A Quantitative Analysis of Response Elimination and Resurgence Using Rich, Lean, and Thinning Schedules of Alternative Reinforcement

Mary M. Sweeney
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A QUANTITATIVE ANALYSIS OF RESPONSE ELIMINATION
AND RESURGENCE USING RICH, LEAN, AND THINNING
SCHEDULES OF ALTERNATIVE REINFORCEMENT

by

Mary M. Sweeney

A thesis submitted in partial fulfillment
of the requirements for the degree

of

MASTER OF SCIENCE

in

Psychology

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UTAH STATE UNIVERSITY
Logan, Utah

2012
ABSTRACT

A Quantitative Analysis of Response Elimination and Resurgence Using Rich, Lean, and Thinning Schedules of Alternative Reinforcement

by

Mary M. Sweeney, Master of Science

Utah State University, 2012

Major Professor: Dr. Timothy A. Shahan
Department: Psychology

A common approach to the treatment of instrumental problem behavior is the introduction of an acceptable alternative source of reinforcement. However, when alternative reinforcement is removed or reduced, the target behavior tends to relapse. The relapse of a target response following the removal of alternative reinforcement has been termed resurgence. Shahan and Sweeney developed a quantitative model of resurgence based on behavioral momentum theory that captures both the disruptive and strengthening effects of alternative reinforcement on the target response. The quantitative model suggests that although higher rates of alternative reinforcement result in faster response elimination, lower rates of alternative reinforcement result in less relapse when removed. The present study was designed to examine the possibility that good target response suppression and less relapse could be achieved by beginning with a higher (rich) rate of alternative reinforcement and gradually thinning it such that a lower (lean) rate of alternative reinforcement is ultimately removed. Furthermore, the data
obtained were generated to provide insight into how thinning rates of alternative reinforcement might be incorporated into the quantitative model of resurgence. Results suggest that rich rates of alternative reinforcement were more effective than lean or thinning rates of alternative reinforcement at response suppression during treatment, but when alternative reinforcement was discontinued, the group that experienced rich rates exhibited a substantial increase. Although lean and thinning rates of alternative reinforcement were not as effective at response suppression during treatment as rich rates, they still resulted in substantial decreases in the target response. Furthermore, removal of lean rates of alternative reinforcement did not result in substantial increase in the target response. Advantages and disadvantages of rich, lean, and thinning alternative reinforcement rates are discussed with respect to target response suppression and sensitivity to the end of treatment, and an alternative response rate is discussed. Although a small modification to the quantitative model was able to similarly account for data produced by rich, lean, and thinning alternative reinforcement, as it currently stands the model is unable to account for the finding that alternative reinforcement may not always serve as a disruptor relative to a no alternative reinforcement control.
PUBLIC ABSTRACT

A Quantitative Analysis of Resurgence in Rich, Lean, and
Thinning Schedules of Alternative Reinforcement

by

Mary M. Sweeney, Master of Science
Utah State University, 2012

Mary M. Sweeney, graduate student in the Experimental and Applied
Psychological Science graduate program, proposed and completed this thesis in partial
fulfillment for the requirements of the degree of Master of Science of Psychology. This
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Many problem behaviors, such as aggression or self-injury in persons with
intellectual or developmental disabilities (IDD) or drug abuse can be maintained by the
consequences of those behaviors. For example, many instances of aggression in
individuals with IDD may be engaged in order to access caregiver attention, or to avoid
undesirable tasks. Drug taking is another behavior maintained by its consequences (the
intoxicating effects of drugs, or relief from withdrawal). Consequences that increase the
probability of the behavior that produced them have been termed “reinforcers”. One
popular and effective treatment of problem behavior maintained by reinforcers is to
introduce an alternative source of reinforcement. For example, an individual with IDD
might be taught to use communication cards to receive caregiver attention (rather than
engaging in problem behavior to do so), or a drug user might be given monetary vouchers or prizes in exchange for drug abstinence. Unfortunately, if these alternative sources end (as treatment ends) the problem behavior in question often relapses.

The present project was designed to assess relative advantages and disadvantages of different rates of treatment, high rates of treatment, low rates of treatment, and treatment that starts at a high rate but decreases across time (thinning rates). The project took place in a basic animal laboratory with rat subjects to help reduce extraneous variables and have better control over the experiment than in treatment settings. The target response (analogous to problem behavior) was pressing a lever to produce food, and the alternative response (analogous to the treatment behavior) was nose poking to produce food. The project was motivated by both applied implications of these treatment types, as well as providing a potential challenge for a recent quantitative framework of relapse.

Results suggest that high rates of treatment were more effective than low or thinning rates of treatment at response suppression during treatment, but when alternative reinforcement was discontinued, the group that experienced high rates exhibited a substantial increase. Although low and thinning rates of treatment were not as effective at response suppression during treatment as high rates, they still resulted in substantial decreases in the target response. Furthermore, removal of low rates of alternative reinforcement did not result in substantial increase in the target response. Advantages and disadvantages of high, low, and thinning treatment rates are discussed with respect to target response suppression, sensitivity to the end of treatment, and treatment response rate are discussed.
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Mary M. Sweeney
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CHAPTER I

INTRODUCTION

The replacement of a dangerous or undesirable behavior with a socially acceptable alternative is a common treatment of instrumental problem behaviors. Alternative sources of reinforcement are often used to reduce behaviors such as self-injury in persons with developmental disabilities or to encourage drug abstinence in substance abusers. Despite treatment efforts, such problem behaviors are characteristically persistent and prone to relapse.

Basic researchers have developed an animal model analogous to relapse following treatment using alternative reinforcement, termed resurgence. In resurgence, an instrumental target behavior is trained during Phase I and then placed on extinction in Phase II (i.e., no reinforcement is available for the response). Also during Phase II, a second, alternative response is introduced and reinforced. When the alternative response is also placed on extinction during Phase III, there is an increase in the first behavior (i.e., relapse). Relapse following this type of intervention is therefore a point of interest for both clinicians and basic scientists.

Recent work has attempted to integrate resurgence into behavioral momentum theory. This theory offers a framework for understanding the persistence of instrumental behavior under disruption. Shahan and Sweeney (2011) developed a quantitative model of resurgence based on behavioral momentum theory that captures the specific effects of alternative reinforcement on persistence and relapse of an instrumental response. The model captures the fact that although higher rates of alternative reinforcement result in faster response elimination than lower rates of alternative reinforcement, lower rates of
alternative reinforcement result in less relapse when they are removed. Therefore, in
order to have a treatment that reduces behavior quickly and results in less relapse, one
might one might initially use a higher rate of alternative reinforcement but gradually thin
(i.e., reduce) it such that a lower rate of alternative reinforcement is ultimately removed.

Although alternative reinforcement thinning is common practice in applied
settings in order to reduce relapse following the end of treatment, increases in problem
behavior are often observed when alternative reinforcement is thinned. Furthermore,
thinning techniques vary across laboratories and participants. As such, it may be useful
to examine this phenomenon in a basic laboratory setting with fewer extraneous
variables. The two objectives of this research were to (a) examine the effects of fixed
high rates, fixed low rates, and thinning rates of alternative reinforcement on response
elimination and resurgence; and (b) evaluate Shahan and Sweeney’s (2011) quantitative
model of resurgence based on the data collected.

This experiment compared the response elimination and relapse of a target
behavior across four groups that varied with respect to alternative reinforcement rate
during Phase II (treatment). One group began with a higher rate (hereafter referred to as
a rich rate) of alternative reinforcement that was thinned daily such that a lower rate
(hereafter referred to as a lean rate) of reinforcement was removed upon the advancement
to Phase III. Three comparison groups were also studied. The first comparison group
had alternative reinforcement fixed to the first (richest) rate experienced by the thinning
group. Another comparison group had alternative reinforcement fixed to the last
(leanest) rate experienced by the thinning group. These two comparisons tested model
predictions regarding the effect of fixed alternative reinforcement rates and also provided
data to judge the relative advantage or disadvantage of thinning compared to fixed schedules of reinforcement. Finally, all groups were compared to a control group in which alternative reinforcement was never introduced.
CHAPTER II

BACKGROUND AND SIGNIFICANCE

Instrumental Problem Behavior

The instrumental learning process, during which organisms learn the relationship between a response and its consequences, is not constrained to advantageous behavior. Many problem behaviors are maintained by access to food, toys, social interaction, money, or drugs. Take for example, an institutionalized man with an intellectual disability who injures himself and others in order to access toys and food that staff use to “calm him down” (Hanley, Iwata, & Thompson, 2001, p. 20). Self-injurious behavior (SIB) and aggression are common obstacles for caregivers and clients with developmental disabilities, with prevalence of SIB ranging from 1.7% to 41% (Cooper et al., 2009a) and physical aggression ranging from 2.1% to 27.9% (Cooper et al., 2009b) with typically much higher rates in institutionalized populations (e.g., Saloviita, 2000).

