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# Effect of signaled reinforcement on response variability

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## Abstract

Three experiments examined the effect of signaling reinforcement on rats' lever pressing on contingencies that reinforced variable responding to extend the exploration of signaled reinforcement to a schedule that has previously not been examined in this respect. In Experiment 1, rats responding on a lag-8 variability schedule with signaled reinforcement displayed greater levels of variability (*U* values) than rats on the same schedule lacking a reinforcement signal. In Experiment 2, rats responding on a differential reinforcement of least frequent responses schedule also displayed greater operant variability with a signal for reinforcement compared with rats without a reinforcement signal. In Experiment 3, a reinforcement signal decreased the variability of a response sequence when there was no variability requirement. These results offer empirical corroboration that operant variability responds to manipulations in the same manner as do other forms of operant response and that a reinforcement signal facilitates the emission of the required operant.

**KEYWORDS**

differential reinforcement of least frequent responses schedule, lag schedule, operant variability, rat, signaled reinforcement

Presenting a brief stimulus (500 ms) simultaneously with, or just prior to, the delivery of reinforcement and following the response that leads to reinforcement on a free-operant schedule affects the rate and pattern of responding relative to an unsignaled reinforcement condition (Reed et al., 1988; Roberts et al., 1984; Sizemore & Lattal, 1978). Depending on the schedule contingency, the signal acts to elevate rates of responding, such as on variable ratio and differential reinforcement of high rate schedules (Reed, 1989a; Reed et al., 1988; Tarpy & Roberts, 1985), but reduces them on others, such as variable interval and differential reinforcement of low rate schedules (Roberts et al., 1984; Tarpy & Roberts, 1985).

Many factors contribute to this signaled-reinforcement effect (see Iversen, 1981; Reed, 1989b; Sizemore & Lattal, 1978; Williams, 1991), but several explanations have suggested that the reinforcement signal facilitates learning about aspects of the schedule requirements (Fedorchak & Bolles, 1986; Roberts et al., 1984). The aspects of the schedule requirements that are learned about, however, are subject to debate (cf. Reed, 1989b; Roberts et al., 1984). Some accounts suggest that the signal facilitates learning about the nature of the operant response preceding reinforcement—sometimes called "molecular accounts" (e.g., Reed, 1989b)—and some views suggest that the signal facilitates learning about the nature of the overall schedule requirements—sometimes called "molar accounts"(e.g., Roberts et al., 1984). Both views suggest that the signal facilitates emission of responding that is associated with reinforcement, and it is perfectly possible that under some conditions molecular and under other conditions molar aspects of the contingency exert a greater influence over performance and are the subject of signal's facilitatory effect on learning (Reed, 2015).

The action of signaled reinforcement on has been studied on many schedules but not in relation to variability schedules (Page & Neuringer, 1985). A critical difference on those latter schedules from other schedules studied to date is that the operant is "abstract" in nature rather than being defined by a particular topology of responding (e.g., a longer or a shorter interresponse time, IRT). Whether this scheduling difference has implications for the way in which behavior will interact with aspects

of the contingency, like the presence of a signal for reinforcement, has received no or scant attention and is the primary purpose of the current series of studies. In addition, if a brief reinforcement signal does affect responding on a variability schedule, it may serve to address some theoretical issues regarding the action of such other manipulations, although this is not the major purpose of the current set of studies.

Operant variability has been noted in many species including rats, pigeons, and humans (Blough, 1966; Manabe et al., 1997; Morgan & Neuringer, 1990; Page & Neuringer, 1985; Pryoret al., 1969; see also Neuringer, 2012; Silbaugh et al., 2021 for reviews). In a typical experiment, the emission of a sequence of responses is reinforced (e.g., pressing a left [L] or right [R] manipulanda, such as LLRR, RLRL, RLLR), but only when the current sequence varies from previous sequences according to some criterion (Page & Neuringer, 1985). For example, if the current sequence had not been recently emitted frequently, it would lead to reinforcer delivery, but if this sequence was recently common, then reinforcement would be withheld.

When behavior on such a variability contingency is compared with that on a schedule delivering identical patterns of reinforcers, but lacking the variability rule, subjects emit a greater variety of sequences (Morgan & Neuringer, 1990; Page & Neuringer, 1985; Pryor et al., 1969; but see Schwartz, 1982). There are several manners in which variability contingencies can be arranged that operate in ways that are subtly different from one another such as "lag" (Page & Neuringer, 1985), differential reinforcement of least frequent responses (Blough, 1966; Shimp, 1967), threshold (Denney & Neuringer, 1998; Doughty & Galizio, 2015), and percentile (Machado, 1989, 1992) schedules (see Lee et al., 2007; Neuringer, 2002). A relatively straightforward way to reinforce variability is seen in the differential reinforcement of least frequent responses schedules, which reinforce only the least frequent response (Blough, 1966; Shimp, 1967). That is, only one response (the one that is least frequent) is eligible for reinforcement on a given trial. On the other hand, percentile schedules (Machado, 1989, 1992) allow great experimental control over variables but are relatively complex. These schedules adjust the reinforcement criterion according to the present displayed variability, and the criterion can be increased or decreased as a consequence of the behavioral variability displayed in order to hold constant the probability that the next variability score will exceed the criterion.

Two widely employed schedules in the study of operant variability are the lag (Page & Neuringer, 1985) and threshold (Denney & Neuringer, 1998; Doughty & Galizio, 2015) schedules. Lag schedules reinforce responses if those responses differ from a specified number of preceding responses. Whereas, threshold schedules reinforce responses only if they occur relatively less frequently than other responses do. Both schedules have been shown to increase variability across similar response types (Neuringer, 2012; Silbaugh et al., 2021), and both have been subject to a range of possible explanations for their effects on increased variability (Lee et al., 2007). These schedules will be the focus of this series of experiments, as they differ in that lag schedules involve the tracking of a specified number of recent response types, whereas threshold schedules require tracking of all responses emitted (Lee et al., 2007; Silbaugh et al., 2021).

The current series explored the effects of signaled reinforcement on different types of variability contingency: one study employing a lag schedule and another using a threshold schedule. The final study explored the effects of signaled reinforcement on the emission of responses sequences when there was no variability contingency. The results may offer both further empirical exploration of the lag schedules and some theoretical insight into possible mechanisms of action of reinforcement signals on free-operant schedules.

# EXPERIMENT 1

The first experiment examined the effect of a brief signal for reinforcement for food-deprived rats that were lever pressing on a lag schedule. In a "lag schedule," response sequences lead to a reinforcer if they differ from a specified number of the preceding sequences. For example, on a lag-8 schedule, response sequences receive a reinforcer when they differ from all of the last eight sequences emitted. Lag schedules have been shown to increase variability when the response topology is defined as an interresponse time (IRT; Schoenfeld et al., 1966), sequences of left–right responses (Page & Neuringer, 1985), or verbal responses (Lee et al., 2002).

In the current experiment, rats were exposed to a lag-8 schedule while required to emit four responses across two levels (L and R), based on that described by Mook and Neuringer (1994). There were 16 possible L/R combinations (e.g., LRLR, RRLL, RLLR, etc.). Food was delivered if the current four-response L/R sequence differed from all of the last eight sequences emitted. Half of the rats received these contingencies alone (unsignaled reinforcement), and half received a brief (500 ms) tone presented simultaneously with reinforcement (signaled reinforcement), based on that described by Reed et al. (1988). Following training, the signaling contingencies were reversed for the two groups, so the signaled group received no signal and the unsignaled group received a signal.

## Method

### Subjects

Sixteen male Lister rats, three months old at the start of training, with a free-feeding body-weight range of

320–355 g, served as subjects. The rats were naïve with respect to lever pressing and all schedules and stimuli used in the experiment. The rats were housed in groups of four and were maintained at 85% of their free-feeding weight (established over 2 weeks on ad lib food prior to the experiment) throughout the experiment. The subjects were housed in groups of four, with water constantly available in the home cage.

## Apparatus

Four identical operant conditioning chambers (Campden Instruments Ltd.) were used. Each chamber was housed in a light- and sound-attenuating case, ventilated by a fan that provided background masking noise (65-dB [A] above background). Each chamber had two levers, both of which could be retracted from the chamber. Reinforcement consisted of one 45-mg food pellet, and this was delivered to a food hopper that was covered by a clear, Perspex, hinged flap that was located centrally between the two response levers. A speaker was mounted on the roof of the chamber, through which a 105-dB (A) tone could be delivered.

## Procedure

The rats received two sessions of magazine training on a random time (RT) 60-s schedule, with the levers retracted. They then received two, 20-min sessions of lever-press training with a continuous reinforcement (CRF) schedule operating on both levers (i.e., a multiple CRF CRF schedule). Subjects were then placed on a concurrent random interval (RI) 30-s RI 30-s schedule for six sessions. Each session lasted 20 min.

