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Effects of reinforcer distribution during response elimination on resurgence of an instrumental behavior

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Abstract

Resurgence has commonly been viewed as the recovery of an extinguished instrumental behavior that occurs when an alternative behavior that has replaced it is also extinguished. Three experiments with rat subjects examined the effects on resurgence of the temporal distribution of reinforcement for the alternative behavior that is presented while the first response is being eliminated. Experiments 1 and 2 examined resurgence when rich rates of reinforcement at the onset of response elimination became leaner over sessions (i.e., forward thinning) and when lean rates became richer (i.e., reverse thinning). Both procedures weakened resurgence compared to that in a group that received the richest rate during all sessions. However, forward thinning was more effective than reverse thinning at reducing the resurgence effect. Experiment 3 found that final resurgence was eliminated when the alternative behavior was reinforced and extinguished in alternating response elimination sessions. The results are consistent with the hypothesis that reinforcer delivery during response elimination provides a contextual stimulus for the extinction of the original behavior; its removal during resurgence testing causes ABC renewal to occur. The results are less consistent with an alternative account that emphasizes the removal of response disruption caused by alternative reinforcement (Shahan & Sweeney, 2011). Other theoretical and applied implications are discussed.

Keywords

Resurgence; extinction; operant conditioning; behavioral momentum; relapse

Resurgence is said to occur when an extinguished behavior returns after an alternative behavior that replaced it is also extinguished. In a typical resurgence experiment, a target response (R1, e.g., pressing a lever) is first reinforced. In a second phase, R1 is no longer reinforced (i.e., extinguished) while a new response (R2, e.g., pressing a second lever) is introduced and provides an alternative source of reinforcement. Finally, in a third phase, R2 is also placed on extinction. The key result is that behavior on R1 often returns, or resurges (e.g., Bouton & Schepers, 2014; Leitenberg, Rawson, & Bath, 1970; Lieving & Lattal, 2003; Sweeney & Shahan, 2013a,b; Winterbauer & Bouton, 2010, 2012; Winterbauer, Lucke, & Bouton, 2013). Resurgence also occurs when reinforcers in Phase 2 are presented in a

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manner that is not contingent on R2 responding; that is, responding also resurges when noncontingent Phase-2 reinforcers are discontinued (Winterbauer & Bouton, 2010). The resurgence phenomenon is consistent with other relapse phenomena (e.g., renewal, spontaneous recovery, and reinstatement) in suggesting that extinction does not cause unlearning or erasure of the original behavior (for a review, see Bouton, 2004; Vurbic & Bouton, 2014). Rather, extinction of an instrumental response may involve learning not to perform the response in a particular context (Todd, 2013).

Animal experiments examining renewal, spontaneous recovery, and reinstatement may be important in helping understand the mechanisms of relapse that often occurs following clinical therapies in humans (Bouton, 2002; Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014; Vervliet, Craske, & Hermans, 2013). Resurgence experiments may be especially important for understanding relapse that can occur after the conclusion of contingency management (CM) therapy. CM has been an especially successful treatment for several types of drug abuse (Higgins, Silverman, & Heil, 2008). In CM, a new behavior, incompatible with drug taking (e.g., abstinence, work, or pro-social behavior), is introduced and reinforced with prizes, vouchers, or monetary awards contingent on continued abstinence from drugs (DeFulio, Donlin, Wong, & Silverman, 2009; Fisher, Green, Calvert & Glassgow, 2004). Research has also found that CM can successfully promote patient compliance by providing reinforcement for adherence to physician-directed health regimens (Raiff & Dallery, 2010; Rosen, Fox, & Gill, 2007; Stanger et al., 2013). However, in CM treatments, like resurgence experiments, the original behavior (i.e., drug taking, or behavior associated with poor health outcomes) is susceptible to relapse when alternative reinforcement is discontinued (e.g., Silverman, Kaminski, Higgins, & Brady, 2011).

At least three explanations of resurgence have been proposed. According to Leitenberg et al. (1970), resurgence occurs because rapid acquisition of the new behavior (i.e., R2) prevents the animal from making the original response (i.e., R1). Response competition precludes the original behavior from being truly extinguished during Phase 2. In Phase 3, when the source of competition is removed, resurgence occurs because R1 responding is not completely extinguished. Although this mechanism may contribute to resurgence under some conditions, resurgence can be observed when experimental groups make a large and presumably sufficient number of R1 responses to learn extinction during response elimination (e.g., see Bouton & Schepers, 2014). Moreover, Winterbauer and Bouton (2010) observed significant resurgence in several experimental groups that made a similar or even greater number of R1 responses during Phase 2 than control groups that did not resurge.

Alternatively, Winterbauer and Bouton (2010) suggested that resurgence occurs because the context changes when reinforcers are discontinued at the start of Phase 3. They suggested that resurgence of R1 may therefore be a special form of ABC renewal (Bouton, Todd, Vurbic, & Winterbauer, 2011; Todd 2013; Todd, Winterbauer, & Bouton, 2012). Previous research suggests that reinforcer (pellet) deliveries in the background can provide a distinct context that can control conditioning and extinction performance (e.g., Bouton, Rosengard, Achenbach, Peck & Brooks, 1993; Bouton 1991, 2002). Thus, when pellets are discontinued during resurgence testing, there is potentially a change in context. On this view, conditions that provide fewer reinforcers during Phase 2 while R1 is being extinguished may weaken

resurgence by allowing more generalization between its extinction and the extinction conditions that prevail during resurgence testing. In contrast, a high rate of reinforcers delivered during Phase 2 may provide a very distinct contextual change when all reinforcement is removed during the test. Resurgence is indeed weakened or eliminated if Phase 2 involves extremely lean rates of alternative reinforcement (Leitenberg, Rawson, & Mulick, 1975; Sweeney & Shahan, 2013b). Resurgence is also weakened by procedures that decrease or thin the rate of alternative reinforcement over the series of Phase 2 sessions (Winterbauer & Bouton, 2012, Sweeney & Shahan, 2013b). Theoretically, that result could occur because the animal learns that R1 is extinguished under conditions containing few reinforcers. In addition, thinning attenuates the abruptness of the contextual change that occurs between Phases 2 and 3.

In related experiments, Bouton and Schepers (2014) observed weakened resurgence when an abstinence contingency was associated with R1 responding during Phase 2. For example, in their Experiment 2, one group of rats received a treatment in which each R1 response resulted in a 45-s time-out that made alternative reinforcement for R2 unavailable. As a result of this abstinence contingency, the distribution of reinforcers delivered during Phase 2 resembled that of a thinning schedule in reverse. That is, early in Phase 2, when rats made many R1 responses, there were consequently many time-outs, and R2 responses produced few reinforcers. Then, in later sessions, when rats made fewer R1 responses, R2 was reinforced at a higher rate, similar to the rate in a control group without the abstinence contingency that was simply reinforced on a VI 10-s schedule. For a third group, R2 responses were reinforced only after a yoked animal from the abstinence contingency group had earned one. The yoking procedure provided the same distribution of reinforcers that the abstinence contingency group received without the negative contingency between R1 and reinforcement. At test, the abstinence contingency and yoked groups exhibited similar resurgence that was weaker than that of the VI 10-s control group. This result suggested that the reinforcer distribution resembling a thinning procedure in reverse, and not the abstinence contingency per se, was responsible for weakening the resurgence. Consistent with a contextual account, learning that R1 is extinguished in periods when there are few reinforcers, even early in Phase 2, may be sufficient to increase generalization between the extinction of R1 and the resurgence testing conditions.

A third account of resurgence is provided by Shahan and Sweeney (2011), who developed a quantitative model of resurgence based on behavioral momentum theory (e.g., Nevin & Grace, 2000). The Shahan-Sweeney model suggests that any source of alternative reinforcement (contingent on responding or not) introduced during Phase 2 has two effects on performance of the original behavior. First, it disrupts performance of the original response. Second, it somewhat paradoxically also contributes to a process that strengthens the original response (e.g., Nevin & Grace, 2000). On this view, the currently nonreinforced R1 response is strengthened by any alternative reinforcement delivered in the context, but the strengthening effects (i.e., resurgence) are not observed until the disrupting reinforcers are first removed. To date, quantitative simulations of the model have used alternative reinforcement at its terminal programmed rate (i.e., the programmed rate in the session immediately before the resurgence test) to predict the strength of resurgence. This approach makes predictions consistent with the resurgence-weakening effects produced by lean

reinforcement rates and thinning procedures described above (Sweeney & Shahan, 2013b; Winterbauer & Bouton, 2012). However, it cannot explain why reverse thinning treatments (e.g., Bouton & Schepers, 2014) reduce resurgence if the experimental and control groups receive equivalent programmed reinforcement rates in the last Phase 2 session. To address this sort of complication, Sweeney and Shahan (2013b) suggested that under certain circumstances using the mean rate of reinforcement throughout Phase 2 (i.e., average programmed rate over all Phase 2 sessions), instead of the rate from the final session, might provide a better fit to the data.

The current experiments were designed to further contrast predictions of the Shahan-Sweeney model and the context hypothesis by manipulating the temporal distribution of alternative reinforcers delivered during Phase 2. Experiment 1 was designed to replicate the weakened resurgence observed after a reverse thinning schedule (Bouton & Schepers, 2014). As noted earlier, the original Shahan-Sweeney (2011) model has difficulty explaining the effects of reverse thinning, because it exclusively emphasizes reinforcement rate in the final session of Phase 2. Experiment 2 then compared resurgence following reverse and forward thinning procedures that had the same average programmed rates of reinforcement over Phase 2. Here, the amended Shahan-Sweeney model (Shahan & Sweeney, 2013b) would predict no difference in resurgence. Experiment 3 provided a further opportunity to compare resurgence in groups that received similar average reinforcement rates during Phase 2. But in this case they differed in whether the rat had the opportunity to experience extended periods in which R1 was extinguished while alternative reinforcers were also unavailable for R2. The contextual hypothesis, but not the Shahan-Sweeney model, predicts that this procedure should further weaken resurgence.

