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Effects of Differential Rates of Alternative Reinforcement on Resurgence of Human Behavior: A Translational Model of Relapse in the Anxiety Disorders

Brooke M. Smith
Utah State University

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EFFECTS OF DIFFERENTIAL RATES OF ALTERNATIVE REINFORCEMENT ON RESURGENCE OF HUMAN BEHAVIOR: A TRANSLATIONAL MODEL OF RELAPSE IN THE ANXIETY DISORDERS

by

Brooke M. Smith

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

MASTER OF SCIENCE in

Psychology

Approved:

Michael P. Twohig, Ph.D.
Major Professor

Timothy A. Shahan, Ph.D.
Committee Member

Gregory J. Madden, Ph.D.
Committee Member

Mark R. McLellan, Ph.D.
Vice President for Research and Dean of the School of Graduate Studies

UTAH STATE UNIVERSITY
Logan, Utah
2015
ABSTRACT

Effects of Differential Rates of Alternative Reinforcement on Resurgence of Human Behavior: A Translational Model of Relapse in the Anxiety Disorders

by

Brooke M. Smith, Master of Science
Utah State University, 2015

Major Professor: Michael P. Twohig
Department: Psychology

Behavioral and cognitive-behavioral psychotherapies utilizing exposure are considered the gold standard in anxiety disorder treatments. Despite their success, relapse remains problematic, especially over long-term follow up periods. Basic researchers traditionally conceptualize the mechanism of exposure as Pavlovian extinction, but this may overlook the important role of operant processes in the treatment and relapse of anxiety. Resurgence, in which a previously extinguished behavior returns following the extinction of another behavior that has replaced it, is a promising model of operant relapse. Nonhuman research on resurgence has shown that, while higher rates of alternative reinforcement result in faster and more comprehensive extinction of target behavior, they also result in greater resurgence. This somewhat paradoxical finding could
have important implications for clinicians treating anxiety, as higher rates of alternative reinforcement may have the unintended side effect of producing greater relapse of avoidance if access to positive reinforcement later becomes unavailable. The current study took a translational approach to investigating the effects of rich and lean rates of alternative reinforcement on extinction and magnitude of resurgence in typically developing humans using a computerized task. Three groups (Rich, \( n = 18 \); Lean, \( n = 18 \); Control, \( n = 10 \)) underwent acquisition of a target response. Target responding was then placed on extinction while varying rates of reinforcement for an alternative behavior were delivered. Resurgence was assessed under extinction conditions for all groups. Results indicated that the rich rate of alternative reinforcement facilitated extinction while the lean rate ultimately had a detrimental effect on extinction. Within groups, Rich and Lean experienced significant resurgence, while Control did not. Effect sizes were large. Between groups, Rich resurged more than Lean and Control. Effect sizes were again large. There was no significant difference in resurgence between Lean and Control. Implications for the treatment of anxiety disorders and future research directions are discussed.

(68 pages)
PUBLIC ABSTRACT

Effects of Differential Rates of Alternative Reinforcement on Resurgence of Human Behavior: A Translational Model of Relapse in the Anxiety Disorders

by

Brooke M. Smith, Master of Science
Utah State University, 2015

Brooke M. Smith, graduate student in the Combined Clinical, Counseling, and School Psychology program at Utah State University, proposed and completed this thesis in partial fulfillment of the requirements of Master of Science in Psychology.

Anxiety disorders are the most common psychological disorders in the world, and they place a substantial burden on society. Although successful psychological treatments for anxiety are available, many people still experience relapse once treatment ends. When consequences that follow a behavior increase its future probability, this is termed “reinforcement.” One common treatment approach for anxiety is to attempt to decrease problematic behavior, such as avoidance, by decreasing its reinforcement, while at the same time increasing reinforcement for healthy replacement behaviors. A phenomenon that basic researchers have used to study relapse in animals is known as “resurgence.” Resurgence occurs when reinforcement of a problem behavior is removed and an alternative behavior is taught. When reinforcement for the alternative behavior is also
removed, the original problem behavior tends to relapse. Previous research with nonhumans has shown that the more frequently reinforcement is delivered for an alternative behavior, the greater relapse a subject will experience if that reinforcement eventually becomes unavailable. This could be counterintuitive to clinicians treating anxiety. However, this aspect of resurgence has yet to be shown in typically developing humans.

The current study investigated the effects of high and low rates of alternative reinforcement on the elimination and relapse of a target behavior in typically developing humans using a computerized task. Three groups received three different rates of reinforcement for an alternative behavior, and resurgence was tested. Results suggest that target behavior decreased the most quickly and completely in those who received higher rates of reinforcement, but this also resulted in more relapse once reinforcement was removed. The implications for the treatment of anxiety disorders and future research directions are discussed.
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Brooke M. Smith
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CHAPTER I
STATEMENT OF THE PROBLEM

Behavioral and cognitive-behavioral (CBT) psychotherapies are considered the gold standard in anxiety disorder treatments and typically result in medium to large effect sizes (Butler, Chapman, Forman, & Beck, 2006; Norton & Price, 2007; Olatunji, Cisler, & Deacon, 2010). Despite these encouraging outcomes, response rates remain relatively low, ranging from less than one-half to two-thirds of those treated with CBT or behavior therapy (Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012). Of those successfully treated, relapse occurs for some during typical follow-up periods of 6 to 12 months (Hiss, Foa, & Kozak, 1994), with some studies showing over half of participants relapsing during long-term follow-up periods of more than one year (Durham, Chambers, Macdonald, Power, & Major, 2003).

Basic and translational researchers have traditionally conceptualized the mechanism of exposure in terms of Pavlovian learning processes, but this approach may overlook the important role of operant processes in the maintenance, treatment, and relapse of anxiety. Anxiety disorders are characterized by avoidance of fear-inducing stimuli, an operant behavior, and recent research shows that toleration of fear, rather than the reduction of it (the goal of Pavlovian extinction procedures), may be the key component to successful exposure therapy (Craske et al., 2008). In addition, there is evidence that residual agoraphobic avoidance could be a major factor contributing to relapse (Craske, Brown, & Barlow, 1991; Ehlers, 1995).
The phenomenon known as “resurgence,” in which a previously extinguished behavior returns following the extinction of another behavior that has replaced it (Lieving & Lattal, 2003), has been used as a model for investigating the elimination and relapse of operant problem behavior in individuals with developmental disabilities following treatment with differential reinforcement of alternative behavior (DRA) (Lieving, Hagopian, Long, & O'Connor, 2004; Lionello-DeNolf, Dube, & McIlvane, 2010; Volkert, Lerman, Call, & Trosclair-Lasserre, 2009; Wacker et al., 2013). Resurgence may also provide a promising laboratory analogue of treatment and relapse within the anxiety disorders. Behavioral and cognitive-behavioral therapies for anxiety disorders often focus on the elimination of avoidance behavior while simultaneously teaching engagement with positively reinforcing life activities as an alternative to avoidance. This process is similar to DRA, with the exception that avoidance behaviors are negatively reinforced, while alternative behaviors are positively reinforced.

In addition, basic animal research on resurgence has shown that, while higher rates of alternative reinforcement result in faster and more comprehensive elimination of the target behavior, they also result in greater resurgence of that behavior once reinforcement has been decreased or removed (Leitenberg, Rawson, & Mulick, 1975; Sweeney & Shahan, 2013; Winterbauer & Bouton, 2012). This finding could have important implications for clinicians treating anxiety, as higher rates of alternative reinforcement, while more effectively reducing undesired avoidance, may have the unintended side effect of producing greater relapse of avoidance if access to positive reinforcement is later reduced or becomes unavailable. Such an effect could be
counterintuitive to clinicians. However, this particular finding within the resurgence literature has yet to be investigated in typically developing humans or with respect to a positively reinforced alternative behavior and its effects on the elimination and relapse of negatively reinforced avoidance behavior.

A better understanding of the effects of varying rates of positive reinforcement of alternative behavior on the elimination and relapse of both positively and negatively reinforced target behavior is necessary to furthering our knowledge of basic processes of elimination and relapse in operant behavior as well as to design treatments that better address the issue of clinical relapse in anxiety disorders. The purpose of the current study was to take a translational approach to investigating the effects of high and low rates of positive reinforcement of alternative behavior on response elimination and magnitude of resurgence of positively reinforced target behavior in humans using a computerized task. The following questions were addressed:

1. Does the loss of reinforcement for a recently reinforced alternative behavior result in the relapse of previously eliminated target behavior in humans?