The rats were then transferred to the lag variability schedule. A trial commenced with the insertion of both levers into the chamber. A trial comprised four lever presses across the left and right levers, in any combination (i.e., there were 16 possible sequences of four responses). For reinforcement, the current sequence had to differ from each of the eight just-preceding sequences (i.e., a lag-8, variability contingency). When this variability criterion was satisfied, a food pellet was presented and the levers were retracted. This was followed by a 3-s intertrial interval (ITI). Following the ITI, both levers were reinserted and the next trial started. If the current response sequence repeated any of the previous eight sequences, then no reinforcer was delivered, the levers were retracted, and there was a 3-s ITI. The first sequence of each session was always reinforced, as there were no sequences previously emitted in the session. On the next trial, emission of any of the 15 other sequences could receive reinforcement, and so on. Sessions terminated after 100 trials.

For one group (Group Sig-Unsig), successful satisfaction of the lag-8 contingency in Phase 1 resulted in the presentation of a 500-ms 105-dB(A) tone simultaneously with the delivery of reinforcement. There was no tone presented on unsuccessful trials. In Phase 2, Group Sig-Unsig did not receive the tone reinforcement signal. For the other group (Group Unsig-Sig), the reinforcement signaling contingencies were reversed so that they received no signal in Phase 1 and a signal in Phase 2. There were 40 sessions of training for both groups in both Phases.

Given recent discussions concerning appropriate measures to capture variability (Nergaard & Holth, 2020), two indices were calculated for the current series of studies. The *U* value was used to measure of sequence variability in each session, as is commonly employed in variability studies. The *U* value was calculated as follows:

$$-\sum[\mathrm{RF}j \times \log 2(\mathrm{RF}j)]/\log_2(16), \quad \text{for } j = 1 \text{ to } 16,$$

where RF*j* refers to the relative frequency of occurrence of each of the 16 possible sequences. Values for *U* can vary between 0, indicating that one or more sequences were highly likely and others tend not to occur, and 1, indicating each of the sequences occurred with approximately equal frequency. In addition, the percentage of sequences emitted that met criterion for variability (percentage qualifying for reinforcement) was also calculated.

## Results and discussion

The mean *U* values were calculated for the last six sessions of each phase of training for all rats, and these values are shown in Figure 1. Inspection of these data suggests that *U* values were generally greater under conditions of signaled reinforcement than under conditions of unsignaled reinforcement. For Group Sig-Unsig, *U* values were higher (indicating more variability) for four of eight rats with signaled reinforcement in Phase 1 compared with the unsignaled reinforcement in Phase 2. Another three of eight rats showed little difference between the phases, and one rat showed higher *U* values in Phase 2 (unsignaled) than in Phase 1. For Group Unsig-Sig, all eight rats showed higher *U* values in Phase 2 (signaled) than in Phase 1 (unsignaled).

Figure 2 shows the group mean *U* values for both groups in both phases of the experiment. Inspection of these data shows that, in both phases, the group that received signaled reinforcement had a higher *U* value than the group receiving unsignaled reinforcement. There was a decrease in the *U* value for Group Sig-Unsig from Phase 1 (signaled reinforcement) to Phase 2 (unsignaled reinforcement) and an increase in the *U* value for Group Unsig-Sig between Phase 1 (unsignaled) and Phase 2 (signaled).

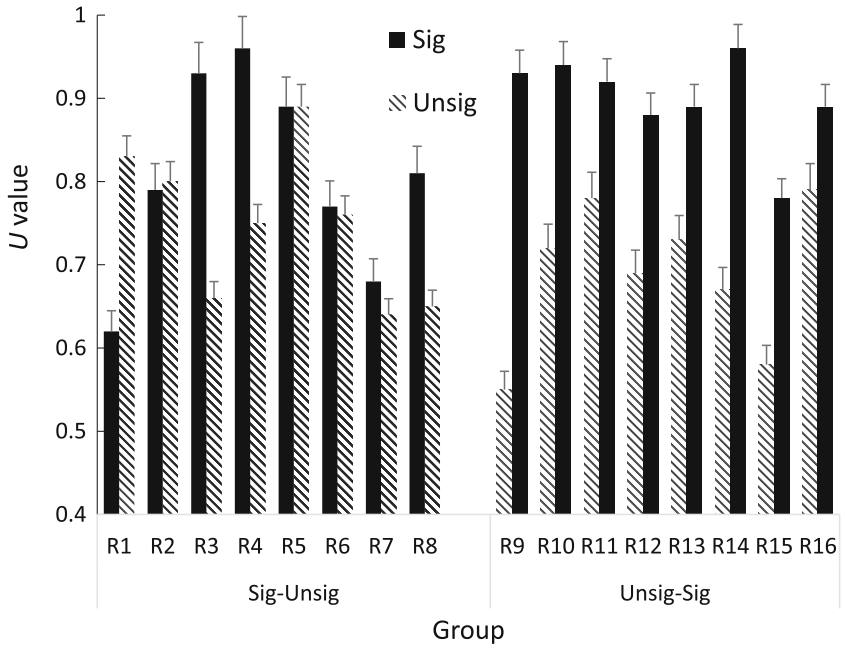


**FIGURE 1** Experiment 1: Mean U values for the last six sessions in each phase for all rats in Group Sig-Unsig and Unsig-Sig on the lag schedule. Error bars = 95% CI.

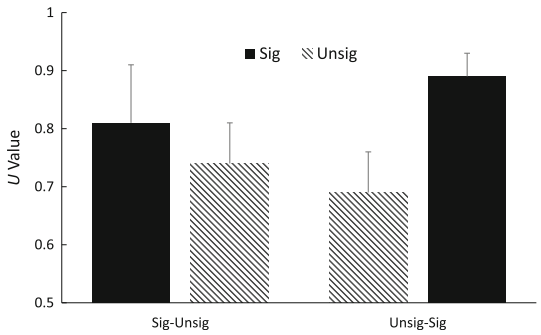


**FIGURE 2** Experiment 1: Group mean U values in each phase for Group Sig-Unsig and Unsig-Sig on the lag schedule. Error bars = 95% CI.

A two-factor repeated measures analysis of variance (ANOVA), with group as a between-subject factor and phase as a within-subject factor, was conducted on these data. The results of this analysis are reported below, along with effect sizes and 95% confidence limits and the appropriate Bayes statistic. The Bayesian statistics are reported because when conclusions may be based on comparing significant with nonsignificant effects, it is as well to have some corroboration that the null effects are real and not due to power issues. These analyses revealed a significant main effect for phase, $F(1, 14) = 5.96$, $p = .028$, $\eta^2_p = .299$, 95% CI [.000, .566], $p(H_1/D) = .850$, no main effect for group, $F < 1.00$, $\eta^2_p = .017$, 95% CI [.000, 267], $p(H_0/D) = .999$, but a significant interaction between the factors, $F(1, 14) = 18.85$, $p < .001$, $\eta^2_p = .574$, 95% CI [.164, .744], $p(H_1/D) = .999$. Simple effect analyses revealed a significant difference between the groups for Phase 1, $F(1, 14) = 6.85$, $p = .020$, $\eta^2_p = .329$, 95% CI [.006, .58611], $p(H_1/D) = .850$, and for Phase 2, $F(1, 14) = 11.25$, $p = .005$, $\eta^2_p = .446$, 95% CI [.058, .664], $p(H_1/D) = .962$. There was no difference between the phases for Group Sig-Unsig, $F(1, 14) = 1.75$, $p = .207$, $\eta^2_p = .111$, 95% CI [.000, .408], $p(H_0/D) = .615$, but there was a significant phase difference for Group Unsig-Sig, $F(1, 14) = 28.75$, $p < .001$, $\eta^2_p = .673$, 95% CI [.287, .803], $p(H_1/D) = .997$.

The mean percentage of sequences reinforced was calculated for the last six sessions of each phase of training for all rats, and these values are shown in Figure 3. Inspection of these data suggests that more sequences were reinforced (indicating greater variability) under conditions of signaled reinforcement than under conditions of unsignaled reinforcement. For Group Sig-Unsig, percentages were higher for four of eight rats with signaled reinforcement in Phase 1 compared with the unsignaled reinforcement in Phase 2. Another, three of eight rats showed little difference between the phases, and one rat showed a higher percentage of reinforced sequences in Phase 2 (unsignaled) than in Phase 1. For Group Unsig-Sig, five of eight rats showed higher percentages reinforced in Phase 2 (signaled) than in Phase 1 (unsignaled), and three of eight rats showed little difference between the phases.

