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CONTROL OVER RESPONSE NUMBER BY A TARGETED PERCENTILE SCHEDULE: REINFORCEMENT LOSS AND THE ACUTE EFFECTS OF d-AMPHETAMINE

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Two fixed-consecutive-number-like procedures were used to examine effects of acute *d*-amphetamine administration on control over response number. In both procedures, rats were required to press the left lever at least once and then press the right lever to complete a trial. The consecutive left-lever presses on each trial comprised a "run." Under the targeted percentile schedule, reinforcement was provided if the current run length was closer to the target length (16) than half of the most recent 24 runs. This differentially reinforced run length while holding reinforcement probability constant at .5. A second group acquired the differentiation under the targeted percentile schedule, but were then shifted to a procedure that yoked reinforcement probability by subject and run length to that obtained under the targeted percentile schedule. The two procedures generated practically identical control run lengths, response rates, reinforcement probabilities, and reinforcement rates. Administration of *d*-amphetamine disrupted percentile responding to a greater degree than yoked control responding. This disruption decreased reinforcement frequency less in the former than the latter procedure. The similar baseline responding under these two procedures suggests that this difference in sensitivity was due to behavioral adjustments to drug prompted by reduction of reinforcement density in the yoked control but not the percentile schedule. These adjustments attenuate the drug's effects under the former, but not the latter, procedure.

Key words: percentile schedules, response differentiation, fixed consecutive number schedules, response number, reinforcement loss, *d*-amphetamine, lever press, rats

In assessing a drug's behavioral effects, it is desirable to evaluate not only possible effects on overall response output, generally measured as response rate, but also more subtle changes in the structure of behavior, such as changes involving stimulus control and/or differentiation of values along a response dimension. The fixed consecutive number (FCN) procedure (Mechner, 1958) is one such differentiation procedure. It involves differential reinforcement of two-response (R_A and R_B) sequences termed "runs." If, on a particular trial, the subject emits n or more R_As before making an R_B, that is, if the current run length exceeds the minimum requirement n, then reinforcement is provided. Runs shorter than the criterion generally are not reinforced.

The FCN procedure has a venerable history in behavioral pharmacology because of the potential for separating drug effects on response rate from those on response accuracy. That is, unlike differentiation of temporal aspects of responding (e.g., interresponse time, response duration), in which changes in the distribution of response values almost inevitably are correlated with changes in overall response rate, differentiation of control by response number can occur relatively independent of changes in response rate. This feature increases the utility of FCN procedures as behavioral baselines by increasing the available dimensions along which drug effects may be observed.

One potential source of concern when using an FCN or any traditional differentiation procedure as a baseline against which to evaluate drug effects is that drug-induced disruptions in the run distribution will produce correlated changes in reinforcement probability and rate, generally decreasing criterional run frequency and consequently reinforcement density. Increasing doses of a wide variety of drugs, including various opioid agonists and antagonists (e.g., Bronson & Moerschbaecher, 1987; Picker, Heise, & Dykstra, 1987), the nonopi-

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oid analgesics clonidine and *l*-nantradol (Picker & Dykstra, 1988), the anticonvulsants clonazepam, ethosuximide (Picker, Leibold, Endsley, & Poling, 1986a) valproic acid, phenytoin, phenobarbital, diazepam (Picker, Leibold, Endsley, & Poling, 1986b), methsuximide, and mephenytoin (Schlinger, Wilkenfield, & Poling, 1988), the stimulants amphetamine (Laties, 1972), methamphetamine, caffeine, and methylphenidate (Mechner & Latranyi, 1963), and the anticholinergic scopolamine (Laties, 1972) all decrease the relative frequency of runs reinforced under FCN procedures.

These decreases in reinforcement density are potentially important because they have been implicated in modifying both acute (e.g., Smith & McKearney, 1977) and chronic (cf. Goudie & Demellweek, 1986) effects of amphetamine and other drugs. Smith and McKearney (1977) reported that the response-rate increases initially produced by *d*-amphetamine under a schedule in which only interresponse times (IRTs) longer than 30 s produced food diminished and ultimately disappeared with successive, acute administrations. They suggested that this attenuation resulted from the restoration of reinforcement frequency that occurred as a consequence. Schuster, Dockens, and Woods (1966) noted a similar diminished effect of chronic *d*-amphetamine administration (i.e., tolerance) dependent on whether drug administration initially decreased reinforcement frequency. Such differential tolerance correlated with reinforcement loss is termed *learned* or *behaviorally mediated* tolerance (see Goudie & Demellweek, 1986, for a review).

