2014

The Effects of Reinforcer Distribution During Response Elimination on Resurgence of an Instrumental Response

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EFFECTS OF REINFORCER DISTRIBUTION DURING RESPONSE ELIMINATION ON THE RESURGENCE OF AN EXTINGUISHED INSTRUMENTAL RESPONSE

A Thesis Presented

by

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to

The Faculty of the Graduate College

of

The University of Vermont

In Partial Fulfillment of the Requirements
For the Degree of Master of Arts
Specializing in Psychology

October, 2014

Accepted by the Faculty of the Graduate College, The University of Vermont, in partial fulfillment of the requirements for the degree of Master of Arts, specializing in Psychology.

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June 26, 2014

ABSTRACT

Resurgence is the relapse of an extinguished instrumental behavior that can occur when an alternative behavior that was introduced to replace it is itself extinguished. In a typical resurgence experiment, rats are trained to make a response (R1) for food reinforcers. In a second phase, responses on R1 are no longer reinforced, but a new response (R2) is introduced and responses on it are reinforced. During a third phase, reinforcement for R2 is removed and behavior on R1 often returns (or "resurges") despite remaining on extinction.

The current experiments were designed to examine the effects of the temporal distribution of reinforcers delivered during Phase 2 on later resurgence. The role of these alternative reinforcers is central to theories that have been proposed to account for resurgence. The experiments provided a special opportunity to contrast predictions made by the Shahan-Sweeney Model (Shahan & Sweeney, 2011) and a contextual account of resurgence (Winterbauer & Bouton, 2010).

Experiments 1 and 2 examined resurgence when alternative reinforcement during Phase 2 was delivered according to the same set of daily reinforcement schedules presented in different orders. That is, one group received rich reinforcement rates that were gradually thinned to leaner ones (Group Thinning) and another group received lean rates that were gradually increased to richer ones (Group Reverse Thinning). Both procedures weakened resurgence compared to that in a group that received the richest rate (a variable interval, or VI 10-s schedule that arranged for a reinforcer to be available for a response every 10s on average) during all of the Phase 2 sessions. However, the forward thinning procedure was more effective than the reverse thinning procedure at eliminating the resurgence effect.

Experiment 3 examined resurgence when alternative reinforcement was only available for R2 during every other session. The results indicated that daily alternations of a VI 10-s schedule with an extinction schedule for R2 weakened resurgence compared to groups that either received the same average rate over the entire phase (VI 17.5-s) or that received the same terminal rate (VI 10-s) in every session.

The Shahan-Sweeney model cannot account for several of the current results. Instead, the results are most consistent with a contextual account of resurgence. That is, resurgence can be conceptualized as an ABC renewal effect in which extinguished R1 behavior returns when an animal is removed from an extinction "context" provided by R2 reinforcement. Lean reinforcement rates at any time during Phase 2 allow the animal to learn to inhibit R1 under conditions that generalize to the extinction conditions that prevail during the resurgence test. The results also suggest that experience with alternating extinction sessions or lean reinforcement rates close to the final resurgence test are especially effective at eliminating the resurgence effect.

ACKNOWLEDGEMENTS

I would like to thank my advisor, Mark Bouton, for his patience, guidance, and unwavering support throughout this entire process. Additionally, I would like to thank a long list of members of the Bouton lab and Biobehavioral cluster, past and present, for their advice and friendship.

I would like to thank my family for their support in the transition to and pursuance of my training at the University of Vermont.

I would also like to thank the members of my committee for their time and support.

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Resurgence is said to occur when an extinguished behavior returns after an alternative behavior that replaced it is also placed on extinction. In a resurgence experiment, during Phase 1, a target response (R1, e.g., pressing a first lever) is reinforced. Then 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). The resurgence of R1 is consistent with other relapse phenomena (e.g., renewal, spontaneous recovery, and reinstatement) in suggesting that the extinction of a behavior does not occur because it has been unlearned or erased (for a review, see Bouton, 2004). Rather, extinction of an instrumental response may involve learning to not 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. 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 (Silverman, Kaminski, Higgins, & Brady, 2011).

At least three theories have attempted to describe mechanisms that contribute to resurgence. Further understanding of these mechanisms may inform CM designs and lead to a reduction in relapse that often occurs following clinical treatments. According to Leitenberg, Rawson, and Bath (1970), resurgence occurs because alternative reinforcement results in rapid acquisition of the new behavior (i.e., R2), which prevents the animal from making the original response (i.e., R1). They suggest that response competition precludes the original behavior from being truly extinguished during Phase 2. In Phase 3, when the source of competition (alternative reinforcement) is removed, resurgence occurs because R1 responding is not completely extinguished. This view suggests that the extinction of R1 will be enhanced and resurgence weakened when less response competition allows animals to make a sufficient amount of non-reinforced (extinction) responses during Phase 2. However, 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 compared with control groups that did not resurge. These results cannot be explained by a response competition explanation of resurgence.

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, 2011; Todd 2013; Todd, Winterbauer, & Bouton, 2012). ABC renewal experiments typically utilize three distinct physical contexts that often contain different visual, tactile, and olfactory stimuli. In Context A, an animal acquires a behavior that is then extinguished in a second context (Context B). When testing then occurs in a novel third context (Context C), the original behavior returns or renews. Renewal in Context C indicates that removal from the extinction context (Context B) is sufficient for extinguished behavior to return.

It is important to note that in a resurgence experiment all three phases are typically conducted within the same physical context. However, previous research suggests that pellet deliveries in the background can also provide a distinct context for conditioning and extinction (Bouton, Rosengard, Achenbach, Peck & Brooks, 1993; Bouton 1991, 2002). Thus, when pellets are discontinued during resurgence testing, there is potentially a change in context. This type of analysis suggests that 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. Several results are consistent with the prediction that resurgence is weakened or eliminated if Phase 2 involves extremely lean rates of alternative reinforcement (Leitenberg, Rawson, & Mulick, 1975; Sweeney &

Shahan, 2013b). Research has also produced weakened resurgence in procedures that decrease or "thin" reinforcement over the series of Phase 2 sessions (Winterbauer & Bouton, 2012, Sweeney & Shahan, 2013b). More specifically, early in Phase 2, Winterbauer and Bouton (2012) provided reinforcement for R2 at a high rate, which was gradually reduced to a very lean rate over subsequent Phase 2 sessions. When reinforcement was discontinued during the resurgence test, groups treated with the thinning procedure exhibited less resurgence than controls that were not thinned. The contextual hypothesis suggests that thinning allows the animal to learn that R1 is extinguished in the "context" of infrequent R2 reinforcement. Perhaps equally important, it attenuates resurgence by reducing the abruptness of the contextual change that occurs between Phase 2 and Phase 3. That is, the lean reinforcement rate at the conclusion of a thinning procedure is more similar to the extinction conditions that prevail during the resurgence test.