Figure 4 shows the group-mean percentage of reinforced sequences for both groups in both phases of the experiment. Inspection of these data shows that, in both

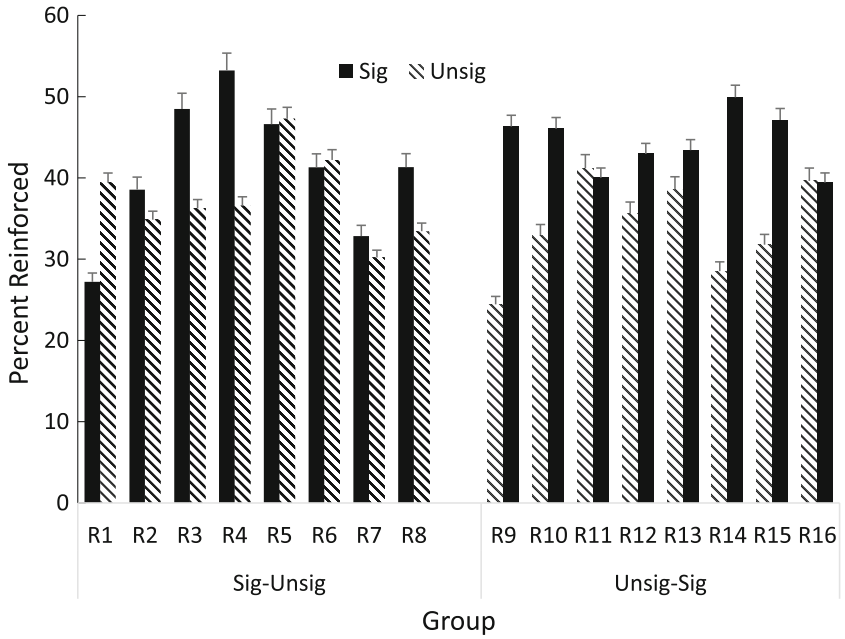


**FIGURE 3** Experiment 1: Mean percentage reinforced sequences for the last six sessions in each phase for all rats in Group Sig-Unsig and Unsig-Sig on the lag schedule. Error bars = 95% CI.

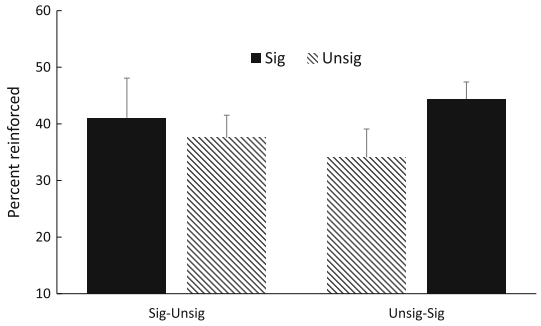


**FIGURE 4** Experiment 1: Group mean percentage sequences reinforced in each phase for Group Sig-Unsig and Unsig-Sig on the lag schedule. Error bars = 95% CI.

phases, the group that received signaled reinforcement had a higher percentage of reinforced sequences than the group that received unsignaled reinforcement. There was a decrease in the percentage of reinforced sequences for Group Sig-Unsig from Phase 1 (signaled reinforcement) to Phase 2 (unsignaled reinforcement) and an increase in the percentage of reinforced sequences for Group Unsig-Sig between Phase 1 (unsignaled) and Phase 2 (signaled). A two-factor repeated measures ANOVA (Group × Phase), along with effect size and 95% confidence limits and the appropriate Bayes statistic, revealed no main effect for phase, $F(1, 14) = 2.20$, $p = .161$, $\eta^2_p = .136$, 95% CI [.000, .433], $p(H_0/D) = .555$, or group, $F < 1$, $\eta^2_p = .001$, 95% CI [.000, 008], $p(H_0/D) = .999$, but a significant interaction between the factors, $F(1, 14) = 9.74$, $p = .008$, $\eta^2_p = .410$, 95% CI [.039, .641], $p(H_1/D) = .944$. Simple effect analyses revealed a significant difference between the groups for Phase 1, $F(1, 14) = 5.51$, $p = .034$, $\eta^2_p = .282$, 95% CI [.001, .554], $p(H_1/D) = .779$, and for Phase 2, $F(1, 14) = 5.15$, $p = .039$, $\eta^2_p = .269$, 95% CI [.001, .544], $p(H_1/D) = .755$. There was no difference between the phases for Group Sig-Unsig, $F(1, 14) = 1.36$, $p = .263$, $\eta^2_p = .089$, 95% CI [.000, .383], $p(H_0/D) = .663$, but there was as significant phase difference for Group Unsig-Sig, $F(1, 14) = 10.41$, $p < .001$, $\eta^2_p = .426$, 95% CI [.047, .652], $p(H_1/D) = .771$.

Both $U$ values and percentages of sequences reinforced indicate that a signal presented along with reinforcement tends to increase the level of variability exhibited on a lag schedule. This effect is less pronounced when the signal is removed after having previously been experienced. These data suggest that the signal improves learning of the variability contingency when on a lag schedule rather than causing increased repetition of the sequence that was just reinforced. These data stand in contrast to those presented by Reed et al. (1991) using a four-response sequence with rats, where the reinforcement signal tended to increase repetition of the preceding sequence. However, a major difference between that study and the current experiment is that there was no variability constraint for reinforcement in the study by Reed et al. (1991). Under those circumstances it has been shown that rats and pigeons tend not to respond variably but to repeat the preceding reinforced sequence (cf. Page & Neuringer, 1985; Schwartz, 1982). It might also be noted that in several studies of variability, the standard condition is to signal reinforcement, as in the current study (see Mook & Neuringer, 1994). The $U$ values noted here with signaled

reinforcement were highly similar to those previously reported in those studies.

## EXPERIMENT 2

An alternative to lag variability schedules are threshold schedules that involve scheduling reinforcement only for responses that occur at a relatively lower frequency than other responses. The frequency of all possible responses is calculated, and any current response that has a particularly low frequency of past emission leads to the delivery of a reinforcer; current responses above this frequency criterion are not reinforced. Such a schedule has increased variability of IRTs (Blough, 1966), responses sequences (Neuringer et al., 2000), and verbal responses (Duker & van Lent, 1991). Experiment 2 examined the effect of reinforcement signals on a threshold schedule.

Rats were exposed to a threshold schedule, based on that described by Denney and Neuringer (1998; although excluding the weighting depending on relative recency to differentiate the schedule more strongly from the lag schedule used in Experiment 1). Food was delivered if the current four-response L/R sequence was in the lowest half of response sequences in terms of frequency—that is, if eight sequences had higher rates of emission. Half of the rats received unsignaled reinforcement, and half received a brief (500 ms) tone reinforcement signal (Reed et al., 1988). These contingencies were then reversed such that rats that previously received signaled reinforcement now received unsignaled reinforcement and vice versa.

### Method

#### Subjects and apparatus

Sixteen, experimentally naive male Lister rats, housed and maintained as described in Experiment 1, served in the current experiment. The apparatus was as described in Experiment 1.

#### Procedure

The rats received two sessions of magazine training (RT 60 s), two sessions of a multiple CRF CRF schedule, and six sessions on a concurrent RI 30-s RI 30-s schedule, as described in Experiment 1. The rats were then transferred to a threshold variability schedule. A trial commenced with the insertion of both levers into the chamber and comprised four lever presses across the left and right levers (i.e., there were 16 possible sequences of four responses). The threshold schedule reinforced relatively infrequent sequences. Given there were 16 possible response sequences, reinforcement was delivered for the current sequence only if its relative frequency was less than a criterion value. The relative frequency of a sequence was defined as the number of times it had occurred during the current session, divided by total occurrences of all possible sequences. A reinforcer was provided only if the relative frequency of the sequence was below 50%. The first sequence of each session was always reinforced, as there were no sequences previously emitted in the session. On the next trial, emission of any of the 15 other sequences could receive reinforcement, and so on.

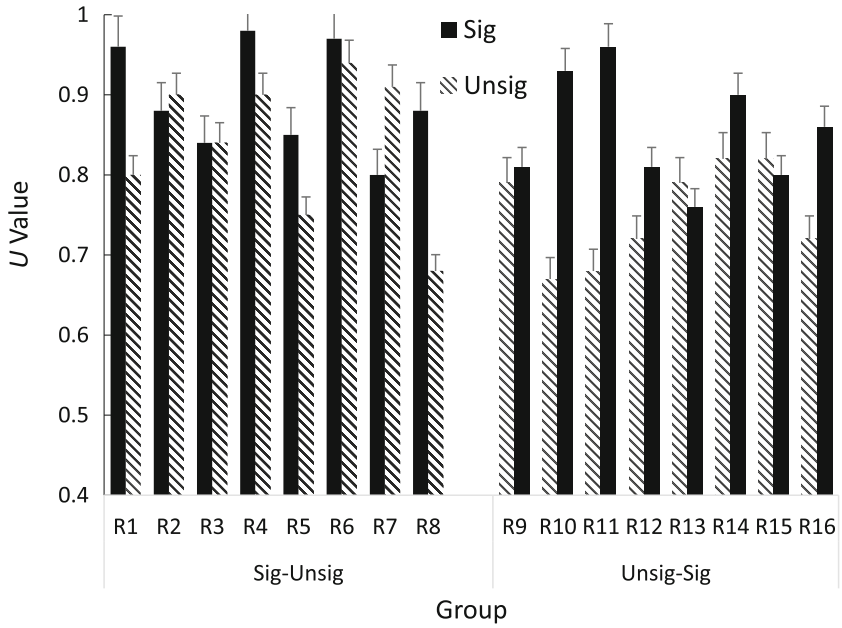


**FIGURE 5** Experiment 2: Mean U values for the last six sessions in each phase for all rats in Group Sig-Unsig and Unsig-Sig on the threshold schedule. Error bars = 95% CI.