The term *behavioral tolerance* often is invoked when tolerance develops more slowly under schedules in which response and reinforcement rate are relatively independent (e.g., fixed- or variable-interval), compared to other schedules that program stricter correlations between response and reinforcer rates (e.g., ratio or IRT $> t$). Unfortunately, these schedules and the resultant behavior differ along a number of dimensions other than reinforcement loss associated with disruptions in responding (e.g., IRT contingencies, baseline response rates, baseline reinforcement rates and probabilities, etc.); hence, it is never entirely clear that reinforcement loss is the sole contributor to differential tolerance development. The two procedures used here, under nondrug conditions, generated comparable run distributions, response rates, reinforcement probabilities, and reinforcement rates. They differed in whether drug-induced disruptions in run length resulted in a decrease in reinforcement probability. Hence, the procedures allow differential drug effects as a function of associated reinforcement loss while equating a number of potential confounding variables.

To effect this comparison, a percentile reinforcement schedule (Galbicka, 1988) was used. Percentile schedules may be thought of as "titrating" differentiation procedures. As response values change along the dimension of interest, the cardinal value segregating criterional from noncriterional responses is modified such that it always defines a specified percentile of the subject's recent response distribution. Increasingly differentiated performance leads to a constant criterional response probability. Criterional and noncriterional responses can then be reinforced with associated conditional reinforcement probabilities. In the present study, these were 1.0 and 0, respectively, making criterional and reinforced runs isomorphic. By controlling criterional response probability in this manner, overall reinforcement probability is equally controlled.

The specific percentile schedule used here was a targeted percentile (see Galbicka & Platt, 1989). Under this variation, response values are first segregated according to whether they fall above or below a particular ("target") value. Then differentiation occurs as before, with the direction dictated by the relation between the current value and the target. Values less than the target are differentiated towards greater values, whereas values greater than the target are shaped towards lesser ones. Hence, as responding differentiates, the run distribution concentrates in the vicinity of the target.

Targeted percentile and FCN procedures differ in that the former programs upper and lower bounds on criterional runs, whereas FCN procedures generally specify only a lower bound. The percentile criteria remain constant relative to the current run distribution, rather than fixed at one cardinal value as in FCN procedures. This controls criterional response probability during the acquisition and maintenance of the differentiation. Further, drug-induced run-length disruptions do not change the expected reinforcement probability, because the schedule references the criterion value relative to the current (disrupted) run distri-

bution, to maintain control over criterional-run probability.

The comparison (yoked control) condition was designed to generate comparable rates and patterns of behavior and reinforcement under nondrug conditions, but to reduce reinforcement density following drug administration. After acquisition of the target under the percentile schedule, subjects were switched to a procedure that maintained the same general bounds on run lengths reinforced by yoking reinforcement probability for different length runs to probabilities derived from previous performance under the percentile schedule. Because the reinforcement probability distribution for the yoked control procedure was derived from asymptotic percentile performance, and the latter reinforced runs closest to the target, the reinforcement probability distributions peaked at the target, dropping with increasing deviation from the target.

Hence, whether the current run was considered criterional depended, under the percentile procedure, on its value relative to the current response distribution, whereas under the yoked control procedure, reinforcement probability for each run length was fixed at a particular value yoked to previous percentile performance. At asymptote, the two procedures are practically indistinguishable, and the transition between them produces little behavioral effect. Only an outside agent that disrupts run lengths, such as drug administration, produces a behaviorally relevant distinction between the two procedures. Unlike the percentile arrangement, shifting the run distribution away from the target necessarily decreases reinforcement density under the yoked control procedure.

To the extent that decreased reinforcement probability sets the occasion for compensatory responses that offset the drug effect, run length under the yoked control procedure might appear less sensitive to the drug. That is, drug-induced run-length disruptions under the yoked control procedure will immediately be translated into decreased reinforcement. This might then provide the impetus for subsequent behavioral changes to recover reinforcement density. Under the percentile arrangement, drug-induced disruptions in performance never decrease reinforcement density, and as such they do not prompt any behavioral adjustment to the drug, thereby allowing drug effects to appear at lower doses. The present study tested this possibility with acute administration of *d*-amphetamine.

METHOD

Subjects

Eight male Sprague-Dawley rats, initially weighing between 300 and 400 g, were maintained, through restricted postsession feeding of rat chow, at a body weight of 350 g. Subjects were individually housed with continuous access to water in acrylic rack-mounted cages lined with pine bedding. The colony room housing the rack was maintained on a 12:12 hr light/dark cycle (onset time: 6:00 a.m.).