Problem behaviors, of course, are not limited to persons with disabilities. Drug taking is an instrumentally learned behavior maintained by the effects of drugs. If drug taking escalates into drug dependence, the resulting addiction can cause harm to the user, his or her family, as well as burden society through loss of productivity, health expenses, and the cost of drug-related crimes. The Office of National Drug Control Policy (2004) estimated that in 2002 alone, drug abuse cost the United States $180.9 billion. This figure does not include any costs to the U.S. that result from legal drugs, such as alcohol and tobacco. It is clear from these examples that treatment of instrumental problem behaviors should be an area of great concern for psychological research.
Treatments Using Alternative Reinforcement

One popular and effective approach to treatment of problem behavior is the introduction of an alternative source reinforcement. Contingency management, for example, provides incentives to recovering substance abusers contingent on drug abstinence. Often the alternative reinforcement takes the form of monetary-based vouchers that can be exchanged for prosocial activities in an attempt to compete with the effects of drugs (e.g., Budney, Higgins, Delaney, Kent, & Bickel, 1991). Another treatment involving alternative reinforcement used in children and adults with disabilities is functional communication training (Carr & Durand, 1985). Typically, this involves withholding the consequence maintaining problem behavior and providing that reward contingent on a novel communicative response. For instance, a child with problem behavior maintained by access to social reinforcers could be taught to say, “Talk to me, please” (Volkert, Lerman, Call, & Trosclair-Lasserre, 2009, p. 148), in order to receive attention. This method is often quite effective at reducing the target (problem) response, but if the subject encounters intermittent reinforcement for the alternative response, or alternative reinforcement is removed altogether, the target behavior often relapses (e.g., Volkert et al., 2009).

Resurgence

Relapse following the removal of alternative reinforcement has been termed resurgence (Epstein & Skinner, 1980). Procedures analogous to alternative reinforcement treatments in humans also produce relapse in animals (e.g., Leitenberg, Rawson, & Bath, 1970; Leitenberg, Rawson, & Mulick, 1975; Lieving & Lattal, 2003; Winterbauer &
Bouton, 2010), suggesting that the resurgence phenomenon is common across species. The typical experimental preparation used to study resurgence involves three distinct phases. The first phase trains an animal in an operant chamber to emit a target response, such as a lever press. Next, the target response is placed on extinction and an alternative source of reinforcement is introduced—for example, pulling a chain to produce food rather than pressing the lever. Lever pressing decreases as chain pulling increases. In the third phase, chain pulling is also placed on extinction and target responding increases (i.e., resurgence) even though reinforcement is still unavailable for the lever press.

Resurgence is not unique to food reinforcement; the phenomenon occurs when the target response is maintained by alcohol (Podlesnik, Jimenez-Gomez, & Shahan, 2006), cocaine (Quick, Pyszczynski, Colston, & Shahan, 2011) and negative reinforcement (Bruzek, Thompson, & Peters, 2009). Thus, despite the temporary effectiveness of treatments that provide an alternative source of reinforcement in reducing target behavior, behavior maintained by a wide variety of reinforcers persists following the removal of alternative reinforcement.

Shahan and Sweeney (2011) recently developed a quantitative model that incorporates the resurgence phenomenon into behavioral momentum theory (e.g., Nevin, 1992). Behavioral momentum theory highlights the important role of the Pavlovian stimulus-reinforcer relationship in determining both the rate of extinction and relapse of a target response. Before discussing the specifics of the quantitative model of resurgence put forward by Shahan and Sweeney, behavioral momentum theory will be described in more detail.
Behavioral Momentum Theory

Behavioral momentum theory suggests there are two separable aspects of instrumental behavior—response rate and resistance to change. Response rate, the theory contends, is determined by the response-reinforcer contingency. Resistance to change, on the other hand, is controlled by the Pavlovian stimulus-reinforcer relationship between the stimulus-context in which the behavior occurs and reinforcement received in that context. More specifically, a response that occurs in a stimulus-context with a rich rate of reinforcement will be more resistant to disruption than a response that occurs in a context with a relatively leaner rate of reinforcement. The effect of baseline reinforcement rate on resistance to change has been observed across a variety of subjects, including humans (e.g., Cohen, 1996; Mace et al., 1990), rats (e.g., Grimes & Shull, 2001), pigeons (e.g., Nevin, 1974), and goldfish (l'gaki & Sakagami, 2004). The phenomenon has also been reported using food and nonfood reinforcers, such as alcohol (e.g., Shahan & Burke, 2004), cocaine (Quick & Shahan, 2009) and extra credit points (Cohen, 1996). In addition, the effect is reliable across disruptors, including extinction, satiation (e.g., Nevin, 1974) or distraction (Mace et al., 1990).

An important aspect of the Pavlovian stimulus-reinforcer relationship that determines resistance to change is that the source of reinforcement does not matter, it matters only that reinforcement is delivered within the stimulus-context in which the operant behavior occurs. For example, Nevin, Tota, Torquato, and Shull (1990) demonstrated that added noncontingent reinforcement and reinforcement contingent on an alternative response both increased relative resistance to change even though they decreased predisruption response rates. Such effects of alternative reinforcement on
response rates and resistance to change bolsters the argument for the separation of the response-reinforcer contingency—which is degraded by noncontingent reinforcement—and the Pavlovian stimulus-reinforcer relationship through which all reinforcement in the context increases resistance to change.

Nevin and Grace (2000) have developed a quantitative model of behavioral momentum theory that predicts how baseline reinforcement rate affects resistance to extinction. The augmented extinction model is:

$$\log \left( \frac{B_t}{B_0} \right) = \frac{-t(c + dr)}{r^b}$$

(1)

where $B_t$ is response rate at time $t$ in extinction, $B_0$ is the pre-extinction response rate, $c$ represents the disruptive effect of ending the instrumental response-reinforcer contingency, $d$ scales disruptive impact of the removal of reinforcers, $r$ is the rate of reinforcement in the stimulus-context during baseline (whether reinforcement is response dependent, independent, or contingent on another response), and $b$ represents sensitivity to reinforcement. Parameters $c$ and $d$ are free to vary and are estimated using a least squares regression fit to the data that uses the independent variable ($r$), the data obtained ($B_0$, $B_t$), and a fixed value of sensitivity ($b = .5$; Nevin, 2002). As time ($t$) in extinction increases, proportion of baseline responding decreases as a result of the disruptive effects of extinction captured by parameters $c$ and $dr$. As it appears in the denominator, $r$ counteracts the disruption of the broken contingency and the removal of reinforcement and represents the strength of the stimulus-reinforcer relationship established by baseline reinforcement rates.
Podlesnik and Shahan (2009, 2010) extended the augmented extinction model to account for relapse of instrumental behavior following extinction. Their experiments trained subjects to respond in a two-component multiple schedule preparation. In one component, the subjects received a rich schedule of reinforcement and in the other component subjects received a relatively lean schedule of reinforcement. Responding in the context with a rich history of reinforcement was more resistant to extinction and showed greater relapse following extinction than responding in the context with a relatively more lean history of reinforcement. The effect was consistent across three different relapse phenomena: reinstatement, renewal, and resurgence. Reinstatement involved the delivery of a small number of response-independent or response-dependent reinforcers following the extinction of the instrumental target response. The deliveries of reinforcement, reinforcement-related cues, or stressors have commonly been used to reinstate a previously reinforced then extinguished behavior (e.g., Anker & Carroll, 2010; Reid, 1958). Renewal is the relapse phenomenon in which the response is trained in context A but extinction occurs in context B. When the organism is returned to context A, or introduced to a novel context C, the response that had decreased in context B relapses (e.g., Bouton, Todd, Vurbic, & Winterbauer, 2011). Podlesnik and Shahan (2009) utilized the house light, which provides general chamber illumination in the pigeon’s experimental chamber, to determine context. During training, the house light was steady, but during extinction, the house light was flashing. When the experimenters made the light constant again, responding that had been extinguished relapsed. They also examined resurgence following the removal of alternative reinforcement in using the three-phase procedure that has been described previously.
Podlesnik and Shahan (2010) proposed an extension of the augmented extinction model that accounted well for the extinction and relapse from a number of experiments. The extended behavioral momentum model is,

\[
\log\left(\frac{B_2}{B_0}\right) = \frac{-t(mc + ndr)}{r^n}
\]

(2)

where all terms are as in Equation 1, with the addition of two parameters \(m\) and \(n\) that lessen the disruptive impact of \(c\) and \(d\) (which represent breaking the response-reinforcer contingency and removal of reinforcement, respectively), when the relapse manipulation occurs. During extinction, \(m\) and \(n\) are equal to 1 such that the disruptors operate as they typically do in the augmented extinction model. When the experimental conditions change from those previously present during extinction, \(m\) and \(n\) take values less than 1, thereby characterizing relapse as a reduction in the disruptive impact of extinction.

