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Laboratory Models of Treatment Relapse and Mitigation Techniques

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Behavioral treatments arranging differential reinforcement effectively treat severe problem behavior while interventions are underway. However, many events challenge these treatments clinically, thereby producing relapse of problem behavior. This paper reviews six laboratory models of treatment relapse for their relevance to understanding the processes underlying treatment relapse, including resurgence, reinstatement, rapid reacquisition, disinhibition, and spontaneous recovery. In addition, we also discuss clinical examples resembling these models and accompanying effects, as well as studies examining the combined effects of these laboratory models. Finally, we describe approaches developed in laboratory studies to mitigate treatment relapse using a variety of approaches involving both antecedent and consequence-based interventions. This research provides a foundation from which basic and clinical researchers can collaborate to establish more durable treatments for problem behavior.

Keywords: laboratory models, treatment relapse, behavioral treatment, relapse mitigation

Reinforcement-based behavioral treatments are remarkably effective in eliminating problem behavior. However, translational research reveals these treatments could make behavior more persistent and susceptible to relapse. This paper reviews research relevant to understanding factors influencing relapse of problem behavior. Specifically, we review laboratory models of relapse used to simulate environmental factors contributing to breakdowns in efficacy of behavioral treatments for problem behavior, as well as potential approaches to mitigating these problems.

Behavioral Treatments for Problem Behavior

The most commonly used treatment for problem behavior is differential reinforcement of alternative behavior (DRA). The effectiveness of DRA has been demonstrated across a wide range of problematic behavior, including aggression, pica, and self-injury (see Petscher, Rey, & Bailey, 2009, for a review). Typically, the first step in implementing DRA is to identify the reinforcer maintaining problem behavior through assessment procedures, including functional analyses (Iwata, Dorsey, Slifer, Bauman, & Richman, 1994). Next, DRA delivers the functional reinforcer at a high rate contingent upon an appropriate behavior and is typically implemented in combination with extinction of the problem behavior (Hagopian, Fisher, Sullivan, Acquisto, & LeBlanc, 1998). A commonly used form of DRA is Functional Communication Training (FCT), which trains a verbal communication response vocally or through other means (e.g., iPad, PECS) to request the reinforcer previously maintaining problem behavior (Durand & Carr, 1991; Fisher, Kuhn, & Thompson, 1998; Tiger, Hanley, & Bruzek, 2008). For example, self-injury maintained by escape from instructional demands can be decreased with FCT by training the client to request breaks from those demands. Thus, FCT teaches functional behaviors likely to be reinforced in the natural environment, providing the individual

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with socially appropriate approaches to obtaining reinforcers.

DRA has several advantages compared with the use of extinction alone, in addition to training the alternative response. Sometimes dangerous side effects of extinction (i.e., emotional behavior, extinction bursts, aggression) can be reduced using differential-reinforcement procedures (e.g., Fisher, Piazza, & Roane, 2011). Additionally, extinction procedures alone might be more sensitive to treatment-integrity errors, such as providing the reinforcer following problem behavior (e.g., Bouton & Trask, 2016). Further, the effectiveness of DRA has been demonstrated without extinction, as the use of extinction may not be feasible for dangerous behaviors (Athens & Vollmer, 2010).

Despite the efficacy of DRA and its advantages over the use of extinction alone, clinical studies typically do not demonstrate long-term maintenance and generalization of treatment effects (see Pritchard, Hoerger, & Mace, 2014a, for a discussion). Treatment relapse, defined as a return of problem behavior previously eliminated when a treatment is challenged, is commonly observed when using DRA procedures (Mace & Critchfield, 2010; Nevin & Wacker, 2013; Schieltz, Wacker, Ringdahl, & Berg, 2017). Understanding and developing solutions for mitigating treatment relapse benefits both individuals engaging in problem behavior, their caregivers, and society (see Podlesnik & Kelley, 2017, for a discussion). Fortunately, translational researchers are examining approaches to mitigate treatment relapse.

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Laboratory Models of Treatment Relapse

Certain events such as changes in context (e.g., new caregivers, transitioning from clinic to school) and changes in reinforcement (e.g., integrity errors, thinning schedule of alternative reinforcement) may make problem behavior likely to return. Laboratory models of relapse simulate these and other variables as a platform from which basic and applied researchers can examine factors contributing to treatment relapse. Furthermore, these models can serve as testing grounds for approaches to mitigate relapse when challenges to behavioral treatments occur (see Bouton, Winterbauer, & Todd, 2012; Podlesnik & Kelley, 2015).

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Table 1 shows six laboratory models of treatment relapse examining different variables potentially contributing to relapse. In all these models, the acquisition of a target response occurs when reinforced in Phase 1. Phase 1 simulates the acquisition and maintenance of problem behavior in a natural environment through contact with a reinforcement contingency (e.g., self-injury to escape from demands; aggression to access attention). In Phase 2, introducing extinction of target responding decreases the response, typically to near-zero levels. Phase 2 simulates the treatment condition designed to decrease problem behavior while the intervention is in place. Phase 3 introduces a manipulation causing the target response to return, usually transiently and below levels observed in Phase 1. Thus, Phase 3 simulates challenges to the behavioral treatment that can precipitate treatment relapse. These laboratory models

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Table 1
Laboratory Models of Treatment Relapse

Procedure	Phase 1: Acquisition	Phase 2: Treatment	Phase 3: Relapse test
Resurgence	R1+	R1−; R2+	R1−; R2−
Reinstatement	R1+	R1−	R1−; Free (or R2+)
Rapid reacquisition	R1+	R1−	R1+
Renewal	A: R1+	B: R1−	A or C: R1−
Disinhibition	R1+	R1−	R1−; novel stimuli
Spontaneous recovery	R1+	R1−	Time off . . . R1−

Note. In laboratory models of treatment relapse, acquisition in Phase 1 involves the reinforcement of a target response (R1+). Phase 2 simulates treatment through the elimination of target responding in Phase 2 with extinction (R1−). Phase 3 constitutes the relapse test, which differs across models. See text for details.

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largely stem from laboratory research examining fundamental learning processes involved in conditioning and extinction (see Bouton et al., 2012; Lattal, St. Peter, & Escobar, 2013; McConnell & Miller, 2014, for reviews). Specifically, the recurrence of target responding in Phase 3 reveals that elimination of target responding with extinction in Phase 2 did not destroy or erase the relations learned during reinforcement in Phase 1.

The models appearing in Table 1 bear some resemblance to factors influencing long-term effectiveness of DRA treatments. In the following sections, we will describe research examining these laboratory models of treatment relapse and their relevance to DRA treatment. Some models have been examined more extensively than others for their relevance to understanding DRA. Therefore, the review of some laboratory models will require the omission of much relevant research while others still require a great deal of further examination and translation.

Resurgence

Resurgence is a laboratory model of relapse receiving the most attention because of procedural and apparent functional similarities with behavioral treatments (see St. Peter, 2015). Resurgence has been defined in numerous ways (see Lattal et al., 2017) but occurs when reducing or eliminating reinforcement for an alternative response increases a previously reinforced and extinguished response. The attention given to resurgence by applied researchers and clinicians stems from the fact that the most typical resurgence procedure integrates DRA plus extinction of the target response into Phase 2. As Table 1 shows, the defining feature of the standard resurgence procedure is that Phase 2 involves extinction of the target response while simultaneously reinforcing an alternative response (i.e., DRA with extinction). Epstein (1983) coined the term "resurgence" over a decade after Leitenberg and colleagues first demonstrated the phenomenon (Leitenberg, Rawson, & Bath, 1970). Nevertheless, Leitenberg et al. introduced this line of research explicitly to examine whether behavioral treatments employing alternative sources of reinforcement decrease the total number of target responses when compared with introducing extinction of target responding alone. Resurgence effects suggest they might not.