Apparatus

Experimental sessions were conducted in four identical Coulbourn Instruments (Model E10-10) modular operant conditioning chambers (29.5 by 24 by 27.5 cm) configured for rats. The instrument panel of each chamber contained two response levers mounted 7 cm on either side of an aperture framing a food cup (6.25 by 3.5 cm) into which 45-mg food pellets could be delivered via operation of a solenoid-driven pellet dispenser. The left lever in each chamber was retractable (Coulbourn Instruments, Model E23-05), a feature not utilized in the present study; the right one was a standard rat lever (Coulbourn Instruments, Model E21-03). These levers required between 0.15 and 0.30 N to operate. Force requirements on each lever were not the same; however, subjects were always studied in the same chamber. Centered in a plane 3 cm above each lever were three stimulus lights (Sylvania 28ESB) 1.5 cm apart, center to center, covered with red, green, or yellow translucent caps. Mounted above the food aperture and behind the front wall was a heavy-duty relay that produced a click when operated. The floor of each chamber was composed of parallel stainless steel rods (0.5 cm diameter) spaced 1.8 cm center to center. The chambers were housed inside light- and sound-attenuating enclosures. Sound from a ventilating fan on each chamber, as well as white noise continuously present in the room, helped further mask extraneous sounds. Stimuli were programmed and data collected and analyzed by a PDP® 11/73 minicomputer in an adjacent room operating under SKED11® software (Snapper & Inglis, 1985).

Sessions also were monitored via Gerbrands (Model C-3SH) cumulative response recorders. A set of FORTRAN subroutines, which operated interactively with SKED11® to evaluate the percentile equations on-line, is available from the first author.

Procedure

All subjects progressed through a series of pretraining phases designed to produce a behavioral sequence comprised of one or more responses on the left lever followed by a response on the right lever. In order, these phases included (a) reinforcing approximations to and subsequent lever pressing on either lever; (b) reinforcing presses on the nonpreferred lever; (c) reinforcing either left- or right-lever presses on a trial, chosen with equal probability and signaled by illuminating the green light above that lever; (d) reinforcing a heterogeneous chain of a left-lever press in the presence of a green light above that lever followed by a right-lever press in the presence of a green light above that lever; (e) decreasing the reinforcement probability at the end of the chain from 1.0 to .5; and (f) increasing the number of responses required in the first component of the chain from one to eight. As a final step in pretraining, trial onset was signaled by illuminating both green lights. Pressing the left lever at least once followed by a right-lever press (a run) was reinforced with a probability of .5. Right-lever presses prior to left-lever presses produced no experimentally arranged consequences. All trials were separated by 3-s timeouts during which the chamber was dark and responses produced no experimentally programmed consequences. Subjects were exposed to this final procedure for 11 sessions. During this and all subsequent conditions, sessions were comprised of 100 trials or 30 min (excluding timeouts), whichever came first, and were conducted 5 days per week.

All subjects next were exposed to a targeted percentile schedule (for a detailed description, see Galbicka & Platt, 1989). To equate reinforcement probability to that from the previous phase, the percentile schedule arranged that half of all runs be classified as criterional, and all of these and only these were reinforced. Accomplishing this involved comparing the current run with all runs within the last 24 trials on the same side of the target (16) as the current run. Denoting the number of comparisons m, the current run was considered criterional if it exceeded at least k of the m runs, where

$$k = (m + 1)(1 - w), \qquad (1)$$

in order to observe a criterional run with the specified probability of $w = .5$. The current run exceeded the comparison if the current run was closer to the target of 16. Thus, a run less than the target had to be longer than the comparison, whereas one greater than the target had to be shorter than the comparison run to exceed the comparison run.

After determining whether the current run met criterion, the distribution was updated to include only the most recent 24 runs (i.e., the current run replaced the oldest comparison run). Comparison runs were carried across sessions except during the first session and during sessions involving vehicle or drug administration, when the distribution was cleared of all values. In those cases, the first run was reinforced with a probability of .5, and subsequent ones were evaluated according to Equation 1, in which m was incremented from 1 to a maximum of 24 across the first 24 trials.

When subjects simultaneously demonstrated stable mean run lengths (minimal variability and no discernible trends for 10 consecutive sessions) in the vicinity of the target, they were divided into two groups. Because of the generally good control exerted over each subject's responding, assignment was not random. Rather, the first 4 subjects were switched to the yoked control procedure, and the rest remained on the targeted percentile schedule. For each yoked control subject, the run distributions during the last five sessions were combined into one distribution. For each run length a reinforcement probability was determined by dividing the number of reinforced runs of that length by the total number of runs of that length. This reinforcement probability distribution assigned reinforcement, for that subject only, during all subsequent sessions. Runs longer than those observed under the targeted percentile schedule were never reinforced. Subjects were exposed for an additional five sessions either to the yoked control procedure or to the percentile procedure, as appropriate to their group assignment, prior to the administration of the drug. Longer exposure to the yoked control procedure was deemed unnecessary, because in no case did run lengths