A Model of Resurgence

Despite the successes of Podlesnik and Shahan's relapse model, the application of Equation 2 to resurgence revealed some shortcomings. First, the values of \(c\) produced by the least squares regression fit to data were unusually large compared to typical extinction values of \(c\). In other words, when alternative reinforcement was introduced, extinction happened faster and the \(c\) parameter was being inflated in order to account for behavior that was eliminated more quickly. Because the disruptive impact of breaking the response-reinforcer contingency is likely no greater in resurgence, \(c\) should not have changed, and the model was clearly missing an important variable.
Shahan and Sweeney (2011) updated the relapse model and suggested that the missing variable is alternative reinforcement rate. The resulting resurgence-specific model is

\[
\log\left(\frac{B}{B_0}\right) = \frac{-t(kR_o + c + dr)}{(r + R_o)^k}
\]

where terms are as in Equation 1, \(R_o\) is alternative reinforcement rate and free parameter \(k\) scales the disruptive impact of alternative reinforcement. Equation 3 allows alternative reinforcement to play two roles. First, in the numerator, alternative reinforcement serves as a disruptor. The higher the rate of alternative reinforcement, the more quickly the target behavior will be decreased in extinction. However, the inclusion of \(R_o\) in the denominator allows alternative reinforcement to strengthen the stimulus-reinforcer relationship of the context in the same manner as baseline reinforcement.

When alternative reinforcement is removed, \(R_o\) in the numerator decreases to zero, and thus Equation 3 predicts an increase in the target response; that is, relapse, because of the decrease in disruption. The degree of resurgence depends on the strength of the stimulus-reinforcer relationship determined by the denominator—the additive value of baseline reinforcement rate and alternative reinforcement rate during extinction.

**Modeling Resurgence Phenomena**

Equation 3 accounts well for known findings in the resurgence literature and fits the existing data well. One such finding is that longer exposure to extinction plus alternative reinforcement generates less resurgence following alternative reinforcement removal. Leitenberg and colleagues (1975, Experiment 4) used four groups of rats with
equal length of baseline and reinforcement frequency in order to test the effect of 3 days, 9 days, and 27 days of exposure to extinction plus alternative reinforcement before its ultimate removal and also compared them with an extinction control that never received alternative reinforcement. The resurgence effect was largest in the group with only 3 days exposure to extinction plus alternative reinforcement, with those rats that received 9 days demonstrating a visually (but not statistically) significant decrease in resurgence. Rats with 27 days of alternative reinforcement did not show statistically significant responding during Phase III when compared with the extinction control group; consistent with model predictions that resurgence should decrease with lengthier exposures to extinction plus alternative reinforcement.

Equation 3 captures the effect of extended exposure to extinction on subsequent resurgence through its use of time since baseline \((t)\) as a factor that increases the impact of disruptors over time. As time in extinction and exposure to alternative reinforcement increase, \(t\) becomes larger, and consequently the larger numerator predicts continued decreases in behavior until the removal of alternative reinforcement has less impact and results in very little resurgence. Figure 1 uses typical model parameter values to illustrate this prediction using the exponentiated version of Equation 3. The exponentiated version avoids the use of log-transformed data and allows the inclusion of zero values common in response elimination procedures. The exponentiated version is:

\[
\frac{B_t}{B_0} = 10^{-\frac{-t(kR_e+c+dr)}{(r+R_w)^h}} \tag{4}
\]

where all terms are as in Equation 3. Another empirically supported prediction of Equation 3 is that resurgence should decrease across repeated exposures to extinction
Figure 1. Simulation produced by Equation 4 using baseline reinforcement rates of variable-interval (VI) 60 seconds and alternative reinforcement rate of VI 30 seconds and increasing time in extinction with exposure to alternative reinforcement as presented in Shahan and Sweeney (2011).
with alternative reinforcement when extinction integrity is maintained. In other words, when subjects are not returned to baseline contingencies of reinforcement, it continues to grow (and consequently relapse diminishes) with each implementation and removal of alternative reinforcement. This suggests that the first removal of alternative reinforcement would produce a large increase in target responding, but when alternative reinforcement is reintroduced and removed a second time, resurgence should be smaller. Figure 2 simulates expected results under these circumstances as predicted by Equation 3. There are two investigations that explicitly investigated and support this prediction. Wacker et al. (2011) provide an applied example of successive conditions of alternative reinforcement implementation and removal. The authors alternated conditions of extinction plus alternative reinforcement (from functional communication training) with conditions of typical extinction of problem behavior of children in their home environments. They found, across eight participants, that the magnitude of resurgence decreased with each removal of alternative reinforcement (i.e., repeated extinction in the absence of functional communication training).

Another study by Quick and colleagues (2011) investigated resurgence of cocaine seeking in rats following the removal of alternative food reinforcement for nose pokes during extinction. They introduced and removed alternative food reinforcement twice while keeping the extinction of cocaine seeking in place. Relapse during the second resurgence test was significantly smaller than the first resurgence test, as predicted by the simulation in Figure 2. These two studies help to demonstrate the generality of the model’s behavioral momentum principles when applied to resurgence across very different circumstances.
Figure 2. Simulation produced by Equation 4 using baseline reinforcement rates of variable-interval (VI) 60 seconds and alternative reinforcement rate of VI 30 seconds and repeated introductions ($R_a$) of alternative reinforcement as presented in Shahan and Sweeney (2011). Reprinted with permission from JEAB.

The most pertinent resurgence phenomenon to the present investigation is the effect of different alternative reinforcement rates on extinction and relapse. Equation 3 allows for the effects of rich versus lean alternative reinforcement rate on extinction and resurgence. That is, while rich rates of alternative reinforcement eliminate target responding more quickly than do lean rates, subsequently removing rich rates produces greater relapse. Leitenberg and colleagues (1975, Experiment 3) examined extinction and relapse of pigeons with equal baseline rates of reinforcement that were then treated with rich, lean, or no alternative reinforcement during extinction. The lean alternative reinforcement group showed no statistically significant relapse following the removal of alternative reinforcement, but extinction was much slower than the rich alternative.
reinforcement group and was not statistically different from the group that received no alternative reinforcement. Figure 3 shows response elimination and relapse for the rich and lean groups as well as the fit of Equation 3 to the data. These findings, and Equation 3, suggest that it is the removal of alternative reinforcement that produces resurgence and the stimulus-reinforcer relationship of the context that determines the degree of relapse.

**Thinning Alternative Reinforcement**

Equation 3 suggests that resurgence could be reduced by using lean rather than rich rates of alternative reinforcement. Leitenberg and colleagues (1975, Experiment 3) showed that while a lean rate of alternative reinforcement produced little resurgence, it also took longer to eliminate the target response. These data suggest that for very dangerous problem behavior, for which immediate reduction is important, a lean schedule of alternative reinforcement may not be plausible. Another possibility would be to begin with a rich schedule of alternative reinforcement and gradually thin (i.e., reduce) it so that only a very lean rate is removed in the final phase. Schedule thinning is common practice in applied settings before the completion of treatment in order to make treatment more practical outside the clinic (e.g., Hagopian, Contrucci, Kuhn, Long, & Rush, 2005; Hagopian, Toole, Long, Bowman, & Lieving, 2004).

On the other hand, current basic (Lieving & Lattal, 2003) and applied data (e.g., Fisher, Thompson, Hagopian, Bowman, & Krug, 2000; Hagopian, Fisher, Sullivan, Acquisto, & LeBlanc, 1998; Hagopian et al., 2001; Volkert et al., 2009) indicate that downward shifts in alternative reinforcement rate typically produce resurgence of the
**Figure 3.** Results from Leitenberg et al., (1975, Experiment 3) and a least squares regression fit of Equation 4 to these data. Circles represent obtained data and the dotted and solid lines represent model predictions with the given values of $c$ and $k$. $a$, $d$ and $b$ are fixed to .001 and .5, respectively. Y-axis displays proportion of baseline response rates and X-axis shows sessions since baseline. Alternative reinforcement was removed after the tenth session of extinction. Reprinted with permission from JEAB.