In an early and representative example of resurgence, Leitenberg, Rawson, and Mulick (1975) reinforced target-lever pressing in rats during Phase 1 according to a variable-interval (VI) 30-s schedule of food presentations. Once target responding occurred at high rates across daily sessions, Phase 2 examined DRA by arranging reinforcement for pressing a different lever according to a fixed-ratio (FR) 10 schedule and extinction of target responding. Target response rates reached near-zero levels while alternative response rates increased across sessions. Finally, extinction of alternative responding produced an increase in target responding followed by a progressive decline across sessions. An important insight from these findings was that there was no overall benefit of DRA compared with extinction alone—there were approximately equal numbers of target responses between Phases 2 and 3, regardless of whether DRA was introduced and eliminated versus when extinction of target responding occurred from the onset of Phase 2.

The findings of Leitenberg et al. (1975) with rats are representative of the most common preparation of resurgence, which is the recurrence of target responding upon introducing extinction of both target and alternative responding in Phase 3. Similar findings have been observed in a range of species under laboratory conditions, including Siamese fighting fish (da Silva et al., 2014), zebrafish (Kuroda, Mizutani, Cançado, & Podlesnik, 2017a, 2017b), pigeons (e.g., Craig & Shahan, 2016; Podlesnik & Kelley, 2014), monkeys (Mulick, Leitenberg, & Rawson, 1976), typically developing humans (e.g., Kuroda, Cançado, & Podlesnik, 2016), and individuals with developmental disabilities (e.g., Reed & Clark, 2011). Resurgence also has been examined under a range of less traditional procedures and situations, including resurgence of response sequences (e.g., Bachá-Méndez, Reid, & Mendoza-Soylovna, 2007; Okouchi, 2015; Reed & Morgan, 2006), within-session assessments (Bai, Cowie, & Podlesnik, 2017; Lattal et al., 2017), under conditions of maintained reinforcement for target responding (e.g., Bouton, Thrailkill, Bergeria, & Davis, 2017), manipulating psychological state via mindfulness training (e.g., McHugh, Procter, Herzog, Schock, & Reed, 2012), with derived stimulus

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relations (Doughty, Cash, Finch, Holloway, & Wallington, 2010; Wilson & Hayes, 1996), with negatively reinforced behavior (Alessandri & Cançado, 2017; Bruzek, Thompson, & Peters, 2009), and in Pavlovian-conditioning procedures (Epstein & Skinner, 1980; Kearns & Weiss, 2007; Lindblom & Jenkins, 1981). Thus, the resurgence is a robust and general phenomenon.

One limitation of most laboratory models of DRA is the availability of alternative reinforcement goes unsignaled but often is signaled when implementing FCT (see Tiger et al., 2008). Therefore, assessing whether resurgence occurs in clinical situations is necessary to establish the relevance of basic studies of resurgence for understanding relapse with DRA treatments generally. The generality of resurgence has been demonstrated with problem behavior in clinical studies of DRA with individuals diagnosed with developmental disabilities (e.g., Berg et al., 2015; Fuhrman, Fisher, & Greer, 2016; Hoffman & Falcomata, 2014; Lambert, Bloom, Samaha, Dayton, & Rodewald, 2015; Lieving, Hagopian, Long, & O'Connor, 2004; Volkert, Lerman, Call, & Trosclair-Lasserre, 2009; Wacker, Harding, Berg, Lee, et al., 2011; Wacker, Harding, Morgan, Berg, et al., 2013). For example, Volkert et al. used functional analyses to identify the function of aggressive problem behavior across three children and found it to be socially maintained by positive and negative reinforcement. Initially, all instances of problem behavior were reinforced across sessions to simulate natural reinforcement contingencies. Next, FCT with extinction resulted in an increase in alternative behavior (card pulling, signing, vocal request) and decreases in problem behavior. Finally, extinction of both alternative and problem behavior typically resulted in a resurgence of problem behavior defined as an increase in rate compared with rates of problem behavior observed during FCT. Therefore, these findings demonstrate the resurgence of clinically relevant problem behavior when the FCT treatment is challenged with extinction (see Nevin & Wacker, 2013; Schieltz et al., 2017, for relevant discussions).

The similar procedures and findings between basic and clinical research lends support for the use of the resurgence procedure for examining factors contributing to the long-term effectiveness of DRA treatments. The removal of alternative reinforcers in Phase 3 of a resurgence procedure simulates treatment-integrity errors resulting in the omission of reinforcers delivered contingent upon the appropriate alternative response (see Podlesnik & Kelley, 2015; St. Peter, 2015, for discussions). In clinical situations, it is unlikely that treatment integrity would be so compromised as to remove of alternative reinforcement so completely but the manipulation serves to provide a robust effect from which to assess factors contributing to resurgence. For example, greater training reinforcement rates tend to produce greater resistance to DRA and greater resurgence (e.g., Kuroda, Cançado, & Podlesnik, 2016; Podlesnik & Shahan, 2009; see Podlesnik & DeLeon, 2015, for a review; but see Craig & Shahan, 2016; Nevin et al., 2016). In addition, a number of studies demonstrate that greater rates of alternative reinforcement more rapidly decrease target responding but produce greater resurgence than lower alternative reinforcement rates (e.g., Bouton & Trask, 2016; Cançado, Abreu-Rodrigues, & Aló, 2015; Craig, Nall, Madden, & Shahan, 2016; Craig & Shahan, 2016; Leitenberg et al., 1975; Sweeney & Shahan, 2013b; see also Pritchard, Hoerger, Mace, Penney, & Harris, 2014b, for clinically relevant behavior). Unfortunately, reviewing all the variables contributing to resurgence is outside the scope of the present paper (see Podlesnik & Kelley, 2015; Pritchard et al., 2014a; Shahan & Craig, 2017, for reviews).

Resurgence also has been demonstrated under circumstances in which alternative reinforcement was not completely withdrawn (e.g., Greer, Fisher, Saini, Owen, & Jones, 2016; Hagopian, Toole, Long, Bowman, & Lieving, 2004; Schepers & Bouton, 2015; Sweeney & Shahan, 2013b; Volkert et al., 2009; Winterbauer & Bouton, 2012; see also Jarmolowicz & Lattal, 2014). Thinning alternative-reinforcement rate is a strategy frequently used to make DRA treatments more manageable to caregivers and service providers compared with continuous-reinforcement schedules (see Hagopian, Boelter, & Jarmolowicz, 2011; Hanley, Iwata, & Thompson, 2001, for reviews). However, laboratory and clinical research cited above reveals the reduction in alternative reinforcement rate can result in increases in target responding above levels observed when DRA and extinction were in effect. Similar findings have been

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observed when reducing the magnitude of alternative reinforcement with pigeons (Craig, Browning, Nall, Marshall, & Shahan, 2017). Therefore, reducing alternative reinforcement in any way, either by design or through omission errors, appears to produce resurgence of target responding (see Lattal et al., 2017, for a relevant discussion). A "worsening" of conditions of alternative reinforcement produces resurgence (Shahan & Craig, 2017).