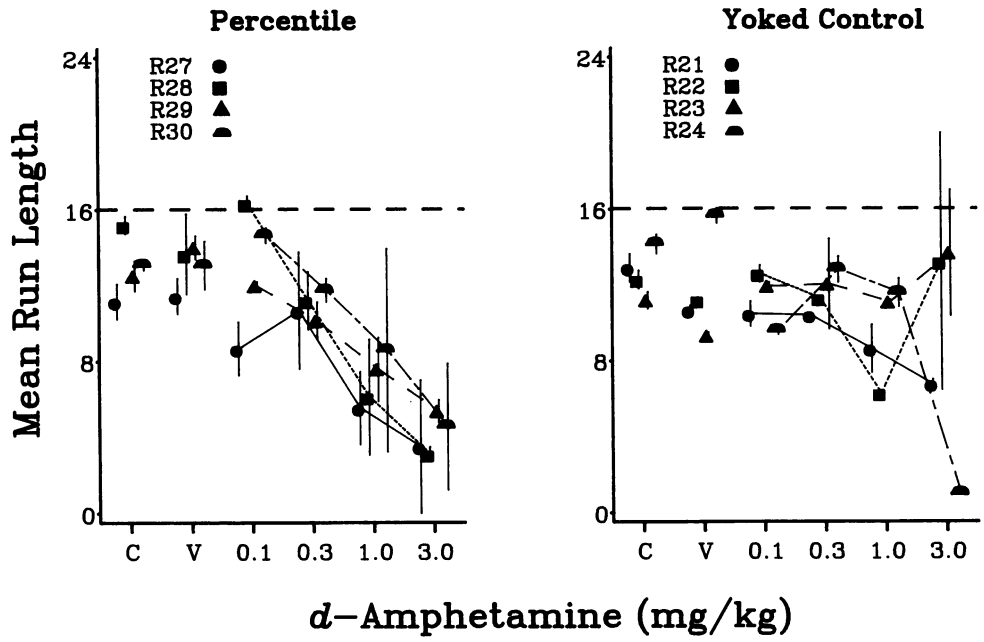

Fig. 1. Effects of *d*-amphetamine on mean run length. Results from the percentile group are presented in the left panel, and those from the yoked control group are presented in the right panel. Individual subjects in each group are represented by different symbols. Points above C represent noninjection control sessions; above V, vehicle injection. Points (and vertical bars) are means (±*SEM*) for control (10 sessions) and vehicle or drug administrations (two sessions). The horizontal dashed line indicates the target. Points have been slightly displaced along the horizontal axis to avoid overlap.

deviate from previous values: they were not expected to, because the same reinforcement probability distribution prevailed during the previous 5 days.

Acute administration of *d*-amphetamine sulfate followed. Mondays and Thursdays served as noninjection (control) days, with drug or vehicle injections on Tuesday and Friday. Although exposed to similar contingencies on Wednesday to maintain a 5-day work regimen, no injections occurred on this day and data collected from these sessions are not included in the analysis here. Drug doses (0.1, 0.3, 1.0, and 3.0 mg/kg) were dissolved in 0.9% NaCl and injected i.p. in a volume of 1 mL/kg 5 min prior to the start of experimental sessions. Each dose was administered twice, along with two vehicle injections. The order of doses was random except that all subjects received the same dose on any particular day and all doses were administered once prior to the second determination. Drug effects were considered reliable if the standard errors of measures obtained following vehicle and following drug injection did not overlap.

RESULTS

All subjects emitted short runs (M = 3.22; range, 1–8) during final pretraining. Across 25 to 50 sessions under the targeted percentile procedure, runs gradually increased and approached the target. Mean control run lengths for the targeted percentile group (Subjects R27 through R30) averaged 13.24 (range, 11.46–15.51). Those for the yoked control group (Subjects R21 through R24) were very similar, averaging 12.90 (range, 12.38–13.98).

Figure 1 shows mean run length for individual subjects in both groups under control conditions and following vehicle or *d*-amphetamine injections. Comparable control run lengths for the two groups are shown by the overlap of points in the far left of each panel. Vehicle injection (points above V) produced unsystematic effects with both groups. Increasing doses of *d*-amphetamine differentially affected run length in the two groups. Under the targeted percentile schedule, run length consistently decreased in a dose-related fashion, whereas effects under the yoked control pro-