Experiment 1

The first experiment expanded on the Bouton and Schepers (2014) experiments, in which rats earned reinforcement for R2 responses in Phase 2 only after a period of required abstinence from responding on R1. The design is shown in Table 1. During the first phase, all groups received reinforcement for R1 responses on a Variable Interval (VI) 30-s schedule, in which a reinforcer was available every 30 s on average. During Phase 2, all groups then received extinction of R1. Group VI 10-s earned reinforcers for R2 according to a VI 10-s schedule. Group Reverse Thinning was reinforced for R2 using a series of different VI schedules that yielded approximately the same daily number of reinforcers received by the 45-s Negative Contingency group from Bouton and Schepers (2014). During the final test, reinforcers in both groups were discontinued. In this way we re-examined the effects of reverse thinning on resurgence.

Two additional groups (Groups VI 10-s Yoked and Reverse Thinning Yoked) received a pellet that was not contingent on responding whenever a yoked rat in one of the other groups earned one during Phase 2. The yoked groups were included primarily to allow us to assess the practicality of using noncontingent Phase-2 reinforcers in subsequent experiments, which would allow complete experimenter control over pellet delivery. They also provided a further possible test of the Shahan-Sweeney model, which predicts that whether reinforcers delivered during the response elimination phase are response-contingent or non-contingent

should not affect rates of R1 responding during Phase 2 or the amount of resurgence in the test, although, as noted above, previous results already suggest that resurgence can be observed in either response-contingent or non-contingent (yoked) groups (Winterbauer & Bouton, 2010).

Method

Subjects—The subjects were 32 female Wistar rats (n = 8) obtained from Charles River, Inc. (St. Constance, Quebec). The rats were approximately 85–95 days old at the start of the experiment and were individually housed in suspended stainless steel cages in a room maintained on a 16:8-h light:dark cycle. At the beginning of the experiment, all rats were food deprived to 80% of their free-feeding weight and maintained at that level throughout the experiment with a single feeding following each day's session.

Apparatus—Conditioning proceeded in two sets of four standard conditioning boxes (Med-Associates, St. Albans, VT; model: ENV-008-VP) that were housed in different rooms of the laboratory. Boxes from both sets measured 31.75 × 24.13 × 29.21 cm (l × w × h) and were housed in sound-attenuation chambers. The front and back walls were aluminum; the sidewalls and ceiling were clear acrylic plastic. There was a 5.08 × 5.08 cm recessed food cup centered in the front wall near floor-level. 4.8 cm stainless steel operant levers (Med Associates model: ENV-112CM) were located to the left and to the right of the food cup, 6.2 cm above the floor. Sixty-dBA background noise provided by ventilation fans, and illumination provided by two 7.5-W incandescent bulbs mounted on the ceiling of the sound-attenuation chamber, remained on for the duration of the experiment. In one set of boxes, the floor consisted of 0.48-cm diameter stainless steel grids spaced 3.81 cm apart and mounted parallel to the front wall. The ceiling and a side wall had black horizontal stripes (3.81 cm wide). In the other set of boxes (also model ENV-008-VP), the floor consisted of alternating stainless steel grids with different diameters (0.48 and 1.27 cm), spaced 1.59 cm. The ceiling and left side wall were covered with dark dots (1.9 cm in diameter). The apparatus was controlled by computer equipment located in an adjacent room. Although the two sets of boxes can provide discriminably different contexts, they were not used in that capacity here. Food reward consisted of 45-mg MLab Rodent Tablets (TestDiet, Richmond, IN).

Procedure—All experimental sessions were 30 min in duration.

Magazine training: On the first day, each rat was assigned to a box and then received a single session in which free pellets were delivered on average every 30 s. The levers were retracted and unavailable during this session.

R1 conditioning (Phase 1): On each of the next 12 days, rats received one session in which R1 presses resulted in pellet delivery on a VI 30-s reinforcement schedule. All sessions began with a 2-min delay before the lever was inserted into the chamber. Following the delay, either the right lever or left lever (counterbalanced) was inserted. No special response shaping was necessary. Sessions ended after 30 min, when the lever was retracted.

Response elimination (Phase 2): On each of the next eight days, the rats received a single session that began with insertion of both the right and left levers after the usual 2-min delay. Rats were randomly assigned to one of four groups, with the restriction that individual boxes and the time of day when sessions occurred were equally represented among the groups. For all groups, R1 presses were recorded but were never reinforced throughout the phase. In Group VI 10-s, R2 presses were reinforced on a VI 10-s schedule. In Group Reverse Thinning, reinforcement was contingent on R2 responses according to schedules that became richer by a factor of four over the first four sessions (Session 1, VI 1200-s; Session 2, VI 300-s; Session 3, VI 75-s; Session 4 VI 19.5-s). During the final four sessions, these rats were then reinforced on a VI 10-s schedule. The remaining groups, Group VI 10-s Yoked and Group Reverse Thinning Yoked, were given a free pellet (not contingent on a lever press response) whenever a rat in Group VI 10-s or Group Reverse Thinning (respectively) earned one. All sessions ended with retraction of the levers at the end of 30 minutes.

Resurgence test (Phase 3): On the final day, all rats received a single 30-min test session in which both levers were inserted after the usual two-minute delay but presses had no scheduled consequences. The session ended with the retraction of both levers.

Data treatment: Response rates were expressed as responses per minute. Data were put through analyses of variance (ANOVAs) with a rejection criterion of $p < .05$. Pairwise comparisons using error terms from the overall ANOVA were then run to assess differences. During resurgence testing, we were primarily interested in separate analyses of the response-contingent and noncontingent groups, because a comparison of the contingent groups allowed us to replicate in the current method Bouton and Schepers's (2014) comparison of response-contingent groups, and a comparison of the noncontingent groups independently allowed us to assess the viability of using noncontingent reinforcers only in the next two experiments. Throughout, we were especially interested in changes in performance between the end of extinction and the resurgence test.

A statistical outlier in Group Reverse Thinning Yoked was removed from the analysis after failing to extinguish responding on R1 lever over Phase 2. During the final Phase 2 session this rat made 58.3 responses per minute on R1. In contrast, the other rats in the group averaged 2.3 responses per minute at this time.

Results

The results are summarized in Figure 1. During the first phase, responding on R1 increased uneventfully. Then, in Phase 2, R1 declined while responding on R2 increased in the groups that received response-contingent reinforcement. During resurgence testing, rats in the response-contingent groups (VI 10-s and Reverse Thinning) each produced a significant resurgence of R1 responding, although the effect was significantly weaker in the Reverse Thinning Group. The groups that had received Yoked pellets during Phase 2 showed a similar pattern, although the results were not as definitive statistically.

Response Acquisition and Elimination—Acquisition of R1 responding is summarized in the upper left panel of Figure 1. R1 increased reliably in all groups over the 12 sessions, $F(11, 297) = 55.28$, $MSE = 37.15$, $p < .001$, $\eta_p^2 = .67$, 95% CI [.60, .71]. Random assignment to groups was successful in the sense that the main effect of Group and the Group × Session interaction did not approach significance, Fs < 1.

As displayed in Figure 1's upper center panel, R1 responding decreased during the response elimination phase. A 2 (Reinforcer Distribution: VI 10-s vs. Reverse Thinning) × 2 (Contingency: Response Contingent vs. Non Contingent) × 8 (Session) ANOVA revealed main effects of Session, $F(7, 189) = 30.03$, $MSE = 14.21$, $p < .001$, $\eta_p^2 = .53$, 95% CI [.41, .59], and Contingency, $F(1, 27) = 9.93$, $MSE = 96.31$, $p < .01$, $\eta_p^2 = .27$, 95% CI [.03, .49]. The main effect of Reinforcer Distribution was not significant, $F < 1$. Significant Session × Reinforcer Distribution, $F(7, 189) = 5.34$, $MSE = 14.21$, $p < .01$, $\eta_p^2 = .17$, 95% CI [.06, .23], and Session × Reinforcer Distribution × Contingency, $F(7, 189) = 2.68$, $MSE = 14.21$, $p < .05$, $\eta_p^2 = .09$, 95% CI [.01, .14], interactions revealed that responding over the phase depended on the distribution of pellets and the overall pattern was dependent on the response contingency. The Session × Contingency interaction was not significant, $F < 1$. A one-way ANOVA examining the effect of contingency during the final Phase 2 session revealed that R1 was more suppressed when alternative reinforcement was contingent on responding (i.e., R2 presses) than when it was not, $F(1, 27) = 9.27$, $MSE = 8.56$, $p < .01$, $\eta_p^2 = .26$, 95% CI [.03, .48].

Turning to R2 (lower middle panel of Figure 1), a 2 (Reinforcer Distribution: VI 10-s vs. Reverse Thinning) × 2 (Contingency: Contingent vs. Non Contingent) × 8 (Session) ANOVA revealed significant main effects of Session, $F(7, 189) = 40.81$ $MSE = 25.46$, $p < .01$, $\eta_p^2 = .60$, 95% CI [.50, .65], and Contingency, $F(1, 27) = 77.66$, $MSE = 774.75$, $p < .001$, $\eta_p^2 = .74$, 95% CI [.53, .83]. The main effect of Reinforcer Distribution was not significant, $F(1, 27) = 2.13$, $MSE = 774.75$, $p = .16$. However, the Session × Reinforcer Distribution, $F(7, 189) = 4.00$, $MSE = 25.464$, $\eta_p^2 = .13$, 95% CI [.03, .19], and the Session × Contingency, $F(7, 189) = 48.08$, $MSE = 25.464$, $p < .001$, $\eta_p^2 = .64$, 95% CI [.55, .69], interactions were reliable. A reliable Session × Reinforcer Distribution × Contingency interaction, $F(7, 189) = 3.94$, $MSE = 25.464$, $p < .001$, $\eta_p^2 = .13$, 95% CI [.03, .19], indicated that differences in R2 responding over the phase depended on pellet distribution and that the overall pattern of results was dependent on the contingency.