Reinstatement and Rapid Reacquisition

Whereas resurgence provides a model of omission errors, another laboratory model of relapse simulates conditions contributing to a different type of threat to treatment integrity—*commission* errors (see Pritchard et al., 2014a). Specifically, *reinstatement* models contribute to our understanding of how delivering the reinforcer previously maintaining target responding can contribute to relapse of previously eliminated target responding (see Reid, 1958, for the first demonstration of operant reinstatement). Table 1 shows a typical reinstatement procedure. Phase 1 arranges reinforcement contingent upon a target response, thereby simulating reinforcement for problem behavior in natural environments. Phase 2 arranges extinction of target responding, simulating treatment by arranging extinction of problem behavior. No alternative response typically is available with reinstatement procedures. In Phase 3, the reinforcer previously maintaining target responding typically is presented response independently (see Reid, 1958; Rescorla & Skucy, 1969) but some studies present a subset of the reinforcing events response dependently. For example, some studies present only a small number of reinforcers response dependently (e.g., Podlesnik & Shahan, 2009), whereas others present only the stimuli previously paired with primary reinforcers in Phase 1, sometimes response dependently (e.g., Acosta, Thiel, Sanabria, Browning, & Neisewander, 2008) or response independently (Sutton et al., 2003). Regardless of how reinstating events are scheduled, reinstatement effects are typically incomplete—target response rates do not reach levels observed in Phase 1. Much of the research examining reinstatement with operant behavior follows decades of research examining reinstatement of Pavlovian conditional responding (see Bouton, 2002, 2004; McConnell & Miller, 2014, for reviews). Operant reinstatement models are employed frequently in basic studies of extinction learning (e.g., Bai et al., 2017; Baker, Steinwald, & Bouton, 1991; Delamater, 1997; Miranda-Dukoski, Bensemann, & Podlesnik, 2016; Ostlund & Balleine, 2007) and laboratory models of drug use (Valles, Rocha, & Nation, 2006) and overeating (e.g., Calu et al., 2013). The variety of different procedures and species examined at least rivals that of resurgence studies, including zebrafish (Kuroda, Mizutani, Cançado, & Podlesnik, 2017b), rats (e.g., Franks & Lattal, 1976), pigeons (e.g., Doughty, Reed, & Lattal, 2004), monkeys (Stretch, Gerber, & Wood, 1971), and humans (e.g., Spradlin, Fixsen, & Girarbeau, 1969; Spradlin, Girardeau, & Hom, 1966).

Clearly, the extinction-only "treatment" during Phase 2 of reinstatement models differs from procedures in place during DRA treatments. Specifically, absent is the reinforcement of an alternative response, that is, DRA. The relevance of reinstatement to understanding relapse with DRA treatments is, first, to demonstrate that reexposure to reinforcers delivered previously could instigate relapse of problem behavior. Second, reinstatement provides a platform from which to examine variables contributing to this type of relapse effect (see Lattal et al., 2013; Pritchard et al., 2014a; Shaham, Shalev, Lu, de Wit, & Stewart, 2003, for reviews). We discuss findings from the reinstatement procedure here to understand how reinforcer delivery following DRA treatment could produce relapse of problem behavior.

Reinstatement effects have been interpreted as the reinforcer delivery in Phase 3 serving as a discriminative stimulus by contacting the reinforcement history established in Phase 1 (see Baum, 2012; Ostlund & Balleine, 2007; Podlesnik & Fleet, 2014; Rescorla & Skucy, 1969; cf. Delamater, 1997). For example, Franks and Lattal (1976) reinforced lever pressing with food in rats. In one condition, Phase 1 maintained high rates of responding according to a variable-ratio (VR) schedule. Following extinction in Phase 2, they arranged response-independent food presentations according to a fixed-time (FT) 30-s schedule during Phase 3. The FT schedule reinstated lever pressing at high rates. In another condition, by contrast, Phase 1 maintained lower rates of responding according to a differ-

ential-reinforcement-of-low-rates (DRL) schedule. Following extinction in Phase 2, arranging the same FT schedule reinstated response rates to lower levels compared with the test following Phase 1 training with the VR schedule. These findings are consistent with the interpretation that the reinstating food presentations increased responding in Phase 3 through discriminative-stimulus effects established in Phase 1 (see also Doughty et al., 2004; Rescorla & Skucy, 1969).

Returning to the role of commission errors during DRA treatment, the reinstatement model isolates the role of the reinforcer contingent upon problem behavior during the acquisition and maintenance of problem behavior. The link between commission errors and reinstatement effects is not as well defined as the link between resurgence and omission errors. Eliminating or reducing alternative reinforcement comprises both omission errors and the primary method for examining resurgence (see St. Peter, 2015). Reinstatement, on the other hand, does not resemble commission errors so closely. Commission errors occur as instances of reinforcing the previously eliminated problem behavior. Reinstatement, however, is typically examined by presenting *response-independent reinforcers* while target responding remains in extinction. Thus, no *response-dependent reinforcement* typically is provided in most standard reinstatement tests (cf. Podlesnik & Shahan, 2009; Acosta et al., 2008), whereas response-dependent reinforcers contingent upon problem behavior generally comprise commission errors (see St. Peter Pipkin, Vollmer, & Sloman, 2010).

We know of no direct evidence suggesting response-dependent and -independent reinstatement procedures differ qualitatively in process (see Baum, 2012; Rescorla & Skucy, 1969). In one empirical demonstration of the similarity between procedures, Podlesnik and Shahan (2009) assessed reinstatement by presenting response-independent or response-dependent reinforcers following extinction with pigeons. For one assessment of reinstatement, they arranged two response-independent food presentations 2 and 8 s into sessions (see also Nevin et al., 2016). The other assessment arranged two response-dependent food presentations contingent upon the first two target responses. Although response-dependent food presentations produced greater reinstatement, reinstatement patterns were nearly identical otherwise. Introducing the reinforcer during Phase 3 in both procedures functions as a discriminative stimulus for the reinforcement history established in Phase 1 (see also Bouton & Swartzentruber, 1991), with the greater response rates with response-dependent reinstatement due to greater response-reinforcer contiguity. These findings imply reinstatement models assessed with response-independent food presentations could be a useful for assessing how commission errors impact problem behavior eliminated with DRA treatment.

The relevance of reinstatement has been demonstrated in several applied studies examining treatment of problem behavior (e.g., DeLeon, Williams, Gregory, & Hagopian, 2005; Pritchard et al., 2014b). DeLeon et al. determined that adult attention maintained aggressive behavior of an individual diagnosed with an unspecified developmental disability In procedures closely resembling basic studies of reinstatement, they arranged continuous reinforcement of aggressive behavior with adult attention in Phase 1. Phase 2 arranged extinction of problem behavior by ignoring all instances of problem behavior (no DRA was present) and aggression decreased to zero levels. In Phase 3, the therapist provided 5 to 10 s worth of attention according to a FT 60-s schedule while continuing to ignore all instances of aggression. Despite extinction remaining in effect in Phase 3, problem behavior reinstated to near Phase 1 levels.

In a related study, Pritchard et al. (2014b) established that aggression and disruption were maintained by attention in a teenage boy diagnosed with a developmental disability. Phase 1 consisted of reinforcing problem behavior according to equal VI 60-s schedules in both of two components of a multiple schedule. In Phase 2, therapists arranged extinction of problem behavior and arranged a modified DRA in both components to maintain programmed rates of alternative reinforcement. They implemented a variable schedule to initiate a prompt to provide a discriminative stimulus for the boy to engage in a communication response to receive access to attention. A communication response resulted in 10-s access to attention. Failure to respond to the prompt resulted in 10-s access to attention response independently. They arranged reinforcement once every 30 s in the

richer component and once every 120 s in the leaner component. They reported to examine reinstatement by reinitiating the VI 60-s schedules of reinforcement for instances of problem behavior. Problem behavior increased in both components but to a greater extent in the richer component, consistent with findings reviewed above observing greater resurgence following greater alternative reinforcement rates with DRA (e.g., Craig & Shahan, 2016; Nevin et al., 2016).

The procedures of Pritchard et al. (2014b) differ from those discussed above in they completely return to reinforcement conditions arranged in Phase 1. Specifically, the so-called "reinstatement" model used by Pritchard et al. technically was an operant *rapid reacquisition* test, as shown in Table 1 (e.g., Crossley, Horvitz, Balsam, & Ashby, 2016; Willcocks & McNally, 2011; Woods & Bouton, 2007; see Bouton et al., 2012, for a review). These procedures are relevant for understanding relapse of problem behavior because commission errors are instances of reinforcing problem behavior (see St. Peter Pipkin et al., 2010). Rapid reacquisition tests reveal that reintroducing the original contingencies following extinction produces faster acquisition compared with initial acquisition, both in studies of operant (Woods & Bouton, 2007) and Pavlovian (Bouton, Woods, & Pineño, 2004) conditioning. Nevertheless, the issue here mirrors the discussion above comparing response-independent and response-dependent reinforcers in reinstatement. Specifically, reintroducing Phase 1 reinforcement conditions in rapid reacquisition is attributed to the discriminative effects of reestablishing the previous reinforcement context as demonstrated by the more rapid acquisition in Phase 3 (see Bouton et al., 2012). Thus, the reinstatement and rapid reacquisition procedures both contribute to our understanding of treatment relapse through discriminative, or signaling, effects of reinforcing events (see Baum, 2012, for a relevant discussion).