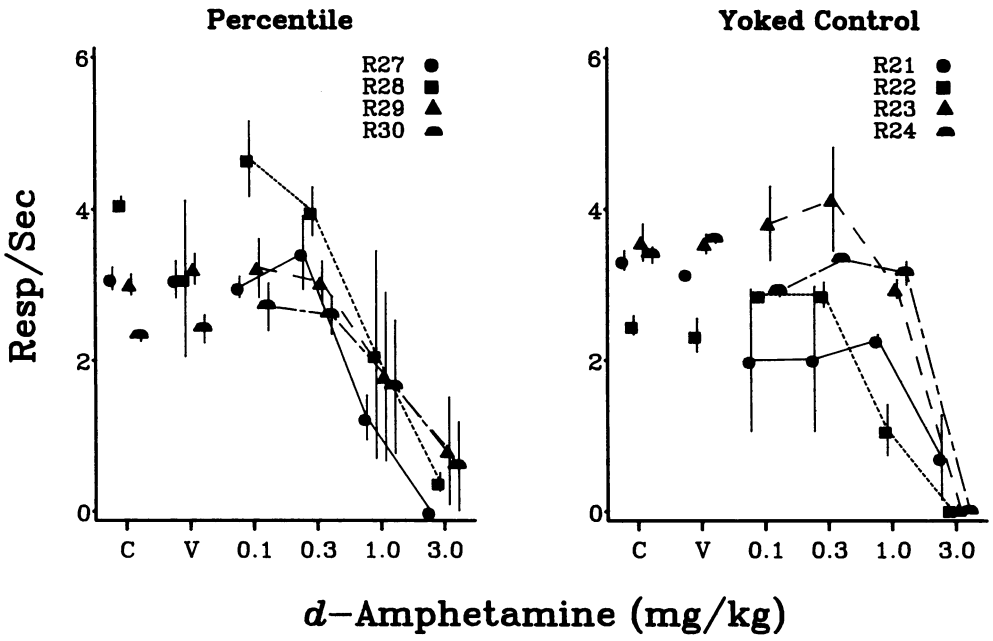

Fig. 2. Effects of *d*-amphetamine on response rate. Plotting conventions as in Figure 1.

cedure were less consistent, with no dose decreasing run length in all subjects.

Figure 2 shows dose-effect curves for response rates of individual subjects. Again, control performances were comparable between the two groups. All percentile subjects showed graded decreases in response rate with increasing doses of *d*-amphetamine; however, only at

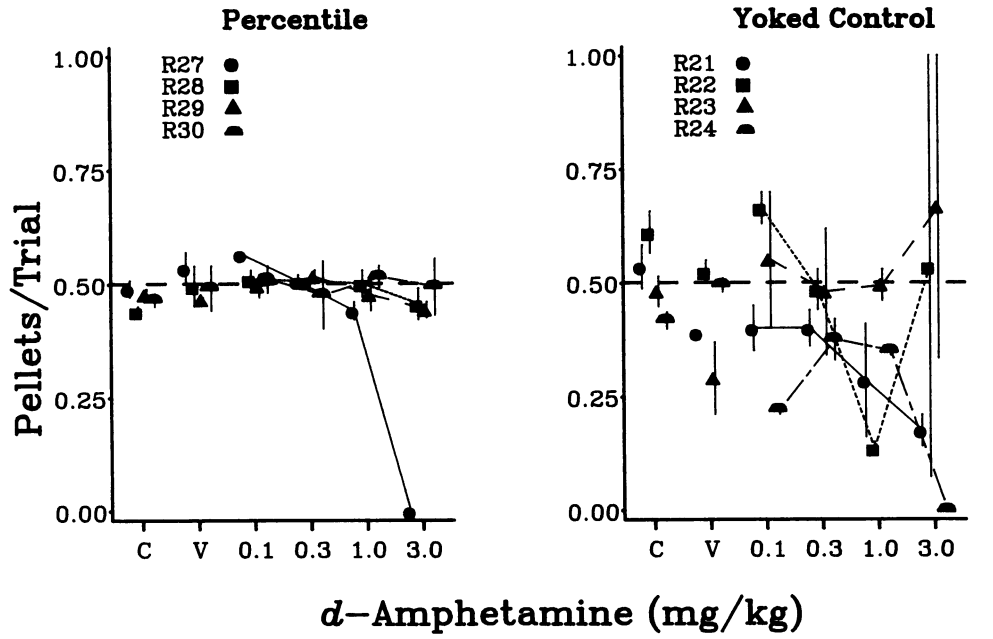

Fig. 3. Effects of *d*-amphetamine on reinforcement probability (pellets per trial). Plotting conventions are the same as in Figure 1, except that the dashed horizontal line represents the nominal probability of reinforcement under the percentile procedure.

