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Stepping Back from 'Persistence and Relapse' to See the Forest: Associative Interference

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Abstract

Historically, there has been considerable interest in a large variety of forms of associative interference. However, various factors including interest in clinical application and perhaps recent funding priorities have resulted in a narrowed focus on one particular instance of interference, extinction, with relative neglect of other types of interference. We have been using the existing literature and conducting new experiments to determine whether there is a consistent set of rules governing the occurrence and persistence of two-phase associative interference across (a) proactive and retroactive interference, (b) cue and outcome interference, (c) the type of training in phase 1 (excitatory, inhibitory, or simple nonreinforcement), and (d) the type of training in phase 2 (excitatory, inhibitory, or simple nonreinforcement). Our hope is that a return to more general questions concerning associative interference might reveal broad truths concerning the nature of forgetting. Identifying global principles of associative interference may also help us better appreciate the nature of extinction, including how it can be enhanced and made more enduring, as well as how it can be minimized and made more fleeting.

Keywords

cue interference; outcome interference; extinction; latent inhibition; relapse

1. Introduction

This paper is an outgrowth of a contribution to a theme of 'Persistence and Relapse' at the 2016 conference of the Society for the Quantitative Analysis of Behavior. For most researchers, such a theme evokes thoughts of some first-learned, reinforced 'target' behavior

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that is then decreased in magnitude or frequency of occurrence as a result of some second-learned training experience, often [but not always] extinction treatment, but extinction is merely one instance of a large family of related phenomena, collectively called associative interference. ‘Persistence’ usually refers to resistance of the first-learned behavior to being decreased, and ‘relapse’ refers to the reappearance of the first-learned behavior without further target training after it has been decreased. In this framework, testing is on the first-learned behavior, which is why in our terminology we would call it the ‘target’ behavior. Here we recast ‘persistence and relapse’ more generally as the stability of that interference.

Associative interference is a decrement in performance on a target learning task resulting from a specific memory conflict between memory of the target training and nontarget training events that have some similarity to target training, but also some unique components. Although under natural conditions, a target memory can suffer interference from multiple interfering memories, for analytic purposes in the laboratory, researchers usually restrict themselves to one target and one interfering memory. As training of each memory can involve multiple trials, in principle the two trial types could be interspersed. However, research suggests that interference is relatively small when the two trial types are interspersed (e.g., Pineno, Ortega, & Matute, 2000). This point is echoed by performance on a partial reinforcement schedule not differing much at asymptote from that on a complete reinforcement schedule (although differences in subsequent rates of extinction speak to the complexity of what is encoded during interspersed training trials). Thus, the present discussion is limited to two-phase associative interference, with target training taking place exclusively in phase 1 or exclusively in phase 2, with the interfering association being trained in either the subsequent or preceding phase, respectively. Often this reduces to an initial cue-outcome[1] relationship is presented during phase 1, and further training in phase 2 in which either the cue or the outcome has been replaced with a different one. A deficit in one’s ability to express the target cue-outcome association results from experience with similar (cue or outcome) but different (outcome or cue) information. That is, the components that are unique seem to disrupt one another’s influence on behavior. Presenting the cue from the target association at test presumably directly (for common cues) or indirectly (for common outcomes where the target cue activates a representation of the outcome which in turn activates the interfering association) initiates activation of both associations which results in the two types of training events being in conflict for control of behavior, (i.e. interference). Associative interference is such a basic phenomenon that it is observed in most every species that has been examined to date. Hence, here we freely mix data from multiple species, primarily humans and rats. Note that here we use ‘associative interference,’ which is the topic of this paper, to refer to the detrimental interaction of two associations that are independently trained, as distinct from ‘cue [or outcome] competition’ which refers to the detrimental interaction of two associations that are trained in compound (e.g., overshadowing; see Miller & Escobar, 2002, for elaboration).

[1] *Outcome* here refers to any stimulus that sequentially follows a cue, which is an antecedent stimulus in a sequential stimulus dyad. An outcome can be either biologically significant in which case we would refer to it as an unconditioned stimulus (US) or neutral in valence. To assess the activation of a neutral outcome by a cue after completion of phases 1 and 2 of a typical two-phase associative interference experiment, something must be done to give the representation of the outcome a response potential. Often this is done by pairing it with an unconditioned stimulus as in a sensory preconditioning procedure.

1.1 Order of training

Memories for initial events (i.e., first-learned relationships concerning stimuli) are often seen to be especially well preserved. That is, they are relatively resistant to disruption by later experiences (i.e., persistence). Behavior reflecting first-learned memories also have a tendency that, when they are disrupted, they often reappear at some later time (i.e., relapse). The disrupting treatment can take many forms, but often it is additional training that is in some way inconsistent with the initial learning. As long as the disruptive treatment is an additional learning event that contains at least some common feature with the target event, the situation can be viewed as a case of two-phase retroactive associative interference.