For one group (Group Sig-Unsig), successful satisfaction of the threshold contingency resulted in the presentation of a 500-ms 105-dB(A) tone simultaneously with reinforcement delivery, but not on unsuccessful trials, in Phase 1. In Phase 2, no reinforcement signal was presented. The other group (Group Unsig-Sig) received the tone reinforcement signal in Phase 2 but not in Phase 1. There were 40 sessions of training for both groups.

## Results and discussion

The mean *U* values for the last six sessions of each phase of training for all rats are shown in Figure 5. For Group Sig-Unsig, four of eight rats showed higher *U* values (indicating more variability) with signaled reinforcement in Phase 1 than with unsignaled reinforcement in Phase 2, two of eight rats showed little difference between the phases, and two of eight rats showed higher *U* values in Phase 2 (unsignaled) than in Phase 1. For Group Unsig-Sig, five of eight rats showed higher *U* values in Phase 2 (signaled reinforcement) than in Phase 1 (unsignaled reinforcement), with three of eight rats showing no difference in *U* values between the phases.

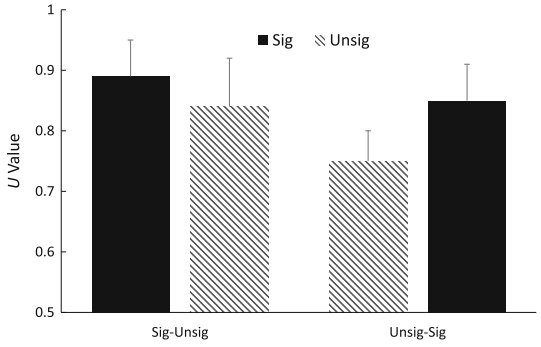


**FIGURE 6** Experiment 2: Group mean U values in each phase for Group Sig-Unsig and Unsig-Sig on the threshold schedule. Error bars = 95% CI.

Figure 6 shows the group mean *U* values for both groups in both phases of the experiment. Inspection of these data shows that in Phase 1 the group that received signaled reinforcement had a higher *U* value than did the group that received unsignaled reinforcement. There was a decrease in the *U* value for Group Sig-Unsig from Phase 1 (signaled reinforcement) to Phase 2 (unsignaled reinforcement) and an increase in *U* value for Group Unsig-Sig between Phase 1 (unsignaled) and Phase 2 (signaled). A two-factor repeated measures ANOVA (Group × Phase) revealed a significant main effect for group, $F(1, 14) = 7.23$, $p = .018$, $\eta^2_p = .341$, 95% CI [.009, .594], $p(H_1/D) = .876$, no significant main effect of phase, $F < 1$, $\eta^2_p = .047$, 95% CI [.000, 318], $p(H_0/D) = .715$, and a significant interaction, $F(1, 14) = 8.22$, $p = .012$, $\eta^2_p = .370$, 95% CI [.021, .614], $p(H_1/D) = .912$. Simple effect analyses revealed a significant difference between the groups for Phase 1, $F(1, 14) = 16.60$, $p < .001$, $\eta^2_p = .542$, 95% CI [.132, .729], $p(H_1/D) = .889$, but not for Phase 2, $F < 1$, $p > .70$, $\eta^2_p = .014$, 95% CI [.000, .258], $p(H_0/D) = .727$. There was no difference between the phases for Group Sig-Unsig, $F(1, 14) = 2.37$, $p = .167$, $\eta^2_p = .144$, 95% CI [.000, .441], $p(H_0/D) = .600$, but there was as significant phase difference for Group

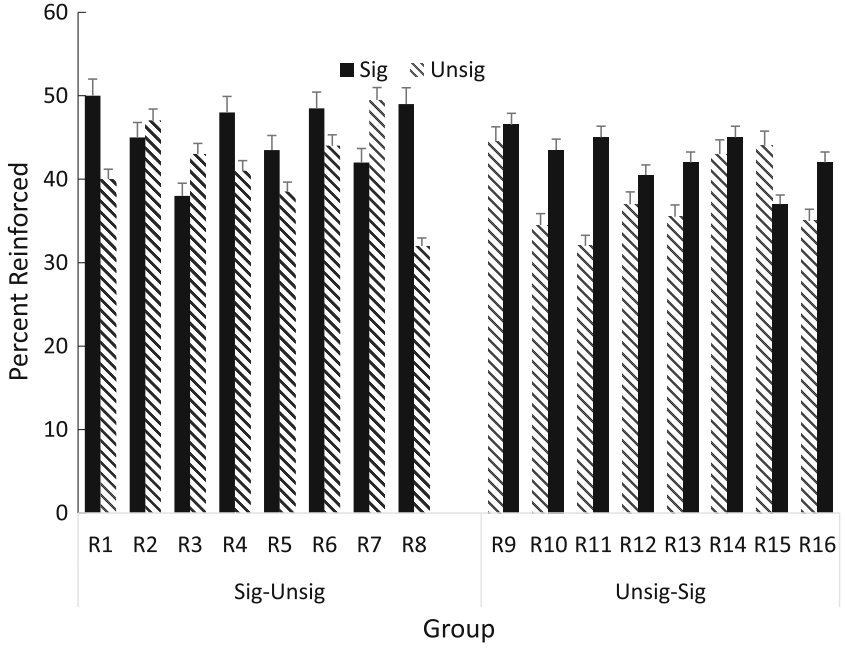


**FIGURE 7** Experiment 2: Mean percentage reinforced sequences for the last six sessions in each phase for all rats in Group Sig-Unsig and Unsig-Sig on the lag schedule. Error bars = 95% CI.

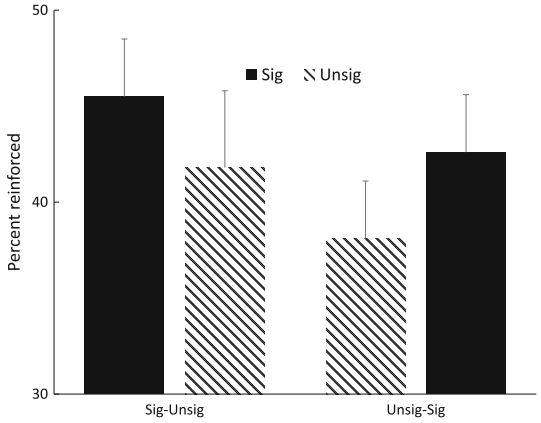


**FIGURE 8** Experiment 2: Group mean percentage sequences reinforced in each phase for Group Sig-Unsig and Unsig-Sig on the lag schedule. Error bars = 95% CI.

Unsig-Sig, $F(1, 14) = 6.02$, $p = .044$, $\eta^2_p = .300$, 95% CI [.001, .566], $p(H_1/D) = .698$.

The mean percentage of sequences reinforced was calculated for the last six sessions of each phase of training for all rats, and these values are shown in Figure 7. Inspection of these data suggests that more sequences were reinforced (i.e., there was greater variability) under conditions of signaled reinforcement than under conditions of unsignaled reinforcement. For Group Sig-Unsig, percentages were higher for four of eight rats with signaled reinforcement in Phase 1 compared with the unsignaled reinforcement in Phase 2. Another, two of eight rats showed little difference between the phases, and two of eight rats showed a higher percentage of reinforced sequences in Phase 2 (unsignaled) than in Phase 1. For Group Unsig-Sig, five of eight rats showed higher percentages reinforced in Phase 2 (signaled) than in Phase 1 (unsignaled), two of eight rats showed little difference between the phases, and one rat showed a higher percentage of reinforced sequences in the unsignaled phase.

Figure 8 shows the group mean percentage of reinforced sequences for both groups in both phases of the experiment. Inspection of these data shows that in both phases the group that received signaled reinforcement had a higher percentage of reinforced sequences than did the group that received unsignaled reinforcement. There was a decrease in the percentage of reinforced sequences for Group Sig-Unsig from Phase 1 (signaled reinforcement) to Phase 2 (unsignaled reinforcement) and an increase in the percentage of reinforced sequences for Group Unsig-Sig between Phase 1 (unsignaled) and Phase 2 (signaled). A two-factor repeated measures ANOVA (Group × Phase), along with effect size and 95% confidence limits and the appropriate Bayes statistic, revealed no main effect for phase, $F < 1$, $\eta^2_p = .004$, 95% CI [.000, .139], $p(H_0/D) = .796$, a significant effect of group, $F(1, 14) = 5.13$, $p = .029$, $\eta^2_p = .268$, 95% CI [.000, 542], $p(H_1/D) = .999$, and a significant interaction between the factors, $F(1, 14) = 5.13$, $p = .039$, $\eta^2_p = .282$, 95% CI [.001, .554], $p(H_1/D) = .807$. Simple effect analyses revealed a significant difference between the groups for Phase 1, $F(1, 14) = 4.87$, $p = .045$, $\eta^2_p = .258$, 95% CI [.001, .534], $p(H_1/D) = .775$, but not for Phase 2, $F < 1$, $\eta^2_p = .007$, 95% CI [.000, .212], $p(H_0/D) = .999$. There was no difference between the phases for Group Sig-Unsig, $F(1, 14) = 1.01$, $p > .30$, $\eta^2_p = .067$, 95% CI [.000, .357], $p(H_0/D) = .586$, but there was a significant phase difference for Group Unsig-Sig, $F(1, 14) = 4.56$, $p = .050$, $\eta^2_p = .246$, 95% CI [.000, .526], $p(H_1/D) = .503$.

