) between the acute and chronic regimen at either 3-s (95% CI on the difference = −32.59, 31.39) or 9-s (95% on the difference = −33.35, 23.75) food duration. Again, the vehicle-control data appeared comparable under all conditions.

Fig. 2 (Panel B) depicts the mean number of responses (±1 SE) for all pigeons under all experimental conditions. The food duration by regimen by dose interaction was not significant, $F(3, 12) = 0.19$, $p = .90$, MSE = 146.42, indicating that the form of the significant regimen by dose interaction [$F(3, 12) = 5.58$, $p = .01$, MSE = 868.40] did not differ as a function of duration of food delivery. The significant regimen by dose interaction demonstrates that tolerance developed to the effects of cocaine, with fewer responses occurring under acute vs. chronic conditions across intermediate doses.

Because the dose–effect curves for the two food durations overlapped (and there was no significant main effect for food duration — see below), their data were combined for comparisons of acute vs. chronic conditions. The test of acute vs. chronic conditions at 3.2 mg/kg was significant, $t(9) = -4.56$, $p = .001$, two-tailed, SE = 15.19, MD = −69.32, (95% CI = −103.68, −34.96), as was the test at 5.6 mg/kg, $t(9) = -3.99$, $p = .003$, two-tailed, SE = 10.67, MD = −42.55 (95% CI = −66.69, −18.41). As with the percent of trials with a response, the dose–response curves for the number of responses under both the 3-s and 9-s food-delivery durations greatly overlapped (and the two conditions did not significantly differ), so their data were combined to calculate the ED_{50} values for the acute and chronic conditions. The ED_{50} for the acute phase was 2.13 mg/kg (95% CI = 1.23, 3.16 mg/kg) and the ED_{50} for the chronic phase was 6.05 (95% CI = 4.65, 8.27). The larger chronic value further confirms that tolerance developed to the effects of cocaine.

Neither the dose by food duration [$F(3, 12) = 2.04$, $p = .16$, MSE = 358.14] nor the regimen by food duration [$F(1, 4) = 0.16$, $p = .71$, MSE = 427.58] interactions were significant. Therefore, duration of food delivery did not significantly modulate the effects of cocaine (insignificant dose by delivery interaction) or the development of tolerance (insignificant regimen by delivery interaction). The main effects of regimen approached significance, $F(1, 4) = 5.09$, $p = .087$, MSE = 3770.60. The main effect of food duration also failed to reach significance, $F(1, 4) = 2.79$, $p = .17$, MSE = 1366.47. In contrast, the main effect of dose was significant, $F(3, 12) = 9.22$, $p = .002$, MSE = 2378.94, with dose-dependent reductions observed in the number of responses.

To summarize these findings with respect to the number of responses: (a) tolerance developed to the effects of cocaine (significant regimen by dose interaction, increased ED_{50} values from acute to chronic conditions); (b) food duration did not modulate the effects of the other independent variables (no significant interactions involving this factor) and responding did not differ across the two durations (no significant main effect of this factor); (c) the drug administration regimen did not, by itself, influence the percent of trials with at least one response (no significant main effect); and (d) cocaine (ignoring regimen) reduced the number of responses in a dose-dependent manner, but this main effect must be qualified due to significant regimen by dose interaction.

4. Discussion

There is considerable, although not invariant, empirical support for the notion that frequency of reinforcement is a major determinant in resistance to disruption by non-pharmacological variables, such as extinction and pre-feeding (Nevin, 1992; Nevin and Grace, 2000), but findings are inconsistent when drugs are used as disruptors. Some studies have shown larger drug effects under conditions of more frequent reinforcement than under similar conditions with less frequent reinforcement (e.g., Egli et al., 1991; Harper, 1999; Hoffman et al. 1987; Hughes and Branch, 1991; Nickel and Poling, 1990; Nickel et al., 1991), but other studies have failed to find such an effect (e.g., Branch, 1990; Cohen, 1986; Jimenez-Gomez and Shahan, 2007; Jones et al., 1995; Pinkston and Branch, 2004; Poling et al., 1996, 2000; Schama and Branch, 1989). When frequency of reinforcement did appear to modulate drug effects, its influence was often apparent in the development of greater tolerance under conditions with more frequent reinforcement, not in the occurrence of smaller acute effects under those conditions (e.g., Nickel & Poling, 1990; Nickel et al., 1991; Porritt et al., 2007).