Resurgence, reinstatement, and rapid reacquisition focus on how reinforcement contingencies come to influence the likelihood of treatment relapse and are relevant to the effects of treatment-integrity errors with DRA treatments (see Kelley, Lerman, & Van Camp, 2002; Shirley, Iwata, Kahng, Mazaleski, & Lerman, 1997; St. Peter Pipkin et al., 2010; Worsdell, Iwata, Hanley, Thompson, & Khang, 2000, for relevant clinical examples). Specifically, they model breakdowns in treatment integrity through omission and commission errors involving reinforcement contingencies. Therefore, these studies could be argued to be pursuing processes relevant to *reinforcer control* over treatment relapse (see Podlesnik & Kelley, 2014, 2015).

Contingencies, however, are not the only influence on treatment effectiveness. Other relapse models focus more on how antecedent variables come to influence relapse, primarily in the form of contextual variables present during and after treatment (see Podlesnik et al., 2017; Pritchard et al., 2014a, for reviews). This research, to which we turn next, pursues processes more relevant to understanding how processes relevant to *stimulus control* contribute to treatment relapse (see Podlesnik & Kelley, 2014, 2015).

Renewal

Severe problem behavior often is treated in clinical settings to make available personnel and resources unavailable under more natural conditions. As a result, behavioral treatments implemented in clinical settings typically are very effective because they can be implemented with high fidelity (see Petscher et al., 2009). Appropriately, successful treatment often comes with less restrictive environments for clients, such as returning to home, school, going on outings, or transitioning to foster care. Renewal models of treatment relapse examine how changes in environmental context influences operant behavior eliminated by extinction (see Podlesnik et al., 2017, for a review). Therefore, renewal models can simulate transitions away from clinical settings in which treatment was implemented successfully and into familiar or novel settings. Findings from studies of renewal allow researchers to understand how those changes influence treatment relapse and provide a platform to examine approaches to improve treatment effectiveness during transitions (see Podlesnik & Kelley, 2017, for a discussion). AQ: 6

Table 1 shows two typical renewal procedures. Phase 1 arranges reinforcement of a response in one Context, A. This phase simulates the acquisition and maintenance of problem behavior due to a reinforcement history under

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natural conditions (e.g., home). Phase 2 arranges extinction of that response in a different Context, B. This phase simulates the initiation of an intervention to eliminate problem behavior in a clinical setting. In Phase 3, returning to Context A or transitioning to a novel Context C results in a transient but reliable increase in responding despite extinction remaining in effect. Returning to Context A simulates returning to a home setting (i.e., ABA renewal) while transitioning to the novel Context C simulates transitioning to a novel setting, such as a foster home (i.e., ABC renewal)—both retain high treatment fidelity during context changes (see Podlesnik et al., 2017, for a detailed discussion and review).

In a representative series of experiments with rats, Bouton, Todd, Vurbic, and Winterbauer (2011) arranged changes in context across phases by changing the location, visual, olfactory, and tactile cues of the operant chambers (see Podlesnik & Kelley, 2015, for a discussion). In Phase 1, they reinforced lever pressing with food according to a VI 30-s schedule in Context A. In Phase 2, they extinguished lever pressing in Context B. In Phase 3, they moved a group of rats back to Context A (i.e., ABA renewal) and others to Context C (i.e., ABC renewal) while extinction remained in effect. In yet another group of rats, they trained and extinguished in the same context, A, across Phases 1 and 2, followed by the renewal test in a novel Context, C (i.e., AAC renewal). In all three groups, responding reliably increased upon changing context in Phase 3, although effects were greatest with the ABA group (see McConnell & Miller, 2014, for a review; cf. Todd, 2013). The relapse effect observed when transitioning to novel contexts with ABC and AAC renewal reveal the robustness and importance of research on renewal. Specifically, relapse occurs through context changes comprising not only of simply returning to contexts previously associated with reinforcement but also when transitioning to contexts previously unrelated to obtaining reinforcement (see Bouton & Todd, 2014; Trask, Thrailkill, & Bouton, 2017, for discussions).

Renewal effects are robust and general across species and experimental situations (see Bouton et al., 2012; McConnell & Miller, 2014; Podlesnik et al., 2017, for reviews). They were initially demonstrated with Pavlovian conditioning (e.g., Bouton & Bolles, 1979) and shown to be relevant to studying factors related to memory (Bouton, 1993), drug use (Marchant, Li, & Shaham, 2013), anxiety (Vervliet et al., 2013), and overeating (Boutelle & Bouton, 2015). Renewal of operant behavior has been demonstrated in zebrafish (Kuroda et al., 2017b), rats (Crombag & Shaham, 2002; Nakajima, Tanaka, Urushihara, & Imada, 2000), pigeons (Berry, Sweeney, & Odum, 2014; Podlesnik & Shahan, 2009), and humans (Kelley, Liddon, Ribeiro, Greif, & Podlesnik, 2015). Using a clinically relevant population, Kelley et al. demonstrated ABA renewal with mastered academic tasks (letter/number tracing, sorting) in two children diagnosed with developmental disabilities. Color of task materials (e.g., experimenter t-shirt, placemat, etc.) defined the contexts across phases. In Phase 1, the response was reinforced in the presence of Context A until responding occurred reliably. Phase 2 introduced extinction in Context B until responding decreased to zero. In Phase 3, they returned to Context A with extinction still in place. Responding increased transiently for both participants. Kelley et al. replicated these effects in a group of pigeons to demonstrate the generality of these effects under laboratory conditions.

A number of applications of DRA and FCT demonstrate instances of renewal of problem behavior when transitioning out of a clinical setting as part of treatment (see Podlesnik et al., 2017, for a review). For example, Schindler and Horner (2005) implemented FCT with extinction in a preschool room to decrease aggression and tantrums in children diagnosed with ASD. Problem behavior initially occurred both at home and in the classroom. Following successful treatment in the preschool classroom, Schindler and Horner assessed whether treatment effects would generalize to the home environment and a different classroom. Instead, problem behavior increased when transitioning to these environments. Therefore, changing environmental context and observing relapse of problem behavior resembles ABA when returning to the home setting and AAC renewal when transitioning to the novel classroom. A number of other studies have observed similar findings in which problem behavior successfully treated with DRA increases when changing to a different setting but maintaining DRA (e.g., Durand & Carr, 1991; Hagopian, González, Rivet,

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Triggs, & Clark, 2011; Pritchard et al., 2016; Wacker et al., 2005).

A related finding from laboratory studies demonstrating the control by stimuli over relapse of operant behavior is called *disinhibition* (e.g., Brimer, 1970a, 1970b, 1972; Hearst, Franklin, & Mueller, 1974; see also Pavlov, 1927). Table 1 shows a typical procedure to study disinhibition. Phase 1 involves reinforcing a response and Phase 2 and 3 extinguish that response. In addition, Phase 3 involves presentation of novel stimuli resulting in temporary increases in responding. For example, Brimer (1970a) reinforced lever pressing in rats according to a VI 150-s schedule with food during Phase 1. Phase 2 introduced extinction of lever pressing until responding reached zero levels. Phase 3 comprised of continued extinction and response-independent presentations of white noise or a light for 1.5 s or 3 min. Increases in response rates occurred with both stimuli but were greater with the longer duration stimuli. Thus, the mere introduction of events can serve to increase extinguished responding.