In related resurgence experiments, Bouton and Schepers (2014) observed weakened resurgence when an abstinence contingency was associated with R1 responding during Phase 2. In the abstinence contingency group, each R1 response resulted in a 45 s time-out in which alternative reinforcement for R2 was made unavailable. As a result of the abstinence contingency, the pellet distribution during Phase 2 resembled that of a thinning schedule in reverse. That is, early in Phase 2, when rats made many R1 responses, which resulted in many time-outs, 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 were 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 actual contingency between R1 and reinforcement. At test, the abstinence contingency and yoked groups exhibited similarly weakened resurgence compared with the VI 10-s control group. This 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 (2010), who developed a quantitative model based on behavioral momentum theory (Nevin & Grace, 2000). The Shahan-Sweeney model suggests that any source of alternative reinforcement (e.g., contingent on responding or not) introduced during Phase 2 has two effects on performance of the original behavior. First, alternative reinforcement is thought to provide a source of disruption to performance of the original response. Second, it somewhat paradoxically also contributes to a process that strengthens it. On this view, all behavior is strengthened by reinforcement delivered in the context, but reinforcement's strengthening effects are not observed until it is discontinued. Therefore, the model suggests that when alternative reinforcement is removed at test, its disruptive force disappears, leaving only the strengthening effects, which are carried forward from Phase 2 to help produce resurgence. To date, quantitative simulations of the model have used alternative reinforcement at its terminal programmed rate (i.e., the programmed rate in a

session immediately before the resurgence test) to predict the strength of resurgence. This has provided predictions consistent with the effects produced by lean rates and reinforcement thinning procedures during Phase 2 as described above (Sweeney & Shahan, 2013a; Winterbauer & Bouton, 2012). However, it cannot explain why reverse thinning treatments (e.g., Bouton & Schepers, 2014) reduce resurgence if the groups' programmed rates are equal in the last Phase 2 session. To address this sort of complication, Sweeney and Shahan (2013a) 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. As described below, one goal of the present experiments was to test this possible modification of the model.

Another prediction of the Shahan-Sweeney model is that merely increasing the number of Phase 2 sessions will weaken resurgence. This is because the model includes a separate variable that results in a prediction that resurgence will decrease as a function of increases in Phase 2 training. This prediction is consistent with the results of Leitenberg, Rawson, and Mulick (1975), who found resurgence was weaker following 27 Phase 2 sessions than after 3 or 9 sessions. However, resurgence did not differ between the groups that received three or nine Phase 2 sessions. And more important, other results have found no effect of increasing the number of Phase 2 sessions on resurgence: Winterbauer, Lucke, and Bouton (2013) observed similar resurgence in groups that received 4, 12, or 36 Phase 2 sessions. Clearly, further research is necessary to determine the effects of additional sessions where R1 is being extinguished. But extending Phase 2 training does not necessarily reduce the resurgence effect.

The current experiments were designed to further test the Shahan-Sweeney model and the context hypothesis by manipulating the distribution of alternative reinforcement during Phase 2. According to Shahan and Sweeney (2010), when the number of sessions is held constant, resurgence is a function of the programmed reinforcement rate during the final Phase 2 session, or as suggested in Sweeney and Shahan (2013a), its average rate throughout the phase. In contrast, the context hypothesis suggests that Phase 2 alternative reinforcement serves as a discriminable extinction context, and that lean reinforcement rates decrease resurgence by increasing generalization between Phase 2 and the extinction conditions that prevail during the resurgence test. These lead to differential predictions in the current series of experiments.

In Experiment 1, I sought to replicate weakened resurgence after a reverse thinning schedule (Bouton & Schepers, 2014). In addition, Experiment 1 compared the effects of response-contingent and non-contingent alternative reinforcement. Experiment 2 compared resurgence following thinning and reverse thinning procedures in which the rate of reinforcement over Phase 2 decreases or increases, respectively. The two procedures were designed to yield identical average rates of reinforcement over Phase 2; therefore, the amended Shahan-Sweeney model 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 they gave the rat the opportunity to experience extended periods where R1 is extinguished while alternative reinforcers were also unavailable for R2. The contextual hypothesis, but not the Shahan-Sweeney model predicts that this procedure should weaken resurgence.

Experiment 1

Experiment 1 expanded on the Bouton and Schepers (2014) experiments, where rats earned reinforcement for R2 responses in Phase 2 only after a period of required abstinence from responding on R1. Those experiments provided evidence that the distribution of earned reinforcers, which resembled a thinning procedure in reverse, was responsible for the weakened resurgence that was observed.

The design of Experiment 1 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 on average every 30 s. 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 number of reinforcers that rats in the 45-s Negative Contingency group from Bouton and Schepers (2014) received over days. In this way I was able to re-examine the effects of reverse thinning. Two additional groups (Group Reverse Thinning Free and Group VI 10-s Free) received a pellet that was not contingent on responding whenever a yoked rat in one of the other groups earned one. The yoked groups served two purposes. First, they provided a further 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 observed. Second, these groups were critical in the design of Experiments 2 and 3. Delivery of non-contingent, as opposed to response-contingent reinforcers would provide greater experimenter control over the number and rate of reinforcers delivered during Phase 2. If non-contingent

reinforcers produce the same effects as response-contingent ones, then Experiments 2 and 3 would utilize non-contingent reinforcement in their experimental designs.

Method

Subjects

The subjects were 32 female Wistar rats 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 a VI 30-s reinforcement schedule. On this schedule, a pellet was available with a 1 and 30 probability during each s. Once the program indicated that one (or more) pellet(s) was available, the next R1 response delivered a single pellet and reset the 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 (n = 8), 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 Free and Group Reverse Thinning Free, 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 primarily expressed as responses per minute. However, because experimental analyses of Behavioral Momentum Theory and the Shahan-Sweeney model have typically measured responding during testing as a proportion of baseline responding (e.g., the final session of Phase 1), additional analyses examined R1 responding during the resurgence test as a proportion of each rat's response rate during the final session of Phase 1. Data were put through analyses of variance (ANOVAs) with a rejection criterion of $p < .05$. Follow-up comparisons were conducted using Fisher's LSD (Least Significant Difference) analyses. Planned t-tests were conducted to examine differences in the rate and increases in the rate of R1 responding during the resurgence test. A statistical outlier in Group Reverse Thinning Free 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. On average, the other rats in the group made 2.3 responses per minute.

Results

The main 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 contingent response 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 results with the groups that had received free pellets during Phase 2 were not quite as clear cut.

Response Acquisition and Elimination. Acquisition of R1 responding is summarized in the upper left panel of Figure 1. R1 responding increased reliably in all groups over the 12 sessions, $F(11, 297) = 55.28$, $MSE = 37.15$, $p < .001$. Random assignment to groups was successful in the sense that the main effect of Group and the Group x 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 4 (Group) x 8 (Session) ANOVA identified a main effect of Session, $F(7, 189) = 30.03$, $MSE = 14.21$, $p < .001$. There was also a main effect of Group, $F(3, 27) = 4.76$, $MSE = 96.31$, $p < .01$, and a Group x Session interaction, $F(21, 189) = 2.93$, $MSE = 14.21$, $p < .001$. LSD Analyses indicated that Group VI 10-s responded less than Group VI 10-s Free over the phase ($p = .001$). But Group Reverse Thinning made a similar number of responses as Group Reverse Thinning Free ($p = .42$). Neither Group VI 10-s nor Group VI 10-s Free differed from the corresponding reverse thinning group (ps > .14). Separate one-way

ANOVAs identified differences in R1 responding during Sessions 1, 2, 3, 5, 6, and 8, *F*s > 3.00. Group VI 10-s made fewer R1 responses than Group VI 10-s Free during each of these sessions (*p*s < .05). Group Reverse Thin responded more than Group VI 10-s during Sessions 1 and 2 (*p*s < .05). However, Group Reverse Thin and Group Reverse Thin Free responded similarly in each Phase 2 session (*p*s > .05). During the final session Group VI 10-s Free responded more than either of the groups receiving response contingent reinforcers (*p*s < .01).