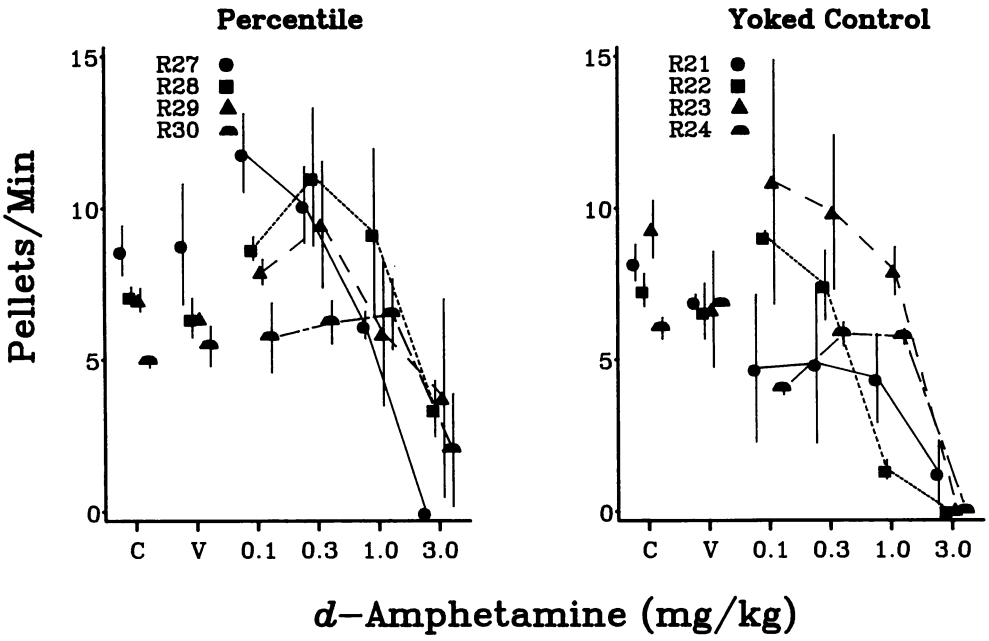

Fig. 4. Effects of *d*-amphetamine on overall reinforcement rate (pellets per minute). Plotting conventions are the same as for Figure 1.

the highest dose were these decreases consistent in all subjects. Yoked control subjects also showed consistent decreases in response rate only at 3.0 mg/kg; however, the dose-effect curves dropped much more precipitously than those for the targeted percentile group.

During control conditions the yoked control procedure generated food probabilities roughly comparable to those obtained under the percentile procedure (see points above C, Figure 3). True to the procedure, the probability of food presentation remained well controlled under the percentile schedule despite drastic changes in run length induced by the drug. Following *d*-amphetamine administration, the only deviation from the programmed probability of food was at the highest dose for R27, which resulted from sessions containing few trials. Food probabilities for yoked control relative to the targeted percentile procedure were much more variable following drug administration. This increase in variability resulted from the small number of trials completed, particularly at higher doses.

Given comparable response rates and reinforcement probabilities during control conditions, it is inevitable that overall food rates were comparable under control conditions across the two groups (Figure 4). All percentile subjects showed at least one reliable increase in pellet rate following *d*-amphetamine. Conversely, only 1 yoked control subject showed a significant increase in pellet rate at any dose (R22 at 0.1 mg/kg).

Figures 5 and 6 provide a more detailed analysis of run length under targeted percentile and yoked control procedures, respectively. Note that control distributions for the two groups were comparable. Percentile subjects showed a gradually greater shift in the entire distribution toward shorter runs with increasing doses of *d*-amphetamine. Two yoked control subjects, R21 and to a lesser extent R22, demonstrated a similar change in the run distribution with increasing doses of *d*-amphetamine. The other subjects showed little effect on the run distribution until the highest dose, when the distributions became very erratic, primarily because of the small number of trials comprising each distribution.

DISCUSSION

The targeted percentile and yoked control procedures successfully maintained runs concentrated in the vicinity of the target. Differentiation of longer runs under the percentile procedure presents an enigma to behavioral accounts that emphasize molar reinforcement parameters in controlling behavior. Prior to