These data, taken together, suggest that a reinforcement signal increases variability exhibited on a threshold schedule. As with Experiment 1, this effect of the reinforcement signal was less pronounced when the signal was removed, having previously been experienced. These data corroborate that the signal improves learning on a variability schedule rather than causing increased repetition of the sequence that was just reinforced. In combination with the data obtained from Experiment 1 using a lag variability schedule, they suggest that the nature of the variability schedule does not affect the tendency of the reinforcement signal to promote that variability.

## EXPERIMENT 3

The preceding two experiments have suggested that a signal presented prior to reinforcement on a variability schedule of either a lag (Experiment 1) or threshold (Experiment 2) nature increases operant variability. To corroborate that these effects are the result of the variability contingency, the third experiment examined the effect of a brief signal for reinforcement on emission of response sequences when there was no variability requirement. On such a schedule, Reed et al. (1991) have demonstrated that a reinforcement signal will increase the emission of one particular sequence and decrease overall variability. As noted in the current Experiment 1, a difference between Experiments 1 and 2 and that of Reed et al. (1991) is that there was no variability constraint for reinforcement in the latter study. The current experiment extended this examination to include a range of reinforcement rates to determine whether this variable might play a role. To this end, groups that potentially could have every sequence reinforced (100% reinforcement) were compared with groups that potentially would have a much lower rate of reinforcement (25% reinforcement). These values were selected purely to generate a large difference in the scheduled rate of reinforcement.

### Method

#### Subjects and apparatus

Thirty-two, experimentally naïve male Lister rats, housed and maintained as described in Experiment 1, served in

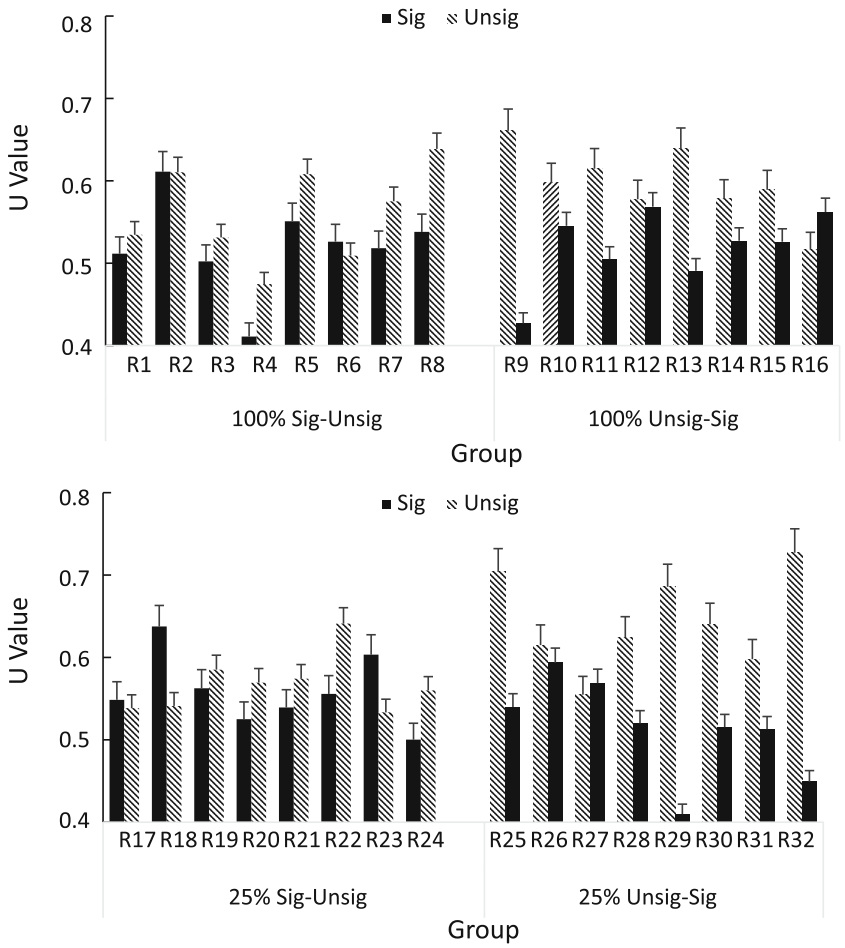


**FIGURE 9** Experiment 3: Mean U values for the last six sessions in each phase for all rats in Groups Sig-Unsig and Unsig-Sig. Top panel: 100% reinforcement groups. Bottom panel: 25% reinforcement groups. Error bars = 95% CI.

the current experiment. The apparatus was as described in Experiment 1.

## Procedure

The rats received two sessions of magazine training (RT 60 s), two sessions of a multiple CRF, CRF schedule, and six sessions on a concurrent RI 30-s RI 30-s schedule, as described in Experiment 1. The rats were then transferred to the four-response contingency, as described in Experiments 1 and 2, except that there was no requirement to vary the response to obtain reinforcement. Thus, four lever presses were required across the left and right levers, in any combination (i.e., there were 16 possible sequences of four responses), for reinforcement, with no variability constraint. When this criterion was satisfied, a food pellet was presented and the levers were retracted. This was followed by a 3-s ITI. Following the ITI, both levers were reinserted and the next trial started.

Four groups of rats responded on this schedule, two (Groups 100%) receiving reinforcement on 100% of trials and two groups (Groups 25%) receiving 100% reinforcement on the first two sessions of training. On sessions 3 and 4, they received 50% reinforcement; each emission of a sequence that fulfilled the criterion for reinforcement had a .50 chance of reinforcement. This was followed by 25% reinforcement for the remaining trials; each sequence fulfilling the criterion has a .25 probability of reinforcement. On nonreinforced trials, no reinforcer was delivered following the fourth response, the levers were retracted, and there was a 3-s ITI. Sessions terminated after 100 trials.

For two groups (Group 100% Sig-Unsig and Group 25% Sig-Unsig), reinforcement in Phase 1 resulted in the presentation of a 500-ms 105-dB(A) tone simultaneously with the delivery of reinforcement. There was no tone presented on nonreinforced trials. In Phase 2, these groups did not receive the tone reinforcement signal. For the other two groups (Group 100% Unsig-Sig and Group 25% Unsig-Sig), these reinforcement-signaling contingencies were reversed so that they received no signal in Phase 1 and a signal in Phase 2. There were 40 sessions of training for both groups in both Phases.

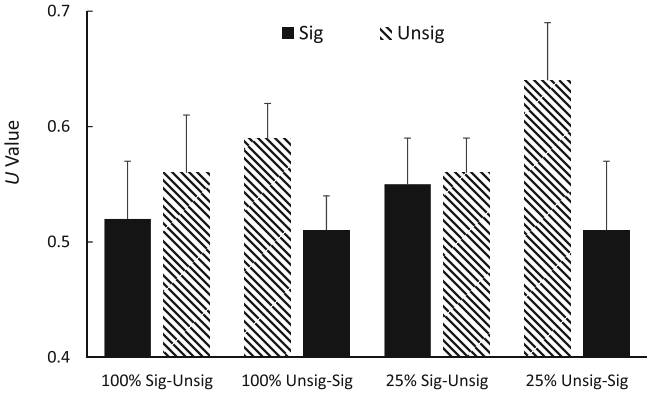


**FIGURE 10** Experiment 3: Group mean U values in each phase for Group Sig-Unsig and Unsig-Sig for all groups. 100% = 100% reinforcement; 25% = 25% reinforcement. Error bars = 95% CI.

## Results and discussion

The mean *U* values for the last six sessions of each phase of training, for all rats, are shown in Figure 9. For Group 100% Sig-Unsig, six of eight rats showed lower *U* values (indicating less variability) with signaled reinforcement in Phase 1 than with unsignaled reinforcement in Phase 2, one rat showed no difference between the phases, and one rat showed a higher *U* value with signaled reinforcement in Phase 1. For Group Unsig-Sig, six of eight rats showed lower *U* values in Phase 2 (signaled reinforcement) than in Phase 1 (unsignaled reinforcement), one rat showed no difference between the phases, and one rat showed a higher *U* value with signaled reinforcement. For Group 25% Sig-Unsig, four of eight rats showed lower *U* values (indicating less variability) with signaled reinforcement in Phase 1 than with unsignaled reinforcement in Phase 2, two of eight rats showed no difference between the phases, and two of eight rats showed higher *U* values with signaled reinforcement. For Group Unsig-Sig, six of eight rats showed lower *U* values in Phase 2 (signaled reinforcement) than in Phase 1 (unsignaled reinforcement), and two of eight rats showed no difference between the phases.