Turning to R2 (lower middle panel of Figure 1), a 4 (Group) x 8 (Session) ANOVA indicated that responding increased over the eight Phase 2 sessions, $F(7, 189) = 40.81$, $MSE = 25.46$, $p < .001$. The main effect of Group, $F(3, 27) = 27.51$, $MSE = 774.75$, $p < .001$, and the Group x Session interaction, $F(3, 27) = 46.77$, $MSE = 61.15$, $p < .001$, were reliable. LSD analyses found that Group VI 10-s made more R2 responses than Group Reverse Thinning ($p < .05$) over the entire phase. Group VI 10-s and Group Reverse Thinning also made more R2 responses than either of the groups that received non-contingent pellets (*p*s < .001).

Resurgence Test. Responding on R1 during the resurgence test (compared with the last Phase 2 session) is shown in the upper right panel of Figure 1. A 4 (Group) x 2 (Session) ANOVA indicated a reliable main effect of Session, $F(1, 27) = 14.61$, $MSE = 6.16$, $p = .001$, as well as a main effect of Group, $F(3, 27) = 6.89$, $MSE = 10.29$, $p < .001$. The Group x Session interaction was not significant, $F(3, 27) = 1.37$, $MSE = 6.16$, $p = .27$. Planned t-tests confirmed significant resurgence of R1 responding during the test compared to Session 8 of Phase 2 in Group VI 10-s, $t(7) = 4.66$, $p < .01$, and Group Reverse Thinning, $t(7) = 3.54$, $p < .01$. In contrast, a significant resurgence effect was not produced in either Group VI 10-s Free, $t(7) = 1.09$, $p = .31$, or in Group Reverse Thinning Free, $t(7) = 1.66$, $p = .15$. An LSD analysis of responding during the test session revealed that Group VI 10-s Free made more R1 responses

than Group Reverse Thinning ($p = .001$) or Reverse Thinning Free ($p < .01$). Group VI 10-s responded at a marginally greater rate during the test compared to Group Reverse Thinning ($p = .06$). Two different types of analyses have been used to compare significant resurgence effects between groups. The first compares rates of total responding during the resurgence test. Results using this type of analysis may be difficult to interpret when groups have different response rates during the final Phase 2 session. A second type of analysis uses difference scores to compare relative increases in rate during the resurgence test compared to rates during the final Phase 2 session. An independent t-test of their respective difference scores revealed that the increase in responding during the test was marginally greater in Group VI 10-s than in Group Reverse Thinning, $t(14) = 1.89$, $p = .08$.

A separate analysis of resurgence that used each animal's level of R1 responding expressed as a proportion of its rate during the final session of Phase 1 (i.e., its baseline rate) is shown in Figure 2. Conclusions based on proportion baseline were highly similar to those based on R1 rate (above). A Group x Session ANOVA revealed a main effect of Session, $F(1, 27) = 13.84$, $MSE = 0.01$, $p = .002$, and a main effect of Group, $F(3, 27) = 6.42$, $MSE = 0.01$, $p = .002$. The Group x Session interaction was not significant, $F(3, 27) = 2.18$, $MSE = 0.01$, $p = .11$. Planned t-tests again confirmed significant resurgence effects only in Groups VI 10-s, $t(7) = 4.17$, $p < .01$ and Reverse Thinning, $t(7) = 5.54$, $p = .001$. LSD analyses indicated that Group VI 10 Free responded more during the resurgence test than Group Reverse Thinning ($p = .001$) or Reverse Thinning Free ($p = .004$). Similarly, Group VI 10-s responded more than Group Reverse Thinning ($p < .05$). Independent t-tests examining difference scores revealed that the increase from the final session of Phase 2 to the resurgence test was larger in Group VI 10-s than in Group Reverse Thinning, $t(14) = 2.52$, $p < .05$.

R2 responding during the test is summarized in the lower right panel of Figure 1. A 4 (Group) x 2 (Session) ANOVA indicated that when put on extinction, R2 decreased during the test session, $F(1, 27) = 64.55$, $MSE = 40.90$, $p < .001$. In addition, a main effect of Group, $F(3, 27) = 33.57$, $MSE = 146.45$, $p < .001$, and the Group x Session interaction were also reliable, $F(3, 27) = 26.50$, $MSE = 40.90$, $p < .001$.

Discussion

These results suggest that a reverse thinning schedule programmed to provide similar reinforcement rates as those produced 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; as a result, animals quickly learned to respond on R2 at a high rate, which may have produced greater response competition with R1 (e.g., Hernstein, 1970). Alternatively, it was also possible that any source of alternative reinforcement would accelerate the rate of R1 extinction by providing a disruptive force to its behavior (Shahan & Sweeney, 2011). However, R1 did not extinguish more rapidly in a group that received an identical number of non-contingent reinforcers (i.e., Group VI 10-s Free).

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). In its original form, the Shahan-Sweeney model cannot account for this result. The model predicts similar resurgence in Group VI 10-s and Group Reverse Thinning because each received alternative reinforcement on a VI 10-s schedule during the final Phase 2 session. However, as noted

earlier, Sweeney and Shahan (2013a) 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. Using this type of analysis, the model can 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 Free and Reverse Thinning Free) were not as clear cut. The rapid extinction of R1 in Group VI 10-s did not occur when the same number of reinforcers were delivered non-contingently in Group VI 10-s Free. In addition, a significant resurgence effect was only produced in groups that received response contingent reinforcers during Phase 2. As a result of these findings with non-contingent reinforcers, the next two experiments were conducted using contingent 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 2. 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, 2010). However, the effects of forward thinning 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 are similar to the extinction conditions present during the resurgence test. However, the contextual hypothesis 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 considered in the model. 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, 2013a), reverse thinning and forward thinning procedures should produce similarly attenuated resurgence.

Method

Subjects and apparatus.

The subjects were 32 female Wistar rats 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 Thin (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 subject received a 30 min test session R1 and R2 responses were both available but were never reinforced.