Target behavior. Increases in the target behavior are common observances during alternative reinforcement thinning (Hagopian et al., 1998, Kahng, Iwata, DeLeon, & Wallace, 2000; Lalli, Casey, & Kates, 1995). If increases in the target problem behavior occur during the course of schedule thinning, the high rate of alternative reinforcement is usually re-implemented and thinning attempted again later in treatment (e.g., Hanley et al., 2001). This makes sense from both an intuitive standpoint and from the perspective of our quantitative model. Even though Equation 3 predicts that any reduction in alternative reinforcement rate should result in an increase in the target response, that effect should decrease with longer exposure to extinction plus alternative reinforcement.
This is a result of the role of \( t \) (time in treatment) in Equation 4, as illustrated in the model simulations displayed in Figures 1 and 2.

There are several thinning techniques used in the applied literature. One technique is to create a multiple schedule that contains one component in which alternative reinforcement is available and one component in which it is not (Hanley et al., 2001; Hagopian et al., 2005). Another possibility is to continue to reinforce each alternative response, but attempt to train delay tolerance by increasing the delay between the response and the reinforcer (Hanley et al., 2001, Hagopian et al., 1998). A third technique is to immediately reinforce the alternative response, but decrease availability of the alternative response materials, such as a functional communication card (Roane, Fisher, Sgro, Falcomata, & Pabico, 2004). A fourth technique is to increase the response requirement if the alternative response is reinforced on a ratio schedule (Lalli et al., 1995; Volkert et al., 2009). Even though these techniques differ with respect to response-reinforcer contingency, they all reduce the rate of alternative reinforcement over time.

Because behavioral momentum theory argues that neither the source of reinforcement nor the response-reinforcer contingency are the driving force behind the persistence of behavior, the predictions of Equation 3 during treatment lapses should still apply to these schedule-thinning techniques. Even so, the variations in alternative reinforcement thinning across laboratory, participant, and treatment type impose extraneous variables that may make generalization difficult. It may, therefore, be useful to examine alternative reinforcement thinning in an experimental setting in order to compare the effects of rich, lean, thinning, and no alternative reinforcement on response elimination and resurgence.
CHAPTER III
PURPOSE

The purpose of the present experiment was to compare rich, lean, and thinning schedules of alternative reinforcement and use the results to provide an empirical basis for the evaluation of Equation 3. In order to assess the relative advantage of thinning alternative reinforcement compared to other treatments, it is necessary to compare both rate of response elimination and the degree of relapse for subjects that receive fixed rich, fixed lean, and no alternative reinforcement (see Figure 4 for Equation 4's predictions). These groups allowed comparison of target response elimination across, rich, lean, and thinning alternative reinforcement relative to each other and the control group.

Equation 4 has never been used to predict performance during schedule thinning. There are two clear approaches one might take to incorporate the effect of thinning alternative reinforcement into Equation 3: adding reinforcement rates experienced across temporal epochs or averaging them. Because the current model adds alternative reinforcement rate to baseline reinforcement rate, the most straightforward extension would be to add each rate of reinforcement to the denominator after the organism has experienced it and to change $R_a$ in the numerator to reflect the alternative reinforcement rate in effect for that session. However, that might result in an unusually large value of the denominator, thus inflating the stimulus-reinforcer relationship. A better approach might be to average all rates of reinforcement experienced in the context. Averaging and adding alternative reinforcement rates in the denominator of Equation 3 have very different predicted outcomes (as seen in Figure 5). Table 1 provides a mathematical illustration of adding versus averaging alternative reinforcement rate using Equation 3.
Figure 4. Predictions of Equation 4 for the fixed rich, fixed lean, and control group. Predictions are based on the same values of $c$ and $k$ obtained in the fit to Leitenberg et al. (1975, Experiment 3). Y-axis displays proportion of baseline response rates and X-axis shows sessions since baseline. Alternative reinforcement is removed after the tenth session of Phase II.

with the proposed experimental parameters. The pattern of response elimination data in the thinning group will be important—similarity to the additive, average or neither prediction of Equation 3 will inform our characterization of the effects of thinning alternative reinforcement during Phase II.

Because treatments using alternative reinforcement often involve training socially appropriate responses, it is not only the rate of the target behavior that concerns us but also the rate of the alternative response. Schedule thinning is often motivated by the undesirably high rate of alternative responding produced by fixed rich schedules of alternative reinforcement (e.g., Hanley et al., 2001). Thus, fixed-lean to thinning-
Figure 5. Predictions of Equation 4 using additive or average reinforcement rates in the denominator. Predictions are based on the same values of c and k obtained in the fit to Leitenberg et al. (1975, Experiment 3). Y-axis displays proportion of baseline response rates and X-axis shows sessions since baseline. Alternative reinforcement is removed after the tenth session of Phase II.

Schedules might produce a more reasonable alternative response rate (depending on the utility of the alternative response). The persistence of alternative responding in Phase III (i.e., treatment lapse) could offer comparative advantages or disadvantages of alternative reinforcement schedule during Phase II (i.e., treatment). According to behavioral momentum theory, the group with the richest rate of alternative reinforcement during Phase II would likely be the most resistant to disruption during Phase III. Given the potential impact of alternative reinforcement schedule on both target and alternative response during extinction and resurgence, it is important to examine the effects of schedule thinning on resurgence in an experimental setting in order to inform both our
Table 1

Averaging and Adding Alternative Reinforcement in Equation 3

<table>
<thead>
<tr>
<th>Method</th>
<th>Phase II Day 1</th>
<th>Phase II Day 10</th>
</tr>
</thead>
</table>
| Average | \[
\log \left( \frac{B_t}{B_0} \right) = \frac{-1 \times (k \times 360 + c + d \times 80)}{(80 + 360 \times \frac{1}{2})^b}
\] | \[
\log \left( \frac{B_t}{B_0} \right) = \frac{-10 \times (k \times 36 + c + d \times 80)}{(80 + 360 + 180 + 120 + 90 + 72 + 60 + 51.43 + 45 + 40 + 36 \times \frac{1}{11})^b}
\] |
| Add | \[
\log \left( \frac{B_t}{B_0} \right) = \frac{-1 \times (k \times 360 + c + d \times 80)}{(80 + 360)^b}
\] | \[
\log \left( \frac{B_t}{B_0} \right) = \frac{-10 \times (k \times 36 + c + d \times 80)}{(80 + 360 + 180 + 120 + 90 + 72 + 60 + 51.43 + 45 + 40 + 36)^b}
\] |

Note. Equation 3 is shown below for ease of reference. Baseline reinforcement rate is 80 per hour (VI 45 s, \( r = 80 \)). Alternative reinforcement rate as a disruptor in the numerator changes from 360 per hour (VI 10 s, \( R_a = 360 \)) to 36 per hour (VI 100 s, \( R_a = 36 \)). Only the alternative reinforcement rate for that session is included in the numerator. Each session’s alternative reinforcement rate is either averaged together with \( r \) in the denominator or added to \( r \) in the denominator.

\[
\log \left( \frac{B_t}{B_0} \right) = \frac{-t(kR_a + c + dr)}{(r + R_a)^b}
\]  \hspace{1cm} (3)
quantitative theory of resurgence and perhaps provide the first step to translate this experiment's findings to applied behavioral treatments.
CHAPTER IV

METHOD

Design

This experiment used a mixed repeated measures group design with four groups, one for each level of the betwee-subjects independent variable (alternative reinforcement rate during Phase II). One group experienced alternative reinforcement thinning (Thinning), a second group received fixed rich alternative reinforcement (Rich), a third group received fixed lean alternative reinforcement (Lean), and finally the control group (Control) received no alternative reinforcement during Phase II. The groups and experimental phases are shown in Table 2. Because the dependent variables (1) target response rate and (2) alternative response rate were examined across Phases II and III, there is an additional within subjects independent variable of session.