Resurgence Test—Responding on R1 during the resurgence test (compared with responding during the last Phase 2 session) is shown in the upper right panel of Figure 1. A 2 (Session: Ext 8 vs. Resurgence Test) × 2 (Reinforcer Distribution: VI 10-s vs. Reverse Thinning) × 2 (Contingency: Response Contingent vs. Non Contingent) ANOVA indicated reliable main effects of Session, $F(1, 27) = 14.61$, $MSE = 6.16$, $p = .001$, $\eta_p^2 = .35$, 95% CI [.08, .55], Reinforcer Distribution, $F(1, 27) = 10.52$, $MSE = 10.29$, $p < .01$, $\eta_p^2 = .28$, 95% CI [.04, .50], and Contingency, $F(1, 27) = 8.02$, $MSE = 10.29$, $p < .01$, $\eta_p^2 = .23$, 95% CI [.02, .45]. No interactions reached significance, Fs < 2.15. We then conducted Group × Session ANOVAs that tested the effects in the response-contingent and non-contingent groups separately. The ANOVA isolating Groups VI 10-s and Reverse Thinning revealed a Session effect, $F(1,14) = 34.17$, $MSE = 3.55$, $p < .001$, $\eta_p^2 = .71$, 95% CI [.34, .83], a Group

effect, $F(1,14) = 14.87$, $MSE = 1.45$, $p = .002$, $\eta_p^2 = .52$, 95% CI [.11, .71], and a marginally significant group × session interaction, $F(1,14) = 3.55$, $MSE = 3.55$, $p = .08$, $\eta_p^2 = .20$, 95% CI [.00, .56]. Both groups increased responding from extinction to the resurgence test, $ps < .02$, suggesting resurgence. However, they differed during the test session, $p = .01$, but not during the last session of extinction, $p = .10$, indicating weaker resurgence in the Reverse Thinning group. A similar ANOVA on the noncontingent groups found no session effect or group × session interaction, $F(1, 13) < 1.72$. The group effect was reliable, $F(1, 13) = 5.03$, $MSE = 19.81$, $p < .05$, $\eta_p^2 = .28$, 95% CI [.00, .56]. Neither group had a significant increase in responding between extinction and the test, $ps > .17$. The yoked groups did differ during the resurgence test, $p = .03$, but not during the final extinction session, $p = .21$.

Interpreting between-group comparisons of responding during the test can be complicated when there are group differences at the end of Phase 2. We therefore also compared the groups' relative increases in rate during the test compared to rates during the final Phase 2 session. An independent t-test of difference scores (Test – Phase 2, Session 8) revealed that the increase in responding during the test was marginally greater in Group VI 10-s than in Group Reverse Thinning, $t(14) = 1.89$, $p = .08$, $\eta^2 = .20$, 95% CI [.00, .49]. In Contrast, Group VI 10-s Yoked did not produce a greater increase in rate than Group Reverse Thinning Yoked during the test, $t(13) = .68$, $p = .51$.

R2 responding during the test is summarized in the lower right panel of Figure 1. A 2 (Session: Ext 8 vs. Resurgence Test) × 2 (Reinforcer Distribution: VI 10-s vs. Reverse Thinning) × 2 (Contingency: Response Contingent vs. Non Contingent) ANOVA indicated reliable main effects of Session, $F(1, 27) = 64.54$, $MSE = 40.90$, $p < .001$, $\eta_p^2 = .71$, 95% CI [.47, .80], and Contingency, $F(1, 27) = 97.95$, $MSE = 146.45$, $p < .001$, $\eta_p^2 = .78$, 95% CI [.60, .86]. The Session × Contingency interaction was also significant, $F(1, 27) = 78.06$, $MSE = 40.90$, $p < .001$, $\eta_p^2 = .74$, 95% CI [.53, .83]. No other main effects or interactions were significant, $Fs < 1.48$.

Discussion

The results suggest that a reverse thinning schedule with reinforcement rates based on those earned with an abstinence contingency in Bouton and Schepers (2014) had similar effects on responding during Phase 2 and in the resurgence test. First, during Phase 2, responding on R1 decreased more rapidly in Group VI 10-s than in Group Reverse Thinning. Presumably, this occurred because the VI 10-s schedule initially provided a greater rate of alternative reinforcement; the animals therefore quickly learned to respond on R2 at a high rate, which may have produced greater response competition with R1 (see Leitenberg et al., 1970). Alternatively, any source of alternative reinforcement might theoretically accelerate the rate of R1 extinction (e.g., Herrnstein, 1970; Shahan & Sweeney, 2011). However, R1 did not extinguish more rapidly in a group that received an identical rate of non-contingent reinforcers (i.e., Group VI 10-s Yoked).

Second, during the resurgence test, the response-contingent VI 10-s and reverse thinning groups each showed a significant resurgence of R1 responding. However, resurgence was weaker following the reverse thinning schedule (see also Experiment 2; Bouton & Schepers, 2014). As noted earlier, in its original form, the Shahan-Sweeney model cannot account for

this result, because Groups VI 10-s and Group Reverse Thinning received alternative reinforcement programmed on the same VI 10-s schedule during the final session of Phase 2. However, as also noted earlier, Sweeney and Shahan (2013b) suggested that using the average programmed rate during Phase 2 might allow better fits to the data in situations where the rate of alternative reinforcement changes considerably over the sessions. If it used the average programmed rate, the model would correctly predict weakened resurgence in Group Reverse Thinning.

The results are also consistent with a contextual hypothesis. On that view, rats in Group Reverse Thinning learned to inhibit R1 responding during the early Phase 2 sessions in a context with few available reinforcers, which generalized more completely to the extinction conditions that prevailed during the final resurgence test.

Results with non-contingent reinforcers (Groups VI 10-s Yoked and Reverse Thinning Yoked) deserve additional comment. Contingent and non-contingent groups that received the same distribution of pellets during Phase 2 had similar patterns of responding during the resurgence test; there was no interaction based on the response-contingency factor. However, we did not observe statistically significant increases in responding from extinction to testing in the yoked groups, perhaps because noncontingent reinforcers yielded performance that was relatively variable and the experiment lacked the necessary statistical power. Winterbauer and Bouton (2010, Experiment 4) have already shown that resurgence can be created with removal of noncontingent reinforcers (that were yoked to a fixed-ratio 10 schedule). For practical reasons, the next two experiments were conducted using response-contingent Phase 2 reinforcement like that in the present Groups VI 10-s and Reverse Thinning.

Experiment 2

Experiment 2 compared the amount of resurgence after the reverse thinning procedure used in Experiment 1 with that of a forward thinning procedure (e.g., Winterbauer & Bouton, 2012). Recall that, in contrast to reverse thinning, a forward thinning procedure gradually decreases the rate of alternative reinforcement over sessions. The present thinning and reverse thinning schedules were also designed to provide identical average programmed reinforcement rates over Phase 2. The details of the design of Experiment 2 can be seen in Table 1. Previous results suggest that resurgence is weakened after both thinning and reverse thinning procedures relative to groups receiving a constant schedule of reinforcement (Bouton & Schepers, 2014; Winterbauer & Bouton, 2012), but the effects of forward and reverse thinning have never been compared.

Experiment 2 included groups that received either thinning (i.e., Group Thinning), reverse thinning (i.e., Group Reverse Thinning), or a standard VI 10-s treatment (i.e., Group VI 10-s) during Phase 2. The context hypothesis predicts less resurgence after either type of thinning procedure because they each provide experience with R1 being extinguished in a context with very lean rates of alternative reinforcement that is similar to the extinction conditions present during the resurgence test. However, it also predicts less resurgence after forward thinning than reverse thinning because low rates immediately prior to the test also

reduce the abruptness of the contextual change between Phase 2 and the resurgence test. In contrast, the Shahan-Sweeney model makes different predictions depending on how the rate of alternative reinforcement is calculated. If the model uses the programmed rate during the final Phase 2 session, as was originally proposed, it predicts that only the thinning procedure will weaken resurgence. If the model alternatively uses the average programmed rate over all sessions (Sweeney & Shahan, 2013b), reverse thinning and forward thinning procedures should produce equally attenuated resurgence.

Method

Subjects and apparatus—The subjects were 32 female Wistar rats (*n*s ranged from 8 to 12) of the same age and from the same stock as those described in Experiment 1. The apparatus was also the same as Experiment 1.

Procedure

Magazine Training and R1 conditioning: These sessions proceeded exactly as described in Experiment 1.

Response elimination (Phase 2): On each of the next eight days, the rats received a single session that began with insertion of both the right and left levers after the usual 2-min delay following placement in the chambers. Rats were randomly assigned to one of three groups with the restriction that individual boxes and the time of day when sessions occurred were equally represented among the groups. For all groups, R1 presses were recorded but never reinforced throughout the phase. In Group VI 10-s (n = 8), R2 presses were reinforced on a VI 10-s schedule. In Group Reverse Thinning (n = 12), reinforcement was contingent on R2 responses on schedules that became richer by a factor of 4 over the first four sessions (Session 1, VI 1200-s; Session 2, VI 300-s; Session 3, VI 75-s; Session 4 VI 19.5-s). During the last four sessions, these rats were reinforced on a VI 10-s schedule. Group Thinning (n =12) received the same schedules as Group Reverse Thinning, but in the opposite order. Thus, during the first four sessions R2 responses were reinforced on a VI 10-s schedule. During the final four sessions their schedule was thinned each session by a factor of 4 (Session 5, VI 19.5-s; Session 6, VI 75-s; Session 7, VI 300-s; Session 8, VI 1200-s).