Results

The main results are summarized in Figure 3. During the first phase, groups similarly acquired R1 responding over twelve sessions. During Phase 2, responding on R1 decreased when it was placed on extinction while responding on R2 increased; R1 responding

extinguished more rapidly in Groups that initially received rich rates for R2 responses (i.e, Group VI 10-s and Group Thinning). During the test, only Groups VI 10-s and Reverse Thinning exhibited a significant resurgence of R1 responding. 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 3. Responding increased over the 12 sessions, $F(11, 319) = 77.54$, $MSE = 25.95$, $p < .001$. The main effect of Group and the Group x 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 3). A 3 (Group) x 8 (Session) ANOVA revealed a main effect of Session, $F(7, 203) = 60.58$, $MSE = 4.11$, $p < .001$, main effect of Group, $F(2, 29) = 12.75$, $MSE = 12.69$, $p < .001$, and a Group x Session interaction, $F(14, 203) = 33.06$, $MSE = 4.11$, $p < .001$. LSD analyses indicated that 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 that were conducted to decompose the significant interaction revealed that the groups differed during the first 3 and the last 3 sessions of Phase 2, minimum $F(2, 31) = 12.52$, $MSE = 3.28$, $p < .001$. During Sessions 1-3, Group Reverse Thinning made more R1 responses that the other groups (ps $< .001$). During Sessions 6-8, Group Thinning conversely made more responses than the other groups (ps $< .001$). 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 3), a 3 (Group) x 8 (Session) ANOVA indicated that R2 responding changed over the eight Phase 2 Sessions, $F(7, 203) = 52.64$, $MSE = 69.51$, $p < .001$. The main effect of Group, $F(2, 29) = 4.01$, $MSE = 1146.37$, $p < .05$, and the Group x Session interaction, $F(14, 203) = 26.31$, $MSE = 69.51$, $p < .001$, were also reliable. Group VI 10-s made more R2 responses over Phase 2 than either of the other groups (ps < .05). Group Reverse Thinning and Group Thinning made similar amount of responses during Phase 2 ($p = .83$). In order to decompose the significant interaction, separate one-way ANOVAs were conducted. They revealed Group differences during Sessions 1, 2 3, 7, and 8, minimum $F(2, 29) = 3.72$, $MSE = 246.14$, $p < .05$. 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 (ps < .001). Conversely, during Sessions 7 and 8, Group Thinning, which was now receiving the lean rates for R2, made fewer responses than either group (ps < .001).

Resurgence Test. As illustrated in the upper right panel of Figure 3, rats in Group VI 10-s and Group Reverse Thin each exhibited a resurgence of R1 responding during the test session. In contrast, Group Thinning appeared to have a weaker effect. A 3 (Group) x 2 (Session) ANOVA indicated a reliable main effect of Session, $F(1, 29) = 27.95$, $MSE = 0.725$, $p < .001$, a main effect of Group, $F(1, 29) = 5.03$, $MSE = 0.725$, $p < .001$, and a significant Group x Session interaction, $F(1, 29) = 32.02$, $MSE = 0.725$, $p < .001$. Paired t-tests confirmed significant resurgence in Groups VI 10-s, $t(7) = 11.43$, $p < .001$, and Reverse Thinning, $t(11) = 3.89$, $p < .01$. In Contrast, R1 responding in Group Thinning *decreased* between the extinction and test sessions, $t(11) = -4.33$, $p = .001$. LSD analyses indicated that

Group Reverse Thinning made marginally fewer responses during the resurgence test than Group VI 10-s ($p = .06$). An independent t-test comparing difference scores (Test – Phase 2, Session 8) revealed that the difference between the significant resurgence effects in Group VI 10-s and Group Reverse Thinning did not reach significance, $t(18) = 1.71$, $p = .11$. However, because Experiment 1 and Experiment 2 contained identical groups which each produced trends that suggested Group VI 10-s produced a larger resurgence effect than Group Reverse Thinning, an additional analysis was conducted pooling the groups in the two experiments. This analyses provided strong support that Group VI 10-s indeed made a greater amount of R1 responses than Group Reverse Thinning at test, $t(34) = 3.48$, $p = .001$. 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, $t(34) = 2.65$, $p = .01$.

Resurgence data expressed as a proportion of baseline is presented in Figure 4. The Group x Session ANOVA revealed a main effect of Session, $F(1, 29) = 21.88$, $MSE = .001$, $p < .001$, a main effect of Group, $F(2, 29) = 5.89$, $MSE = .003$, $p < .01$ and a Group x Session interaction, $F(2, 29) = 34.35$, $MSE = .001$, $p < .001$. Planned t-tests again confirmed significant resurgence effects in Group VI 10-s, $t(7) = 10.03$, $p < .001$, and Group Reverse Thinning, $t(11) = 3.49$, $p < .01$. In contrast, responding again decreased in Group Thinning, $t(11) = 4.51$, $p = .001$. LSD analyses confirmed that Group VI 10-s responded more during the test than Group Reverse Thinning ($p < .05$) or Group Thinning ($p < .05$). Independent t-tests confirmed that the significant resurgence effect was a larger increase in rate in Group VI 10-s than Group Reverse Thinning, $t(18) = 2.41$, $p < .05$.

R2 responding in the resurgence test is summarized in the lower right panel of Figure 3. A 3 (Group) x 2 (Session) ANOVA revealed an effect of Session, $F(1, 29) = 176.66$, $MSE = 47.81$, $p < .001$, a main effect of Group $F(2, 27) = 33.31$, $MSE = 210.87$, $p < .001$ and a Group x Session interaction, $F(2, 29) = 25.24$, $MSE = 47.81$, $p < .001$. LSD analyses indicated that Group Thinning made fewer R2 responses during the test session than either of the other groups, (ps < .001).

Reinforcers earned during Phase 2. Figure 5 summarizes the number of pellets the different groups earned in the sessions of Phase 2. A Group x Session ANOVA identified a main effect of Session, $F(7, 203) = 49.43$, $MSE = 130.79$, $p < .001$, a main effect of Group $F(2, 29) = 290.11$, $MSE = 366.69$, $p < .001$, and a reliable Group x Session interaction, $F(14, 203) = 469.38$, $MSE = 130.79$, $p < .001$. LSD analyses indicated that Group VI 10-s earned more reinforcers than Group Reverse Thinning or Group Thinning over Phase 2 ($p < .001$). In addition, that Group Thinning earned marginally fewer pellets than Group Reverse Thinning ($p = .05$). At the end of Phase 2 the average number reinforcers earned per session were 159.39, 95.94, and 90.19 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, the reverse thinning schedule attenuated but did not eliminate the resurgence effect. Additionally, the forward thinning procedure was effective at completely eliminating resurgence. Each of these 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.

The Shahan-Sweeney model also correctly predicts that the thinning procedure will weaken resurgence compared with a constant VI 10-s schedule. However, the model's ability to explain the other results depends on the method of calculating Phase 2 reinforcement. Because Group VI 10-s and Group Reverse Thinning received identical terminal programmed rates (thus predicting equal resurgence using that analysis), the original model cannot predict weakened resurgence in Group Reverse Thinning. Moreover, 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 order to account for the weaker resurgence observed in Group Thinning, the model must again be considered using the terminal rate of alternative reinforcement.

It may be important to note that Group Reverse Thinning received approximately 6% more reinforcers on average per session during Phase 2 than Group Thinning (95.94 vs. 90.19). However, it seems unlikely that this 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%.

Thus, it is more likely that receiving the lean rates closer to the test (and not the marginal difference in the number of reinforcers earned) weakened resurgence in Group Thinning.

Experiment 3

Experiment 3 provided a further test of how the distribution of reinforcement throughout Phase 2 influences resurgence. As previously mentioned, the contextual hypothesis suggests that resurgence can be weakened when Phase 2 conditions allow the animal to learn that responses are not reinforced in a pellet context similar to that which will prevail during the resurgence test. As noted earlier, this approach is consistent with the effects of our thinning and reverse thinning procedures.