Figure 10 shows the group mean *U* values for all groups in both phases of the experiment. Inspection of these data shows that in Phase 1 groups that received signaled reinforcement had a lower *U* values than did groups that received unsignaled reinforcement. There was an increase in the *U* value for Group 100% Sig-Unsig from Phase 1 (signaled reinforcement) to Phase 2 (unsignaled reinforcement) and a decrease in *U* value for Group 100% Unsig-Sig and Group 25% Unsig-Sig between Phase 1 (unsignaled) and Phase 2 (signaled).

A three-factor repeated measures ANOVA (Reinforcement level × Counterbalancing × Phase) revealed a significant main effect for phase, $F(1, 28) = 8.43$, $p = .007$, $\eta^2_p = .231$, 95% CI [.020, .453], $p(H_1/D) = .926$, but not of counterbalancing, $F(1, 28) = 2.05$, $p = .167$, $\eta^2_p = .067$, 95% CI [.000, 279], $p(H_0/D) = .665$, or reinforcement level, $F(1, 28) = 3.65$, $p = .066$, $\eta^2_p = .115$, 95% CI [.000, 321], $p(H_0/D) = .523$. There was a significant interaction between counterbalancing and phase, $F(1, 28) = 21.56$, $p < .001$, $\eta^2_p = .435$, 95% CI [.151, .615], $p(H_1/D) = .912$, but not between counterbalancing and reinforcement level, $F < 1$, $\eta^2_p < .001$, 95% CI [.000, 001], $p(H_0/D) = .999$, phase and reinforcement level, $F(1, 28) = 2.55$, $p = .144$, $\eta^2_p = .083$, 95% CI [.000, .300], $p(H_0/D) = .614$, or all three factors, $F < 1$, $\eta^2_p = .006$, 95% CI [.000, .150], $p(H_0/D) = .825$. Simple effect analyses conducted on each counterbalancing condition revealed a significant difference between the groups for Phase 1, $F(1, 28) = 10.20$, $p = .003$, $\eta^2_p = .267$, 95% CI [.035, .484], $p(H_1/D) = .950$, but not for Phase 2, $F(1, 28) = 3.60$, $p = .068$, $\eta^2_p = .114$, 95% CI [.000, .3371], $p(H_0/D) = .520$. There was no difference between the phases for Group Sig-Unsig, $F(1, 28) = 1.00$, $p > .30$, $\eta^2_p = .035$, 95% CI [.000, .226], $p(H_0/D) = .769$, but there was as significant phase difference for Group Unsig-Sig, $F(1, 28) = 18.19$, $p < .001$, $\eta^2_p = .393$, 95% CI [.116, .584], $p(H_1/D) = .996$.

The mean percentage of sequences qualifying for reinforcement (note that only 25% of these received reinforcement in Groups 25%) for the last six sessions of each phase of training for all rats are shown in Figure 11. For Group 100% Sig-Unsig, seven of eight rats had a lower percentage of sequences qualifying for reinforcement (indicating less variability) with signaled reinforcement in Phase 1 than with unsignaled reinforcement in Phase 2, and one rat showed no difference between the phases. For 100% Group Unsig-Sig, five of eight rats had a lower percentage of sequences qualifying for reinforcement with signaled reinforcement (Phase 2) than with unsignaled reinforcement (Phase 1), two rats showed no difference between the phases, and one rat had a higher percentage of sequences qualifying for reinforcement with signaled reinforcement. For Group 25% Sig-Unsig, four of eight rats had a lower percentage of sequences qualifying for reinforcement with signaled reinforcement in Phase 1 than with unsignaled reinforcement in Phase 2, two of eight rats showed no difference between the phases, and two of eight rats had a higher percentage of sequences qualifying for reinforcement with signaled reinforcement. For Group Unsig-Sig, five of eight rats had a lower percentage of sequences qualifying for reinforcement with signaled reinforcement (Phase 2) than with unsignaled reinforcement (Phase 1), and three of eight rats showed no difference between the phases.

Figure 12 shows the group mean percentage of sequences qualifying for reinforcement for all groups in both phases of the experiment. Inspection of these data shows that in Phase 1 groups that received signaled reinforcement had a lower percentage of sequences qualifying for reinforcement than did groups that received unsignaled reinforcement. There was an increase in the percentage of sequences qualifying for reinforcement for Group 100%

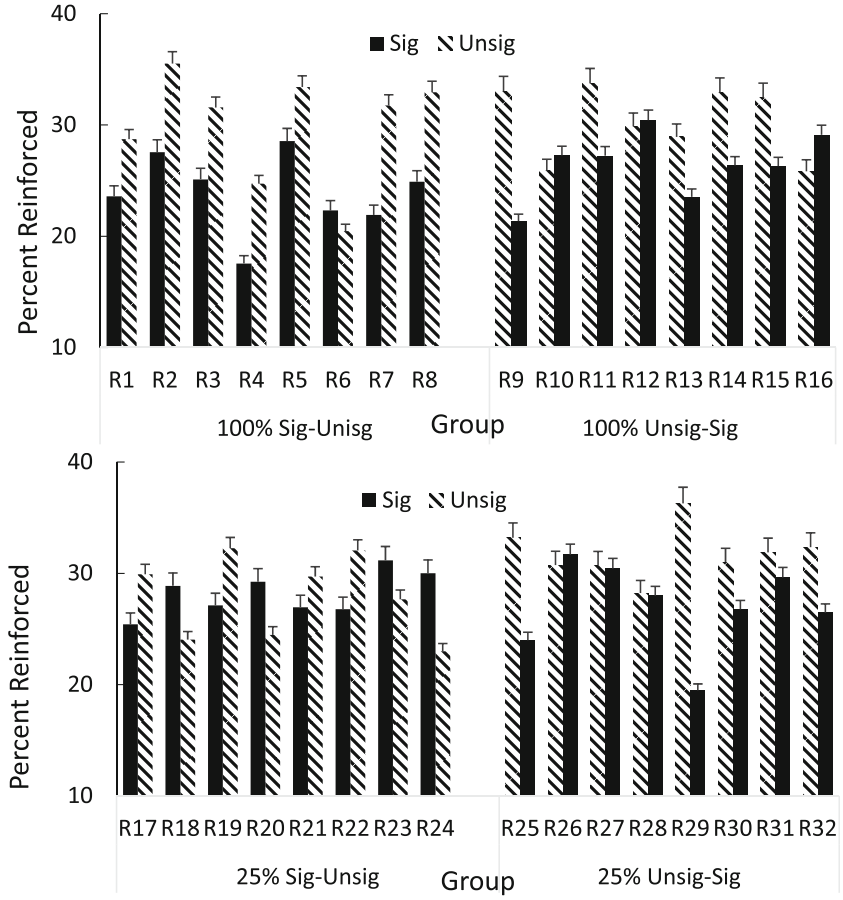


**FIGURE 11** Experiment 3: Mean percentage of sequences qualifying for reinforcement for the last six sessions in each phase for all rats in Groups Sig-Unsig and Unsig-Sig. Top panel: 100% reinforcement groups. Bottom panel: 25% reinforcement groups. Error bars = 95% CI.

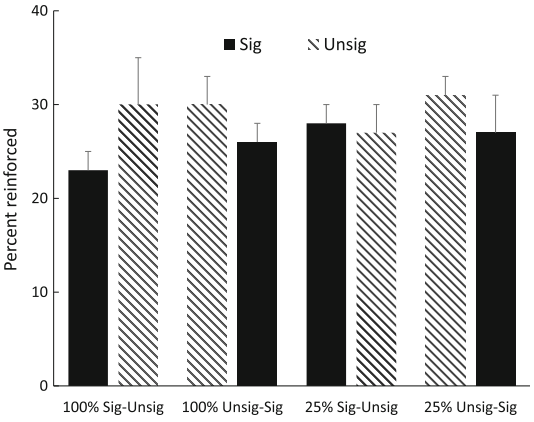


**FIGURE 12** Experiment 3: Group mean percentage of sequences qualifying for reinforcement in each phase for Group Sig-Unsig and Unsig-Sig for all groups. 100% = 100% reinforcement; 25% = 25% reinforcement. Error bars = 95% CI.

Sig-Unsig from signaled reinforcement (Phase 1) to unsignaled reinforcement (Phase 2) and a decrease in the percentage of sequences qualifying for reinforcement for Group 100% Unsig-Sig and Group 25% Unsig-Sig between Phase 1 (unsignaled) and Phase 2 (signaled).