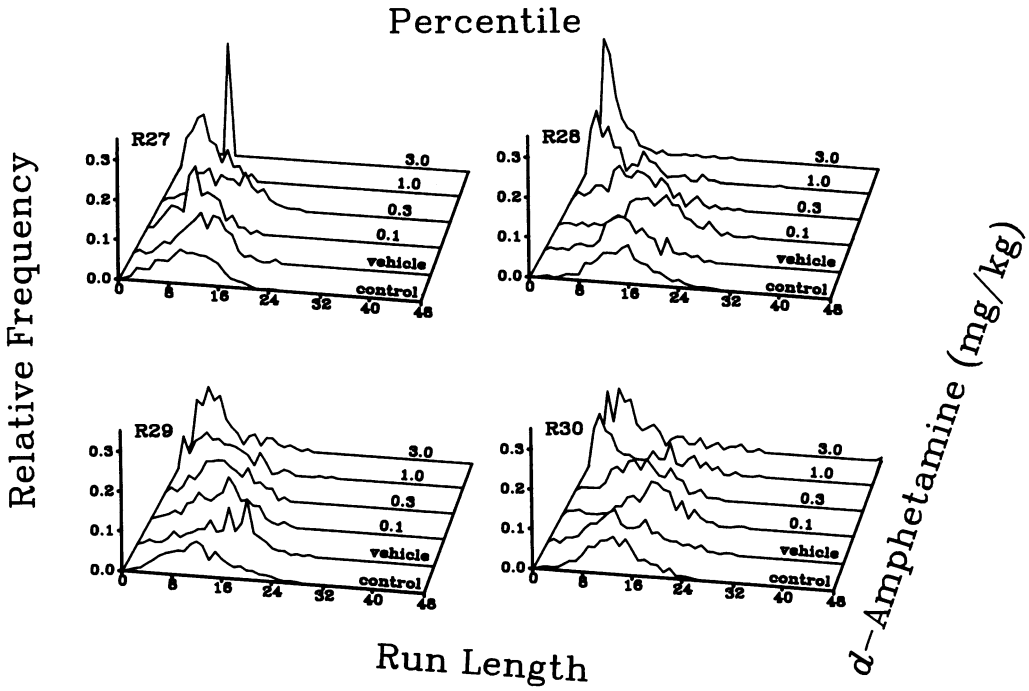

Fig. 5. Run length distributions for percentile group subjects. Each set of distributions represent data from the subject noted in the upper left corner. The relative frequency (vertical axes) of different length runs (left to right) is plotted for each treatment (front to back). Control distributions were cumulated across 10 noninjection control sessions; vehicle and drug distributions were cumulated across two determinations of each value.

and during differentiation, runs were reinforced with a probability of .5. Further, early in training, runs were relatively short; hence, trials were short and reinforcement rate was relatively high. Increasing run length certainly would not shorten trial durations, and for most subjects lengthened them, thereby decreasing overall reinforcement rate. Thus, runs more closely approximating the target under the percentile schedule increased neither overall reinforcement probability nor rate, yet the differentiation was acquired.

The local reinforcement contingencies in percentile schedules provide a mechanism for the observed control over run length. Only runs closer to the target than previous ones were ever reinforced, and each trial provided an opportunity to get closer still. Hence, the present data add to previous reports (e.g., Galbicka & Platt, 1984, 1986, 1989; Platt, 1979) demonstrating that local reinforcement contingencies substantially alter behavior in the absence of associated changes in molar reinforcement parameters.

The control over response number under the present procedures was not identical to that engendered under FCN procedures, in which the mode of the distribution is most commonly found at a value slightly greater than criterion. Both procedures used here generated run distribution modes slightly shorter than the target. The FCN procedure specifies as criterion the minimum reinforced value; therefore, the distribution mode often exceeds this value. Under the present procedures, the target specifies the peak of the reinforcement probability distribution. Hence, not only do runs shorter than the target produce reinforcement, but the longest runs also only produce food early in the differentiation under the percentile schedule (i.e., when there are no comparison runs on the far side of the target) and never under the yoked control procedure. These factors will tend to shorten the distribution mode relative to an equal-valued FCN schedule.

Decreased run lengths and response rates following acute doses of *d*-amphetamine under both of the present procedures generally replicate effects reported under standard FCN procedures (e.g., Laties, 1972), as well as a host of reports that amphetamine decreases high rates of responding (see McKearney &

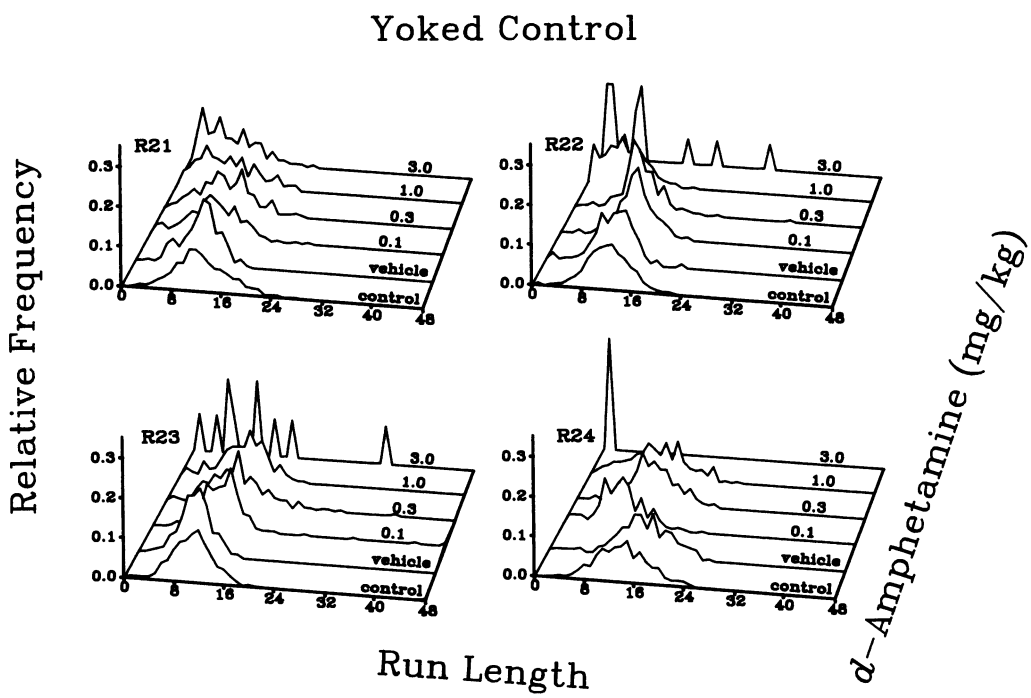

Fig. 6. Run length distributions for each subject in the yoked control group, plotted with the same conventions as in Figure 5.