2. What are the effects of high and low rates of reinforcement for an alternative behavior on the elimination of simultaneously extinguished target behavior in humans?

3. What are the effects of high and low rates of reinforcement for a recently reinforced alternative behavior on the magnitude of relapse of previously eliminated target behavior once alternative reinforcement is removed in humans?
CHAPTER II
REVIEW OF THE LITERATURE

Anxiety Disorders

Anxiety disorders are the most common class of psychological disorders both in the United States (Kessler, Berglund, et al., 2005; Kessler, Chiu, Demler, & Walters, 2005) and worldwide (Kessler et al., 2007). In a review of prevalence studies from 1980 to 2004, one-year and lifetime prevalence rates for all anxiety disorders were estimated at 10.6% and 16.6% respectively (Somers, Goldner, Waraich, & Hsu, 2006). They are also the most economically costly of all psychological disorders, with costs in 1990 estimated between 42 and 47 billion dollars in the United States, or up to 31.5% of the total economic cost of all psychological disorders (Greenberg et al., 1999; Rice & Miller, 1998). Not only do anxiety disorders place a substantial burden on society, but individuals with anxiety suffer a significantly lower quality of life than nonclinical samples (Olatunji, Cisler, & Tolin, 2007). In addition, these disorders often follow a chronic course in many clients (Shear, 1995).

In a naturalistic 1-year follow-up study of individuals with panic disorder, 92% who had been diagnosed with current panic disorder continued to experience panic attacks over the follow-up period, and 41% of initially remitted patients relapsed (Ehlers, 1995). Maintenance and relapse rates were not significantly related to whether an individual had received treatment or not. Even when individuals do receive treatment and achieve remission from an anxiety disorder, this does not necessarily mean that they will
maintain their gains over the long term. In one study, 42% of individuals who had achieved full remission of their symptoms relapsed at least once within eight years (Yonkers, Bruce, Dyck, & Keller, 2003).

Exposure-based behavioral and CBT psychotherapies are typically considered the gold standard psychosocial interventions for anxiety disorders, resulting in medium to large effect sizes (Butler et al., 2006; Norton & Price, 2007; Olatunji et al., 2010) and having more enduring effects than pharmacological treatments (Hollon, Stewart, & Strunk, 2006). Despite this success, however, response rates are still relatively low, ranging from less than one-half (Beidel, Frueh, Uhde, Wong, & Mentrikoski, 2011; McDonagh et al., 2005) to approximately two-thirds (Newman et al., 2011; Öst, Thulin, & Ramnero, 2004) at posttreatment. Of those who do respond to treatment with CBT or behavior therapy, clients still experience relapse, particularly during long-term follow-up periods extending beyond one year (Vervliet, Craske, & Hermans, 2013). Although long-term follow-up data are limited, Durham et al. (2003) conducted an 8-14 year follow-up study of two randomized clinical trials comparing CBT with non-CBT (including pharmacological treatments, placebo control groups, and non-CBT psychotherapies) for participants with a diagnosis of generalized anxiety disorder (GAD). The first study was conducted at a university in the United Kingdom and consisted of participants recruited from primary care settings. Using Jacobson criteria for clinically significant change (Jacobson & Truax, 1991), 80% of participants in the CBT condition were classified as recovered at 6-month follow-up. At long-term follow-up, however, over half of these (56%) had relapsed. In the second study, conducted at a separate university in the UK and
using participants with a GAD diagnosis recruited from both primary and secondary care, 26% of CBT participants were recovered at 6-month follow-up. Of these participants, 63% had relapsed by long-term follow-up. Long-term outcomes in both studies for participants in non-CBT groups did not significantly differ from those receiving CBT.

One factor contributing to long-term relapse could be the finding that many anxiety disordered individuals who complete successful treatment for their disorder often continue to experience substantial residual symptoms. Craske et al. (1991) conducted a 24-month follow-up of patients receiving exposure-based CBT for panic disorder. While 81% of participants had been panic-free for the month prior to the follow-up assessment, only 50% of those had met criteria for both panic-free status and mild clinical severity ratings. In other words, half of participants who had achieved panic-free status were still experiencing considerable symptomatology at long-term follow-up. Most notably, participants continued to experience agoraphobic avoidance, a symptom known to be related to maintenance of panic disorder in treated individuals and recurrent panic attacks in remitted individuals (Ehlers, 1995). Brown and Barlow (1995) have suggested that the typical cross-sectional approach to follow-up measurement in studies of panic disorder can conceal important ideographic differences in response and tends to overestimate the long-term success of treatment. Longitudinal measurement methods, requiring more stable outcomes over the follow-up period, reveal that many clients who are considered panic-free at follow-up actually experience a considerable return of symptoms over the follow-up period that are not detected via the cross-sectional method of measurement. Despite the sometimes large effect sizes achieved with exposure-based CBT and behavior
therapy for anxiety, response rates are less than ideal, and long-term relapse remains a problem.

**Mechanisms of Exposure**

The majority of basic and translational researchers investigating treatment and relapse in anxiety have focused on Pavlovian processes, conceptualizing the mechanism of exposure as Pavlovian extinction of self-reported fear and physiological “fear responses,” such as heart rate and electrodermal response (Lipp, 2006). Likewise, the predominant conceptualization of relapse has been the Pavlovian relapse phenomenon known as “return of fear,” which includes renewal, reinstatement, and spontaneous recovery (Rachman, 1979; Vervliet et al., 2013). However, this approach may overlook the important role of operant processes in the maintenance, treatment, and relapse of anxiety disorders. Anxiety disorders are characterized by avoidance of fear-inducing stimuli (Barlow, 2004), which is an operant behavior. In addition, recent research has shown that the reduction of self-reported fear and physiological fear responses (the goals of Pavlovian extinction procedures) may not be the key component to successful exposure therapy. Toleration of fear, rather than the reduction of it, has been shown to be more predictive of therapeutic outcomes (Craske et al., 2008). Therefore, successful treatment of anxiety might be better conceptualized as the result of operant extinction of avoidance responding than Pavlovian extinction of fear responding. From this perspective, relapse would also result from operant processes.
Resurgence

One way to conceptualize the relapse of operant behavior is through the phenomenon known as resurgence, in which a previously extinguished behavior returns following the extinction of an alternative behavior that has replaced it (Leitenberg, Rawson, & Bath, 1970; Lieving & Lattal, 2003). Resurgence has been used as a model for investigating the elimination and relapse of problem behavior in individuals with developmental disabilities following treatment with DRA. Although not explicitly required, DRA procedures typically consist of reinforcement of an alternative behavior that is in the same functional class as the target problem behavior (Cooper, Heron, & Heward, 2007). In applied studies investigating resurgence, target and alternative behaviors have functioned to gain access to both positive reinforcement (e.g., Lieving et al., 2004) and negative reinforcement (e.g., Wacker et al., 2013). Some studies have looked at positively and negatively reinforced behaviors within the same study but, for each behavior, the function of the alternative behavior has always been the same as that of the target behavior (Marsteller & St. Peter, 2012; Volkert et al., 2009).

Resurgence may provide a promising laboratory analogue of treatment and relapse within the anxiety disorders. Behavioral and cognitive-behavioral therapies for anxiety disorders often focus on the elimination of avoidance behavior while simultaneously teaching engagement with positively reinforcing life activities as an alternative to avoidance (Barlow, 2004; Hayes, Strosahl, & Wilson, 2011). This process
is similar to DRA, with the exception that avoidance behaviors are negatively reinforced, while alternative behaviors are positively reinforced.