Given the likelihood of individuals contacting a range of stimuli in any setting, the implication of disinhibition for treatment with DRA is clear. Problem behavior eliminated with DRA and extinction could relapse if the individual becomes exposed to novel stimuli, which could include new staff or loud noises (see Lerman & Iwata, 1996, for a discussion). Nevertheless, there are relatively few demonstrations of operant disinhibition. Therefore, additional exploration of the factors contributing to disinhibition and the relevance to behavioral treatment is warranted.

Spontaneous Recovery

The final relapse model we discuss is spontaneous recovery. As with many of the other models, Pavlov (1927) first described spontaneous recovery using respondent-conditioning procedures and has been used extensively to assess learning processes in extinction (see Rescorla, 2004, for a review). Spontaneous recovery of operant behavior occurs with time off from experimental sessions following extinction of a previously reinforced response. Table 1 shows the typical procedure for examining spontaneous recovery in laboratory situations. Phases 1 and 2 are identical to reinstatement and disinhibition, with reinforcement of target responding in Phase 1 and extinction of target responding in Phase 2. Once response rates reach near-zero rates in Phase 2, experimental sessions are no longer conducted for a specified amount of time. Upon reintroducing sessions of extinction after time off in Phase 3, spontaneous recovery occurs when response rates transiently increase.

In a laboratory study with rats, Bernal-Gamboa, Gámez, and Nieto (2017) demonstrated spontaneous recovery of lever pressing maintained by food as part of a larger study. Phase 1 reinforced target responding according to a VI 30-s schedule and extinguished target responding during Phase 2. Immediately after and five days after the final extinction session, tests for spontaneous recovery occurred during extinction. Response rates increased only during the test session following five days off from sessions, indicating spontaneous recovery. Spontaneous recovery has been demonstrated in laboratory models across a range of species, including rats (e.g., DiCiano & Everitt, 2002; Graham & Gagné, 1940; Rescorla, 1997; Troisi, 2003), pigeons (Rescorla, 2006; Thomas & Sherman, 1986; Zeiler, 1971), monkeys (e.g., Murphy, Miller, & Finocchio, 1956), and humans (e.g., López-Romero, García-Barraza, & Vila, 2010).

In a clinically relevant example, Lerman, Kelley, Van Camp, and Roane (1999) determined through functional analysis that access to positive reinforcers in the form of toys maintained screaming in a 21-year-old woman diagnosed with developmental disabilities. Instances of screaming were reinforced according to a FR-1 schedule in both of two components of a multiple schedule. They conducted three sessions each day all of one component type. Next, DRA in the form of hand clapping plus extinction of screaming was in place. Hand clapping resulted in 10-s access to toys in one component and 60-s access to toys in the other component. Although screaming eventually decreased to near-zero proportions of sessions in both components, spontaneous recovery of screaming accompanied the first session of each day in both components. Moreover, spontaneous recovery was greater in the component arranging DRA producing shorter access to toys. Thus, time off from treatment in the absence of context changes or breakdowns in DRA-treatment in-

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tegrity can produce increases in problem behavior in ways consistent with laboratory studies of spontaneous recovery.

Combinations of Relapse Procedures

Thus far, we reviewed laboratory and clinical research relevant to understanding events producing relapse of operant behavior through the models shown in Table 1. These models are useful for isolating particular variables likely contributing to instances of relapse. However, instances of treatment relapse under natural or clinical conditions likely reflect some combination of the variables comprising Table 1. Some laboratory studies examined the role of multiple processes in relapse by assessing combinations of these procedures. These studies assess whether combinations of variables could exacerbate relapse of problem behavior beyond any one variable on its own.

Resurgence and Reinstatement

Treatment-integrity errors with DRA treatments can take the form of failing to reinforce appropriate behavior (i.e., omission errors) or reinforcing inappropriate problem behavior (i.e., commission errors; see St. Peter Pipkin et al., 2010; Vollmer, Roane, Ringdahl, & Marcus, 1999). We discussed above how resurgence procedures simulate omission errors while reinstatement and rapid reacquisition procedures simulate commission errors (see also Podlesnik & Kelley, 2015; Pritchard et al., 2014a). Several studies are relevant to examining the effect of combining omission and commission errors (Bouton & Trask, 2016; Liggett, Nastri, & Podlesnik, in press; St. Peter Pipkin et al., 2010).

In a laboratory study with rats, Bouton and Trask (2016) reinforced pressing a target lever in Phase 1 according to a VI 30-s schedule, which resulted in one type of food pellet, or outcome (O1). In Phase 2, they extinguished target responding and reinforced a different lever according to a VI 30-s schedule with a different type of food pellet (O2). In Phase 3, they arranged extinction of both levers across groups. In the two relevant groups for these purposes, one group only received extinction (typical resurgence) while the other group also received response-independent presentations of O1 (resurgence and reinstatement tests). They found no difference in the increase in target response rates between the two groups, suggesting the O1 presentations did not contribute to relapse beyond resurgence due to withdrawing O2 presentations. Thus, combining the reinstatement procedure (i.e., commission errors) with the resurgence procedure did not enhance the overall relapse effect above resurgence alone (i.e., omission errors). Bouton and Trask attributed the lack of a combined effect due to generalization between the O2 and O1 reinforcers, which they confirmed in a following experiment. These findings suggest that combinations of procedures do not necessarily imply enhanced relapse (see also Sweeney & Shahan, 2015).

In contrast, translational and clinical work shows combinations of omission and commission errors do not enhance relapse of problem behavior over commission errors alone (e.g., St. Peter Pipkin et al., 2010; Vollmer et al., 1999). In a laboratory study with university students, St. Peter Pipkin et al. arranged conditions in which simulated DRA treatment on a computer screen by reinforcing an appropriate response and extinguished a previously reinforced target response. They simulated treatment integrity errors by (a) increasing the probability of reinforcement for target responses (i.e., commission errors), (b) decreasing the probability of reinforcement for alternative responding (i.e., omission errors), and (c) combining commission and omission errors. In contrast to the findings of Bouton and Trask (2016), St. Peter Pipkin et al. found omission errors did not contribute to increases in target responding when combining omission and commission errors. Commission errors appeared to be primarily responsible for relapse in target responding. These findings were consistent with a subsequent experiment with clinically relevant behavior in a child diagnosed with developmental disabilities (see also Vollmer et al., 1999).

Also investigating whether resurgence and reinstatement procedures combine to enhance relapse over either procedure alone, Liggett et al. (in press) arranged a laboratory study with three children diagnosed with developmental disabilities. In the first phase, they reinforced placing a ball in a box as the target response with access to small bites of preferred food to simulate reinforcement of problem behavior un-

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der natural conditions. In the next phase, they provided response-independent access to toys and arranged extinction of the target response to simulate treatment. Finally, they alternated between treatment and three relapse tests. They examined (a) reinstatement by presenting three response-independent food presentations, (b) resurgence by removing access to toys, and (c) a combination of response-independent food and removing toys. Unlike Bouton and Trask (2016) and St. Peter Pipkin et al. (2010), Liggett et al. observed greater relapse effects when combining the procedures than with either alone, with resurgence resulting in greater relapse than reinstatement when testing those procedures alone.

The findings reviewed above suggest multiple aspects of contingencies could potentially contribute to relapse of problem behavior with DRA treatments. It is likely that particular aspects of the variables comprising those contingencies and other environmental events will determine the ultimate contribution of errors of treatment integrity to relapse of problem behavior. Regarding contingencies, St. Peter Pipkin et al. (2010) arranged ratio schedules of reinforcement for commission errors but suggested other reinforcement schedules might result in different effects. In addition, discriminability of response-reinforcer relations between target and problem behavior could influence effects of treatment-integrity errors (see Davison & Nevin, 1999, for a relevant framework). Finally, longer DRA training at high treatment integrity could modulate the effects of different treatment-integrity errors (see Sweeney & Shahan, 2013a; Wacker et al., 2011). In terms of other environmental events, aspects like contextual changes, as observed in studies of renewal, could contribute to the influence of errors in treatment integrity. We turn here next.