Subjects

Subjects consisted of 32 experimentally naïve male Long-Evans rats purchased from Charles River (Portage, MI, USA) commercial laboratory animal supplier. The rats were approximately 71-80 days upon arrival at the Utah State University Laboratory Animal Research Center (LARC). Rats were housed in a temperature controlled colony room with a 12h light, 12h dark cycle with lights on at 7:00 am. Subjects were individually housed with free access to water while not in the experimental session. Each day, subjects were run in groups of four. Each rat experienced his experimental session at approximately the same time from one day to the next. They were maintained at
Table 2

Summary of Experimental Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Phase I</th>
<th>Phase II</th>
<th>Phase III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thinning</td>
<td>A: VI 45 s</td>
<td>A: EXT B: Daily 10 s increases in VI 10-100 s</td>
<td>A: EXT B: EXT</td>
</tr>
<tr>
<td>Rich</td>
<td>A: VI 45 s</td>
<td>A: EXT B: VI 10 s</td>
<td>A: EXT B: EXT</td>
</tr>
<tr>
<td>Lean</td>
<td>A: VI 45 s</td>
<td>A: EXT B: VI 100 s</td>
<td>A: EXT B: EXT</td>
</tr>
<tr>
<td>Control</td>
<td>A: VI 45 s</td>
<td>A: EXT B: EXT</td>
<td>A: EXT B: EXT</td>
</tr>
</tbody>
</table>

Note. Description of experimental conditions across groups where A is the target lever press, B is the alternative nose poke, and EXT means no food available for a response. Group Thinning received alternative reinforcement decreases, group Rich received fixed rich alternative reinforcement, group Lean had fixed lean alternative reinforcement, and group Control did not receive alternative reinforcement.

approximately 80% of their free feeding weight using supplemental, immediately postsession feedings. Animal housing and care was conducted in accordance with the regulations of the Utah State University Institutional Animal Care and Use Committee (IACUC; protocol #1098). From beginning to end, the study contained 3 cohorts of rats. The first cohort (n = 8) began in June, 2011, the second cohort (n=8) began in August, 2011, and the third cohort (n = 16) began in October, 2011.

Apparatus

Experimental sessions took place in four Med Associates modular rat operant chambers (30 cm x 24 cm x 21 cm) enclosed in sound-attenuating cubicles. Each chamber contains two aluminum response walls and two Plexiglas walls. On response wall is a curved nose poke wall containing five evenly spaced 2 cm x 2 cm apertures. The apertures are equipped with lights to signal an active hole and photobeams to detect nose entries. Only the leftmost nose poke aperture was used in this experiment. The
opposite wall houses the food receptacle (5 cm x 5 cm) centered between two retractable levers. Each food pellet was a 45 mg grain-based pellet (Bio-Serv, dustless precision pellet, Frenchtown, New Jersey, USA). Food pellet deliveries were accompanied by an audible click, and the food magazine light was illuminated for 3 s, but all other lights in the chamber (if any were on) were turned off for 3 s. This 3-s interval for food consumption did not count toward session time or the variable interval timer. No house light was used in order to make the nose poke light more salient during Phases II and III. Above each lever is a circular opaque light that was illuminated above the target response throughout Phases I, II, and III. The location of the target lever (on the left or on the right) was counterbalanced across subjects.

**Procedure**

**Magazine Training**

Prior to Phase I, subjects experienced food magazine training consisting of 30-min sessions of variable-time (VT) 60 s food pellet deliveries. Both the left and the right lever were extended into the chamber (but not illuminated). Presses to the levers were recorded but had no programmed consequences. Magazine training consisted of at least two sessions, and was continued, if necessary, until the rat consumed all of the pellets. Magazine training lasted for no more than four sessions for all subjects.

**Phase I**

Immediately following magazine training, subjects moved to a baseline (Phase I) for a fixed time of 10 sessions. During Phase I, a press to the target lever produced food
on a variable-interval (VI) 45 s schedule across all groups. That is, pressing the target lever produced food after the chosen interval had elapsed. Each interval was randomly selected (without replacement) from exponential distribution of ten intervals, the average of which was 45 s (Flesher & Hoffman, 1962). Presses on the inactive lever, nose-poke entries to any of the nose poke apparatuses, as well as head entries into the food magazine were recorded but produced no programmed consequences. At the end of Phase I, average target response rates were calculated for the last five days of baseline. Rats were then assigned to groups in order to make average Phase I response rates approximately equal across groups.

**Phase II**

During Phase II, four things were true for all groups (a) the target lever remained available and illuminated, but pressing it no longer produced food (i.e., extinction of the target response); (b) the inactive lever and nose poke responses were also available and had no programmed consequences; (c) the alternative response nose poke was illuminated; and (d) Phase II lasted a fixed time of 10 sessions. For the Rich group, poking in the illuminated nose poke (the alternative response) produced food on a VI 10 s schedule throughout Phase II. For the Lean group, the alternative response produced food on a VI 100 s schedule throughout Phase II. For those rats in the Thinning group, the alternative response produced food on a VI 10 s on the first day of Phase II, and each subsequent day saw increases in the VI by 10 s per day such that on the tenth and final session of Phase II, the alternative response produced food on a VI 100 s. Rats in Group 4 (Control) served as an extinction control in which the nose poke was illuminated but poking never produced food.
Phase III

During the third phase, both the target response and the alternative response were illuminated but neither produced food. This was true across all groups. Phase III lasted for 4 days.

Justification

The VI schedules for baseline reinforcement for all groups and alternative reinforcement for the fixed rich group were adopted from Podlesnik and Shahan (2009). The VI 100 s of the Lean group is a richer schedule than the fixed lean group of Leitenberg et al. (1975, Experiment 3), because one could argue that the very lean schedule of VI 240 s would cause the animals to undergo considerable extinction before encountering alternative reinforcement. Furthermore, the VI 240 s group showed no statistically significant difference from control group that received no alternative reinforcement.
CHAPTER V

RESULTS

Phase I

One subject was excluded from the experiment after failing to acquire the target response. In all other subjects, acquisition of the target response proceeded normally. After the final session of Phase I, average target response rate (using the last 5 days) was calculated for each subject. Subjects were then randomly assigned to one of the four experimental groups, with the constraint that average target response rates not differ reliably between groups. A one-way ANOVA comparing Phase I (last 5 days) target response rates confirmed there was not a significant difference between groups, $F(3, 27) = 0.005, p = .99$. The leftmost panel of Table 3 displays the mean target response rate, alternative response rate (nose poke 1), inactive response rate, response rate in the other nose pokes (nose pokes 2-5) as well as obtained food rate for the last 5 days of Phase I across groups.

Phase II

Group means for target, alternative, inactive and other nose poke response rates as well as obtained food rates averaged across all of Phase II can be found in the center panel of Table 3. Figure 6 displays mean target response rates (with standard error) for each group across the 10 days of Phase II. Target responding decreased the most rapidly in the Rich group, and target response rates in this group stayed relatively low throughout Phase II. Although the Lean group did see a decrease in target response rate during
Table 3

Mean Target, Alternative, Inactive, and Other Nose Poke Response Rates, and Obtained Food Rates During Phases I, II, and III

<table>
<thead>
<tr>
<th>Group</th>
<th>Phase I Responses per minute</th>
<th>Phase II Responses per minute</th>
<th>Phase III Responses per minute</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Target</td>
<td>Alt</td>
<td>Inactive</td>
</tr>
<tr>
<td>Thinning</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>13.24</td>
<td>0.02</td>
<td>1.23</td>
</tr>
<tr>
<td>(SD)</td>
<td>(8.70)</td>
<td>(0.02)</td>
<td>(1.13)</td>
</tr>
<tr>
<td>Rich (n = 8)</td>
<td>13.51</td>
<td>0.02</td>
<td>0.74</td>
</tr>
<tr>
<td>Mean</td>
<td>(4.71)</td>
<td>(0.02)</td>
<td>(1.11)</td>
</tr>
<tr>
<td>Lean (n = 8)</td>
<td>13.31</td>
<td>0.03</td>
<td>1.42</td>
</tr>
<tr>
<td>Mean</td>
<td>(7.14)</td>
<td>(0.03)</td>
<td>(1.24)</td>
</tr>
<tr>
<td>Control (n = 7)</td>
<td>13.63</td>
<td>0.04</td>
<td>0.88</td>
</tr>
<tr>
<td>Mean</td>
<td>(7.45)</td>
<td>(0.06)</td>
<td>(0.44)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Phase II, it was a more gradual decrease and target response rates did not ultimately decrease to a low level that was comparable to the Rich group. On the first day of Phase II, when the Thinning group and the Rich group experienced the same condition, target response rates of the Thinning group and the Rich group were approximately equal. However, the subsequent daily VI increases slowed the rate of target response decrease in the Thinning group such that the mean target response rate did not markedly change from session 2 to session 10 of Phase II. When the Thinning group experienced the same VI for the alternative response as the Lean group, their target response rates were approximately equal. The Control group showed faster decreases in the target response than the Lean group (but slower than the Rich group), and ultimately target response rates in the Control group were approximately equal to the Rich group and lower than the
Thinning group and the Lean group. A repeated-measures ANOVA returned significant main effects of Group, \(F(3, 27) = 7.96, p < .001\), and Session in Phase II, \(F(9, 243) = 51.18, p < .001\). The test also showed Group x Session interaction, \(F(27, 243) = 2.22, p < .001\), capturing that the pattern of target response rate across Phase II was different across groups.