Relatively few studies have examined whether reinforcer magnitude modulates drug effects. In one, Lamb and Ginsburg (2008) examined the effects of fluvoxamine, a serotonin reuptake inhibitor, and desipramine, a norepinephrine reuptake inhibitor. In their study, pigeons were exposed to a multiple fixed-interval (FI) 300-s schedule with 2-, 4-, or 8-s food deliveries in separate components. Reinforcer magnitude did not consistently influence the effects of fluvoxamine (1–56 m/kg) or desipramine (0.3–10 mg/kg) on overall response rates. When response rates in each consecutive tenth of the fixed interval were determined and drug rates were expressed as a percentage of control rates, rate-dependent effects were observed.

In general, fluvoxamine increased lower control rates (which occurred early in the interval) more than higher control rates. These rate-dependent effects were stronger in the components with 2- and 4-s food deliveries than in the component with 8-s food deliveries. Lower doses of desipramine had similar rate-dependent effects only in the component with 2-s food deliveries, whereas higher doses also had rate-dependent effects in the components with 4- and 8-s food deliveries. Overall, their findings suggest that increasing the reinforcer magnitude (defined as duration of food delivery) attenuated the rate-dependent effects of fluvoxamine and disipramine. Similar results with the same drugs were obtained in a subsequent study in which rats responded under a multiple FI 300-s schedule in which 2 or 10 food pellets were delivered in different components (Ginsburg and Lamb, 2008). Reinforcer magnitude did not, however, modulate the effects of fluvoxamine or desipramine in pigeons exposed to a multiple FR 30 schedule with 2-, 4-, and 8-s food deliveries in different components (Lamb and Ginsburg, 2005).

The findings of Lamb and Ginsburg (2005, 2008) and Ginsburg and Lamb (2008) demonstrate that reinforcer magnitude can modulate acute drug effects, albeit in a somewhat subtle and limited fashion. The findings of other experiments, however, provide no evidence of such modulation. For example, in a study with pigeons, Hughes et al. (2005) found that tolerance to the rate-reducing effects of morphine developed more readily under short FR schedules than under long ones, even when duration of access to food was adjusted to produce an approximately equal unit price (number of responses

required to produce a given amount of food). The results of Hughes et al. are comparable to those obtained by Nickel and Poling (1990) under similar conditions in which the duration of access to food was not adjusted. Therefore, reinforcer magnitude did not appear to modulate tolerance to morphine in pigeons exposed to FR schedules of food delivery. It is noteworthy that the failure of reinforcer magnitude to influence the development of tolerance to morphine was observed with FR schedules of food delivery, and under these schedules duration of food delivery failed to influence the acute effects of fluvoxamine and desipramine in the study by Lamb and Ginsburg (2005). Thus, available evidence suggests that reinforcer magnitude does not affect acute or chronic drug effects under FR schedules.

In the present study reinforcer magnitude did not significantly (a) produce differences in automaintained responding following administration of vehicle, or (b) modulate the acute effects of cocaine or the development of tolerance to those effects. That is, acute and chronic drug effects on both response measures in the present study were comparable with 3- and 9-s food deliveries. These findings appear to be inconsistent with a behavioral momentum analysis of drug action.

It is unclear why manipulating frequency of food delivery in the study by Porritt et al. (2007) influenced tolerance to cocaine's effects on automaintained responding in pigeons, whereas manipulating duration of food delivery failed to do so in the present study. Both manipulations should affect the momentum of responding (Nevin, 1974), but as noted previously prior studies have yielded inconsistent results concerning the influence of each variable on acute and chronic drug effects under other procedures. At present, it appears that multiple and poorly understood contextual and procedure variables influence whether frequency and magnitude of reinforcement influence drug effects, and that the behavioral momentum metaphor does not generally predict acute or chronic drug effects.

Porritt et al. (2007) suggested that automaintained key-pecking in pigeons is a workable assay for studying behavioral momentum, but only their study and the present investigation used the procedure for that purpose. Both examined cocaine as the disruptor of behavior. To our knowledge, no one has investigated the effects of non-pharmacological perturbations, such as pre-feeding, on automaintained responding under conditions that would allow results to be interpreted in terms of behavioral momentum. Examining the effects of such variables has yielded substantial support for a behavior momentum analysis of response strength under schedules of operant reinforcement (Nevin and Grace, 2000). It is of interest to examine their effects under conditions comparable to those of the present study and of the experiment reported by Porritt et al. (2007). If automaintained responding is a feasible baseline for studying behavioral momentum, then pre-feeding and other non-pharmacological disruptors should produce substantially greater disruption under conditions involving weaker CS–US pairings or lesser US magnitudes. If these disruptors do not do so, then this might call into question the utility of the procedure for examining behavioral momentum. Regardless, additional research examining the application of behavioral momentum analyses to responding under classical conditioning paradigms has practical and theoretical importance and should therefore continue (Savastano and Miller, 2004).

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