**Resurgence in Human Operant Preparations**

Analogue studies of resurgence involving human operant preparations have been somewhat limited and have primarily focused on positively reinforced target and alternative behaviors. For example, Doughty, Cash, Finch, Holloway, and Wallington (2010) investigated the effects of varying lengths of training history on resurgence in three college students. Using a match-to-sample procedure, participants were trained to emit different target responses by using a mouse to click on arbitrary stimuli on a computer screen. All responses earned points on a fixed ratio 1 (FR 1) schedule of reinforcement. Different target responses received differing lengths of training during baseline, such that selecting B2 in the presence of A2 was trained for 100 trials, and selecting D2 in the presence of C2 was trained for 50 trials. Selections of B3 and D3 in the presence of A2 and C2 respectively were considered alternative responses and were trained for 50 trials each, during which time the previously reinforced responses of selecting B2 and D2 were extinguished. When B3 and D3 responses were subsequently placed on extinction, resurgence of B2 responding was observed across all three participants, while resurgence of D2 responding was observed in only one of three participants, possibly reflecting the longer training history of B2 as compared to D2. Resurgence was defined as more responses to target comparison stimuli than to other comparison stimuli to which responding had never been reinforced in the presence of A2.
or C2. Doughty et al.’s (2010) experiment demonstrates resurgence of positively reinforced target responding in a human operant match-to-sample paradigm. Others have also demonstrated similar results in human operant preparations utilizing positive reinforcement (Dixon & Hayes, 1998; Doughty, Kastner, & Bismark, 2011; McHugh, Procter, Herzog, Schock, & Reed, 2012; Wilson & Hayes, 1996).

Bruzek, Thompson, and Peters (2009) investigated the resurgence of a negatively reinforced target response following the removal of negative reinforcement for an alternative response. In this experiment, participants were undergraduate college students who were trained to emit target and alternative caregiving responses toward a baby doll in a simulated caregiving context. Reinforcement consisted of escape from a recorded infant cry, and duration of caregiving responses constituted the primary dependent measures. In Experiment 1, seven participants were required to engage in the target caregiving response for three seconds in order to terminate crying. After meeting acquisition criteria, the target response was placed on extinction, and crying occurred regardless of participant response. Participants then acquired the alternative caregiving response. After meeting acquisition criteria for this response, both target and alternative responses were placed on extinction. Five of seven participants demonstrated resurgence of the target response relative to a control response that was never reinforced.

In Bruzek et al.’s (2009) Experiment 2, the effects of length and recency of training on resurgence were examined. Eight undergraduate students were trained to engage in one of four variations of toy play toward a baby doll. Again, correct responding resulted in escape from infant crying. Acquisition criteria for the first target response
consisted of engaging in toy play for 5 continuous minutes on three consecutive sessions. This was done in order to establish a longer training history for the first target response. Participants then acquired the second target response. Acquisition criteria for this response consisted of engaging in toy play with a different toy for 5 continuous minutes on only one session. The second target response therefore had a shorter, but more recent, training history than the first. Target responses 1 and 2 were then placed on extinction, and infant crying continued regardless of participant response. A third, alternative, playing response was then trained according to the parameters established in Experiment 1. After acquisition, this response was also placed on extinction, and six of eight participants showed a resurgence effect. For five participants, target response 1 resurfaced more than target response 2 or a control response that had never been reinforced. For one participant, both target responses resurfaced equally. Experiments 1 and 2 both demonstrate resurgence of negatively reinforced responding in a human operant preparation. As in the case of Doughty et al. (2010) and the DRA treatment studies reviewed previously, both target and alternative behaviors were in the same functional class.

**Different Functional Classes**

While researchers conducting human applied and analogue resurgence studies have yet to attempt to train an alternative response that is functionally different from the target response, Podlesnik, Jimenez-Gomez, and Shahan (2006) did just that in rats, using alcohol as the target reinforcer and food pellets as the alternative. In this study, four male
Long-Evans rats were trained to press a lever to self-administer alcohol on a random ratio 25 (RR 25) schedule of reinforcement for 25 sessions. Following acquisition of the target response, lever pressing was placed on extinction and a chain was introduced into the experimental chamber. Chain pulling was then reinforced on a RR 10 schedule for 10 sessions, using food pellets as reinforcers. In the last phase of the experiment, both lever pressing and chain pulling were placed on extinction, and higher rates of responding were observed on the lever previously correlated with alcohol than on an inactive lever that had never been correlated with reinforcement. Therefore, although both target and alternative behaviors were positively reinforced, Podlesnik et al.’s (2006) study demonstrates resurgence of a target behavior that is functionally different from the alternative behavior following removal of alternative reinforcement. This same phenomenon has also been demonstrated in rats trained to press a lever for cocaine (Quick, Pyszczynski, Colston, & Shahan, 2011).

The studies reviewed thus far demonstrate that resurgence occurs with humans in applied and analogue settings when both target and alternative behaviors are maintained through positive reinforcement and when both target and alternative behaviors are maintained through negative reinforcement. In addition, resurgence has been shown to occur in animal models when target and alternative behaviors are in separate functional classes. However, in order to apply a resurgence model to the treatment and relapse of anxiety, resurgence must also be demonstrated when a target behavior is negatively reinforced and an alternative behavior is positively reinforced. This has yet to be investigated in either humans or animals.
Varying Rates of Alternative Reinforcement

Another important aspect of resurgence when attempting to apply it to the treatment and relapse of anxiety comes from basic animal research showing that, while higher rates of alternative reinforcement result in faster and more comprehensive elimination of target behavior, they also tend to result in greater resurgence of that behavior once reinforcement has been decreased or removed. For example, Leitenberg et al. (1975, Experiment 3) used a between-groups design to compare the effects of varying rates of alternative reinforcement on the resurgence of target behavior in 30 pigeons. In this experiment, pigeons were randomly assigned to one of three equal groups: Group H (high rates of alternative reinforcement), Group L (low rates of alternative reinforcement), and a control group receiving no alternative reinforcement. During Phase 1, pigeons were trained to peck Key A for food on a variable interval 120-second schedule (VI 120s) for five days. After acquiring the target response, Phase 2 began, in which pecking on Key A was placed on extinction, and Key B was introduced into the experimental chamber. Pecking on Key B was then reinforced at different rates for the three different groups: a VI 30s schedule for Group H, a VI 240s schedule for Group L, and no alternative reinforcement for the control group. Group H showed greater suppression of Key A responding during Phase 2 than did the other two groups, which
did not significantly differ from one another. When alternative reinforcement was removed during Phase 3, Group H also showed more resurgence of Key A responding than the other two groups, which again did not significantly differ. Therefore, although higher rates of alternative reinforcement resulted in more effective elimination of target responding, they also resulted in greater relapse once reinforcement was removed.

Winterbauer and Bouton (2010) found similar results in their Experiment 2, in which 31 Wistar rats were trained to press a lever (L1) for pellets on a random interval 10-second schedule (RI 10s) for five sessions. During Phase 2, rats were split into three experimental groups: 12 rats in Group RI 10 received reinforcement on a RI 10s schedule for pressing a second lever (L2), 12 rats in Group RI 30 received reinforcement on a RI 30s schedule for L2 presses, and seven rats in Group EXT received no alternative reinforcement. Following acquisition of L2 responding in Groups RI 10 and RI 30, responding on both levers was placed on extinction during Phase 3. Response elimination proceeded more quickly in Group RI 10 during Phase 2 than in Group RI 30, but did not differ from Group EXT. In Phase 3, target responding resurged in both Group RI 10 and Group RI 30. Visual inspection of the data indicate that resurgence was greater in Group RI 10 than Group RI 30, however, a post hoc test directly comparing resurgence between these groups was not reported, making it difficult to determine whether this effect was statistically significant. In sum, while a rich rate of alternative reinforcement was more effective than a lean rate at decreasing target responding, it was no more effective than no alternative reinforcement and resulted in more resurgence once reinforcement was removed. In addition, the lean rate of alternative reinforcement given to group RI 30
seems to have impeded elimination of target responding during Phase 2, while still resulting in some resurgence during Phase 3. These results could also have been affected by the shift between Phase 1 and Phase 2 rates of reinforcement, which remained the same for the group receiving a rich rate of alternative reinforcement, but decreased from Phase 1 (RI 10s) to Phase 2 (RI 30s) for the group receiving a lean rate of reinforcement.