In a recent paper, Sweeney and Shahan (2013a) reported other results that might be consistent with a contextual hypothesis of resurgence. In one condition, animals received alternating Phase 2 sessions in which R2 was reinforced on a VI 60-s schedule or was placed on extinction. According to the context hypothesis, the extinction sessions should weaken later resurgence because they provide animals with opportunities to learn that R1 is not reinforced in a context of pellet distribution that is identical to the one that prevails during the resurgence test (extinction). In fact, that is what Sweeney and Shahan (2013a) found; R1 responding did not resurge in a final test after alternating exposure to alternating sessions of VI 60-s and extinction during Phase 2.

However, Sweeney and Shahan (2013a) also reported a similar lack of resurgence in a separate condition in which reinforcement was delivered on a VI 60-s schedule during all five of the Phase 2 sessions. This result is not consistent with the context hypothesis, because it suggests that the alternating extinction sessions had no effect on the amount of resurgence during the test. However, Sweeney and Shahan (2013a)

concluded that these results were consistent with predictions of the Shahan-Sweeney model; as noted earlier, resurgence is expected to decrease as the number of any type of Phase 2 session increases. However, the absence of resurgence in a condition that received so little Phase 2 training (5 sessions) is surprising. One possible explanation is that the result was a consequence of the fact that animals in the experiment were not naïve; they had been used in previous conditioning experiments. Animals with extensive conditioning histories may have previously learned about the extinction conditions that exist during a resurgence test. These facts complicate the interpretation of the lack of resurgence in the group that received alternating sessions of VI 60-s and extinction. Therefore, Experiment 3 was designed to further the issue using procedures that have become more standard in our laboratory

The design of Experiment 3 is summarized in Table 3. 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 Shahan-Sweeney model, these groups should produce equal resurgence. That is, the alternating extinction sessions should have no effect on resurgence. 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 programmed averages, the average reinforcement version of the model predicts equal resurgence in Group Average and Group Alternating.

Method

Subjects and apparatus

The subjects were 32 female Wistar rats 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.

Data treatment. Analyses were conducted in exactly the same way as in Experiments 1 and 2. A significant outlier ($Z = 3.13$, Field, 2005) in Group Alternating was excluded from the analyses. During the resurgence test, this animal responded on R1 at 73% of its rate during baseline (the final Phase 1 session). The remaining animals in Group Alternating, on average, responded at only 6% of their baseline rate.

Results

The main results are summarized in Figure 6. All rats acquired lever responding on R1 during the first phase. During Phase 2, responding on R1 decreased and R2 increased over sessions in Groups VI 10-s and Group 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 Group VI 10-s and Group 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 6. R1 responding increased over the twelve Phase 1 sessions, $F(11, 308) = 61.39$, $MSE = 30.81$, $p < .001$. Random assignment to groups was successful in that the main effect of Group and a Group x 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 6). This was confirmed by 3 (Group) x 7 (Session) ANOVA that identified a main effect of Session, $F(6, 168) = 37.07$, $MSE = 2.91$, $p < .001$, a main effect of Group, $F(2, 28) = 8.70$, $MSE = 10.84$, $p = .001$, and a Group x Session interaction, $F(12, 168) = 6.24$, $MSE = 2.91$, $p < .001$. Follow up analyses indicated that Group Alternating responded more over Phase 2 Sessions than Group Average ($p < .05$) 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 $F(2, 29) = 3.42$, $MSE = 7.66$, $p < .05$. Follow up analyses revealed that in Session 1, Group Average made more responses than Group Alternating ($p < .05$). 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 during these sessions in the Alternating Group.

A 3 (Group) x 7 (Session) ANOVA on R2 responding (lower, middle panel) revealed a Session effect $F(6, 168) = 63.46$, $MSE = 41.20$, $p < .001$, a main effect of Group, $F(2, 28) = 5.63$, $MSE = 1170.27$, $p < .01$, and a Group x Session interaction, $F(12, 168) = 20.40$, $MSE = 41.20$, $p < .001$. LSD analyses indicated that Group VI 10-s and Group Average responded similarly on R2 (ps $>.05$), but that each group made more R2 responses than Group Alternating (ps $< .05$). 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 $F(2, 28) = 10.05$, $MSE = 129.08$, $p = .001$. Group Alternating made fewer responses than the other groups in each of these sessions (ps < .001).

Resurgence Test. As illustrated in the upper right panel of Figure 6, 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 produce a similar increase. A 3 (Group) x 2 (Session) ANOVA indicated a reliable main effect of Session, $F(1, 28) = 50.72$, $MSE = 3.40$, $p < .001$, and a significant Group x Session interaction, $F(2, 28) = 30.83$, $MSE = 3.20$, $p = .001$. The main effect of Group did not reach significance, $F(2, 29) = 2.43$, $MSE = 3.90$, $p = .11$. Planned t-tests confirmed the significant resurgence effects in Group VI 10-s, $t(7) = 5.76$, $p = .001$, and Group Average, $t(11) = 7.53$, $p < .001$, and no effect in Group Alternating, $t(10) = 0.53$, $p = .61$. LSD analyses indicated that responding was similar in Group VI 10-s and Group Average during the Final Resurgence Test ($p = .62$), and that each made more R1 responses than Group Alternating, largest ($p = .001$). The analysis of difference scores revealed that Group VI 10-s and Group Average produced a similar increase in rate over the previous session, $t(18) = .98$, $p = .34$.

Figure 7 shows the resurgence data expressed as a proportion of baseline responding. A 3 (Group) x 2 (Session) ANOVA indicated a significant main effect of Session, $F(1, 28) = 63.63$, $MSE = .001$, $p < .001$ and a significant Group x Session interaction, $F(2, 28) = 12.12$, $MSE = .002$, $p < .001$. The main effect of Group was not significant, $F(2, 28) = 2.38$, $MSE = .004$, $p = .11$. Independent t-tests indicated that responding was similar in Group VI 10-s and Group Average during the final resurgence test, $t(18) = .64$, $p = .53$ and that each made more

R1 responses than Group Alternating, smallest $t(17) = 4.58$, $p < .001$. Planned t-tests again confirmed a significant resurgence effects in Group VI 10-s, $t(7) = 6.80$, $p < .001$ and in Group Average, $t(11) = 10.29$, $p < .001$. Group Alternating did not produce a similar effect, $t(10) = .56$, $p = .59$. Independent t-tests in this analysis again showed that the significant increases in responding during the resurgence tests were similar in Group VI 10-s and Group Average, $t(18) = 1.56$, $p = .14$.

R2 responding during the final resurgence test is summarized in the lower right panel of Figure 6. A 3 (Group) x 2 (Session) ANOVA indicated a main effect of Session, $F(1, 28) = 127.35$, $MSE = 120.13$, $p < .001$, and a main effect of Group $F(2, 28) = 3.92$, $MSE = 278.12$, $p < .05$. The Group x Session interaction was not reliable, $F(2, 28) = 1.31$, $MSE = 120.13$, $p = .29$.