Figure 7 displays mean alternative response rates (with standard error) for each group across Phase II. The Rich group showed a steady increase in alternative response rate across Phase II. The Lean group acquired the alternative response more slowly than the Rich group, and ultimately (on the final day of Phase II) the Lean group mean alternative response rate was less than half the alternative response rate of the Rich group. When the Thinning group experienced the same VI for the alternative response as the Rich group (on the first day of Phase II) their alternative response rate was approximately equal to the Rich group. Although the alternative response rate in the Thinning group was higher than the Lean group in the beginning of Phase II, the mean alternative response rate did not increase substantially from session 2 to session 10 of Phase II. By the end of Phase II, the Thinning group showed similar alternative response rates to the Lean group. Alternative response rates for the Control group remained negligible throughout Phase II. A repeated measures ANOVA showed significant main effects of Group [\(F (3, 27) = 10.92, p < .001\)] and Session in Phase II [\(F (9, 243) = 21.18, p < .001\)] as well as a Group x Session interaction [\(F (27, 243) = 7.31, p < .001\)] on alternative response rate.
Figure 7. Mean and standard error of alternative response rate for each group across Phase II.

A one-way ANOVA comparing the target response rate across groups on the last day of Phase II revealed a significant difference between groups, $F(3, 27) = 6.35, p < .01$. A follow-up analysis using Tukey’s Multiple Comparison Test further showed that the Lean group and the Thinning group had significantly higher mean target response rates than both the Rich group and the Control Group. However, the Thinning group was not significantly different from the Lean group and the Rich Group was not significantly different from the Control Group. Another one-way ANOVA comparing alternative response rate across groups on the last day of Phase II revealed a significant difference between groups, $F(3, 27) = 9.60, p < .001$. Tukey’s Test further showed that the Rich group had significantly higher mean alternative response rates than the Thinning group,
the Lean group, and the Control Group. There were not any statistically significant
differences in alternative response rate between the Thinning group, the Lean group, and
the Control group.

Phase III

Figure 8 displays the mean target response rate on the last day of Phase II
compared to the first day of Phase III for each group. The Rich Group saw a statistically
significant increase from the last day of Phase II to the first day of Phase III \([t(7) = 3.65,\]
\(p < .01\]). Neither the Lean group, \(t(7) = 1.09, p = .31\), nor the Thinning group, \(t(7) =
0.57, p = .58\), nor the Control group, \(t(6) = 1.33, p = .23\), showed a significant difference
between the final day of Phase II and the first day of Phase III. A one-way ANOVA
comparing the target response rate across groups on the first day of Phase III revealed a
significant difference between groups, \(F(3, 27) = 4.18, p < .05\). A subsequent Tukey’s
Test showed that the only statistically significant difference was between the Rich group
and the Control Group; however the data are visually ordered such that Rich > Thinning
> Lean > Control.

Figure 9 displays the mean alternative response rates for each group on the last
day of Phase II and the first day of Phase III. A one-way ANOVA comparing
alternative response rate across groups on the first day of Phase III revealed a significant
difference between groups, \(F(3, 27) = 10.7, p < .001\). Tukey’s Test showed that the Rich
group had significantly higher alternative response rates than the Thinning Group, the
Lean Group, and the Control group. However, there were no significant differences
between the Thinning group, the Lean group, and the Control group.
Figure 8. Mean and standard error of target response rate for each group on the last day of Phase II and the first day of Phase III.

Given the visual difference, but not statistically significant difference between group means for target and alternative response, individual subject data on the last day of Phase II and the first day of Phase III are displayed in Figure 10 (target response rate) and Figure 11 (alternative response rate) to look for overall tendencies that may be obscured by the calculation of mean response rates. The individual subject data suggest that the target response rate on the last day of Phase II was indeed generally lower in the Rich group and the Control group, whereas an elevated, wider range of target response rates was observed in the Lean and Thinning groups. Target response rates on the first day of Phase III were comparable in the Rich, Lean, and Thinning groups, with the visually higher mean rate in the Rich group likely being the result of one subject with a particularly high target response rate on the first day of Phase III. The change in target
response rate from the last day of Phase II to the first day of Phase III is markedly different across groups. In the Rich group, all subjects increased target responding on the first day of Phase III relative to the last day of Phase II. In the Lean group and the Thinning group, some subjects increased, some decreased, and some stayed approximately the same. In the Control group, some subjects decreased and others stayed approximately the same.

Individual alternative response rates were also considered (see Figure 11). Three subjects in the Rich group had very high alternative response rates, but the remaining 5 subjects responded similarly to the Lean and Thinning groups. Therefore, it is likely that the statistically significant difference in alternative response rate obtained on the last day of Phase II is the result of the 3 rats that had very high alternative response rates. Clearly the groups that received alternative reinforcement (Rich, Lean, and Thinning) showed
Figure 10. Individual subject data for the target response on the last day of Phase II and the first day of Phase III. Each subject is depicted by its two data points and a connecting line.

Individual alternative response rates were also considered (see Figure 11). Three subjects in the Rich group had very high alternative response rates, but the remaining 5 subjects responded similarly to the Lean and Thinning groups. Therefore, it is likely that the statistically significant different in alternative response rate obtained on the last day of Phase II is the result of the 3 rats that had very high alternative response rates. Clearly, the groups that received alternative reinforcement (Rich, Lean, and Thinning) showed substantially higher alternative response rates than the control group, even though for the Lean and Thinning group this difference was not statistically significant. On the first
Figure 11. Individual subject data for the alternative response on the last day of Phase II and the first day of Phase III. Each subject is depicted by its two data points and a connecting line.

day of Phase III, subjects in the rich group showed similar alternative response rates as the Lean and Thinning groups, with the exception of 1 subject.

**Model Fits**

The exponentiated version of Equation 3 (i.e., Equation 4) was used in order to allow zero values, avoid log-transformed data and to make the parameter values comparable to those obtained in previous fits to resurgence data (i.e., Shahan & Sweeney, 2011). For all of the following fits, parameters $b$ and $d$ were fixed to values of 0.5 and
0.001, respectively, as in Shahan and Sweeney (2011). Only parameters $k$ and $c$ were free to vary.

First, a least squares regression fit of Equation 4 using Frontline Systems Solver for Mac Excel 2008 was conducted on the data obtained during Phases II and III for the Rich, Lean, and Control groups (the groups with constant schedules of alternative reinforcement). These groups were fit first because Equation 4 has been fit to constant schedules of alternative reinforcement previously with no adjustments (Shahan & Sweeney, 2011). Before solving, the starting value of parameter $k$ was .05 and the starting value of $c$ was .89 (the values obtained in the fit of Leitenberg et al., 1975, Experiment 3). Figure 12 illustrates the obtained data (open and closed circles, open triangles) as well as the obtained parameter values and model fits (dotted, solid, and dashed lines). Although the Rich group demonstrated faster response elimination and greater resurgence than either the Lean or the Control group (as predicted by Equation 4) and an adequate $R^2$ value of .88 was obtained, there are several major inconsistencies with model fits and obtained data. First, the model predicts that the Control group should have slower response elimination than the Lean group, but in fact the Control group showed faster response elimination than the Lean group. Furthermore, the model systematically underpredicts later Phase II data in both the Rich group and the Lean group. Finally, the model systematically underpredicts target responding in Phase III for the Lean group.

Next, the two methods of incorporating reinforcement rates into the denominator of Equation 3 outlined previously (i.e., adding each reinforcement rate experienced and averaging them; see Table 1) were compared by fitting each technique to the obtained
Figure 12. Least squares regression fit of Equation 4 to data from the Rich, Lean, and Control groups. Single filled circles (Rich), open circles (Lean), and open triangles represent obtained data, whereas the solid (Rich), dotted (Lean), and dashed (Control) lines represent the model predictions.