In Winterbauer and Bouton (2010, Experiment 1), rates of reinforcement were also shifted between Phases 1 and 2, but in the opposite direction. Using 32 Wistar rats split into three experimental groups, they showed that rats shifting from an RI 30s schedule of target reinforcement during Phase 1 up to a richer RI 10s schedule of alternative reinforcement during Phase 2 decreased their target responding in Phase 2 more quickly than rats who remained on a RI 30s schedule in both phases. During Phase 3, when alternative reinforcement was removed, both groups showed similar levels of resurgence. While results from both experiments are somewhat difficult to interpret due to the shifting rates of reinforcement between Phases 1 and 2, it would appear that, on the whole, rich rates of alternative reinforcement were more effective at facilitating elimination of the target response than lean rates, while resulting in either the same or more resurgence during Phase 3. Additionally, lean rates of alternative reinforcement, especially when there was a downshift in reinforcement rate between Phases 1 and 2, actually produced less effective elimination of the target response than an extinction condition, while still resulting in resurgence of target behavior during Phase 3.

Sweeney and Shahan (2013) also showed similar effects using 32 Long-Evans rats split into four experimental groups: Rich \((n=8)\), Lean \((n=8)\), Thinning \((n=8)\), and
Control \((n = 7)\). During Phase 1, all rats received food reinforcement for the target response (pressing a lever) on a VI 45s schedule of reinforcement for ten days. Rats were then randomly assigned to one of the four experimental groups, with the constraint that average target response rates did not significantly differ between groups. During Phase 2, also ten days, target responding was placed on extinction and an alternative response (nosepoke) was reinforced at varying rates for each of the four groups. The Rich group received reinforcement on a VI 10s and the Lean group on a VI 100s. The Thinning group began Phase 2 on a VI 10s, which increased by 10 seconds each day, until reaching a VI 100s by the last day of Phase 2. Rate of reinforcement was thereby gradually thinned throughout Phase 2 for this group. The Control group did not receive alternative reinforcement. During Phase 3, all responses were placed on extinction.

Results indicated that target responding was suppressed most rapidly in the Rich group and least rapidly in the Lean group. For the Thinning group, response suppression proceeded at a similar rate as the Rich group for the first session of Phase 3 but, once the schedule began to be thinned beginning at session 2, suppression slowed. By session 4, an increase in target responding was observed in this group, analogous to an early resurgence effect. At the end of Phase 2, responding in both Thinning and Lean groups was comparable. Similarly, although response suppression in the Rich group occurred rapidly for the first two sessions of Phase 2, by the end of Phase 2, both the Rich and Control groups were responding at approximately equal rates, which were significantly less than the other two groups. During Phase 3, target responding resurged most in the
Rich group, whereas no resurgence effect was shown for the Lean, Thinning, or Control groups.

The finding that the Thinning and Lean groups had similar target response rates on the last day of Phase 2, both of which were significantly higher than either the Rich or Control groups, could indicate that thinning alternative reinforcement is ultimately no more effective at suppressing target responding than is reinforcing alternative behavior on a fixed thin schedule. In addition, both fixed thin and thinning schedules of alternative reinforcement may actually be less effective than no alternative reinforcement at all, as evidenced by significantly more target responding on the last day of Phase 2 in both these groups as compared to the Control group, a finding that may also be related to the downshift in reinforcement rate from Phases 1 to 2, as was seen in Winterbauer and Bouton (2010) Experiment 2. Although target responding was suppressed most rapidly in the Rich group, this was the only group to show a statistically significant resurgence effect during Phase 3.

Winterbauer and Bouton (2012) had a similar finding with respect to thinning and rich schedules of alternative reinforcement. In their Experiment 1, 32 Wistar rats were trained to press a lever on an RI 30s schedule of reinforcement for 12 sessions during Phase 1. Following this phase, rats were randomly assigned to one of the three experimental groups, with the constraints that the groups did not reliably differ on baseline response rates or daily runs. During Phase 2, which lasted four sessions, responses on the first lever were placed on extinction, while groups Resurge ($n = 11$) and Thinning ($n = 11$) were trained to press a newly inserted second lever on a RI 20s
schedule. Beginning on the second session, group Thinning’s schedule doubled halfway through each session, ending on a RI 160s schedule by the final session of Phase 2. Group Extinction ($n = 10$) received no alternative reinforcement. Responding on both levers was placed on extinction for all groups during Phase 3.

Target response suppression proceeded most rapidly in the group receiving the highest rate of alternative reinforcement (group Resurge) during Phase 2. While group Thinning showed more rapid suppression of target responding than group Extinction for the first four sessions of Phase 2, target responding increased slightly during session 3 and more substantially during session 5, ending at significantly higher levels than the other two groups, whose target responding was comparable. As in Sweeney and Shahan (2013), the thinning group showed an early resurgence effect once the schedule of alternative reinforcement began to be thinned. Group Resurge showed a resurgence effect from the last session of Phase 2 to the first session of Phase 3, as did group Extinction, although to a lesser extent, indicating some spontaneous recovery. Group Thinning did not show a resurgence effect during Phase 3. However, as mentioned earlier, this group showed early resurgence of target responding during Phase 2, and therefore their average level of target responding on the first session of Phase 3 was similar to group Resurge. Like Sweeney and Shahan (2013), thinning of alternative reinforcement was less effective in reducing target responding than no alternative reinforcement, due mainly to the early (Phase 2) resurgence effect demonstrated by the Thinning group.

A second experiment by Winterbauer and Bouton (2012), in which alternative reinforcement was thinned at either the same rate as in Experiment 1 (group Stepped), or
more gradually (group Gradual), also showed early resurgence in both thinning groups. Again, suppression of target responding was most rapid in the group receiving the highest rate of alternative reinforcement (group Resurge). Results from both experiments suggest that, while higher rates of alternative reinforcement produce more rapid suppression of target responding than no alternative reinforcement, they also result in resurgence of responding once reinforcement is removed. As was the case in Sweeney and Shahan (2013), reducing (thinning) alternative reinforcement does not ameliorate the problem. Rather, resurgence occurs once the alternative schedule begins to thin. Reinforcement on a fixed lean schedule also does not appear to be a viable solution, as lean schedules of alternative reinforcement can actually have a detrimental effect on the elimination of target responding, while still sometimes resulting in resurgence (e.g., Sweeney & Shahan, 2013; Winterbauer & Bouton, 2010, 2012).

It is worth noting that not all studies have found support for the relationship between rich rates of alternative reinforcement and greater resurgence. One study that did not show this relationship was conducted with 24 children diagnosed with Autism Spectrum Disorder (ASD; Reed & Clark, 2011). The children were taught to emit different play sequences as target and alternative behaviors, and target behavior was extinguished before alternative behavior was trained. Alternative reinforcement length and rate was manipulated in Phase 2 using three groups: a group receiving reinforcement on a variable ratio 4 schedule for 60 minutes (VR-4 60 min), a group receiving reinforcement on a variable ratio 4 schedule for 30 minutes (VR-4 30 min), and a group receiving reinforcement on a variable ratio 2 schedule for 30 minutes (VR-2 30 min). The
latter two groups, therefore, received varying rates of alternative reinforcement over the same length of time. During a final extinction test, resurgence occurred in the VR-4 30 min group and not in the VR-2 30 min group. In other words, more resurgence occurred in the group receiving lean rates of alternative reinforcement than in the group receiving rich rates, which is the opposite of the majority of findings in the literature (see also CanÇado & Lattal, 2013).

Although the literature is not completely consistent with regard to the effects of alternative reinforcement rate on resurgence, the majority of studies have found that higher rates of reinforcement for the alternative behavior result in greater resurgence. Such paradoxical findings could have important implications for clinicians treating anxiety, as higher rates of alternative reinforcement, while more effectively reducing problematic avoidance behavior, may have the unintended side effect of producing greater relapse of avoidance if access to positive reinforcement is later reduced or becomes unavailable. In addition, the use of either lean or thinning schedules of alternative reinforcement does not appear to offer a viable solution to this problem. Such effects could be counterintuitive to clinicians. However, this particular finding within the resurgence literature has yet to be investigated in typically developing humans or with respect to a positively reinforced alternative behavior and its effects on the elimination and relapse of negatively reinforced avoidance behavior.