Resurgence and Renewal

Laboratory studies of renewal (e.g., Bouton et al., 2011) and clinical findings (e.g., Schindler & Horner, 2005) suggest that treatment implemented in one context (e.g., clinical setting) can relapse when transitioning back to a pretreatment setting (i.e., ABA renewal) or to a novel setting (i.e., ABC renewal) even when treatment integrity remains high. Furthermore, several studies suggest combinations of contextual changes and omitting reinforcement of alterative responding can exacerbate relapse compared with either alone. In a laboratory study with rats, Trask and Bouton (2016) reinforced a target response according to a VI 30-s schedule with one type of food pellet (O1) in Context A during Phase 1. In Context B during Phase 2, they arranged extinction of target responding and presented a different type of food pellet (O2) response independently. During relapse tests, they continued O2 reinforcers for one group and removed them for another group. These two groups were further subdivided and assessed either by remaining in Context B or returning to Context A. Relapse was greatest when removing alternative reinforcers and returning to Context A, intermediate when only removing reinforcement or only changing context, and absent with retaining O2 and remaining in Context B. Thus, combining resurgence and renewal conditions enhanced resurgence beyond either alone (e.g., Kearns & Weiss, 2011; Kincaid, Lattal, & Spence, 2015; King & Hayes, 2016; Nakajima, Urushihara, & Masaki, 2002; see also Podlesnik & Kelley, 2014, 2015). As with combinations of resurgence and reinstatement (e.g., Bouton & Trask, 2016), however, combining procedures does not always enhance relapse compared with either procedure on their own (see Kearns & Weiss, 2007; Sweeney & Shahan, 2015).

Similar findings that combining resurgence and renewal can increase relapse has been observed clinically. In children diagnosed with developmental disabilities, Durand and Carr (1991) reduced problem behavior maintained by escape using FCT in a classroom setting. Problem behavior occurred at low rates for all participants while in effect in an original classroom and for two participants upon transitioning to a novel classroom after a break. For the third participant, entering the new classroom resulted in increases in problem behavior. Clarity of the communication response degraded, resulting in the teacher repeating instructions rather than reinforcing the communication response by providing help with tasks. Thus, the combination of context change and lack of reinforcement for appropriate behavior (omission errors) might have contributed to greater relapse of problem behavior than either in isolation. Retraining the participant to speak the communication response clearly in the novel classroom

again reduced problem behavior. This particular case reveals the potential complexity in isolating causes of treatment relapse—these findings suggest contextual changes could alter treatment efficacy through (a) renewal of the problem behavior, (b) poor generalization of the appropriate response to novel contexts, (c) omission of reinforcement for engaging in the appropriate response, or (d) a combination of these events.

Renewal and Rapid Reacquisition

In laboratory studies with rats, changing context can enhance reacquisition of an extinguished response compared with remaining in the extinction (treatment) context. In two experiments with rats, Todd, Winterbauer, and Bouton (2012a) reinforced lever pressing according to VI 30-s schedules during Phase 1 and extinguished lever pressing in Phase 2. Phase 3 reintroduced reinforcement of lever pressing according to a FR-5 schedule. Also in Phase 3, Experiment 1 returned to the training context (ABA) and Experiment 2 transitioned to a novel context (AAB). Both experiments compared contextual changes in Phase 3 with a control of remaining in the extinction context. In both experiments, responding reacquired faster when changing context compared with remaining in the extinction context (see also Willcocks & McNally, 2011). These findings suggest that transitioning out of the treatment context can increase the impact commission errors during DRA treatments.

Other than the rapid-reacquisition model, all these laboratory models of treatment relapse typically produce only transient increases in target behavior. This might suggest to some the processes underlying these models are of little clinical significance for the long-term efficacy of behavioral treatments. However, any increase in problem behavior increases the likelihood of commission errors, thereby further exacerbating the severity of relapse in problem behavior. It is for this reason that developing approaches to mitigate relapse during DRA treatments is an important pursuit for translational and clinical researchers (see Podlesnik et al., 2017; Podlesnik & Kelley, 2017, for relevant discussions).

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Procedures to Mitigate Treatment Relapse

A number of approaches have been developed to mitigate renewal through laboratory and clinical research. Typically, these approaches examine mitigation through a single type of relapse model at a time. Nevertheless, additional research could show the approaches reviewed below could be effective when combinations of environmental factors contribute to treatment relapse of problem behavior.

Extending Duration of DRA Treatment

One approach demonstrating effectiveness clinically is to extend the duration of DRA treatment (see Shahan & Craig, 2017, for a detailed discussion). In a resurgence procedure with rats, Leitenberg et al. (1975) arranged three, nine, or 27 sessions of DRA during Phase 2. Resurgence decreased with longer durations of DRA and the 27-session group was not different from an extinction control group. Similarly in a clinical study, Wacker et al. (2011) implemented FCT with eight children diagnosed with developmental disabilities and exhibiting destructive behavior (e.g., self-injury, aggression, property destruction) maintained at least in part by escape from demands identified with functional analysis. Following functional analyses, they implemented FCT and extinction of problem behavior. Introducing occasional extinction probes intermixed with sustained FCT generally showed reduced resurgence of problem behavior the longer FCT was in effect. In a laboratory study with rats, however, Winterbauer, Lucke, and Bouton (2013) found no differences in resurgence in Phase 3 after four, 12, and 36 sessions of DRA in Phase 2. Therefore, longer DRA treatments at minimum have no effect upon resurgence but might make DRA-treatment effects more durable (see also Laborda & Miller, 2013; Thomas, Vurbic, & Novak, 2009, for related findings).

Serial-DRA Training

Multiple topographies of behavior can occur as part of a response-class hierarchy both with clinically relevant problem behavior (e.g., Lalli & Mace, 1995; Lieving et al., 2004) and in laboratory research (e.g., Bachá-Mendez et al., 2007; Shabani, Carr, & Petursdottir, 2009; see also Bruzek et al., 2009). Specifically, several

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response topographies could potentially resurge when eliminating alternative behavior but the likelihood of resurgence of a given topography depends sequentially on the emission of other topographies. Hierarchies of functionally equivalent responses also have been trained with appropriate responses (e.g., Berg et al., 2015; Carr & Durand, 1985; Hagopian et al., 1998; Hoffman & Falcomata, 2014; Rooker, Jessel, Kurtz, & Hagopian, 2013; Tiger et al., 2008). In a laboratory study with adults with disabilities, Lambert et al. (2015) further observed that training multiple topographies in Phase 2 mitigated resurgence of target responding in Phase 3, when compared with training only a single alternative-response topography. Previously trained alternative responses resurged before target responding. Thus, "serial-DRA training" provides a promising approach to mitigating relapse with DRA treatment while establishing multiple appropriate responses as alternatives to problem behavior for accessing reinforcement (see Bloom & Lambert, 2015; Shahan & Craig, 2017, for discussions).

Punishment

Including punishers during DRA treatment with extinction can make the procedure more effective compared with more typical DRA treatment that includes extinction of target behavior (e.g., Fisher et al., 1993; Hagopian et al., 1998; Wacker et al., 1990). Punishment contingencies (e.g., temporary mild restraint) often are used when typical reinforcement-based DRA is unsuccessful. However, whether inclusion of punishers during DRA mitigates resurgence when removing alternative reinforcement is not entirely clear. In a laboratory study with rats, Kestner, Redner, Watkins, and Poling (2015) reinforced target-lever pressing according to a VI 30-s schedule in Phase 1. In Phase 2, they reinforced an alternative nose-poke response according to a VI 30-s schedule and extinguished target-lever pressing. One group also received footshock contingent upon all target responses. Phase 3 assessed resurgence by arranging extinction of both responses and removal of shock in the relevant group. Resurgence effects generally were small but arranging punishment in Phase 2 tended to decrease resurgence relative to no punishment in Phase 2. Thus, punishment could effectively reduce relapse during behavioral treatments (see also Boe & Church, 1967, for related findings).