Resurgence Test (Phase 3): As in Experiment 1, on the final day each rat received a 30-min test session in which R1 and R2 responses were both available but never reinforced.

Results

The results are summarized in Figure 2. During the first phase, groups similarly acquired R1 responding over sessions. During Phase 2, responding on R1 decreased when it was placed on extinction. Extinction occurred more rapidly in Groups that initially received rich rates for R2 responses (i.e, Group VI 10-s and Group Thinning). Responding on R2 increased over the entire phase in Groups VI 10-s and Reverse Thinning. In Group Thinning, R2 responding initially increased but then decreased when its reinforcement rate was thinned in sessions 5 through 8. During the test, only Groups VI 10-s and Reverse Thinning exhibited a significant increase in R1 responding (resurgence). However, as in Experiment 1, the resurgence was weaker in the Reverse Thinning Group.

Response acquisition and elimination—Acquisition of R1 responding is summarized in the upper left panel of Figure 2. Responding increased over the 12 sessions, $F(11, 319) = 77.54$, $MSE = 25.95$, $p < .001$, $\eta_p^2 = .73$, 95% CI [.67, .76]. The main effect of Group and the Group × Session interaction were not significant, largest $F(22, 319) = 1.24$, $MSE = 25.95$, $p = .21$.

R1 responding then decreased during Phase 2 (upper middle panel of Figure 2). A 3 (Group) × 8 (Session) ANOVA revealed main effects of Session, $F(7, 203) = 60.58$, $MSE = 4.11$, $p < .001$, $\eta_p^2 = .68$, 95% CI [.60, .72], and Group, $F(2, 29) = 12.75$, $MSE = 12.69$, $p < .001$, $\eta_p^2 = .47$, 95% CI [.17, .62], as well as a Group × Session interaction, $F(14, 203) = 33.06$, $MSE = 4.11$, $p < .001$, $\eta_p^2 = .70$, 95% CI [.61, .73]. Group VI 10-s responded less than Group Thinning ($p = .01$) and Group Reverse Thinning ($p < .001$) over Phase 2. In addition, Group Reverse Thinning made fewer R1 responses than Group Thinning, ($p < .05$). One-way ANOVAs on each session revealed that the groups differed during the first 3 and the last 3 sessions of Phase 2, smallest $F(2, 31) = 12.52$, $MSE = 3.28$, $p < .001$, $\eta_p^2 = .45$, 95% CI [.16, .61]. During Sessions 1–3, Groups VI 10-s and Thinning made fewer R1 responses than Group Reverse Thinning (*p*s < .001). During Sessions 6–8, Group Thinning made more responses than the other two groups (*p*s < .001). This increase in R1 responding toward the end of the forward thinning procedure is similar to findings reported by Winterbauer and Bouton (2012).

Turning to R2 (lower, middle panel of Figure 2), a 3 (Group) × 8 (Session) ANOVA indicated that R2 responding changed over the eight Phase 2 Sessions, $F(7, 203) = 52.64$, $MSE = 69.51$, $p < .001$, $\eta_p^2 = .65$, 95% CI [.56, .69]. The main effect of Group, $F(2, 29) = 4.01$, $MSE = 1146.37$, $p < .05$, $\eta_p^2 = .22$, 95% CI [.00, .42], and the Group × Session interaction, $F(14, 203) = 26.31$, $MSE = 69.51$, $p < .001$, $\eta_p^2 = .64$, 95% CI [.54, .68], were also reliable. Group VI 10-s made more R2 responses over Phase 2 than either of the other groups (*p*s < .05). Group Reverse Thinning and Group Thinning did not significantly differ in the amount of responses over the phase ($p = .83$). In order to decompose the significant Group × Session interaction, separate one-way ANOVAs were conducted. They revealed Group differences during Sessions 1, 2 3, 7, and 8, minimum $F(2, 29) = 3.72$, $MSE = 246.14$, $p < .05$, $\eta_p^2 = .20$, 95% CI [.00, .40]. During Sessions 1, 2, and 3, when its responses were reinforced at the relatively lean rates, Group Reverse Thinning made fewer responses than Group VI 10-s or Group Thinning (*p*s < .001). Conversely, during Sessions 7 and 8, Group Thinning, which was now receiving the lean rates for R2, made fewer responses than either group (*p*s < .001).

Resurgence Test—As illustrated in the upper right panel of Figure 2, rats in Group VI 10-s and Group Reverse Thinning increased their R1 responding during the test session relative to the last extinction session, demonstrating resurgence. In contrast, R1 responding in Group Thinning did not increase, but indeed decreased from the level observed at the end of Phase 2. A 3 (Group) × 2 (Session) ANOVA indicated a reliable main effect of Session, $F(1, 29) = 27.95$, $MSE = 0.725$, $p < .001$, $\eta_p^2 = .49$, 95% CI [.21, .65], a main effect of Group, $F(2, 29) = 5.03$, $MSE = 3.27$, $p < .01$, $\eta_p^2 = .15$, 95% CI [.00, .37], and a significant Group × Session interaction, $F(1, 29) = 32.02$, $MSE = 0.725$, $p < .001$, $\eta_p^2 = .52$, 95% CI [.

25, .68]. Follow up comparisons confirmed significant resurgence in Groups VI 10-s and Reverse Thinning, $p < .001$. In contrast, responding in Group Thinning *decreased* significantly between the extinction and test sessions, $p < .01$. Group VI 10-s made marginally more responses during the resurgence test than Group Reverse Thinning, $p = .06$, but not Group Thinning, $p = .10$. An independent t-test comparing difference scores (Test – Phase 2, Session 8) confirmed that the change in rate was significantly different in Group Thinning and Group Reverse Thinning, $t(22) = 5.56$, $p < .001$, $\eta^2 = .58$, 95% CI [.27, .73]. However, the difference between the resurgence effects in Group VI 10-s and Group Reverse Thinning did not reach significance, $t(18) = 1.71$, $p = .11$. Because Experiment 1 and Experiment 2 contained identical VI 10-s and Reverse Thinning groups an additional analysis was conducted pooling the groups from the two experiments. This analysis indicated that Group VI 10-s indeed made a greater number of R1 responses than Group Reverse Thinning during the test, $t(34) = 3.48$, $p = .001$, $\eta^2 = .27$, 95% CI [.05, .46]. In addition, the significant resurgence effect was a greater increase in responding over the previous session in Group VI 10-s than in Group Reverse Thinning, $t(34) = 2.65$, $p = .01$, $\eta^2 = .17$, 95% CI [.01, .38].

R2 responding in the resurgence test is summarized in the lower right panel of Figure 2. A 3 (Group) × 2 (Session) ANOVA revealed an effect of Session, $F(1, 29) = 176.66$, $MSE = 47.81$, $p < .001$, $\eta_p^2 = .86$, 95% CI [.74, .91], a main effect of Group $F(2, 27) = 33.31$, $MSE = 210.87$, $p < .001$, $\eta_p^2 = .71$, 95% CI [.47, .80], and a Group × Session interaction, $F(2, 29) = 25.24$, $MSE = 47.81$, $p < .001$, $\eta_p^2 = .64$, 95% CI [.37, .75]. Group Thinning made fewer R2 responses during the test session than either of the other groups, ($ps < .001$).

Reinforcers earned during Phase 2—Figure 3 summarizes the number of reinforcers the different groups earned in the sessions of Phase 2. A Group × Session ANOVA identified a main effect of Session, $F(7, 203) = 49.43$, $MSE = 130.79$, $p < .001$, $\eta_p^2 = .63$, 95% CI [.54, .68], a main effect of Group $F(2, 29) = 290.11$, $MSE = 366.69$, $p < .001$, $\eta_p^2 = .95$, 95% CI [.91, .97], and a reliable Group × Session interaction, $F(14, 203) = 469.38$, $MSE = 130.79$, $p < .001$, $\eta_p^2 = .97$, 95% CI [.96, .97]. Group VI 10-s earned more reinforcers than Group Reverse Thinning or Group Thinning over Phase 2, $p < .001$. In addition, Group Thinning earned marginally fewer reinforcers than Group Reverse Thinning, $p = .05$. At the end of Phase 2 the average number reinforcers earned per session were 159.4, 95.9, and 90.2 respectively for Groups VI 10-s, Reverse Thinning, and Thinning. Thus, there was only a 6% difference in the total reinforcers earned for the Thinning and the Reverse Thinning groups.

Discussion

As in Experiment 1, Groups VI-10 and Reverse Thinning both increased their R1 responding from the end of extinction to resurgence testing, demonstrating resurgence; however, the reverse thinning schedule again attenuated that effect. Additionally, the forward thinning procedure was effective at completely eliminating resurgence in the sense that there was a significant decrease, rather than increase, in R1 responding from extinction to test. Although either the original or modified versions of the Shahan-Sweeney model correctly predicts that the forward thinning procedure will weaken resurgence compared

with a constant VI 10-s schedule, neither version of the model can account for the entire pattern of results. Because Group VI 10-s and Group Reverse Thinning received identical programmed terminal rates (thus predicting equal resurgence using that analysis), the original model cannot predict weakened resurgence in Group Reverse Thinning. But if the model is modified to use the average rates (Shahan & Sweeney, 2013a), it incorrectly predicts that resurgence should be equal in the thinning and reverse thinning groups.