A three-factor repeated measures ANOVA (Reinforcement level × Counterbalancing × Phase) revealed no significant main effect for phases, $F < 1$, $\eta^2_p = .028$, 95% CI [.000, .208], $p(H_0/D) = .999$, counterbalancing, $F(1, 28) = 3.00$, $p = .094$, $\eta^2_p = .097$, 95% CI [.000, 317], $p(H_0/D) = .535$, or reinforcement level, $F(1, 28) = 1.73$, $p = .199$, $\eta^2_p = .052$, 95% CI [.000, 265], $p(H_0/D) = .694$. There was a significant interaction between counterbalancing and phase, $F(1, 28) = 16.25$, $p < .001$, $\eta^2_p = .367$, 95% CI [.096, .564], $p(H_1/D) = .996$, and reinforcement level and phase, $F(1, 28) = 4.05$, $p = .050$, $\eta^2_p = .126$, 95% CI [.000, 351], $p(H_1/D) = .598$, but not between counterbalancing and reinforcement level, $F < 1$, $\eta^2_p = .001$, 95% CI [.000, .004], $p(H_0/D) = .999$, or among all three factors, $F(1, 28) = 2.32$, $p = .139$, $\eta^2_p = .077$, 95% CI [.000, 291], $p(H_0/D) = .614$. Simple effect analyses conducted on each counterbalancing condition revealed a significant difference between the groups for Phase 1, $F(1, 28) = 17.24$, $p < .001$, $\eta^2_p = .386$, 95% CI [.106, .575], $p(H_1/D) = .999$,

but not for Phase 2, $F(1, 28) = 3.45$, $p = .073$, $\eta^2_p = .109$, 95% CI [.000, .332], $p(H_0/D) = .515$. There was a difference between the phases for Group Sig-Unsig, $F(1, 28) = 5.05$, $p = .032$, $\eta^2_p = .152$, [.000, .378], $p(H_1/D) = .712$, and for Group Unsig-Sig, $F(1, 28) = 11.92$, $p = .002$, $\eta^2_p = .299$, [.051, .501], $p(H_1/D) = .999$.

These data replicate those from Reed et al. (1991) that demonstrated a reinforcement signal increased the emission of limited numbers of sequences and decreased variability. This effect was noted on both the 100% and 25% reinforcement rate schedules and suggests that reinforcement rate per se may play little part in influencing the effect of the signal. However, the possibility of extinction-induced variability should be considered as a contributor to the effects. The variability noted on the 25% schedules could have been contributed to by intermittent exposure to extinction. This may mean that any differences in variability noted between the 100% and 25% groups may have been due to extinction. It was the case that mean variability was numerically higher in the groups with lower rates of reinforcement (25% versus 100%). In the 25% groups, subjects R17, R18, and R23 had higher variability with signaled reinforcement than with unsignaled reinforcement. Additionally, there was higher variability, relative to most other rats, in the unsignaled phase with subjects R15, R28, R29, R30, and R32. However, it should be noted that these effects were not statistically reliable and it is difficult to know just how much weight to put on these observations.

Whatever the influence of extinction-induced variability, the effect of the signal was clearly similar across the 100% and 25% schedules. In comparison to the opposite pattern of results with respect to the effect of the signal on variability seen in the current Experiments 1 and 2, these data suggest it is the variability contingency that is important in determining the effect of the reinforcement signal. The presence of such a contingency has been noted to change the degree of variability in responding considerably relative to a range of control conditions sequence (Page & Neuringer, 1985). Moreover, in the absence of such a contingency, responding tends to become more stereotypical (Reed et al., 1991; Schwartz, 1982). Thus, the reinforcement signal appears to facilitate the type of learning that the schedule requires.

## GENERAL DISCUSSION

The current experiments examined the effect of signaling reinforcement on variability contingencies. The primary purpose of these studies was to document the effects of signaled reinforcement on a contingency that has previously not been examined in this respect. Variability schedules differ from previously studied schedules (cf. Reed et al., 1988; Roberts et al., 1984) in that their operant is not defined by a particular response topography. It was hoped to explore the manner in which operant variability interacts with other aspects of the contingency, which has received no or scant attention. These data also allow some theoretical insight into possible mechanisms of action of variability schedules and of reinforcement signals on free-operant schedules.

In Experiment 1, rats responding on a lag-8 variability schedule (Page & Neuringer, 1985) with signaled reinforcement displayed greater levels of variability than did rats on the same schedule lacking a reinforcement signal. In Experiment 2, rats responding on a threshold variability schedule (Neuringer et al., 2000) also displayed greater operant variability with signaled reinforcement compared with rats without a reinforcement signal. These effects stand in contrast to the effect of a reinforcement signal on the emission of response sequences when there was no variability requirement (Experiment 3), where the signal reduced operant variability.

These results offer empirical confirmation that operant variability responds to signaled reinforcement in the same way as do other forms of operant response—that is, the signal facilitates the emission of the required operant (Reed et al., 1991). To this extent, the current findings corroborate a range of previous studies that have shown that variability can be an operant that is sensitive to its consequences (e.g., Blough, 1966; Doughty & Galizio, 2015; Pryor et al., 1969; Schoenfeld et al., 1966) and, therefore, can be conditioned in similar manners to other topologically defined responses, like a lever press. In fact, the current findings relating to the effects of signaled reinforcement on variability add to the existing evidence for the relationship of variability to other sources of behavioral control, such as discriminative stimuli and reinforcing consequences, which are characteristics of other operant dimensions (see Neuringer, 2002, for a discussion).

The current results from Experiments 1 and 2 also suggest that a reinforcement signal serves to increase the variability targeted for reinforcement, and it did so on both lag and threshold schedules. This further develops parallels between variability as an operant and other forms of response, as with simple lever presses reinforcement signals have been shown to facilitate learning about the operant (see Reed et al., 1988, 1991). Using a threshold schedule procedure, Doughty and Galizio (2015) suggested that reinforcement served to induce variability of responses emitted closest to the reinforcer, and a signal for reinforcement may serve to enhance this process.

Both forms of variability schedule (lag and threshold) employed in the current series increased variability and have been noted to increase variability across response types that are similar to one another (Neuringer, 2012; Silbaugh et al., 2021). However, one uninvestigated difference between these two variability schedules has been highlighted by Lee et al. (2007), who discussed whether differences in the manners by which variability schedules operate and need to be monitored have any implications for their usage in the clinical arena. For example, it could

be assumed that lag schedules may have more utility because they place fewer demands on the participant's abilities, requiring monitoring of recent, but not all, responses. Whereas threshold and percentile schedules require constant monitoring for all responses to determine whether the current response fits the criteria for reinforcement (Lee et al., 2007; Silbaugh et al., 2021). This is clearly an applied issue of some importance that requires further investigation.

Although it may well be true that these aspects of the variability schedules have practical implications, the current series of studies noted little difference in the manner of implementation of the variability constraint for responding. Albeit a cross-experimental comparison, variability as measured by the *U* value was slightly higher for the threshold schedule (Experiment 2) than for the lag schedule in Experiment 1. However, little should be made of these minor differences (the mean *U* in the unsignaled condition was .72 for the lag schedule, compared with .79 for the threshold schedule), which could reflect the operation of many variables differing across the two current studies. The interaction between the variability schedule and signaling reinforcement was highly similar in the current Experiments 1 and 2. In both the lag and threshold schedules, the reinforcement signal increased operant variability. It is the case that the relative effect was greater for the lag schedule in Experiment 1 (increasing the *U* value by approximately 15%) than for the threshold schedule in Experiment 2 (increasing the *U* value by about 8%). Again, it is hard to make much of such a cross-experimental comparison, and this difference may reflect the lower baseline *U* value in the unsignaled condition for the lag schedule. The current exploration of the effects of signaled reinforcement on variability schedules suggests that this operant (i.e., variability) is affected in the same manner as are operants that are defined by their topography (cf. Reed et al., 1988; Roberts et al., 1984).

It is possible to derive a number of theoretical predictions about the nature of the effect of a signal for reinforcement on the two types of variability schedules. These predictions are based on assumptions regarding the influence that reinforcement signals have on learning about the primary aspects of the contingencies and which of these aspects are most prominent in various types of variability schedule. As noted above, if it were assumed that reinforcement signals facilitate learning of the immediately preceding response (Reed, 1989b; Reed et al., 1991) and that lag schedules have a more molecular basis than threshold schedules (as they do not require all responses to be monitored; see Lee et al., 2007), then it could be suggested that signaling reinforcement would facilitate learning about the variability contingency on lag but not on threshold schedules. Alternatively, if reinforcement signals are assumed to facilitate learning about molar aspects of the contingency (Roberts et al., 1984), then they may have more of an influence on threshold than on lag schedules. Of course, this analysis is making a lot of assumptions about the nature of the lag and threshold schedules.

It is unclear whether the current lag-8 schedule could reasonably be termed "molecular," especially as the response unit comprises four responses (making a total of 32 individual responses). Lee et al. (2007) were discussing clinical contents, where lag-1 schedules are often employed and single individual responses (admittedly complex in nature) are the "unit" of response. Moreover, the lag and threshold schedules differ from each other in a number of other ways that may be interacting with the reinforcement signal. For example, in a threshold schedule, repetition of a recently emitted response could be reinforced if that response is still under the threshold value, whereas a lag schedule never reinforces repetition. It may be that the investigation of variability schedules that have clearer or more definable aspects than the lag or threshold schedules may be of use in teasing apart these issues (see Machado, 1989, 1992, 1993). Thus, the differences between these schedules are interesting but their source is currently difficult to isolate. Moreover, neither of the above predictions were borne out by the current data, and the suggestion that the signal facilitates learning about the nature of the operant appears the best description of it action, without having to specify a particular mode of action (molecular or molar).