**Summary**

Given the current literature on the effects of varying rates of alternative
reinforcement on the elimination and relapse of target behavior, researchers should attempt to examine this variable in typically developing humans. In particular, two findings within the nonhuman literature may be especially relevant to clinicians. These are: (a) rich rates of alternative reinforcement, while promoting elimination of target behavior, also tend to result in greater relapse of that behavior once reinforcement has been removed, and (b) lean rates of alternative reinforcement have either no effect or a detrimental effect on rate of elimination of target behavior. Because groups whose schedules of alternative reinforcement are thinned over time may perform similarly to groups on lean schedules of reinforcement, research should include groups with fixed rich and lean schedules of reinforcement. Finally, research should address whether resurgence occurs when a target behavior is negatively reinforced and an alternative behavior is positively reinforced. This question is especially relevant for researchers attempting to translate basic research findings to the treatment of anxiety disorders.
CHAPTER III
METHODS

Purpose

The purpose of this study was to take the first step toward applying a model of resurgence toward anxiety disorders, by using a translational approach to investigate the effects of fixed high and low rates of positive reinforcement of alternative behavior on rate of response elimination and magnitude of resurgence of positively reinforced target behavior in humans using a computerized task. The following questions were addressed:

1. Does the loss of reinforcement for a recently reinforced alternative behavior result in the relapse of previously eliminated target behavior in humans?
2. What are the effects of high and low rates of reinforcement for an alternative behavior on the elimination of simultaneously extinguished target behavior in humans?
3. What are the effects of high and low rates of reinforcement for a recently reinforced alternative behavior on the magnitude of relapse of previously eliminated target behavior once alternative reinforcement is removed in humans?

Research Design

The study progressed in three phases. During Phase 1, participants acquired the target response, during Phase 2, target responding was placed on extinction and
participants acquired the alternative response, and during Phase 3, all responding was placed on extinction.

A between-groups design was used with three experimental conditions. Each group experienced a different rate of alternative reinforcement during Phase 2: a rich schedule (Rich), a lean schedule (Lean), and no alternative reinforcement (Control).

**Participants and Setting**

The study was approved by Utah State University’s Institutional Review Board (IRB). Forty-six undergraduate psychology students, recruited through SONA Systems at Utah State University, served as participants. Exclusion criteria included a motor impairment affecting the dominant hand and nonfluency in English. No participants were excluded. Students received course credit in exchange for participation. Demographic information was missing for one participant. Of the remaining 45 participants, 66.67% were female (33.33% male). The participants’ mean age was 19.87 years ($SD = 2.83$; range = 18-31), 86.7% were single (6.7% married, 4.4% other, 2.2% divorced), 93.3% were White (4.4% Hispanic, 2.2% Asian/Pacific Islander), 77.8% identified their religion as the Church of Jesus Christ of Latter-Day Saints (8.9% agnostic, 4.4% Catholic, 4.4% other Christian, 2.2% atheist, 2.2% other).

Participants were assigned quasi randomly to one of three experimental conditions: Rich ($n = 18$), Lean ($n = 18$), or Control ($n = 10$). The experiment was completed in one 30-minute session. Participants worked independently at a single
workstation located in an office-sized room (approximately 4.2 X 2.4 meters) at Utah State University.

**Materials and Apparatus**

A letter of information, approved by the university’s IRB, was given to participants upon arrival, along with a demographics questionnaire that assessed age, sex, ethnicity, English fluency, marital status, religion, employment status, level of education, level of income, previously diagnosed hearing impairments, previously diagnosed motor impairments, and current alcohol/substance use. A poststudy questionnaire assessing rules generated by participants about the experimental task was given at the conclusion of the experiment.

Each workstation was equipped with a chair, desk, Dell computer and monitor, speakers, a keyboard, and a mouse. The computer ran Microsoft Windows XP operating system and a custom computer program programmed using Microsoft Visual Basic software. Instructions were presented on the computer screen and were as follows:

“Shapes will appear on the screen in front of you. Clicking on the correct shape will earn you points. Your job is to earn as many points as possible. Please let the experimenter know if you have any questions at this time. The experiment will begin when you click ‘OK’.”

Stimuli consisted of a red and a blue rectangle, each approximately 6 cm X 4 cm, 11 cm apart, and centered on the screen (approximately 12 cm from the top of the screen, 8 cm from the bottom of the screen, and 9 cm from each side). Both stimuli remained visible and in the same location on the screen throughout all phases of the experiment.
Responses consisted of mouse clicks on these stimuli, and reinforcement consisted of 2 points per correct response according to the schedule of reinforcement. Each time a stimulus was clicked, whether or not points were earned, a black outline appeared around the stimulus for 500 milliseconds, indicating that a response had been made. Points earned were indicated in green text that flashed in the middle of the screen for 1 second, and total accumulated points were displayed in a box at the top of the screen. Data were collected by the computer program and analyzed using Microsoft Excel and IBM Statistical Package for the Social Sciences (SPSS).

**Measures**

Target responses consisted of mouse clicks on the correct stimulus. Dependent variables were target response rates, analyzed as a proportion of baseline target responding, during Phase 2 (extinction) and Phase 3 (resurgence).

**Procedure**

Participants were recruited through SONA Systems at Utah State University. Upon arrival, each participant was given the letter of information and asked if they had questions about the experiment. They were then instructed by the experimenter or a research assistant to sit down at the workstation in front of the computer monitor. The computer program assigned each participant quasi-randomly to one of three conditions: Rich, Lean, or Control. On-screen instructions were delivered prior to beginning the
experimental task (see materials section), and participants clicked a box to begin the task. The experiment lasted for a total of 7.5 minutes and was delivered in three phases.

**Phase 1**

A description of each phase and condition is presented in Table 1. During Phase 1, all groups acquired the target response. A mouse click on the target stimulus, the red rectangle, resulted in the delivery of 2 points on an RI 2s schedule of reinforcement. In this schedule of reinforcement, the computer randomly determined the availability of reinforcement once per second with a probability of 0.5. Once reinforcement became available, it remained available until the participant emitted a correct response, at which time reinforcement was delivered, and the computer returned to determining the availability of reinforcement in the next second. Point delivery was indicated by text that appeared on the computer screen (i.e., “+2 points”), and the points were added to the box at the top of the screen displaying total accumulated points. A mouse click on the red rectangle did not earn points during this phase. Phase 1 lasted for 180 seconds.

**Phase 2**

During Phase 2, responses to the target stimulus no longer resulted in points for any group. Responses to the alternative stimulus, the blue rectangle, resulted in the delivery of 2 points for groups Rich and Lean. The Rich group received points on an RI 1s schedule of reinforcement, while the Lean group received points on an RI 3s schedule of reinforcement. As in Phase 1, point delivery was indicated by text on the computer screen and total accumulated points were displayed at the top of the screen. The Control
group did not receive points for any responses during this phase. Phase 2 lasted for 180 seconds.

Table 1

*Experimental Phases and Conditions*

<table>
<thead>
<tr>
<th>Group</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rich</td>
<td>A: RI 2s</td>
<td>A: EXT</td>
<td>A: EXT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B: RI 1s</td>
<td>B: EXT</td>
</tr>
<tr>
<td>Lean</td>
<td>A: RI 2s</td>
<td>A: EXT</td>
<td>A: EXT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B: RI 3s</td>
<td>B: EXT</td>
</tr>
<tr>
<td>Control</td>
<td>A: RI 2s</td>
<td>A: EXT</td>
<td>A: EXT</td>
</tr>
<tr>
<td></td>
<td></td>
<td>B: EXT</td>
<td>B: EXT</td>
</tr>
</tbody>
</table>

**Phase 3**

During Phase 3, responses to either stimulus did not result in points for any group.

The box displaying total accumulated points remained visible at the top of the screen.

Phase 3 lasted for 90 seconds.
CHAPTER IV

RESULTS

A total of 46 participants completed the study. There were no missing data. In order to facilitate data analysis, responses were grouped into 30-second bins across all phases. Table 2 displays obtained reinforcement rates for each group during Phases 1, 2, and 3. To confirm that programmed rates of reinforcement resulted in the expected obtained reinforcement, a one-way ANOVA compared obtained reinforcement rate across groups during Phase 2. The test was statistically significant, $F(2, 43) = 109.71, p < .001$. A post hoc Tukey HSD test revealed significant differences between Rich and Lean, Rich and Control, and Lean and Control. Obtained reinforcement rate was not significantly different across groups during Phase 1, $F(2, 43) = 0.92, p = .41$.