In contrast, the pattern of results is consistent with a contextual account of resurgence. According to this view, resurgence is weakened after either thinning procedure because they both allow the animal to learn that R1 is extinguished in a sparse pellet context that is similar to conditions that will be present during the resurgence test. The fact that resurgence is weakened in Group Reverse Thinning indicates that experiencing the lean pellet context even during early Phase 2 sessions is sufficient to generalize to the testing context. However, the fact that resurgence was completely eliminated in Group Thinning suggests that experiencing the lean pellet context closer to the test increases generalization and also reduces the abruptness of the context shift between Phase 2 and the resurgence test.

Two additional aspects of the results deserve discussion. First, Group Forward Thinning's R1 responding increased during the later response elimination (Phase 2) sessions, as the reinforcement rate was being thinned. Such "early resurgence" has been observed before during forward thinning procedures (Winterbauer & Bouton, 2012). Winterbauer and Bouton evaluated several possible accounts of early resurgence, but concluded that it could have resulted from the change of reinforcement context that occurred as the rate of reinforcement decreased. In the present experiment, however, early resurgence meant that the Forward Thinning group entered testing with a higher absolute level of R1 responding than the other groups. Although this complicated a comparison of the groups' absolute levels of R1 responding during the test, the fact that R1 responding did not increase between response elimination and testing can be interpreted as evidence of attenuated resurgence. The fact that responding significantly *decreased* from extinction to testing in that group is difficult to reconcile with the Shahan-Sweeney model, which stipulates that the decrease in reinforcement rate from response elimination to testing must either increase R1 or at best produce no change in it.

It is also worth noting that Group Reverse Thinning received approximately 6% more reinforcers on average per session during Phase 2 than Group Thinning did (95.9 vs. 90.2, respectively). However, it seems unlikely that this small difference was responsible for weaker resurgence in Group Thinning. In the next experiment (i.e., Experiment 3), no difference in resurgence was seen in groups that differed in the reinforcers they earned by 46%. And equally important, a robust difference in resurgence was observed in groups that had equivalent reinforcement rates over Phase 2. Thus, it is more likely Group Thinning's receipt of the lean rates closer to the test is the factor that weakened its resurgence.

Experiment 3

Experiment 3 provided a further test of how the distribution of reinforcement throughout Phase 2 influences resurgence. The design is summarized in Table 1. Like Experiments 1

and 2, Experiment 3 included a control group (Group VI 10-s) that received a VI 10-s reinforcement schedule for responses on R2, which has consistently produced robust resurgence effects even after many Phase 2 sessions (Bouton & Schepers, 2014; Winterbauer & Bouton, 2010). In a second group (Group Alternating), the VI 10-s schedule was alternated with sessions in which alternative reinforcement was not available. According to a contextual hypothesis, these alternating extinction sessions should weaken resurgence relative to Group VI 10-s because they allowed animals to learn that R1 is extinguished in a pellet context identical to the extinction conditions of the resurgence test. In contrast, according to the original Shahan-Sweeney model, these groups should produce equal resurgence, because they receive the same programmed reinforcement rates at the end of Phase 2. If the Phase 2 average rate is used, however, the model predicts less resurgence in Group Alternating. A third group (Group Average) was therefore included to provide a further test of the averaging version of the model. In this group, alternative reinforcement was available on a constant VI 17.5-s schedule, which was the average programmed rate of Group Alternating over all Phase 2 sessions. With equal averages, the average reinforcement version of the model predicts equal resurgence in Group Average and Group Alternating.

It should be noted that Sweeney and Shahan (2013a, Experiment 2) recently reported that animals receiving an alternating series of sessions in which R2 was reinforced and extinguished did not resurge. In one condition, animals received five alternating Phase 2 sessions in which R2 was reinforced on a VI 60-s schedule or was placed on extinction. Consistent with the context hypothesis, R1 responding did not resurge in a final test in which R2 was again extinguished. However, Sweeney and Shahan (2013a) also reported a lack of resurgence in a separate condition in which reinforcement was delivered on a VI 60-s throughout Phase 2, a finding that may be consistent with the possibility that resurgence is prevented by lean schedules of reinforcement (e.g., Leitenberg et al., 1975). Unfortunately, the lack of resurgence in the constant VI 60-s condition makes it uninformative as a control for assessing the effects of alternating reinforcement and extinction on final resurgence. Experiment 3 therefore examined the issue using a control group that received the constant VI 10-s schedule that supported resurgence in the previous experiments.

Method

Subjects and apparatus—The subjects were 32 female Wistar rats (*n*s ranged from 8 to 12) of the same age and from the same stock as those described in Experiments 1 and 2. The apparatus was also the same.

Procedure—*Magazine training and R1 conditioning* proceeded exactly as described in Experiments 1 and 2.

Response elimination (Phase 2): On each of the next seven days, the rats received a single session that began with insertion of both the right and left levers after the usual 2-min delay following placement in the chambers. Rats were randomly assigned to one of three groups with the restriction that individual boxes and the time of day when sessions occurred were equally represented among the groups. For all groups, R1 presses were recorded but never reinforced throughout the phase. In Group VI 10-s (n = 8), R2 presses were reinforced on a

VI 10-s schedule. In Group Alternating (n = 12), R2 responses were reinforced on a VI 10-s schedule and placed on extinction in alternating sessions: during Sessions 1, 3, 5, and 7 the VI 10-s schedule prevailed and during Sessions 2, 4, and 6 R2 responses were not reinforced. For Group Average (n =12), R2 responses were reinforced in every session on a VI 17.5-s schedule, which delivered reinforcers at the average programmed rate that would be received by Group Alternating over the seven Phase 2 sessions.

Resurgence Test (Phase 3): In the final session, R1 and R2 responses were both available but were never reinforced.

Results

The main results are summarized in Figure 4. All rats acquired lever responding on R1 during Phase 1. During Phase 2, responding on R1 decreased and R2 increased over sessions in Groups VI 10-s and Average. In Group Alternating, R2 responding was high when it was reinforced but low during sessions that it was extinguished. However, when alternative reinforcement was removed for the first time in Groups VI 10-s and Average during the final resurgence test, each group exhibited a similarly robust resurgence of R1 responding. Group Alternating showed no such resurgence.

Response acquisition and elimination—Acquisition of R1 responding is summarized in upper left panel of Figure 4. R1 responding increased over the twelve Phase 1 sessions, $F(11, 319) = 61.53$, $MSE = 30.53$, $p < .001$, $\eta_p^2 = .68$, 95% CI [.61, .71]. Random assignment to groups was successful in that the main effect of Group and a Group × Session interaction were not significant, Fs < 1.

R1 responding decreased during Phase 2 when it was placed on extinction and alternative reinforcement was introduced for R2 responses (upper center panel of Figure 4). This was confirmed by 3 (Group) × 7 (Session) ANOVA that identified a main effect of Session, $F(6, 174) = 28.19$, $MSE = 3.70$, $p < .001$, $\eta_p^2 = .49$, 95% CI [.37, .56] a main effect of Group, $F(2, 29) = 9.89$, $MSE = 10.60$, $p = .001$, $\eta_p^2 = .41$, 95% CI [.11, .58] and a Group × Session interaction, $F(12, 174) = 5.21$, $MSE = 3.70$, $p < .001$, $\eta_p^2 = .26$, 95% CI [.11, .32]. Follow up analyses indicated that Group Alternating responded more over Phase 2 Sessions than Group Average ($p < .01$) and Group VI 10-s ($p < .001$). Group Average made marginally more R1 responses than Group VI 10-s ($p = .09$). Separate one-way ANOVAs on each session were conducted to decompose the significant interaction. These revealed group differences during sessions 1, 2, 4, and 6, smallest $F(2, 29) = 3.42$, $MSE = 7.66$, $p < .05$, $\eta_p^2 = .41$, 95% CI [.00, .39]. Follow up analyses revealed that in Session 1, Group Average made more responses than Group VI 10-s ($p < .05$). In Sessions 2, 4, and 6 (tests 1, 2, and 3) Group Alternating made more responses than either of the other groups (ps < .05), which suggests reliable resurgence of R1 during these sessions in the Alternating Group.

A 3 (Group) × 7 (Session) ANOVA on R2 responding (lower, middle panel) revealed a Session effect $F(6, 174) = 60.27$, $MSE = 43.09$, $p < .001$, $\eta_p^2 = .68$, 95% CI [.59, .72] a main effect of Group, $F(2, 29) = 6.71$, $MSE = 1158.53$, $p < .01$, $\eta_p^2 = .32$, 95% CI [.04, .50] and a Group × Session interaction, $F(12, 174) = 18.61$, $MSE = 43.09$, $p < .001$, $\eta_p^2 = .56$, 95% CI [.43, .61]. Group VI 10-s and Group Average responded similarly on R2 (ps >.05), but each

group made more R2 responses than Group Alternating (*p*s < .05). In order to decompose the significant interaction, separate one-way ANOVAs were conducted for each session. These revealed group differences only during Sessions 2, 4, and 6, when R2 responses were not reinforced with pellets for Group Alternating, minimum $F(2, 29) = 11.35$, $MSE = 125.19$, $p < .001$, $\eta_p^2 = .44$, 95% CI [.14, .60]. Group Alternating made fewer responses than the other groups in each of these sessions (*p*s ≤ .001).

Resurgence Test—As illustrated in the upper right panel of Figure 4, Group VI 10-s and Group Average each exhibited a robust resurgence of R1 responding when alternative reinforcement was removed in the final test session. In contrast, Group Alternating did not. A 3 (Group) × 2 (Session) ANOVA indicated a reliable main effect of Session, $F(1, 29) = 41.83$, $MSE = 4.68$, $p < .001$, $\eta_p^2 = .59$, 95% CI [.33, .72] and a significant Group × Session interaction, $F(2, 29) = 4.49$, $MSE = 20.99$, $p < .05$, $\eta_p^2 = .24$, 95% CI [.00, .43]. The main effect of Group was not significant, $F(2, 29) = 1.07$, $MSE = 4.76$, $p = .36$. Follow up comparisons confirmed the significant resurgence effects in Group VI 10-s and Group Average, *p*s < .001. A significant resurgence effect was not produced in Group Alternating, $p = .14$. Group VI 10-s and Group Average did not differ during the final resurgence test, $p = .67$, and each responded more than Group Alternating, *p*s < .05. The alternating procedure thus clearly weakened the resurgence effect. The analysis of Session 7-Test difference scores also confirmed that Group Alternating produced a smaller increase in rate than Groups VI 10-s and Group Average, smallest $t(21) = 3.51$, $p < .01$, $\eta_p^2 = .37$, 95% CI [.06, .59] that produced similar increases over the previous session, $t(18) = .98$, $p = .34$.