Retroactive associative interference refers to the second-learned association disrupting performance that is based on the first-learned association (i.e., test on the first-learned information). It is to be contrasted with proactive interference in which the test is on the second-learned information. Orthogonal to the distinction between proactive and retroactive interference, one can further distinguish between whether the conflict is between unique outcomes sharing a common cue (outcome interference), or between unique cues sharing a common outcome (cue interference). Of the four resultant different forms of associative interference, in recent years retroactive outcome interference has received the broadest attention in the pursuit of understanding extinction (e.g., Miller & Laborda, 2011).

1.2 History

Historically, there was an interest in many forms of associative interference; both retroactive and proactive, with [more commonly] outcomes or [sometimes] cues as the inconsistent elements receiving empirical attention (e.g., Miller & Escobar, 2002; Slamecka & Ceraso, 1960). However, a growing emphasis on applied research has in part motivated most researchers who were interested in associative interference to focus on interference within the extinction/relapse paradigm as it applies to clinical settings. This narrowed approach has surely aided practitioners, who treat various forms of deleterious behavior through one or another form of exposure-like therapy, in improving therapy and reducing the incidence of relapse from therapy. But this strong focus had the consequence of promoting understanding of only one specific type of associative interference (namely, retroactive outcome interference) to the detriment of any other form. The generality of the fundamental conflict that underlies the four forms of associative interference suggests the existence of general principles governing how the mind resolves the inherent conflict between inconsistent memories. Supportive of this view, there are numerous empirical parallels observed across the four forms of interference and observed across a variety of different species and preparations (discussed below). Determining the rules for associative interference in general, assuming that there are any, might inform our understanding of extinction, and more generally illuminate associative interference as a major source of forgetting. In an effort to highlight some of the possible commonalities below we will be drawing extensively from work with humans as well as non-human animal studies. Some of the findings to be discussed are well established, whereas others have relatively sparse empirical support and call for more research.

2. The ubiquitous nature of associative interference in forgetting

The suggested causes of forgetting are innumerable. But in our view, they can be categorized into four principal families of causes: 1) spontaneous decay, 2) inappropriate retrieval cues at test, 3) displacement from working memory by nontarget events at training or test, and 4) associative interference.

2.1 Spontaneous decay

'Spontaneous decay' is an odd name as it suggests that the passage of time alone is a candidate causal agent for forgetting; however, the passage of time alone is not a mechanism. Rather, it is processes, perhaps presently unidentified, that occur as time passes that are responsible. In the case of spontaneous decay of memory, ongoing metabolism, independent of specific experience, is presumably the causal factor. But many, not all, studies have found that so-called spontaneous decay arises in large part from interfering stimulus events that occur during the retention period (i.e., retroactive interference; e.g., Waugh & Norman, 1965) but sometimes prior to training (i.e., proactive interference; e.g., Keppel & Underwood, 1962). Granted, it is impossible to categorically reject the mere passage of time independent of specific stimulus experience as a contributing factor. Yet, it has become little more than a default account. A growing body of data suggests that associative interference is the actual basis of many instances of so-called spontaneous decay. Without acquisition of potentially interfering memories, little spontaneous decay is observed.

2.2 Inappropriate retrieval cues

With respect to 'inappropriate' retrieval cues, Tulving's widely cited encoding-retrieval specificity rule (Tulving & Thomson 1973; Tulving, 1985) correctly states that, all other things being equal, a worse match of retrieval cues with those of target learning will result in worse recall. However, Nairne (2002) modernized this tenet by demonstrating that the critical factor in recall is often not the absolute match between target training cues and retrieval cues, but the match of the retrieval cues to target training cues *relative* to the match of the retrieval cues to cues for interfering memories. Alternatively stated, a retrieval cue is apt to reactivate the target outcome provided there is not a nontarget outcome that is better associated with the retrieval cue. That is, as Tulving posited, the match of retrieval cues at test to those of the target memory is critical, but only relative to the match of the retrieval cues to potentially interfering memories. Thus, the inappropriateness of retrieval cues is a function of the content of potentially interfering memories.

2.3 Displacement from working memory

With respect to displacement or disruption of the target information from working memory during target training or test, there are seemingly clear instances that impact performance through nonassociative means (e.g., concussions). But in many situations, so-called disruption of processing the target information results from processing additional related material as opposed to totally unrelated information. That is, we must distinguish associative interference from general interference with information processing that is not stimulus specific. Critically, even seemingly unrelated nontarget events that occur temporally

proximate to target training or target retrieval share temporal time tags with the target information, and this similarity could evoke associative interference. Forgetting presumably due to displacement from working memory mechanisms seems to arise at least in part from associative interference in which common denominators of the interfering memory and the target memory include the contextual and temporal attributes of these two memories (e.g., Oberauer, Farrell, Jarrold, & Lewandowsky, 2016).