Table 2

*Means and Standard Deviations of Obtained Reinforcement During Phases 1, 2, and 3*

<table>
<thead>
<tr>
<th>Group</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rich (SD)</td>
<td>108.67 (32.91)</td>
<td>262.72 (69.51)</td>
<td>0</td>
</tr>
<tr>
<td>Lean (SD)</td>
<td>118.17 (38.57)</td>
<td>99.00 (29.99)</td>
<td>0</td>
</tr>
<tr>
<td>Control (SD)</td>
<td>129.30 (48.92)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Phase 1

Absolute response rates for the target and alternative responses during Phases 1, 2, and 3 are displayed in Figures 1 and 2. In order to test for group differences in target responding during Phase 1, a one-way ANOVA was conducted comparing target
Figure 1. Medians of absolute target responding for each group across Phase 1, 2, and 3.

Figure 2. Medians of absolute alternative responding for each group across Phase 1, 2, and 3.
responding during the last 90 seconds of Phase 1 in the three groups. The ANOVA was not statistically significant, \( F(2, 43) = 1.58, p = 0.218 \). Despite this nonsignificant result, target responses during Phases 2 and 3 were analyzed as a proportion of baseline responding because it was considered to be a superior measure to absolute response rate, as it controls for all differences between participants in baseline responding. The left panel of Table 3 shows median absolute response rates during the last 90 seconds of Phase 1 (as sums) for target and alternative responses across conditions. Baseline was calculated for each participant by averaging absolute response rates during the last 3 bins (90 seconds) of Phase 1.

### Phase 2

Because proportion of baseline target response rate distributions for the three groups across the six bins of Phase 2 and three bins of Phase 3 were not all normal (most were positively skewed), data are summarized using median as a measure of central tendency and interquartile range (IQR) as a measure of dispersion. The middle panel of Table 3 displays median absolute and proportion of baseline response rates for the target response and median absolute response rates for the alternative response during Phase 2. Figure 3 displays target proportion of baseline responding for the three groups during Baseline, Phase 2, and Phase 3. During the first bin of Phase 2, target responding was similarly suppressed in the Rich and Lean groups, to 24% and 23% of baseline respectively. In the Control group, target responding remained at 59% of baseline.
Table 3

Medians and Interquartile Ranges of Target (Absolute and Proportion of Baseline) and Alternative (Absolute) Response Rates During Phases 1, 2, and 3

<table>
<thead>
<tr>
<th>Group</th>
<th>Phase 1 (last 90s)</th>
<th>Phase 2 (last 90s)</th>
<th>Phase 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Target Absolute</td>
<td>Alt. Absolute</td>
<td>Target Absolute</td>
</tr>
<tr>
<td>Rich (n = 18)</td>
<td>83.00</td>
<td>11.00</td>
<td>1.00</td>
</tr>
<tr>
<td>(IQR)</td>
<td>(33.75)</td>
<td>(21.50)</td>
<td>(5.75)</td>
</tr>
<tr>
<td>Lean (n = 18)</td>
<td>92.00</td>
<td>8.00</td>
<td>21.50</td>
</tr>
<tr>
<td>(IQR)</td>
<td>(52.00)</td>
<td>(12.25)</td>
<td>(24.75)</td>
</tr>
<tr>
<td>Control (n = 10)</td>
<td>107.00</td>
<td>2.50</td>
<td>28.00</td>
</tr>
<tr>
<td>(IQR)</td>
<td>(89.75)</td>
<td>(10.25)</td>
<td>(80.75)</td>
</tr>
</tbody>
</table>

*Figure 3. Medians of proportion of baseline target responding for each group across Baseline, Phase 2, and Phase 3.*
During the second bin of Phase 2, target responding continued to decline to 2% of baseline in the Rich group and 37% of baseline in the Control group, while stabilizing in the Lean group. Following bin 2, target responding stabilized at zero in the Rich group, with Control increasing slightly at bin 4 and then decreasing sharply to 7% of baseline by the end of Phase 2.

To test target response suppression in each group, Friedman tests were conducted on each group with Baseline and the six bins of Phase 2 as levels of the main factor. A Bonferroni correction was used for multiple tests, and statistical significance was accepted at the $p < .0167$ level. Results indicated that Rich, Lean, and Control were significantly different across the different bins from Baseline through Phase 2 (Rich, $\chi^2(6) = 57.61, p < .001$; Lean, $\chi^2(6) = 33.20, p < .001$; Control, $\chi^2(6) = 23.95, p = .001$), indicating that response suppression occurred in all groups.

In order to test for differential suppression across the three groups, a Kruskal-Wallis H test comparing mean ranks of the three groups on the first bin of Phase 2 was used. The test was statistically significant, $\chi^2(2) = 19.52, p < .001$, as was a test comparing the last 90 seconds of Phase 2 across the three groups, $\chi^2(2) = 28.61, p < .001$, and a test comparing the three groups during the last bin of Phase 2, $\chi^2(2) = 15.88, p < .001$. Subsequently, pairwise comparisons were conducted between the three groups on the first bin of Phase 2, the last 90 seconds of Phase 2, and the last bin of Phase 2, using Dunn’s (1964) method with a Bonferroni correction for multiple comparisons. Statistical significance was accepted at the $p < .0056$ level. For the first bin of Phase 2, there were statistically significantly differences between the Rich (mean rank = 17.14) and Control
(mean rank = 39.80) \(p < .001\) and between Lean (mean rank = 20.81) and Control \(p < .001\) groups, but not between the Rich and Lean groups \(p = 0.412\). Comparisons of target responses in the last 90 seconds of Phase 2 revealed statistically significant differences between Rich (mean rank = 35.22) and Control (mean rank = 7.60) \(p < .001\) and between Rich and Lean (mean rank = 20.61) \(p = .001\), but not between Lean and Control \(p = .014\). Comparisons of the last bin of Phase 2 indicated statistically significant differences only between Rich (mean rank = 14.67) and Lean (32.11) \(p < .001\), with no differences between Rich and Control (mean rank = 23.90) \(p = .075\) or Lean and Control \(p = .113\). In sum, results indicated that the target response significantly decreased in all groups from baseline through the end of Phase 2, occurring more quickly in Rich and Lean than in Control (first bin of Phase 2). In the second half of Phase 2, responding was completely suppressed in Rich, stable at about 20% of baseline in Lean, and steeply decreasing in Control. Response suppression was more thorough by the end of Phase 2 in Rich and Control than in Lean (last bin of Phase 2).

**Resurgence**

Figure 4 displays median target response rate as a proportion of baseline during the last bin of Phase 2 and the first bin of Phase 3 in each group. During the last 30 seconds of Phase 2, target responding in Rich and Control were suppressed to below 10% of baseline and Lean to 19% of baseline. During the first 30 seconds of Phase 3, Rich increased to 53% and Lean to 42% of baseline, while Control increased to 27%, indicating some spontaneous recovery of the target response in the Control group.
Resurgence was defined as a statistically significant increase in target responding between the last bin of Phase 2 and the first bin of Phase 3. Again, nonparametric statistics were used because target response rate distributions across the three groups and three bins of Phase 3 did not meet the assumptions of parametric statistics. Exact sign tests were used to test for statistical significance between the last bin of Phase 2 and the first bin of Phase 3 in each group. A Bonferroni correction was used for multiple tests and statistical significance was accepted at the $p < .0167$ level. Of the 18 participants in the Rich group, response rates increased for 17 and showed no change for one from the last bin of Phase 2 ($Mdn = 0$) to the first bin of Phase 3 ($Mdn = 0.535$).

Figure 4. Medians of proportion of baseline target responding for each group during the last bin of Phase 2 and the first bin of Phase 3.
Overall, this represented a statistically significant increase in the median of the differences of 0.478, $p < .001$, $r = 0.85$. Of the 18 participants in the Lean group, 14 increased, one decreased, and three showed no change between the last bin of Phase 2 ($Mdn = 0.184$) and the first bin of Phase 3 ($Mdn = 0.425$). Overall, there was a statistically significant increase in the median of the differences of 0.130, $p = .001$, $r = 0.79$. In the Control group, out of 10 participants, five increased, three decreased, and three showed no change in target responding between the last bin of Phase 2 ($Mdn = 0.069$) and the first bin of Phase 3 ($Mdn = 0.269$). This represented a nonsignificant change in the median of the differences ($Mdn = 0$), $p = 1.0$.