R2 responding during the final resurgence test is summarized in the lower right panel of Figure 4. A 3 (Group) × 2 (Session) ANOVA indicated a main effect of Session, $F(1, 29) = 105.31$, $MSE = 139.01$, $p < .001$, $\eta^2 = .78$, 95% CI [.61, .85] and a main effect of Group, $F(2, 29) = 4.46$, $MSE = 269.19$, $p < .05$, $\eta^2 = .24$, 95% CI [.00, .43]. The Group × Session interaction was not reliable, $F(2, 29) = 0.53$, $MSE = 139.08$, $p = .60$.

Reinforcers earned during Phase 2—The number of reinforcers earned during Phase 2 is summarized in Figure 5. A 3 (Group) × 7 (Session) ANOVA was conducted to determine whether groups differed in the number of reinforcers earned. A main effect of Session, $F(6, 174) = 180.30$, $MSE = 126.21$, $p < .001$, $\eta^2 = .86$, 95% CI [.82, .88] indicated that reinforcers were earned differentially over Phase 2. A reliable main effect of Group, $F(2, 29) = 174.07$, $MSE = 515.82$, $p < .001$, $\eta^2 = .92$, 95% CI [.85, .95] and a significant Group × Session interaction, $F(12, 174) = 213.86$, $MSE = 126.21$, $p < .001$, $\eta^2 = .94$, 95% CI [.92, .94] indicated that group differences depended on the session. Group VI 10-s earned a greater number of reinforcers during Phase 2 than Group Average ($p > .001$) or Group Alternating ($p > .001$). There was a marginally significant difference in reinforcers earned between Group Alternating and Group Average ($p = .06$).

Discussion

The results suggest that resurgence is weakened when alternative reinforcement is only available in every other Phase 2 session. Rats in the alternating group were given three sessions that were identical to the extinction conditions that are present during the final test.

In contrast, a robust resurgence effect was produced in two groups that received constant rates that were either the programmed average rate or the rate during the final Phase 2 session of the alternating schedule.

The finding that resurgence is weaker following the alternating procedure is consistent with previous findings that resurgence is weaker following repeated resurgence tests (Quick, Pyszczynski, Colston & Shahan, 2011; Shahan & Sweeney, 2011; Sweeney & Shahan, 2013a). However, the overall results in this experiment are inconsistent with the Shahan-Sweeney model. That model has difficulties accounting for less resurgence in Group Alternating than in groups that received either the same terminal reinforcement rate (Group VI 10-s) or the same average rate over the entire phase (Group Average). When the model uses the rate from the final Phase 2 session, it incorrectly predicts equal resurgence in Group Alternating (i.e., VI 17.5-s) and Group VI 10-s. When the model alternatively uses the Phase 2 average, it successfully predicts less resurgence in Group Alternating than Group VI 10-s. However, because Group Alternating and Group Average received identical programmed average rates over Phase 2 (i.e., VI 17.5-s) the model cannot account for weakened resurgence in Group Alternating.

A potential problem for both the contextual hypothesis and the Shahan-Sweeney model is that resurgence was similar in Group VI 10-s and Group Average. These schedules provided a different average number of reinforcers during each Phase 2 session (180 vs. 102.8), but each produced similar levels of resurgence during the test. It is possible that both of these reinforcement rates were high enough to yield reliable resurgence effects, without being discriminably different. However, the current results nonetheless suggest that resurgence is weaker in a group that receives pauses in reinforcer availability compared to one that received identical and rich average programmed rates of reinforcement (VI 17.5-s) without pauses.

General Discussion

The results of these experiments suggest that the distribution of reinforcement rates during response elimination sessions can have a powerful impact on the resurgence effect. Experiments 1 and 2 found that resurgence can be weakened with two types of response thinning procedures during Phase 2. In both experiments, resurgence was attenuated following reverse thinning. That is, resurgence at test was reduced when alternative reinforcement was delivered at lean rates during early sessions that were gradually increased over later sessions. In Experiment 2, resurgence was even further reduced (i.e., completely abolished) when the same schedule was simply mirrored in a forward thinning procedure. In this schedule, rich rates were available during early sessions and were gradually thinned to the lean rates. In Experiment 3, resurgence was also eliminated by a schedule that alternated between a rich rate and a zero rate over adjacent sessions. In contrast, groups that received constant rates at either the same average or the same terminal rate as the alternating group each exhibited robust resurgence effects. Together, the results suggest that experiencing lean rates at any point during Phase 2 may reduce resurgence. However, lean rates immediately prior to the test or intermittent removal of alternative reinforcement may have the strongest final effect.

The current results provide some difficulty for the model proposed by Shahan and Sweeney (2011). According to this view, alternative reinforcement delivered during Phase 2 has two effects on the original behavior. First, whether it is delivered contingent on responding or not, it is expected to act as a disruptive force, suppressing the original behavior. Second, it concurrently provides a source of strength that is expressed as resurgence when alternative reinforcement is removed. It is important to note that the model generally expects that greater rates of alternative reinforcement further suppress the original behavior and increase the resurgence effect. Previously, the model has only considered programmed reinforcement rates during the final Phase 2 session. However, Sweeney and Shahan (2013b) suggested that considering the Phase 2 average rate might provide better model fits to resurgence data when the rate changes considerably over sessions. Therefore, when analyzing the current results the model was considered using both the rate during the final session and the Phase 2 average.

When the model uses the programmed rate during the final Phase 2 session, it has difficulties explaining results from all three experiments. In Experiments 1 and 2, it cannot account for weakened resurgence after reverse thinning; the model predicts that groups with equal programmed rates at the end of Phase 2 should demonstrate equal resurgence. In Experiment 3, the model could not predict weaker resurgence in a group that received alternating extinction and VI 10-s sessions than a group that had continuous VI 10; these groups had the same terminal rates. Clearly, resurgence is influenced by more than just the final rate of alternative reinforcement programmed or delivered at the end of Phase 2.

The present results also suggest that resurgence is not merely dependent on the average rate of reinforcement earned during Phase 2 (Sweeney & Shahan, 2013a). Using the average rate, the model does not predict a difference between the thinning groups in Experiment 2. That is, it cannot account for weaker resurgence following a thinning procedure compared to one that delivers the same rates but in the opposite order. In Experiment 3, it also does not predict that resurgence should be weakened following a schedule that receives alternating extinction sessions compared with a group that received the same average rate over the phase. Together these inconsistencies indicate that the Shahan-Sweeney model has difficulty explaining resurgence effects when the rate of alternative reinforcement changes over Phase 2 whether the model is considered using either its terminal or average rate. It remains to be seen whether some other method of calculating reinforcement rate over sessions can be devised to accommodate the present results. Although the finding that forward thinning produced weaker resurgence than reverse thinning (Experiment 2) suggests that recent sessions may be weighted more heavily than early sessions, the fact that alternating sessions of extinction and reinforcement produced weaker resurgence than a procedure with the same average reinforcement rate (Experiment 3) will require a different type of calculation.

The contextual hypothesis suggests that resurgence occurs according to the mechanisms that underlie ABC renewal. It accepts the fact that removal from the context of extinction can be sufficient for an extinguished behavior to return (e.g., Bouton, 2002, 2004) and that factors that increase generalization between the extinction and test phases can reduce renewal (Brooks & Bouton, 1994; Willcocks & McNally, 2014). This sort of analysis is consistent with the current results. It successfully predicts the results of Experiments 1 and 2. That is,

resurgence should be weaker following either type of thinning procedure compared to a group receiving its richest rate over the entire phase. In addition, forward thinning should further weaken resurgence compared to reverse thinning. In Experiment 3, it correctly predicted that alternating extinction sessions should reduce resurgence compared to schedules that received constant rates. It should be noted that both a contextual hypothesis and the Shahan-Sweeney model have difficulty accounting for equal resurgence in Group VI 10-s and Average Groups in Experiment 3. These groups differed substantially in the number of pellets earned during each Phase 2 session. However, it is possible these constant rates were sufficiently rich that the animals would have had difficulty discriminating between them. If discrimination between the rates was difficult it is not surprising that they would produce similar levels of resurgence.

The present experiments were mainly designed to separate the Shahan-Sweeney and contextual accounts of resurgence. However, we also collected data that may help evaluate an even simpler view of resurgence that assumes that R1 is suppressed in Phase 2 by direct competition from R2, and that resurgence might result at least in part from release from that competition when R2 undergoes extinction (cf. Leitenberg et al., 1970). During the tests in each experiment, we correlated each animal's R1 and R2 responding in each group. If R1 and R2 compete, we might expect to find R1 to be negatively correlated with R2. The correlations were generally positive (ranging from +.06 to +.99), though rarely significant; in no case was the correlation negative. If anything, individual animals that responded on R1 also responded on R2. There was thus little evidence of a role of response competition in the present resurgence tests.