Reinforcers earned during Phase 2. The number of pellets earned during Phase 2 is summarized in Figure 7. A 3 (Group) x 7 (Session) ANOVA was conducted to determine whether groups differed in the number of pellet reinforcers earned during Phase 2. A main effect of Session, $F(1, 28) = 196.98$, $MSE = 114.64$, $p < .001$, indicated that reinforcers were earned differentially over Phase 2. A reliable main effect of Group, $F(2, 28) = 177.90$, $MSE = 486.36$, $p < .001$, and a significant Group x session interaction, $F(1, 28) = 228.34$, $MSE = 114.64$, $p < .001$, indicated that group differences in reinforcers earned was dependent on the session. LSD analyses revealed that 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 not a significant difference in reinforcers earned between Group Alternating and Group Average ($p = .11$).

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 weakened 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. The model suggests that the decreases in resurgence are not due to the multiple tests per se, but rather resurgence is weakened with additional Phase 2 sessions. However, the fact that robust resurgence effects were produced in the other two groups that received an identical number of Phase 2 sessions suggests that the additional resurgence tests and not simply additional sessions were responsible for weakening resurgence in Group Alternating. This result is better explained with the contextual hypothesis. According to that view, the three Phase 2 tests provided an opportunity for animals in Group Alternating to learn that R1 was not reinforced in a context that was identical to the one that was present during the final test.

The Shahan-Sweeney model has difficulties accounting for less resurgence in Group Alternating than groups that received either the same terminal rate (Group VI 10-ss) rate or the same average rate over the entire phase (Group Average). When the model

is considered using the rate from the final Phase 2 session it incorrectly predicts equal resurgence in Group Alternating and Group VI 10-s. In addition, it also predicts that Group Average, which received a leaner terminal rate (i.e., VI 17.5-s), would resurge less than Group VI 10-s.

When using the Phase 2 average, the model successfully predicts less resurgence in Group Alternating (i.e., VI 17.5-s) 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 does not predict that resurgence should be less in Group Average.

A potential problem for both the Shahan-Sweeney model and a contextual hypothesis is that resurgence was equal in Group VI 10-s and Group Average. These schedules could provide a quite different amount of reinforcers during each Phase 2 session (180 vs. 102.85) but each produced similar levels of resurgence during the test. It is possible that at relatively high and constant rates these differences have relatively small effects on resurgence. 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 rate (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 the resurgence effect 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 with a rate that alternated between a very rich rate or a rate of zero 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 produced 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 completely eliminate resurgence.

The current results provide some difficulty for the quantitative 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 Experiments 1 and 2. Specifically, it cannot account for weakened resurgence after reverse thinning compared to a group that received a constant rate throughout the phase. Rather, the model predicts that groups with equal programmed rates at the end of Phase 2 should produce equal resurgence. In Experiment 3, the model also cannot predict that resurgence can be eliminated by a schedule that alternates between extinction and a VI 10-s. Instead, the model predicted that the alternating group would resurge at a level similar to that of a group that received the same terminal rate in all sessions. 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 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.

The contextual hypothesis suggests that resurgence occurs according to the mechanisms that underlie ABC renewal. That is, removal from the context of extinction is sufficient for an extinguished behavior to return. But factors that increase generalization between the extinction and test phases have shown to reduce renewal (Brooks & Bouton, 1994; Willcocks & McNalley, 2014). This 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 that received its richest reinforcement schedule 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.

In summary, the current results suggest that resurgence is better explained by the contextual hypothesis than by the Shahan-Sweeney model using 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 of the current results is that CM therapies in humans may more effectively reduce relapse when reinforcement is available at only lean rates at any point, but that lean rates might be more effective when they are given near the conclusion of treatment. In addition, designs that intermittently remove alternative reinforcement during treatment may reduce potential relapse by increasing generalization with the conditions after treatment ends.

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Table 1

Experiment 1 Design

Group	Phase 1	Phase 2	Phase 3
VI 10-s	R1: VI 30 R2: ---	R1: Ext R2: VI 10	R1: Ext R2: Ext
Reverse Thnning	R1: VI 30 R2: ---	R1: Ext R2: VI 1200…VI 10	R1: Ext R2: Ext
VI 10-s Free	R1: VI 30 R2: ---	R1: Ext R2: Yoked VI 10	R1: Ext R2: Ext
Reverse Thinning Free	R1: VI 30 R2: ---	R1: Ext R2: Yoked VI 1200…VI 10	R1: Ext 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. "Free" groups had no response requirements and were delivered a "free" pellet immediately after an animal in the corresponding contingent group had earned one.

Table 2

Experiment 2 Design

Group	Phase 1	Phase 2	Phase 3
VI 10-s	R1: VI 30 R2: ---	R1: Ext R2: VI 10	R1: Ext R2: Ext
Thinning	R1: VI 30 R2: ---	R1: Ext R2: VI 10…VI 1200	R1: Ext R2: Ext
Reverse Thinning	R1: VI 30 R2: ---	R1: Ext R2: VI 1200…VI 10	R1: Ext 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.

Table 3

Experiment 3 Design

Group	Phase 1	Phase 2	Phase 3
VI 10-s	R1: VI 30 R2: ---	R1: Ext R2: VI 10	R1: Ext R2: Ext
Group Alternating	R1: VI 30 R2: ---	R1: Ext R2: VI 10 (Sessions 1,3,5,7); Ext (Sessions 2,4,6)	R1: Ext R2: Ext
Group Average	R1: VI 30 R2: ---	R1: Ext R2: VI 17.5	R1: Ext 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.