Data in the Thinning group as seen in Figure 13. For both methods, the starting value of \( c \) was 1.17 and the value of \( k \) was 0.01—the values obtained in the fit to the constant schedules. The additive denominator approach accounted for more of the variance in the data (\( R^2 = 0.90 \)) than did the averaged denominator approach (\( R^2 = 0.87 \)). Both approaches fall short in terms of visual fit during early Phase II data. Importantly, the additive denominator approach does not predict a large increase from Phase II to Phase III, whereas the averaged approach does. Because a large increase in target responding was not observed in the obtained data and the additive approach accounted for more variance, the additive approach was determined to be the superior method of the two proposed.
Figure 13. Least squares regression fit of Equation 4 to data from the Thinning group using averaged and additive denominators. Single data points represent obtained data for the Thinning group, whereas the dashed lines represent the model predictions.
a single value of \( r \) was also added in the denominator. For example, on day 1 of Phase II, 11 reinforcement rates were added in the denominator for each group: one rate for each day of Phase I and one rate for the first day of Phase II (for the Control group, the value added for the first day of Phase II was 0). Figure 14 displays the obtained data and model fits. Although the variance accounted for increased by 1%, the additive technique did nothing to ameliorate the discrepancies seen with the original application of Equation 4 to constant schedules of alternative reinforcement. For example, the model still predicts that response elimination should occur more rapidly in the Lean group than in the Control group (the opposite was true). The model also continues to systematically underpredict later Phase II data in the Rich and Lean groups as well as underpredicted target response rate during Phase II for the Lean group and the Thinning Group.
Figure 14. Least squares regression fit of Equation 4 using an additive denominator approach. Single data points represent obtained data, whereas the lines represent the model predictions.
CHAPTER VI
DISCUSSION

Both theoretical and applied concerns motivated the comparison of rich, lean, thinning, and no alternative reinforcement during response elimination and relapse. The results of the present study present important implications both for the quantitative model of resurgence put forward by Shahan and Sweeney (2011) as well as for practitioners and researchers using alternative reinforcement in applied settings. One purpose of this experiment was to present a challenge to Equation 3, evaluate its performance, and posit explanations for its shortcomings. In addition, the pattern of results is interpreted with respect to potential applied importance.

Evaluation of Model Fits

There are two major inconsistencies with the fits of Equation 3 to the data obtained. First, there is systematic underprediction of later Phase II data in the Rich and the Lean groups. Second, the model predicts that response elimination should happen more slowly in the Control group than in the Lean group, but the opposite is the case in these data. It is true that adding and averaging are only two methods of incorporating reinforcement rates into the denominator of Equation 3, and that there are other possible methods by which one might integrate reinforcement rates across temporal epochs. One method could be to weigh recently experienced reinforcement rates more heavily than more distantly experienced reinforcement rates (e.g., weighted average or an exponentially weighted average). Unfortunately, this would do nothing to solve the problem of underprediction in both the Lean and the Rich groups. If more recent
experienced reinforcement rates are weighed more heavily than distant ones, the
denominator in the Rich group would increase but the denominator in the Lean group
would decrease. A larger denominator value would increase the predicted persistence of
the target response, however, because weighing would not increase the denominator
across all groups, one would not see across the board increases in the predictions for later
Phase II data.

Differentially weighing distantly versus recently experienced weights would also
not solve the problem that the Control group showed faster response elimination than the
Lean group because no matter what, $R_o$ in the numerator is always treated as an additional
source of disruption. Therefore, the model as it currently stands cannot capture that
alternative reinforcement may not always be an effective disruptor. The results of this
experiment, as well as similar investigations suggest that alternative reinforcement may
not always serve as an effective disruptor, especially at low rates.

Looking again to Leitenberg et al. (1975), Experiment 3, which pitted high rates
of alternative reinforcement, low rates of alternative reinforcement and no alternative
reinforcement against one another for 15 days during Phase II, there is some ambiguity in
the later Phase II data. Although the first 4 days of Phase II show that pigeons in the lean
alternative reinforcement group responded less to the target key than the no alternative
reinforcement control group, the remainder of Phase II showed that the lean group
actually responded more than or approximately the same as the no alternative
reinforcement rate control (and at no point were these differences statistically
significant).
In another study, Winterbauer and Bouton (2010) studied the effects of thinning alternative reinforcement Phase II of a resurgence preparation. In their first experiment, rats that had experienced equal baselines of random-interval (RI) 30 s schedules were separated into three groups prior to Phase II. During Phase II, the first group (comparable to this experiment’s Rich group) received an alternative reinforcement rate of a RI 20 s schedule that remained constant throughout Phase II. The second group (similar to this experiment’s Control group) received no alternative reinforcement. The third group (comparable to the Thinning group in this experiment) began Phase II with an alternative reinforcement rate of a RI 20 s, but halfway through each session, beginning the second session of Phase II, the interval was doubled such that the last rate they experienced on the fourth and final session of Phase II was a RI 160. As in the present experiment, the group that experienced thinning alternative reinforcement rates showed elevated target response rates towards the end of Phase II such that target response rates were greater in the thinning group than they were in the extinction control group.

The Winterbauer and Bouton study (2010) is informative in two ways. First, they eliminated several key alternative explanations for why one might see elevated response rates in a group that experienced alternative reinforcement thinning. For example, their second experiment compared a group with gradual thinning (adding a small constant increment adding up to an increase in the average interval by 20 s daily) versus a group that experienced more abrupt, mid-session, 20 s increases in the average interval. They determined that the abruptness of the interval change did not matter, because both groups showed equally high rates toward the end of Phase II, the same pattern as Experiment 1. They also ruled out explanations by adventitious reinforcement of the target response,
reinstatement of the target response by pellet delivery for the alternative, and the
development of target lever presses as a schedule induced interim behavior. Apart from
the elimination of alternative explanations for high rates of the target behavior during
Phase II, the similar pattern of results obtained in Winterbauer and Bouton also speaks to
the generality of this effect across laboratories and procedural variations.

On the other hand, there is a key shortcoming to the experiments in Winterbauer
and Bouton (2010) that render the results of the present study valuable. Their experiment
was designed to test the hypothesis that resurgence is a form of A-B-C renewal, in which
Phase I is considered context A defined by reinforcement for the target response,
response elimination takes place in Phase II (which is considered context B defined by
reinforcement for the alternative), and Phase III is considered a novel context, C, in
which there is no reinforcement. They proposed that resurgence is the result of
contextual renewal when the rat is exposed to the novel context, C. According to this
formulation, if the context is gradually changed from B to C, the rat should be less
susceptible to resurgence. Their hypothesis was that thinning alternative reinforcement
would reduce resurgence. Instead, what they saw was elevated response rates during
Phase II. Although the increase in target response rate from Phase II to Phase III was
less, the high rates of the target response during Phase II are better characterized by early
rather than reduced resurgence. Their interpretation of the results was that each reduction
of alternative reinforcement was enough of a change in context to induce renewal.
However, this interpretation is not consistent with elevated response rates that we have
seen in the Lean group in our experiment. Winterbauer and Bouton did not have a fixed-
low rate group, only a fixed-high rate group, and as such they cannot account for the
overall rate of alternative reinforcement on target response rate. Their thinning group experienced lower rates of alternative reinforcement overall during Phase II, and thus without a fixed-low rate group we cannot tell whether the elevated response rates were a result of gradually changing alternative reinforcement or lower rates in general. Because our results showed elevated target response rates in both the Thinning group and the Lean group, the elevated target response rates were likely not a result of changing contexts because the context in the Lean group was constant across Phase II. A much more simple explanation is that lower rates of alternative reinforcement (such as those experienced by the Thinning and Lean groups in the present study) are less effective at reducing the target response.

Empirically we can see from the present experiment as well as the data in Winterbauer and Bouton (2010) that lean rates of alternative reinforcement are less effective at response elimination than rich rates or even the control group with no alternative reinforcement, but why might this be the case? Winterbauer and Bouton ruled out reinstatement as an explanation for the increased target response rates, which would be evident in an immediate return to the target response following the delivery of alternative reinforcement. They also ruled out adventitious reinforcement, which would be clear if for some reason alternative reinforcement happened to be preceded closely by the target response more often in the groups with thinning rates rather than with high rates. They found that, if anything, there was more opportunity for adventitious reinforcement in the high rate group than in the thinning group. One possibility is that rather than reinstatement, context change, or adventitious reinforcement, the elevated response rates in the lean and thinning groups might be the result of resurgence on a
smaller time scale. The delivery of alternative reinforcement might be initially
disruptive, but after the anticipated interval to reinforcement has elapsed, alternative
reinforcement is no longer perceived as present. In terms of Equation 3, $R_a$ might be
present in the numerator (as a disruptor) immediately after it is delivered only until the
anticipated interval to the next reinforcer has elapsed. For example, in this experiment,
the interval to the next reinforcer following Phase I might be approximately 45 s. In the
Rich group, $R_a$ would be consistently present in the numerator because alternative
reinforcement is nearly always obtained within the anticipated interval to reinforcement,
therefore we would not expect any resurgence. On the other hand, in the Lean group,
alternative reinforcement is introduced but the next interval to reinforcement nearly
always exceeds the rat's anticipated interval to reinforcement. Thus, from the rat's
perspective, alternative reinforcement has been removed, and we ought to expect
resurgence. In the extinction only Control group, alternative reinforcement is never
introduced (therefore never removed) so we should not expect elevated target response
rates after any particular interval, but we should also not expect as much disruption as the
Rich group. Although the model in its current state is not equipped to operate at such as
time scale, investigations of moment-by-moment resurgence may provide an empirical
basis for deciding at what point (and why) alternative reinforcement is no longer an
effective disruptor.