In summary, the current results suggest that resurgence may be better explained by the contextual hypothesis than by Shahan and Sweeney's (2011) extension of behavioral momentum theory when it uses either of two methods of calculating alternative reinforcement. The results suggest that given similar average rates of Phase 2 reinforcement, two factors may weaken resurgence that occurs when alternative reinforcement is removed. First, lean rates experienced more closely to the test have a bigger impact on reducing the resurgence than those experienced earlier in the phase (Experiment 2). Second, removing alternative reinforcement altogether in alternating Phase 2 sessions might be especially effective at eliminating resurgence. One implication is that CM therapies in humans that intermittently remove alternative reinforcement during treatment may reduce potential relapse by increasing generalization with the conditions after treatment ends. Although the results also suggest that lean rates at the end of response elimination may be very effective, it should be noted that in Experiment 2, as in Winterbauer and Bouton (2012), forward thinning produced an early resurgence of responding toward the end of response elimination that may complicate its specific application to treatment.

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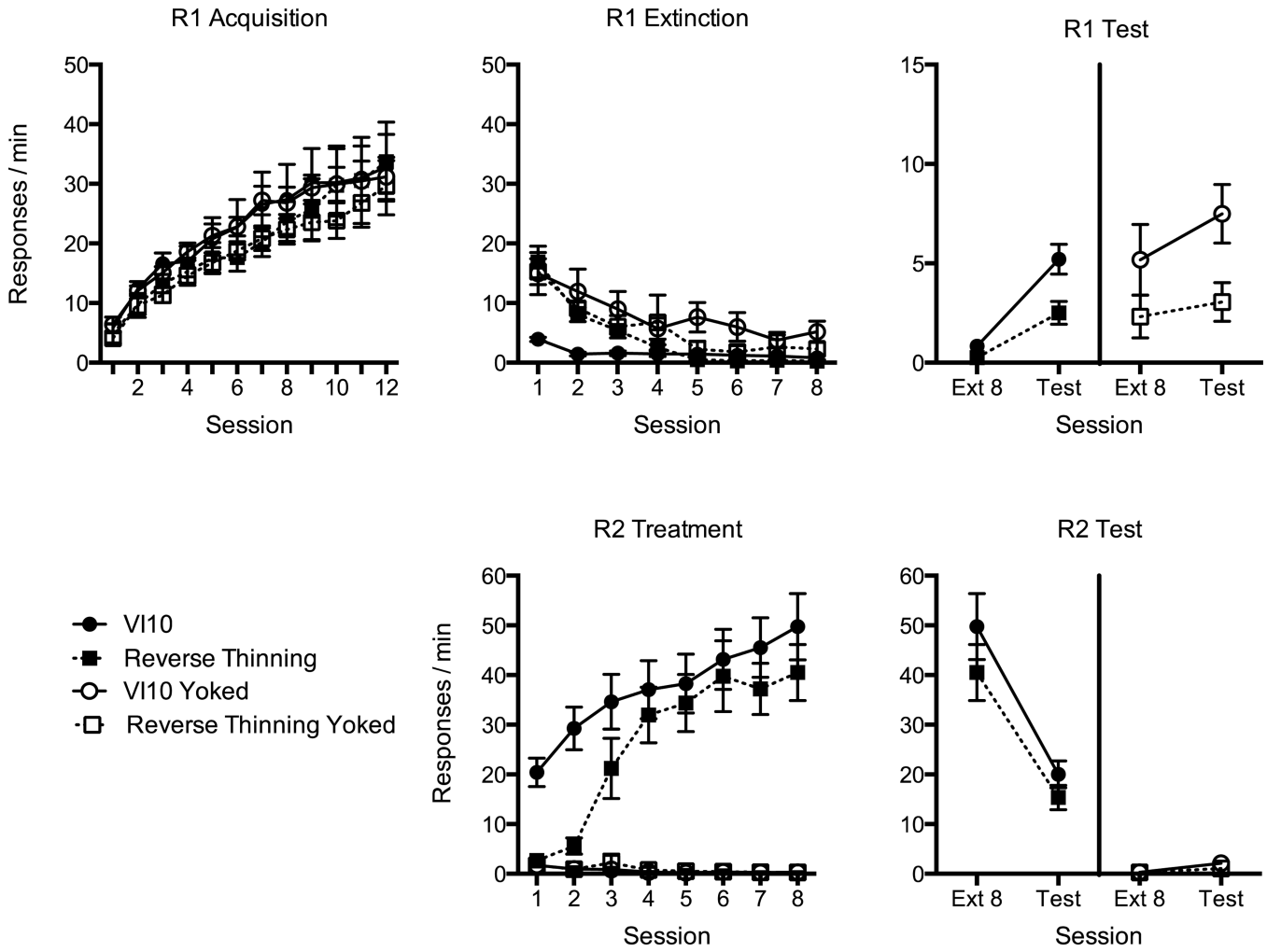

Figure 1.

Results of Experiment 1. The upper panels summarize mean R1 responding (± SEM) during its acquisition (left), extinction (middle), and during resurgence testing compared with the final Phase 2 session (right; note changed y-axis). Corresponding R2 rates are summarized in the lower panels.

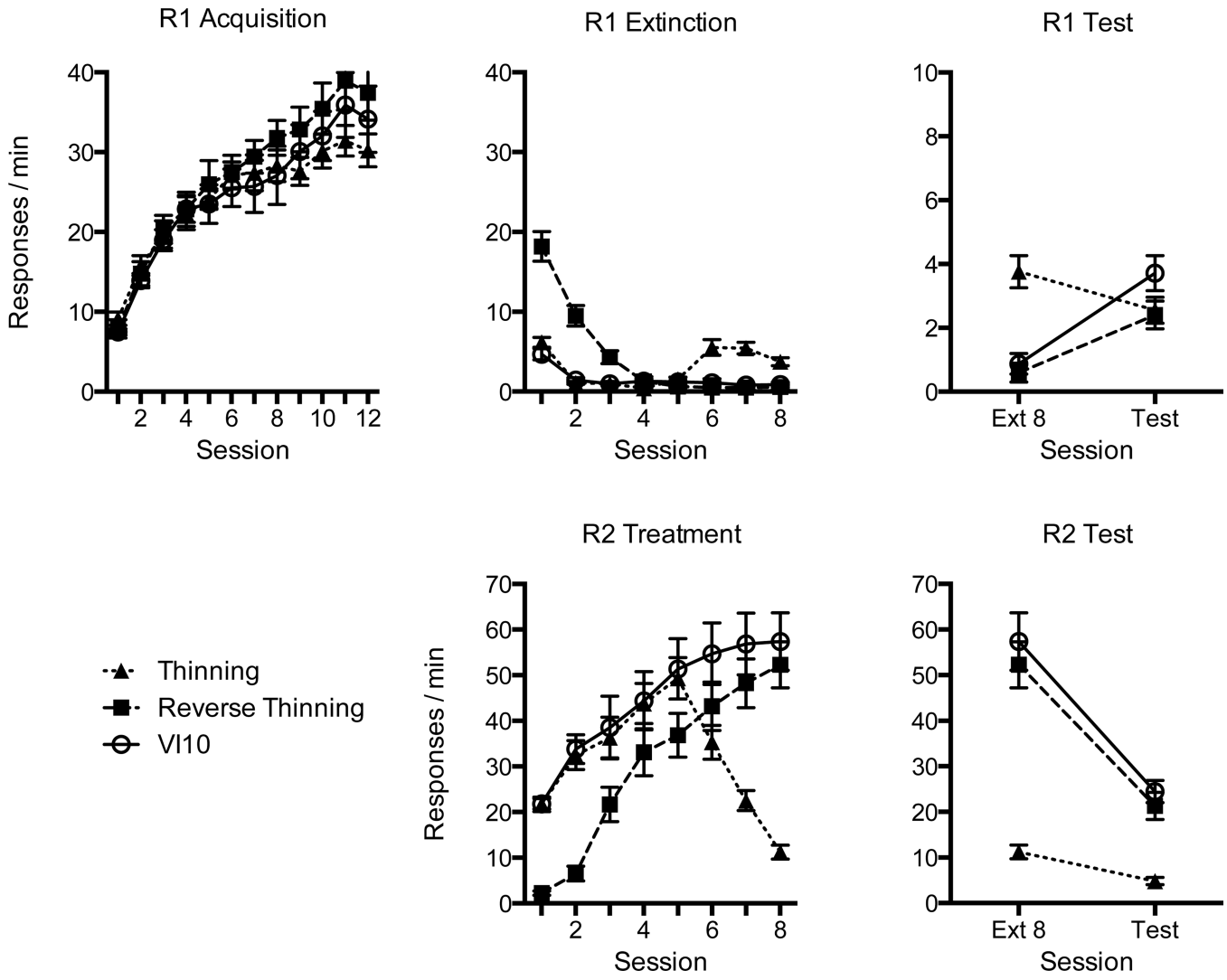

Figure 2.

Results of Experiment 2. The upper panels summarize mean R1 responding (± SEM) during its acquisition (left), extinction (middle), and during resurgence testing compared with the final Phase 2 session (right; note changed y-axis). Corresponding R2 rates are summarized in the lower panels.