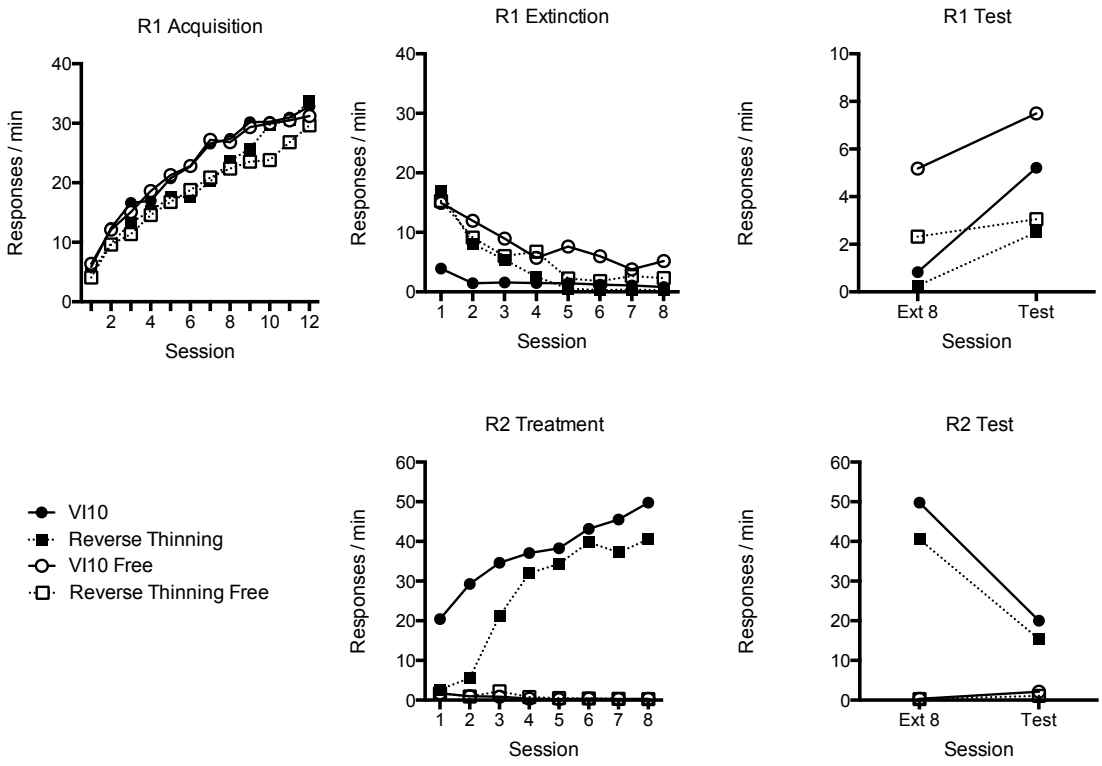

Figure 1. Results of Experiment 1. The upper panels summarize R1 responding during its acquisition (left), extinction (middle), and during the resurgence test compared with the final Phase 2 session (right). R2 rates are summarized in the lower panels during Phase 2 (middle) and during the test compared with its rate during the final Phase 2 session.

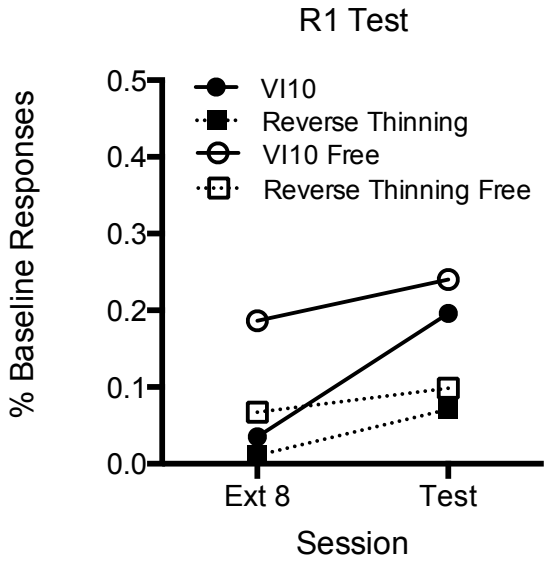

Figure 2. Proportion of baseline responding during the final test relative to the final Phase 2 session in Experiment 1.

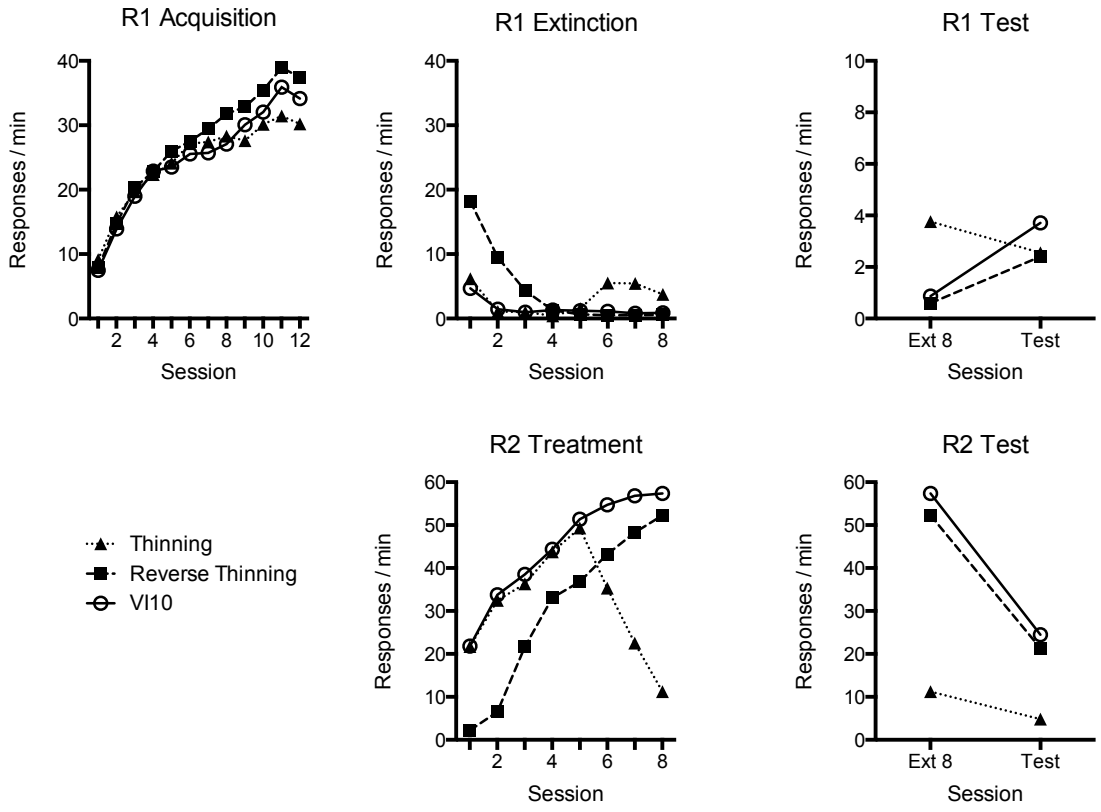

Figure 3. Results of Experiment 2. The upper panels summarize R1 responding during its acquisition (left), extinction (middle), and during the resurgence test compared with the final Phase 2 session (right). R2 rates are summarized in the lower panels during Phase 2 (middle) and during the test compared with its rate during the final Phase 2 session.

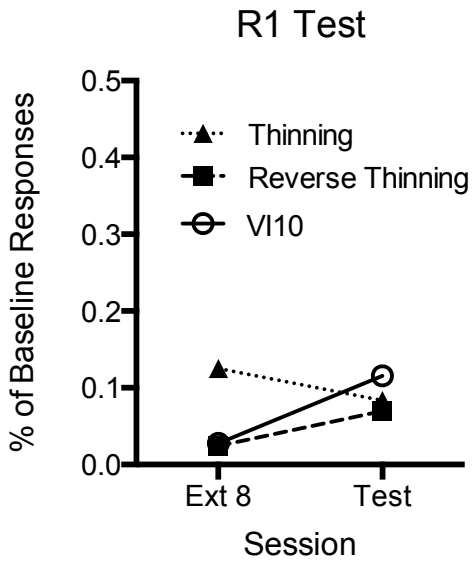

Figure 4. Proportion of baseline responding during the final test relative to the final Phase 2 session in Experiment 2.