A Kruskal-Wallis H test confirmed that resurgence, as measured by difference scores (first bin Phase 3 – last bin Phase 2), was significantly different between the groups, $\chi^2(2) = 17.76$, $p < .001$. Pairwise comparisons using Dunn’s (1964) procedure with a Bonferroni correction (statistical significance accepted at the $p < 0.0167$ level) revealed that resurgence was significantly greater in Rich ($Mdn = 0.48$, $IQR = 0.33$, mean rank = 33.67) than Lean ($Mdn = 0.13$, $IQR = 0.23$, mean rank = 18.58) ($p = .001$, $r = 0.56$) and in Rich than Control ($Mdn = 0$, $IQR = 0.41$, mean rank = 14.05) ($p < 0.001$, $r = 0.70$). No significant difference was found between Lean and Control ($p = 0.391$). Figure 5 shows boxplots of the difference scores in each of the three groups. Overall, results indicated that a statistically significant and large effect of resurgence occurred in the Rich and Lean groups, but not in the Control group. In addition, Rich surged significantly more than Lean and Control, also with large effects. There was no statistically significant difference in resurgence between Lean and Control.
Phase 3

The right panel of Table 3 displays median absolute and proportion of baseline response rates for the target response and median absolute response rates for the alternative response during Phase 3. Following the first bin of Phase 3, responding in the Control group continued to increase in the second bin and then decreased to 26% of baseline by the end of Phase 3. Rich also had a slight increase during the second bin and decreased during the third bin of Phase 2, ending at 54% of baseline by the end of Phase 3, and Lean continued decreasing to similar levels as Control by the end of Phase 3.

![Box plot showing response proportions](image)

*Figure 5.* Target response proportion of baseline difference scores (first bin of Phase 3 – last bin of Phase 2) in each group.
Kruskal-Wallis H tests were conducted in order to determine whether target responding in the three groups differed during Phase 3. Results for the first bin of Phase 3 were not significant ($\chi^2(2) = 2.16, p = 0.34$), nor were results for all of Phase 3 ($\chi^2(2) = 1.83, p = 0.402$) or the last bin of Phase 3 ($\chi^2(2) = 2.64, p = 0.267$), indicating that, although the magnitude of resurgence differed between groups, levels of target responding following relapse were similar in all groups.
CHAPTER V
DISCUSSION

The purpose of the current study was to take the first step in applying a model of resurgence toward human relapse in the anxiety disorders. Clinicians working with anxiety disordered individuals often employ therapeutic techniques aimed at decreasing problem target behaviors while simultaneously increasing positively reinforced alternative behaviors. Two findings within the animal literature on resurgence were considered especially relevant to clinicians treating anxiety. The first was that richer rates of alternative reinforcement, while facilitating faster and more complete suppression of target behavior, also tend to result in greater relapse of that behavior once reinforcement has been removed. The second was that leaner rates of alternative reinforcement seem to have either no effect on the suppression of target behavior or actually have a detrimental effect on suppression. In order to begin to investigate whether such findings also apply to human populations, this study took a translational approach to examining whether resurgence occurs in a typically developing human population and whether rich and lean rates of positive alternative reinforcement affect rate of suppression and magnitude of resurgence.

Results indicated that all three groups showed significant suppression of the target response during Phase 2. In the Rich group, target responding was eliminated the most quickly and thoroughly, with proportion of baseline response rates reaching and
remaining at zero by the third bin of Phase 2. Target responding in the Lean group also rapidly decreased once alternative reinforcement was introduced, but was never fully eliminated, remaining at about 20% of baseline for the duration of Phase 2. Target responding in the Control group was the slowest to decrease but, by the end of Phase 2, was lower than in the Lean group. While the rich rate of alternative reinforcement appears to have facilitated response suppression, the lean rate, while not apparent at first, ultimately had a detrimental effect on suppression.

With regard to the facilitating effect of rich rates of alternative reinforcement on target response suppression, the current findings are consistent with the majority of those in the nonhuman literature (i.e., Leitenberg et al., 1975; Sweeney & Shahan, 2013; Winterbauer & Bouton, 2010, 2012). In each case, rich rates of alternative reinforcement produced faster and more complete suppression of target responding than lean rates and, in all but two studies, than no alternative reinforcement. The findings of Winterbauer and Bouton (2010) Experiment 2, in which Rich and Control extinguished equally, could be due to the reinforcement schedule remaining the same between Phases 1 and 2, while all the other studies cited, including the current study, increased reinforcement rate between Phases 1 and 2 in the Rich group. Based on these findings, it may be necessary to increase the density of alternative reinforcement compared to target reinforcement in order for alternative reinforcement to facilitate suppression. In Sweeney and Shahan (2013), target responding in Rich eventually ended at the same level as Control, but suppression occurred more rapidly in Rich.
The finding that the lean rate of alternative reinforcement had a detrimental effect on suppression of the target response has also been shown previously in the nonhuman literature, but results are more mixed. Sweeney and Shahan (2013) showed that target suppression in the Lean group was more gradual and ended at a higher rate than in Rich and Control. A similar pattern was observed in Winterbauer and Bouton (2010) Experiment 2, with Lean decreasing more gradually than Rich and Control, although, on the last session of Phase 2, all groups were at similar levels of target responding. The pattern in the current study was somewhat different, with response suppression initially occurring rapidly in the Lean group, but then stabilizing, while both Rich and Control continued to decrease. Although it appears that lean rates of alternative reinforcement ultimately impeded response suppression in the current study, this has not consistently been the case in the literature. For example, in Leitenberg et al. (1975, Experiment 3) and Winterbauer and Bouton (2010, Experiment 1), responding in Lean and Control decreased at equal rates. So while results are somewhat mixed concerning the effects of lean rates of alternative reinforcement on suppression of target responding, findings do seem to indicate that they are no more effective at suppressing target behavior than simple extinction procedures and may sometimes impede suppression.

Results also indicated that participants who received both rich and lean rates of alternative reinforcement experienced significant resurgence of target responding once that reinforcement was removed, while those receiving no alternative reinforcement did not. The effect size of resurgence within both groups receiving alternative reinforcement was large, with a larger effect observed for Rich than for Lean. Comparing resurgence
between groups, Rich resurged significantly more than Lean and Control. Effect sizes were large in both cases, but larger between Rich and Control than Rich and Lean. There was not a significant difference between Lean and Control.

The within group finding that resurgence occurs following the removal of rich rates of alternative reinforcement has been observed consistently in the nonhuman literature (Leitenberg et al., 1975; Sweeney & Shahan, 2013; Winterbauer & Bouton, 2010, 2012), and the current study replicates these findings in typically developing humans. Additionally, in the current study, target responding in the group receiving lean rates of alternative reinforcement also resurged, a finding that was observed in Experiments 1 and 2 of Winterbauer and Bouton (2010), but not in Sweeney and Shahan (2013). Within groups, this effect was strong in both the Rich and Lean groups, while the Control group did not show a significant resurgence effect.

However, when resurgence was analyzed as a difference score and compared between groups, resurgence was not significantly greater in Lean than Control, which could be the result of the lower power of nonparametric tests compared to parametric tests, and indicating a somewhat mixed resurgence effect for the Lean group. In the between groups analysis, Rich showed a much clearer effect, significantly greater than both Lean and Control and with large between-groups effects for each comparison, larger for the comparison with Control. This type of between-groups analysis, using difference scores, seems to be unique in the literature and was the result of the use of nonparametric statistics, serving as an alternative to the between groups factor in a mixed-ANOVA. These particular results are therefore difficult to compare to the existing literature,
although Winterbauer and Bouton (2012, Experiment 1), using a mixed-ANOVA, showed that a group receiving richer rates of reinforcement resurged more than Control.