Barrett, 1978). The percentile schedule generated behavior that appeared more sensitive to *d*-amphetamine than did the yoked control procedure. Dose-effect curves for the first group appeared more orderly and, for run length at least, behavior was disrupted at doses lower than those required for the yoked control group. The targeted percentile subjects showed no decrease in reinforcement probability, except for 1 subject at a dose that all but eliminated responding. Similarly, reinforcement rate was decreased only at the highest dose. Indeed, every subject in the percentile group showed at least one instance in which drug *increased* reinforcement rate. Reinforcement probabilities and rates for the yoked control subjects, conversely, showed greater disruption, with only 1 subject maintaining reinforcement densities comparable to control conditions across all but the highest dose of *d*-amphetamine. Thus, when reinforcement density was correlated with run length (yoked control), drug effects were less easily discerned than under the percentile schedule, which programmed a constant probability of reinforcement independent of run length.

These results suggest that the magnitude of a drug's acute behavioral effects may in some instances be attenuated by behavioral adjustments that occur as a result of reinforcement loss associated with drug administration. As such, they extend Smith and McKearney's (1977) observation and indirectly support arguments presented for behavioral tolerance as a function of reinforcement loss (cf. Goudie & Demellweek, 1986). These results imply that when reinforcement criteria "float" with changes in behavior to maintain a constant reinforcement density, drug effects may be revealed in a more orderly manner and at lower doses, because the behavior observed under drug results from a single manipulation—drug administration—not two opposing manipulations—drug administration and concomitant behavioral adjustments prompted by reduced reinforcement density. The almost indistinguishable control run lengths, response rates, and reinforcement patterns under the present procedures eliminate a number of variables to which differential drug effects might otherwise be attributable were disruptions in reinforcement density studied by comparing, for example, variable-interval (VI) and IRT > *t* schedules (e.g., Galbicka, Lee, & Branch,

1980). Although reinforcement density is relatively independent (VI) or dependent (IRT > t) on response rate under these schedules, they generate different overall response rates, reinforcement probabilities, and interreinforcement intervals, all of which might contribute to differences in drug effects observed.

The greater disruption of percentile run length by d-amphetamine cannot be attributed to increased reinforcement of short runs following injections under this procedure, relative to the yoked control, rather than to behavioral adjustments under the latter in response to reinforcement density reduction. Low doses of amphetamine and vehicle did not result in short runs, even though the comparison distribution was cleared prior to these sessions as well, leaving the potential for short runs to be reinforced. Hence, subjects did not learn a conditional discrimination that short runs were reinforced only following injection. Adding the caveat that amphetamine must first reduce run length, and then reinforcement for these short runs maintains their frequency, only clouds the distinction between this and the reinforcement density reduction argument. The most parsimonious account of the difference between the two groups appears to be that reinforcement density reductions prompted subjects in the yoked control group to learn, in the presence of the drug, behavioral adjustments that reestablished run length and thereby restored reinforcement density. That is, because the percentile schedule controls reinforcement probability, decreasing run length generates no adverse effect on reinforcement density. As such, there is no strong differential reinforcement to emit runs approximating control values. The percentile schedule does differentially reinforce longer runs; however, this contingency relates to the current distribution of shortened runs. Shaping may thus occur throughout a session, generating longer runs that still remain short relative to control levels. Under the yoked control procedure, conversely, when drug administration decreases run length, reinforcement density is concomitantly suppressed and only recovers with runs approximating the target (i.e., the reinforcement probability distribution is not anchored to the current run distribution as it is under the targeted percentile schedule). The differential reinforcement contingency under the yoked control procedure tends to generate not merely longer runs but runs approximating the target. Longer runs shaped during the session would help restore reinforcement density and attenuate the effects of drug on run length. This difference between procedures in the local reinforcement contingencies may be revealed only under drug administration, because the disparity in differential reinforcement contingencies under the two procedures increases as the current run distribution is displaced further from the target.

It is clear that the interaction of drug and behavior is more dynamic than generally portrayed. Not only does the historical context differentially affect both acute (see Barrett, 1987; Barrett & Witkin, 1986) and chronic (see Barrett, Glowa, & Nader, 1989) drug effects, so, too, do more immediate changes in the local reinforcement contingencies modulate behavioral effects of drugs. An awareness of the local dynamics of behavioral control can lead to completely new interpretations of drug effects, similar to that provided by Galbicka (1990) in synthesizing drug effects on punishment procedures with those on responding maintained by response-produced shock. New procedures and dependent measures that more adequately capture the local interplay between environmental and pharmacological control of behavior will further our understanding of both. The procedures presented here represent a methodological step in that direction.

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