In terms of the mechanisms of reinforcement signals, a similar range of suggestions could be made about the influence of such a signal on variability schedules. If the reinforcement signals serve to improve the discriminability of the target response (see Fedorchak & Bolles, 1986), then it might have been predicted that they would have no influence on learning on variability schedules, as all responses will be followed by a reinforcement signal at some point and there will be no differential effect. This was not the case in either the current Experiment 1 or Experiment 2. It is difficult to see how a straightforward conditioned reinforcement account could accommodate the current data (see Williams, 1991). A marking hypothesis (Reed, 1989b) might suggest that the signal would serve to increase the memorability of a sequence of responses (see Reed et al., 1991). If this were the case, then it might be expected that this would facilitate performance on a variability schedule by helping to track the responses that have been made. Clearly, this effect would need to be time limited for any marked response; otherwise, the same issue as for the differential outcome effect explanation (Fedorchak & Bolles, 1986), discussed above, would need to be considered.

In summary, the current experiments have shown that a reinforcement signal increases the variability in emission of response sequences when there is a variability requirement. This was the case irrespective of the nature of the variability component. These results offer empirical confirmation that operant variability responds to this manipulation as other forms of operant response have been noted to respond and that a reinforcement signal facilitates the emission of the required operant.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

## ETHICS STATEMENT

The treatment of all nonhuman subjects was in accordance with ethical guidelines.

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## REFERENCES


Blough, D. S. (1966). The reinforcement of least-frequent interresponse times. *Journal of the Experimental Analysis of Behavior*, *9*(5), 581–591. https://doi.org/10.1901/jeab.1966.9-581

Denney, J., & Neuringer, A. (1998). Behavioral variability is controlled by discriminative stimuli. *Animal Learning & Behavior*, *26*(2), 154–162. https://doi.org/10.3758/BF03199208

Doughty, A. H., & Galizio, A. (2015). Reinforced behavioral variability: Working towards an understanding of its behavioral mechanisms. *Journal of the Experimental Analysis of Behavior*, *104*(3), 252–273. https://doi.org/10.1002/jeab.171

Duker, P. C., & Van Lent, C. (1991). Inducing variability in communicative gestures used by severely retarded individuals. *Journal of Applied Behavior Analysis*, *24*(2), 379–386. https://doi.org/10.1901/jaba.1991.24-379

Fedorchak, P. M., & Bolles, R. C. (1986). Differential outcome effect using a biologically neutral outcome difference. *Journal of Experimental Psychology: Animal Behavior Processes*, *12*(2), 125–130. https://psycnet.apa.org/doi/10.1037/0097-7403.12.2.125

Iversen, I. H. (1981). Response interactions with signaled delay of reinforcement. *Behaviour Analysis Letters*, *1*, 3–9.

Lee, R., McComas, J. J., & Jawor, J. (2002). The effects of differential and lag reinforcement schedules on varied verbal responding by individuals with autism. *Journal of Applied Behavior Analysis*, *35*(4), 391–402. https://doi.org/10.1901/jaba.2002.35-391

Lee, R., Sturmey, P., & Fields, L. (2007). Schedule-induced and operant mechanisms that influence response variability: A review and implications for future investigations. *The Psychological Record*, *57*(3), 429–455. https://doi.org/10.1007/BF03395586

Machado, A. (1989). Operant conditioning of behavioral variability using a percentile reinforcement schedule. *Journal of the Experimental Analysis of Behavior*, *52*(2), 155–166. https://doi.org/10.1901/jeab.1989.52-155

Machado, A. (1992). Behavioral variability and frequency-dependent selection. *Journal of the Experimental Analysis of Behavior*, *58*(2), 241–263. https://doi.org/10.1901/jeab.1992.58-241

Machado, A. (1993). Learning variable and stereotypical sequences of responses: Some data and a new model. *Behavioural Processes*, *30*(2), 103–129. https://doi.org/10.1016/0376-6357(93)90002-9

Manabe, K., Staddon, J. E., & Cleaveland, J. M. (1997). Control of vocal repertoire by reward in budgerigars (*Melopsittacus undulatus*). *Journal of Comparative Psychology*, *111*(1), 50–62. https://doi.org/10.1037/0735-7036.111.1.50

Mook, D. M., & Neuringer, A. (1994). Different effects of amphetamine on reinforced variations versus repetitions in spontaneously hypertensive rats (SHR). *Physiology & Behavior*, *56*(5), 939–944. https://doi.org/10.1016/0031-9384(94)90327-1

Morgan, L., & Neuringer, A. (1990). Behavioral variability as a function of response topography and reinforcement contingency. *Animal Learning & Behavior*, *18*(3), 257–263. https://doi.org/10.3758/BF03205284

Nergaard, S. K., & Holth, P. (2020). A critical review of the support for variability as an operant dimension. *Perspectives on Behavior Science*, *43*(3), 579–603. https://doi.org/10.1007/s40614-020-00262-y

Neuringer, A. (2012). Reinforcement and induction of operant variability. *The Behavior Analyst*, *35*(2), 229–236. https://doi.org/10.1007/BF03392281

Neuringer, A. (2002). Operant variability: Evidence, functions, and theory. *Psychonomic Bulletin & Review*, *9*(4), 672–705. https://doi.org/10.3758/BF03196324

Neuringer, A., Deiss, C., & Olson, G. (2000). Reinforced variability and operant learning. *Journal of Experimental Psychology: Animal Behavior Processes*, *26*(1), 98–111. https://doi.org/10.1037/0097-7403.26.1.98

Page, S., & Neuringer, A. (1985). Variability is an operant. *Journal of Experimental Psychology: Animal Behavior Processes*, *11*(3), 429–452. https://doi.org/10.1037/0097-7403.11.3.429

Pryor, K. W., Haag, R., & O'Reilly, J. (1969). The creative porpoise: Training for novel behavior. *Journal of the Experimental Analysis of Behavior*, *12*(4), 653–661. https://doi.org/10.1901/jeab.1969.12-653

Reed, P. (1989a). Marking effects in instrumental performance on DRH schedules. *The Quarterly Journal of Experimental Psychology Section B*, *41*(4b), 337–353. https://doi.org/10.1080/14640748908401200

Reed, P. (1989b). Influence of interresponse time reinforcement on signalled-reward effect. *Journal of Experimental Psychology: Animal Behavior Processes*, *15*(3), 224–231. https://doi.org/10.1037/0097-7403.15.3.224

Reed, P. (2015). Rats show molar sensitivity to different aspects of random-interval-with linear-feedback-functions and random-ratio schedules. *Journal of Experimental Psychology: Animal Learning and Cognition*, *41*(4), 432–443. https://doi.org/10.1037/xan0000065

Reed, P., Schachtman, T. R., & Hall, G. (1988). Potentiation of responding on a VR schedule by a stimulus correlated with reinforcement: Effects of diffuse and localized signals. *Animal Learning & Behavior*, *16*(1), 75–82. https://doi.org/10.3758/BF03209046

Reed, P., Schachtman, T. R., & Hall, G. (1991). Effect of signaled reinforcement on the formation of behavioral units. *Journal of Experimental Psychology: Animal Behavior Processes*, *17*(4), 475–485. https://doi.org/10.1037/0097-7403.17.4.475

Roberts, J. E., Tarpy, R. M., & Lea, S. E. (1984). Stimulus–response overshadowing: Effects of signaled reward on instrumental responding as measured by response rate and resistance to change. *Journal of Experimental Psychology: Animal Behavior Processes*, *10*(2), 244–255. https://doi.org/10.1037/0097-7403.10.2.244

Schoenfeld, W. N., Harris, A. H., & Farmer, J. (1966). Conditioning response variability. *Psychological Reports*, *19*(2), 551–557. https://doi.org/10.2466/pr0.1966.19.2.551

Schwartz, B. (1982). Failure to produce response variability with reinforcement. *Journal of the Experimental Analysis of Behavior*, *37*(2), 171–181. https://doi.org/10.1901/jeab.1982.37-171

Shimp, C. P. (1967). Reinforcement of least-frequent sequences of choices. *Journal of the Experimental Analysis of Behavior*, *10*(1), 57–65. https://doi.org/10.1901/jeab.1967.10-57

Silbaugh, B. C., Murray, C., Kelly, M. P., & Healy, O. (2021). A systematic synthesis of lag schedule research in individuals with autism and other populations. *Review Journal of Autism and Developmental Disorders*, *8*(1), 92–107. https://doi.org/10.1007/s40489-020-00202-1

Sizemore, O. J., & Lattal, K. A. (1978). Unsignalled delay of reinforcement in variable interval schedules. *Journal of the Experimental Analysis of Behavior*, *30*(2), 169–175. https://doi.org/10.1901/jeab.1978.30-169

Tarpy, R. M., & Roberts, J. E. (1985). Effects of signaled reward in instrumental conditioning: Enhanced learning on DRL and DRH schedules of reinforcement. *Animal Learning & Behavior*, *13*(1), 6–12. https://doi.org/10.3758/BF03213358

Williams, B. A. (1991). Marking and bridging versus conditioned reinforcement. *Animal Learning & Behavior*, *19*(3), 264–269. https://doi.org/10.3758/BF03197885


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