**Applied Implications**

The present study was not only motivated by the evaluation of Equation 3, but
also by the clinical significance of comparing rich, lean, thinning, and no alternative
reinforcement during the treatment of a target response. The pattern of results obtained is both consistent with and relevant to applied behavioral treatments using alternative reinforcement, such as contingency management treatments of substance abuse and functional communication training in individuals with intellectual or developmental disabilities.

For example, the ability of treatments using higher rates of alternative reinforcement to produce greater abstinence from the target response relative to treatments using lean rates of alternative reinforcement is also evident in the contingency management literature (Dallery, Silverman, Chutuape, Bigelow, & Stitzer, 2001; Higgins, Heil, Dantona, Donham, Matthews & Badger, 2006; Petry et al., 2004; Silverman, Chutuape, Bigelow, & Stitzer, 1999; Stitzer & Bigelow, 1984). For example, Higgins et al. (2006) conducted a randomized controlled trial in which there were two groups, one receiving vouchers for cocaine abstinence at a high monetary value and one receiving vouchers at a fourfold lower value. Participants in the high value voucher group achieved better treatment retention, longer periods of continuous abstinence during treatment, and superior abstinence at posttreatment follow-up tests than did participants in the low value group. The fact that the high value voucher group also achieved greater abstinence during follow-up may seem contrary to the present findings that there was a marked increase in the Rich group during Phase III. However, when Higgins et al. (2006) compared participants across condition that achieved similar levels of abstinence during treatment, those in the low value group actually achieved better long-term abstinence than participants in the high value group—consistent with the predictions of Equation 3.
In the present study, although target responding in the Rich group was substantially more suppressed by the end of Phase II (mean proportion of baseline response rates were 6% of baseline on the last day of Phase II in the Rich group) than was target responding in the Lean (22% of baseline) and Thinning (23% of baseline), subjects in the Rich group were much more sensitive to a lapse in treatment—reflected in the increases in target response rate seen in Figure 10. Furthermore, the individual subject data for the alternative response suggest that during treatment, rich rates of alternative reinforcement may engender excessive rates of the alternative response in some subjects (in this experiment illustrated by the three subjects with higher response rates) or comparable rates obtained with Lean and Thinning rates. Also, the individual subject data for the alternative response on the first day of Phase III indicate that for most subjects, alternative response rates were no higher in the Rich group during a treatment lapse than in the Lean and Thinning groups. This probably means that if treatment integrity briefly lapsed in an applied setting, those clients that experienced higher rates of reinforcement for the alternative response would be just as likely to continue to engage in the alternative response and be responding appropriately until treatment is restored as those that experienced lean and thinning rates of reinforcement for the alternative response.

These results carry significant implications for practitioners of differential reinforcement of alternative treatments such as functional communication training, who typically utilize high rates of alternative reinforcement to treat problem behavior. In the beginning of treatment, it is common to follow each alternative response (i.e., a fixed-ratio 1 schedule; FR) with access to the reinforcer (e.g., Hanley et al., 2001; Roane et al.,
2004), and only implement thinning of the alternative reinforcement when a low target response criterion has been met. When alternative reinforcement thinning is explicitly studied in an applied setting (typically with single-subject designs) some results favor schedules of alternative reinforcement thinned from rich schedules (Hagopian et al., 2004), others favor lean schedules of alternative reinforcement from the outset of treatment (Hagopian et al., 2004), and many report increased target response rates during lean and thinned schedules of alternative reinforcement (Hagopian et al., 1998, 2004; Kahng et al., 2000; Lalli et al., 1995).

If high levels of alternative reinforcement during treatment can result in relapse following treatment, whereas low levels of alternative reinforcement are ineffective at producing sufficient abstinence, what are potential strategies for increasing long-term effectiveness of treatment? It is clear from the present experiment, Winterbauer and Bouton (in press), and the increases often found in applied literature during reinforcement thinning (e.g., Fisher et al., 2000) that alternative reinforcement thinning alone is not a sufficient solution. Another strategy is to introduce alternative reinforcement during treatment that does not go away as treatment ends or thinning is introduced.

One method by which applied researchers have found to reduce alternative reinforcement without increasing target response rates is to provide competing stimuli (such as toys) during alternative reinforcement thinning (Hagopian et al., 2005). By explicitly arranging differential reinforcement for the functional communication response, they were able to reduce the target behavior effectively. Then, by introducing competing stimuli (toys) that were not removed during alternative reinforcement schedule thinning, they were able to avoid the increases in problem behavior often seen during
thinning. Providing a competing stimulus during thinning of the alternative reinforcement schedule is, in effect, leaving another form of alternative reinforcement in place while another is being thinned. Mathematically, in Equation 3, $R_a$ in the numerator would not decrease (or turn to zero with the end of treatment) with a competing stimulus present, but rather should include the rate of reinforcement for the competing stimulus. Thus the success of competing stimuli in reducing the rate of target behavior during thinning of alternative reinforcement is also consistent with the predictions of Equation 3.

In contingency management, the therapeutic workplace (e.g., Silverman et al., 2002) could be considered a similar strategy. In this employment-based abstinence program, participants are hired to work in data entry training programs. If payment for work and availability of the therapeutic workplace are contingent upon drug abstinence, cocaine abstinence is improved relative to if the therapeutic workplace is provided independently of drug-use (e.g., Silverman et al., 2007; DeFulio, Donlin, Wong, & Silverman, 2009). For example, in a study by Silverman et al. (2002) evaluating the therapeutic workplace intervention with chronically unemployed, pregnant or recently postpartum women, when therapeutic workplace participants were compared to usual care (no job skills) control conditions, the therapeutic workplace participants achieved twice the abstinence rate as the control participants both during the first 6 months of treatment and for 3 years following the start of treatment. The goal of employment-based treatments and job skills training is more than simply to achieve better abstinence during treatment, but also to provide participants with skills that cannot be taken away as treatment ends. In the terms of Equation 3, such treatment strategies attempt to leave
alternative reinforcement in the numerator, allowing disruption of the target response to continue following the end of treatment.

In contingency management, because extinction of the target response (drug taking) is not in place, the no alternative reinforcement control group in the present experiment is not particularly relevant. However, the fact that extinction with no alternative reinforcement was more effective than lean or thinning alternative reinforcement during treatment is quite relevant to many applied scenarios. If introducing an alternative source of reinforcement sometimes results in higher rates of problem behavior than ordinary extinction, treatments that introduce lean rates of alternative reinforcement could be doing more harm than good. Even though rich rates of alternative reinforcement might be effective during treatment, they are, practically speaking, more difficult to implement and in some cases, more expensive. When treatment lapse occurs (such as in the present experiment) the target behavior may relapse to a higher rate than if alternative reinforcement was never introduced. Adding control conditions that contain no alternative reinforcement, only extinction, to applied research would allow us to see when alternative reinforcement is an effective disruptor and when it may actually make problem behavior more persistent than ordinary extinction.

Ordinary extinction also has the advantage of not requiring an alternative response or alternative reinforcement. In some cases, the alternative response may be a very desirable behavior that can occur at high levels without causing any problems. In other situations, alternative responses (such as verbal requests for a break) may not only be impractical to continue outside of treatment at rich or lean rates of alternative reinforcement, but at lean rates of alternative reinforcement could result in higher
problem behavior than if the problem behavior was simply placed on extinction.

Therefore, the desired level of target response suppression and the appropriateness of the alternative response and alternative reinforcement should be carefully considered before implementing treatment.
CHAPTER VII
CONCLUSION

With respect to the quantitative model of resurgence, using the current rate of alternative reinforcement in the numerator and adding each experienced value in the denominator proved superior to the method of averaging each value together in the denominator. However, fits to the data revealed that the model significantly underpredicts later Phase II data in the Rich and Lean groups. Furthermore, the model always treats alternative reinforcement as a disruptor despite evidence that lean of alternative reinforcement may not always result in faster response elimination than no alternative reinforcement. Future research that provides an empirical basis for when (and why) alternative reinforcement is an effective disruptor and when it is ineffective is necessary before Equation 3 can be further modified.

Rich rates of alternative reinforcement were most effective at response suppression during treatment, but the removal of alternative reinforcement during Phase III caused substantial resurgence. Lean and Thinning rates of alternative reinforcement were less effective at response suppression during Phase II, but less sensitive to the lapse in treatment during Phase III. Potential strategies for reducing resurgence during schedule thinning or following the end of treatment include attempting to include forms of alternative reinforcement that are not removed during thinning or after treatment. The choice to implement rich, lean, thinning or no alternative reinforcement during treatment should depend on the prioritization of (A) desired level of target response suppression, and (B) appropriateness of the alternative response and alternative reinforcement.
REFERENCES