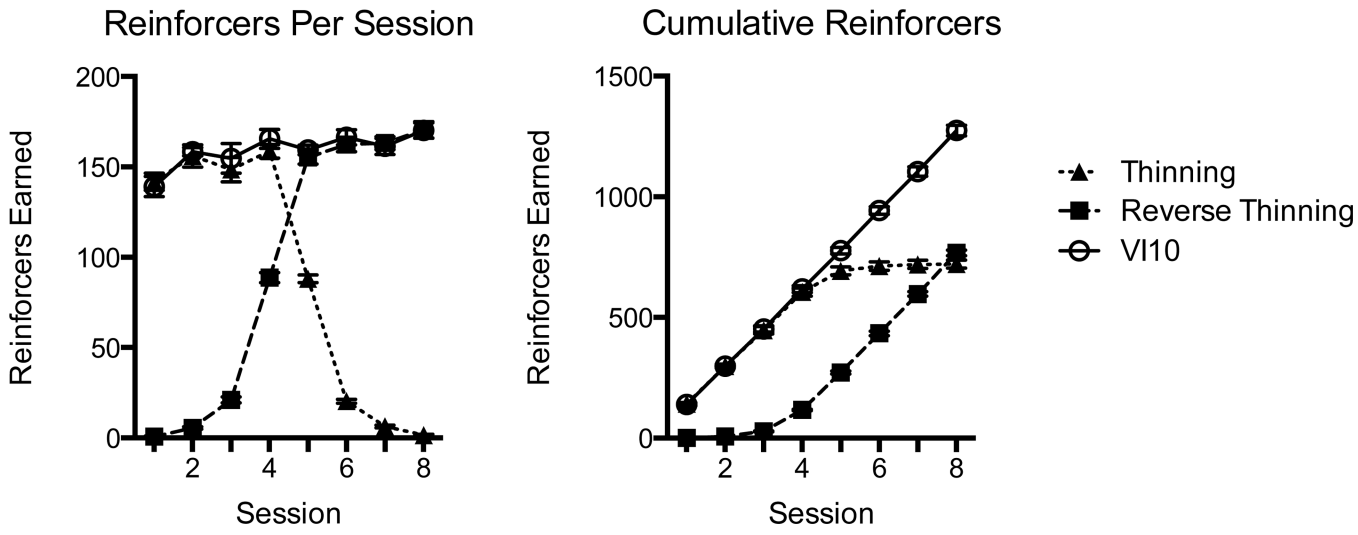

Figure 3.

Mean number of reinforcers earned (± SEM) during each Phase 2 session (left) and the cumulative mean number of reinforcers that had been earned during Phase 2 at each session in Experiment 2.

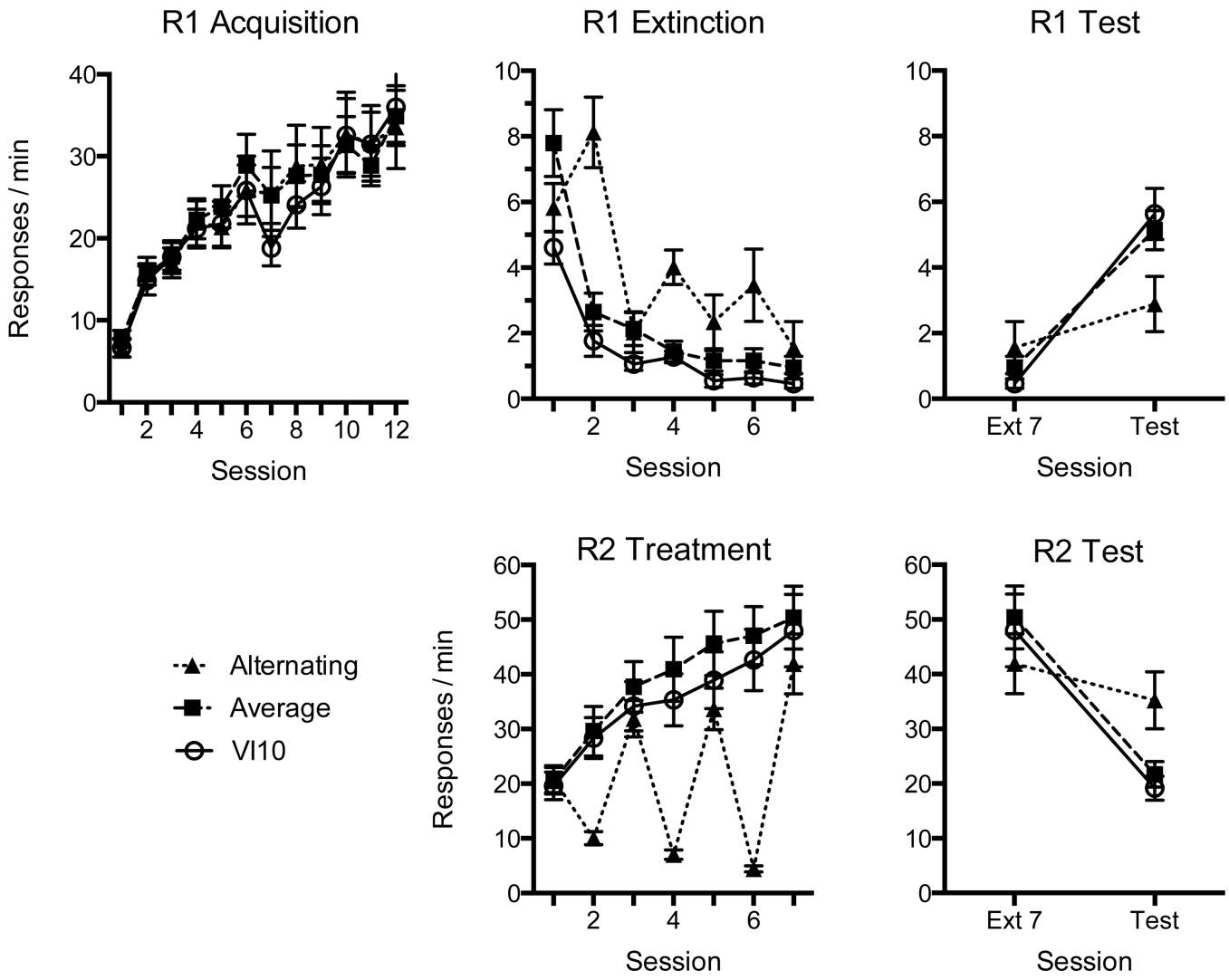

Figure 4.

Results of Experiment 3. The upper panels summarize mean R1 responding (± SEM) during its acquisition (left), extinction and during resurgence testing compared with the final Phase 2 session (middle and right panels respectively; note changed y-axes). Corresponding R2 rates are summarized in the lower panels.

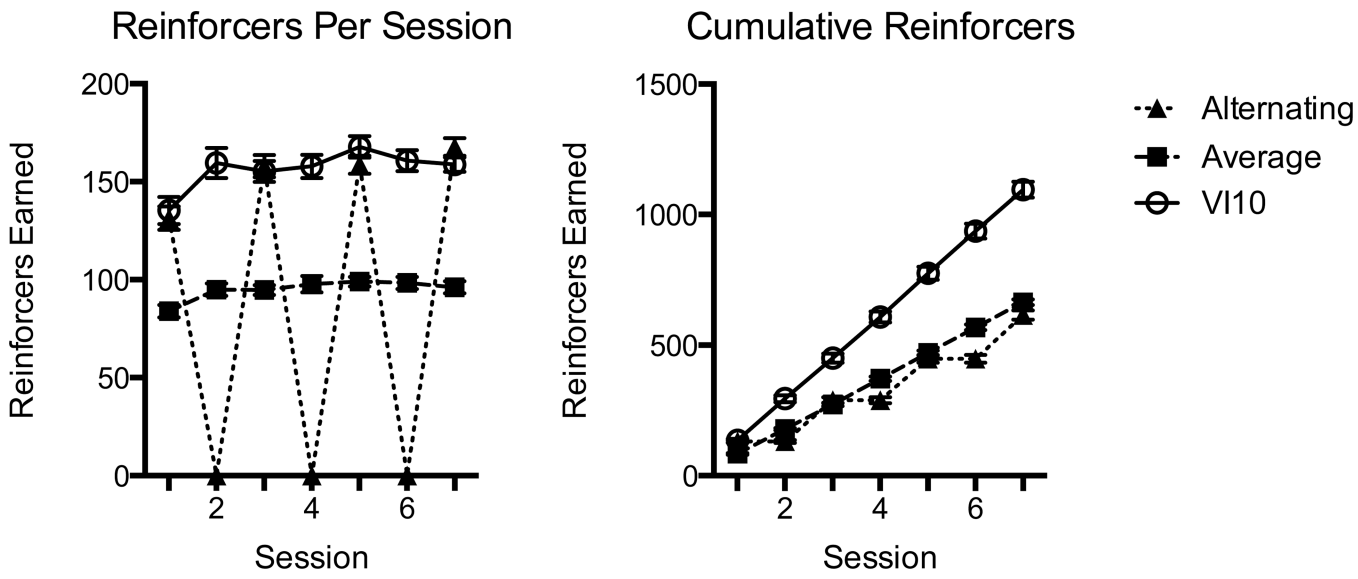

Figure 5.

Mean number of reinforcers earned (± SEM) during each Phase 2 session (left) and the cumulative mean number of reinforcers that had been earned during Phase 2 at each session in Experiment 3.

Table 1

Experimental Design

Experiment	Group	Phase 1	Phase 2	Phase 3
1	VI 10-s	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: VI 10	R2: Ext
	Reverse Thinning	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: VI 1200…VI 10	R2: Ext
	VI 10-s Yoked	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: Yoked VI 10	R2: Ext
	Reverse Thinning Yoked	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: Yoked VI 1200…VI 10	R2: Ext
2	VI 10-s	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: VI 10	R2: Ext
	Thinning	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: VI 10…VI 1200	R2: Ext
	Reverse Thinning	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: VI 1200…VI 10	R2: Ext
3	VI 10-s	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: VI 10	R2: Ext
	Group Alternating	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: VI 10 (Sessions 1,3,5,7); Ext (Sessions 2,4,6)	R2: Ext
	Group Average	R1: VI 30	R1: Ext	R1: Ext
		R2: ---	R2: VI 17.5	R2: Ext

Note: Reinforcer always consisted of pellet delivery. R1 represents the lever available during Phase 1. R2 represents the source of reinforcement during Phase 2. Two levers were always available during Phases 2 and 3. “Yoked” groups had no response requirements and were delivered a “free” pellet immediately after an animal in the corresponding contingent group had earned one.