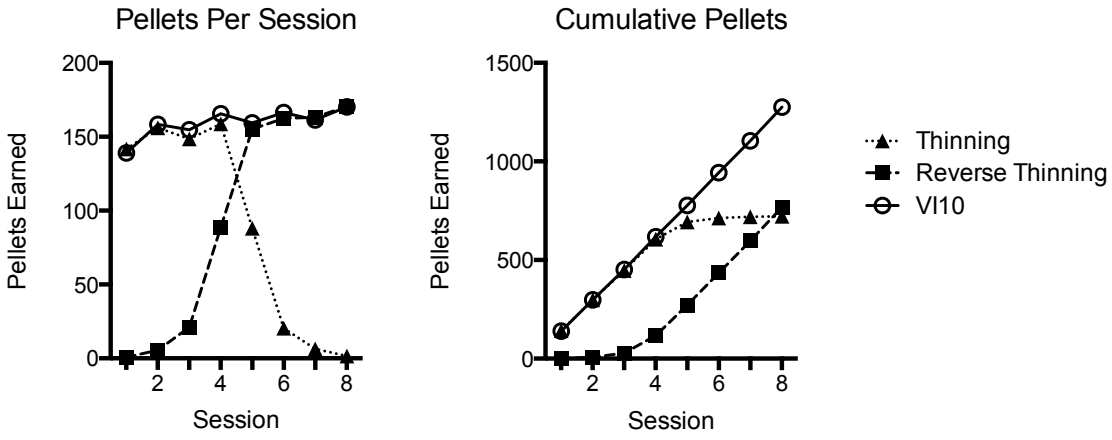

Figure 5. Reinforcers during Phase 2 of Experiment 2. The mean number of pellets earned during each Phase 2 session (left) and the cumulative mean of pellets that had been earned during Phase 2 at each session.

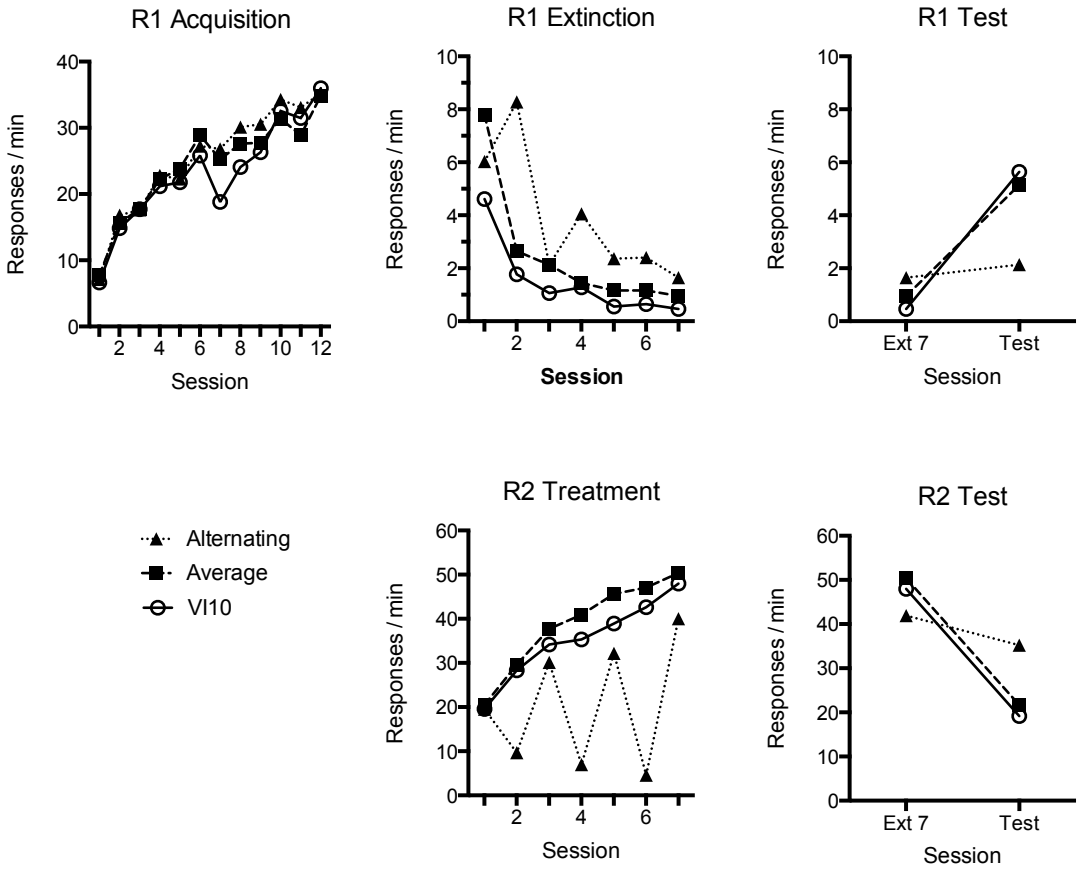

Figure 6. Results of Experiment 3. The upper panels summarize R1 responding during its acquisition (left), extinction (middle), and during the resurgence test compared with the final Phase 2 session (right). R2 rates are summarized in the lower panels during Phase 2 (middle) and during the test compared with its rate during the final Phase 2 session.

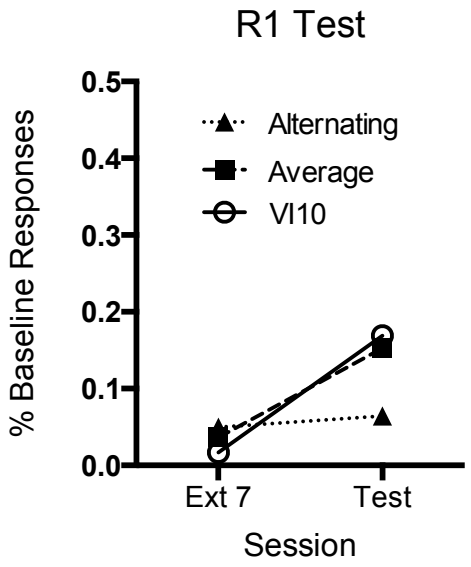

Figure 7. Proportion of baseline responding during the final test relative to the final Phase 2 session in Experiment 3.

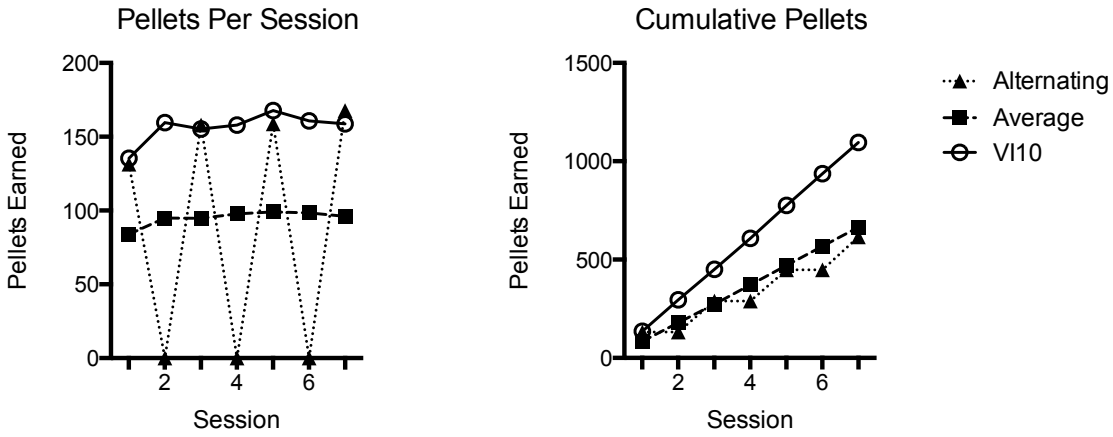

Figure 8. Reinforcers during Phase 2 of Experiment 3. The mean number of pellets earned during each Phase 2 session (left) and the cumulative mean of pellets that had been earned during Phase 2 at each session.