A more common analysis in the literature is the between-groups comparison during Phase 3. The current study showed no significant difference in levels of responding relative to baseline between groups during Phase 3. A similar result was also observed in Sweeney and Shahan (2013), who found no statistically significant difference between groups during the first session of Phase 3. However, their groups were ordered such that Rich > Thinning > Lean > Control. Winterbauer and Bouton (2010, Experiment 2) also showed a similar ordinal position of groups in Phase 3, with Rich > Lean > Control, but a statistical result testing these differences was not reported. Similarly, in the current study, although the differences between groups in the first bin of Phase 3 were not statistically significant, they were ordered such that Rich > Lean > Control. As extinction continued into Phase 3, the group receiving rich rates continued to respond at higher levels than the other two groups. By the last bin of Phase 3, groups were ordered such that Rich > Lean and Control, with Lean and Control approximately equal. While these differences are not statistically significant, they do suggest that richer rates of alternative reinforcement may result in greater levels of responding relative to baseline than leaner rates. This conclusion would be consistent with the above studies that have shown ordinal differences during Phase 3, and it would be consistent with studies showing that groups receiving richer rates of alternative reinforcement consistently show greater resurgence than no alternative reinforcement (Leitenberg et al., 1975; Winterbauer & Bouton, 2010,
2012), and leaner rates of alternative reinforcement sometimes show greater resurgence than no alternative reinforcement (Winterbauer & Bouton, 2010, 2012).

While the majority of the (albeit small) literature shows that varying rates of alternative reinforcement may differentially affect response suppression and resurgence, it is worth noting that some studies have failed to show this relationship. In their Experiment 1, Winterbauer and Bouton (2010) found no difference between resurgence in the Rich and Lean groups. In addition, in a number of manipulations of alternative reinforcement rate and schedule during a response-elimination phase in pigeons, CanÇado and Lattal (2013) did not find systematic effects of response rate following the removal of alternative reinforcement. However, during their response-elimination phases, they primarily employed differential reinforcement of other behavior (DRO) schedules, which have been shown to be less effective at suppressing target behavior than reinforcement of a discrete alternative response (e.g., Mulick, Leitenberg, & Rawson, 1976) and could therefore have affected the results of resurgence.

Another study that did not show a relationship between varying rates of alternative reinforcement and resurgence of target behavior was conducted by Reed and Clark (2011) in children diagnosed with ASD. Results from this study indicated that resurgence occurred in the group receiving leaner rates of alternative reinforcement and not in the group receiving richer rates. The authors suggest that their results may be related to the number of reinforcers experienced in each group, such that greater numbers of experienced reinforcers result in less resurgence, or to the effects of massed trials during acquisition of the target response. Either way, these results are surprising, as the
ordinal positions of their group data are the opposite of what has been observed in the nonhuman literature and the present study, and the hypothesis that more alternative reinforcement results in less resurgence runs counter to the predictions of a recent quantitative theory of resurgence (Shahan & Sweeney, 2011) that has begun to receive some empirical support (Sweeney & Shahan, 2013). While some studies have not shown a consistent effect of varying rate of alternative reinforcement on magnitude of resurgence, the majority of studies have shown that rich rates result in clear resurgence effects. While results are more mixed for lean rates, orderly ordinal relationships have been demonstrated, though they have not always met statistical significance.

The present study adds to the body of literature demonstrating resurgence effects in human operant preparations (Bruzek et al., 2009; Doughty et al., 2010; Doughty et al., 2011; McHugh et al., 2012; Wilson & Hayes, 1996). In addition, findings regarding the effects of varying rates of alternative reinforcement on magnitude of resurgence replicate the majority of findings in the nonhuman literature and represent one of the first extensions of this phenomenon to humans.

**Applied Implications**

The finding that relapse of target behavior occurs following the removal of reinforcement for alternative behavior has implications for the treatment of clinical populations. Because most behavioral and cognitive behavioral treatments for clinical problems target the reduction of problem behavior while simultaneously teaching more effective and healthy replacement behaviors, there is a risk of relapse if opportunities for
reinforcement are subsequently lost. Such an effect makes intuitive sense and is supported by the current experimental findings. What may be less intuitive, however, is the finding that relapse occurs to a greater extent in individuals who have a history of receiving high rates of reinforcement for an alternative behavior. Because high rates of alternative reinforcement more quickly and thoroughly suppress target behavior compared to low rates, it would be a logical choice for clinicians to encourage replacement behaviors resulting in the highest rate of alternative reinforcement available. While this approach may be quite effective in treatment, possibly resulting in a large decrease in problem behavior, the risk of relapse may be greater if life circumstances change and that replacement behavior or source of reinforcement becomes unavailable. Thus one logical clinical option, other than suggesting low rates of reinforcement, is to find multiple sources of reinforcement. Therefore, if one decreases, there would be others to keep the overall rate of alternative reinforcement high.

**Limitations and Future Directions**

The present study had some limitations. First, while investigating resurgence using positively reinforced target and alternative behaviors was a necessary first step in the translation of this phenomenon to humans, it was not the closest approximation of treatment and relapse in anxiety disorders because such disorders are characterized by avoidance behavior. In order to make the most relevant translation possible, response suppression and resurgence should be investigated with respect to negatively reinforced target behavior and positively reinforced alternative behavior. Such a preparation would
more closely approximate problematic avoidance behavior and positively reinforced
replacement behaviors that are typical of anxiety disorders and their treatments.

A second limitation of the current study involved the use of two stimuli that were
not counterbalanced throughout the experiment. While some analogue preparations have
utilized three or more stimuli (e.g., Doughty et al., 2010; Doughty et al., 2011), including
response options that have never been correlated with reinforcement in order to control
for extinction-induced variability, the current study utilized only two response options.
This was done because it was observed during piloting that participants engaged in high
levels of variable responding when given more than two response options. One reason for
this could be that humans are highly verbal and quickly generate rules that exceed the
complexity of the programmed experimental contingencies. In addition, no other
response options were available within the laboratory setting and, during extinction,
participants may have found that it was more entertaining to click on stimuli than simply
to stop responding. For this reason, the experiment was conducted between groups, with a
group of participants serving as the control for extinction-induced variability rather than
an extra response option. Although this was adequate for the current purposes, and
systematic results were observed, the design of future studies would be strengthened by
including more than two response options. Future designs should also consider
counterbalancing the position of stimuli on the screen in order to control for possible side
preferences. This was not done in the current study in an attempt to keep the experimental
procedure as similar as possible to those used with nonhumans.
Although not a limitation of the current study per se, another area for future research with a typically developing human population is to extend these procedures to more closely approximate verbal operant behavior. Because so much of human behavior is verbal, including many problem behaviors and the therapeutic interaction itself, investigating the effects of varying rates of reinforcement on the suppression and relapse of verbal behavior would be an especially important area of inquiry. Also, investigating resurgence effects within clinical populations, both in analogue and applied experimental contexts, may reveal important differences between clinical and nonclinical populations with regard to levels of suppression and relapse of problem behavior.

A final area for future research is the investigation into which variables attenuate the resurgence effect and whether these are feasible to incorporate into treatment protocols. A number of variables have already been explored in the literature, including manipulations of target acquisition (Phase 1) variables, such as length of target response training (Bruzek et al., 2009; Uhl, 1973), target reinforcement rates (da Silva, Maxwell, & Lattal, 2008, Experiment 1), and target response rates (da Silva et al., 2008; Reed & Morgan, 2007). While the effects of these variables on resurgence are arguably better understood than target response suppression or alternative response acquisition (Phase 2) factors, they are less likely to be easily manipulated in a therapeutic context, as they represent an individual’s learning history prior to entering treatment. Some Phase 2 factors that have been explored in the animal literature include thinning schedules of alternative reinforcement (Sweeney & Shahan, 2013; Winterbauer & Bouton, 2012), increasing the length of target response extinction and alternative response acquisition
(Leitenberg et al., 1975, Experiment 4; Lieving & Lattal, 2003), and exposing subjects to alternating alternative response acquisition and extinction conditions (Wacker et al., 2013). Of these, extending target extinction and alternative acquisition, or increasing the length of treatment in a clinical setting, may be a promising possibility. Other possibilities include increasing the number of alternative behaviors and/or sources of alternative reinforcement and assessing the effects of quality and magnitude of alternative reinforcement on level of resurgence.
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