In contrast, Rawson and Leitenberg (1973) found little difference between punished and unpunished target responding when modeling DRA treatment. They observed more rapid decreases when including punishment but no difference in resurgence across groups of rats when comparing DRA with extinction versus DRA with extinction plus punishment of the target response (see also Okouchi, 2015). Therefore, including punishers during DRA treatment might not always result in sustained reductions in problem behavior when challenging the treatment contingencies with challenges, such as omission errors.

Another condition of Rawson and Leitenberg (1973) is also relevant to understanding the role of punishment during treatment. In an additional group of rats receiving only extinction and punishment of target responding in Phase 2, the increase in target responding during the Phase 3 relapse test in extinction was similar to levels of resurgence in the other groups receiving DRA. Therefore, the punishment in the final group functionally signaled the onset of the extinction contingency in Phase 2 as revealed by the relapse effect upon withdrawing the punishment contingency in extinction during Phase 3 (see also Azrin & Holz, 1966; Estes, 1944). The implication is that punishers could serve a discriminative role for nonreinforcement, thereby producing relapse to target responding when removing punishers.

In a similar set of findings with renewal of punished behavior in rats, Bouton and Schepers (2015) reinforced target responding with food according to a VI 30-s schedule in Context A during Phase 1. In Phase 2, they continued to reinforce target responding while introducing a shock-punishment contingency according to a VI 90-s schedule in Context B. In Phase 3, removing the food and shock contingencies accompanied increases in target responding whether returning to Context A or transitioning to a novel Context C. In control groups, response-independent shocks delivered in Phase 2 did not systematically decrease target responding. Therefore, these findings support those of Rawson and Leitenberg (1973) and Azrin and Holz (1966) that events such as shock presented during treatment in Phase 2 could serve a dis-

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criminative function—removing those events could result in relapse of target responding. Thus, additional research is needed to examine the most effective approach to using punishment in the context of DRA treatments to mitigate, and not exacerbate, treatment relapse.

Signaling Alternative Reinforcement

Clinical implementation of DRA tends to arrange high reinforcement rates for engaging in an alternative behavior (e.g., FR 1). Greater alternative rates tend to be more effective in decreasing target responding but several basic and clinical studies reveal greater resurgence with greater alternative reinforcer rates (e.g., Craig & Shahan, 2016; Pritchard et al., 2014b). Therefore, Nevin et al. (2016) examined whether signaling the availability of alternative reinforcement influences the efficacy DRA when in place and during resurgence tests. With both pigeons and participants diagnosed with developmental disabilities, they arranged a fourfold difference in alternative reinforcement rates during DRA between multiple-schedule components. Further, they arranged a signal only when alternative reinforcement was available, consistent with many clinical interventions with DRA (e.g., Betz, Fisher, Roane, Mintz, & Owen, 2013). These procedures, however, were unlike many of the assessments of DRA and resurgence employing free-operant DRA schedules that did not include signals for reinforcement (e.g., Craig & Shahan, 2016; Podlesnik & Kelley, 2014). Overall, they found that signaling alternative reinforcement tended to produce no systematic difference across experiments in effectiveness of DRA when in place and less resurgence when discontinuing the leaner DRA than the richer DRA. They found similar patterns when assessing reinstatement. Therefore, these findings suggest that signaling leaner schedules of DRA reinforcement could reduce the likelihood of relapse following omission and commission errors (see also Bland, Bai, Fullerton, & Podlesnik, 2016, for related findings).

Treatment-Correlated Stimuli

Several laboratory studies mitigated relapse by arranging stimuli paired with extinction. Originally based on studies of respondent conditioning (Brooks & Bouton, 1993, 1994), presenting stimuli during operant extinction contingencies in Phase 2 mitigated relapse during testing in Phase 3 (Bernal-Gamboa, Gámez, & Nieto, 2017; Willcocks & McNally, 2014). In a study with rats partially discussed above, Bernal-Gamboa et al. reinforced a target response according to VI 30-s schedules during Phase 1. In Phase 2, extinction decreased target response rates while presenting a 5-s response-independent tone according to a VT 30-s schedule. During relapse testing either immediately or five days following Phase 2, extinction remained in effect. As described above, the test five days later produced spontaneous recovery of target responding but continuing to present the 5-s tone mitigated spontaneous recovery compared with withdrawing the tone. In addition, Bernal-Gamboa et al. also found the inclusion of the 5-s tone mitigated reinstatement of target responding. In a related study of alcohol self-administration in rats, Willcocks and McNally (2014) also found that 60-s tones throughout an hour-long session paired with extinction mitigated ABA renewal. The tones mitigated renewal whether the tones were presented response independently or dependently with the target response across experiments.

The laboratory findings with extinction cues suggest that including stimuli paired with DRA treatment conditions potentially could mitigate relapse of problem behavior across a range of situations. In an applied example, Fisher, Greer, Fuhrman, and Querim (2015) incorporated a multiple schedule with functional communication training and demonstrated generalization of treatment effects across contexts and therapists (see also Craig, Browning, & Shahan, 2017; Nieto, Uengoer, & Bernal-Gamboa, 2017; Trask & Bouton, 2016, for related findings).

Despite these promising effects with renewal, Willcocks and McNally (2014) found their tones did not reduce the rate of reacquisition of target responding in Phase 3 compared with the absence of the tone. Thus, reestablishing training conditions when returning to Context A appeared to overpower any mitigating effects of the treatment cues. These finding suggest that a complete breakdown in treatment integrity with regard to commission errors will be very disruptive to treatment (see also St. Peter Pipkin et al., 2010; Vollmer et al., 1999). Nevertheless, treatment cues might still increase the effectiveness of DRA treatments consisting of less com-

plete breakdowns in treatment integrity, as modeled with reinstatement by Bernal-Gamboa, Gámez, and Nieto (2017).

Multiple-Context Training

Findings from studies of renewal indicate that effective treatments might not generalize across situations (see Bouton et al., 2012; Podlesnik et al., 2017, for reviews). Therefore, studies of both respondent (e.g., Bouton, García-Gutiérrez, Zilski, & Moody, 2006; Chaudhri, Sahuque, Cone, & Janak, 2008; Gunther, Denniston, & Miller, 1998) and operant (Bernal-Gamboa, Nieto, & Uengoer, 2017) conditioning examined whether establishing extinction effects across multiple contexts could increase generalization of extinction's effects to training and novel contexts (see also Todd, Winterbauer, & Bouton, 2012b). In a laboratory study with rats, Bernal-Gamboa et al. reinforced lever pressing according to a VI 30-s schedule of food reinforcement in Context A during Phase 1. During Phase 2, they arranged extinction across different groups. Some groups received extinction in a single context (B) while others received extinction in three contexts (BCD). They found the multiple context training could mitigate renewal when returning to the training context (A) or transitioning to a novel context (E). However, groups returning to Context A required more extensive multiple-context training to mitigate renewal than those transitioning to Context E. Thus, the likelihood of treatment relapse of problem behavior with DRA treatment likely is going to be more severe and more resistant to relapse-mitigation techniques when returning to training contexts than transitioning to novel contexts (see also McConnell & Miller, 2014).

Combining Relapse-Mitigation Techniques

Falcomata and Wacker (2013) suggested combining techniques to promote generalization of FCT might promote its effectiveness. We suggest a similar approach with regard to the use of relapse-mitigation techniques. Multiple techniques could reduce the likelihood of treatment relapse compared with any one approach alone (see also Podlesnik et al., 2017).

In one clinical example employing treatment-correlated stimuli and multiple-context training along with punishment, Piazza, Hanley, and Fisher (1996) effectively eliminated severe cigarette-butt pica in a teenager diagnosed with developmental disabilities. The participant came from a home of chronic smokers and consumed cigarette butts both at home and around the community for approximately four years. In a clinical setting, it was determined that nicotine maintained pica. Therapists presented a purple card provided to signal treatment was in effects. Treatment consisted of noncontingent access to food while sternly stated "no butts!" when the participant attempted to pick up cigarette butts planted by experimenters.

Once pica was successfully eliminated in the original treatment room, Piazza et al. (1996) implemented treatment by including the purple card across multiple other contexts within the clinical setting (e.g., living area, office). Thus, they employed multiple context training while presenting the purple card as the treatment cue. Compared with a yellow control card, responding remained eliminated in the presence of the purple card across contexts. These effects generalized to a range of other novel settings in the community (e.g., on walks, mall), as well as when returning home.

Although Piazza et al. (1996) did not conduct a component analysis of the controlling treatment variables to determine relative contributions of the treatment components, they provide a useful example of how multiple mitigation techniques could be employed. As Falcomata and Wacker (2013) suggest with generalization techniques, the addition of mitigation techniques needs to be weighed along with feasibility and the possibility that multiple techniques could negatively affect treatment fidelity. These issues could be addressed with more research.

Theoretical Issues

Healthy debate exists about what theoretical framework best accounts for operant relapse effects (see Podlesnik & Kelley, 2015; Shahan & Craig, 2017; Trask, Schepers, & Bouton, 2015). Identifying the theory that best accounts for relapse effects can provide direction for future research and guide clinicians in developing treatments. Given the ongoing discussion detailed in previous reviews, we will only comment on this briefly.

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Context theory suggests all relapse effects are a result of a common set of processes (see Bouton & Todd, 2014; Trask et al., 2017, for reviews). Specifically, all forms of relapse result from environmental events serving as contextual stimuli associated with the available contingencies. Specifically, relapse effects occur because the effects of extinction are context specific, whereas training effects generalize far more readily. Thus, changes in environmental context with renewal models, contingencies with resurgence and reinstatement models, and so on, produce relapse because the extinction effects fail to generalize to the changed situation of the relapse test in Phase 3. This framework benefits from an extensive series of relevant studies and its parsimonious approach to explaining relapse. However, Context Theory has been criticized for its lack of quantitative rigor and falsifiability (see Shahan & Craig, 2017, for a discussion), although counterarguments exist (e.g., Trask et al., 2015).

Behavioral momentum theory, on the other hand, provides a quantitative theoretical framework for explaining relapse effects (see Nevin et al., 2017; Podlesnik & Shahan, 2010; Shahan & Sweeney, 2011). Reinforcement during training conditions in Phase 1 strengthens operant responding. Extinction and other contingencies (e.g., DRA) during Phase 2 serve to disrupt operant behavior. Relapse effects in Phase 3 are attributed to removal of disruptive conditions in Phase 3 (e.g., eliminating DRA, context changes). This theory precisely predicts how reinforcement and disruptive conditions impact relapse but the preponderance of quantitative theoretical tests have come from data sets involving resurgence. Unfortunately, the theory has not done well conceptually or quantitatively when accounting for resurgence (see Craig & Shahan, 2016; Nevin et al., 2017; Podlesnik & Kelley, 2015; Shahan & Craig, 2017, for critical reviews).

Resurgence as Choice (RaC) is a quantitative framework developed around quantitative models of choice (e.g., Baum, 1974; Baum & Rachlin, 1969; Killeen, 1972), which suggests reinforcer effects extending in time using a temporal weighting parameter (Devenport & Devenport, 1994). This framework has two problems. It has yet to be tested rigorously and has been developed in a way that currently is specific to resurgence (see also Bai et al., 2017). Therefore, it remains to be seen whether RaC adequately accounts for resurgence and other relapse effects upon testing.

Treatment Relapse Versus Induced Variability

Thus far, we have considered how extinction contingencies decrease responding and, with resurgence, can increase previously reinforced responses. Treatment relapse typically is defined as the increase in target response rates in Phase 3 (a) above levels observed at the end of Phase 2 and sometimes also (b) above levels observed on an unreinforced control response. The inclusion of a control response that does not get reinforced throughout all phases in some studies serves as a control to measure potential increases in variability induced by extinction or other decreases in reinforcement conditions (see Bolívar, Cox, Barlow, & Dallery, 2017; Kuroda et al., 2017b; Podlesnik, Jimenez-Gomez, & Shahan, 2006; Sweeney & Shahan, 2016). In laboratory studies, extinguishing a previously reinforced response can increase variability along a range of dimensions, such as response location, rate, or force (see Neuringer & Jensen, 2012, for a review). In clinical settings, extinction can change behavior in a number of ways, producing aggression, emotional behavior, extinction bursts, and alternative response topographies (see Lerman & Iwata, 1996, for a review). Therefore, including some form of control response is important for determining whether increases in target responding are, in fact, relapse effects due to the previous reinforcement history.

Many laboratory studies reveal relapse specific to previously reinforced responses, especially in studies using laboratory animals (e.g., Craig & Shahan, 2016; Kuroda et al., 2017b; Podlesnik et al., 2006). Other studies employing human participants, however, find commensurate increases in control responding in studies employing human participants, in particular (e.g., Bolivar et al., 2017; Sweeney & Shahan, 2016). It is unclear whether these differences are attributable to differences in procedure, motivation, learning history, or some interaction. For example, these procedures used to study resurgence in humans arranged responses spatially near one another on a computer screen in comparison to the spatially separated responses

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arranged in operant chambers used with nonhuman animals. Therefore, alternating among responses requires less effort for humans interacting with events on a computer screen. With regard to motivation, these participants earned points only (Bolivar et al., 2017) or points exchangeable for chance of earning a gift card (Sweeney & Shahan, 2016). Therefore, motivation for earning reinforcers is unclear relative to standardized deprivation present in most animal studies of relapse. With regard to learning history, humans typically have a long history of problem solving that includes the capacity to develop and follow rules. This history likely also includes experience interacting with and resolving issues with computers and other electronic devices. Thus, the combination of the procedures employed in studies with humans, complex learning history, and individual differences in motivation all could contribute to inconsistent relapse effects with human participants.

Bolivar et al. (2017) and Sweeney and Shahan (2016) point to the importance of arranging control responses in studies examining relapse processes. We agree. However, we encourage researchers to examine a range of approaches to assess variability beyond engaging in similar unreinforced control responses. As discussed above, extinction in natural conditions induces a range of other historically relevant, emotional, and/or aggressive responses. For these reasons, we interpret relapse effects to be a single form of variability comprising a multidimensional phenomenon (see Liggett et al., in press, for a relevant discussion). Understanding variability in all its dimensions is relevant to understanding behavioral processes evoked during these procedures used to study relapse, as well as for understanding the effects of potential challenges to appropriately implementing behavioral treatments.

Conclusion

We reviewed literature on laboratory models of treatment relapse relevant to understanding factors influencing the long-term effectiveness of DRA treatments (see Table 1). We also provided examples of clinical studies appearing to reflect those processes and procedures examined in the laboratory models. These examples suggest compelling evidence for engaging in research devoted to modeling the procedures employed in clinical situations to provide better understanding of variables involved in producing successful behavioral treatment. Further, these findings also suggest clinical studies could assess the efficacy of laboratory approaches to mitigating relapse. Overall, we argue for integrated translational research to develop conceptually systematic interventions for treating problem behavior (see also Mace & Critchfield, 2010; Podlesnik & Kelley, 2017; Podlesnik et al., 2017).

Finally, there are several different areas of research particularly ripe for additional work on understanding the relevance of these relapse models for DRA treatment. For example, much of the basic research on relapse-mitigation techniques arranges extinction as the "behavioral treatment" during Phase 2 (e.g., Bernal-Gamboa, Gámez, & Nieto, 2017; Bernal-Gamboa, Nieto, & Uengoer, 2017). Assessing the effectiveness of these techniques while modeling DRA treatment could provide greater insight into how to implement during clinical behavioral treatments. In addition, evaluating relevant theoretical frameworks, including Behavioral Momentum Theory, Context Theory, and Resurgence as Choice, is important for developing better understanding of the fundamental behavioral processes involved in relapse. AQ: 9

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