2.4 The centrality of associative interference

Thus, we see that many (not necessarily all) instances of the first three families of sources of forgetting (i.e., so-called spontaneous decay, inappropriate retrieval cues, and displacement from working memory) are actually forms of associative interference. Consequently, it appears that associative interference is the dominant (although not exclusive) source of typical forgetting. To better understand forgetting in general, a more thorough understanding of associative interference would be most informative.

3. What associative interference is and is not

Associative interference refers to a decrement in responding based on a target association as a result of the subject being trained on an inconsistent association. Consider training two separate associations (X-O1 and Y-O2), one in each of two separate phases of training, where X and Y are cues and O1 and O2 are outcomes. If the cues are the same and the outcomes are also the same, then enhanced performance would be expected because the nontarget phase of training is simply more target training. If X and Y are distinctly different and O1 and O2 are also distinctly different, then the YO2 training should have no impact of on responding to X that reflects X's association with O1. It is when the two memories have some elements in common (i.e., cues or outcomes) and other elements which differ (i.e., outcomes or cues) that associative interference is expected to be maximal. For example, X-O1 followed by X-O2 (where O2 differs from O1 and might even consist simply of the absence of O1 as in extinction) would be expected to result in interference. Granted, extra-stimulus features, such as the surrounding context or proximity in time, might serve to generate some degree of similarity, but those would have more muted and peripheral effects than similarities that exist within features of the target association (i.e., cues and outcomes). Additionally, some other part of the two associations could differ (e.g., with respect to the interstimulus interval [ISI], cue duration, or outcome duration; see Savastano & Miller (1998) for a review of the Temporal Coding Hypothesis that posits that associations include information concerning the spatiotemporal relationship of the paired stimulus events), but the elements are otherwise the same. An example of this would be a change in the timing of when the target response is emitted based on learning a new ISI within an otherwise identical X-O1 association. Conversely, interference can be decreased with differing ISIs if other attributes of the two associations also differ. The different cues combined with different ISIs seemingly make the two associations too dissimilar to allow for strong interference (Escobar & Miller, 2003).

3.1 The nature of the interfering training event

But is it training on the interfering association per se that actually decreases behavior indicative of the target X-O1 association? Indeed, counterconditioning is ordinarily more effective at producing a response decrement than mere extinction (Holmes, Leung, & Westbrook 2016; Escobar, Arcediano, & Miller 2001; Pavlov, 1927), but it remains unclear whether this applies to interference effects more generally. In an associative interference situation, it might be irrelevant that the interfering treatment actually consist of training that involves the formation of a novel association between two events. For illustrative purposes, consider the retroactive outcome interference observed when behavior reflecting X-O1 pairings in phase 1 is reduced by X-O2 pairings that occur in phase 2 (i.e., a counterconditioning design). Might exposure to X and O2 unpaired in phase 2 have produced the same decrement in behavior indicative of an X-O1 association at test as X-O2 pairings in phase 2? Obviously X-alone is extinction treatment and hence might be expected to attenuate behavior reflecting X-O1. No doubt, with sufficient X-alone trials, responding indicative of X-O1 will be reduced. But controlled experiments have demonstrated that X-O2 presentations are more often detrimental to behavior indicative of the X-O1 association than are X-alone (and O2 alone) presentations, at least until a floor effect is reached (e.g., Escobar, Arcediano, & Miller, 2001). Similarly, in retrospective cue interference (i.e., X-O1 followed by Y-O1), the Y-O1 pairings have been found to be more detrimental to behavior indicative of the X-O1 pairings than is exposure to Y and O1 unpaired in phase 2 (e.g., Escobar et al., 2001), an observation highly relevant to the selection of clinical behavioral interventions.

Even greater concern for the role of the putatively interfering association need be taken with proactive interference (i.e., X-O2 or Y-O1 in phase 1 followed by target training [X-O1] in phase 2). In proactive interference situations, presenting potentially interfering X-O2 trials in phase 1 in principle constitutes a form of latent inhibition treatment of X with respect to any future X-O2 association, and YO1 treatment in phase 1 in principle allows the establishment of a context-O1 association that is thought to underlie the US-preexposure effect. [The US-preexposure effect itself is often viewed as an instance of blocking of the X-O1 association by the context-O1 association; blocking is ordinarily viewed as a form of cue competition, but evidence indicates that a component of the blocking deficit is sometimes due to proactive interference (e.g., Vadillo, Castro, Matute, & Wasserman, 2008).] The question is whether the X-O2 or Y-O1 treatment in phase 1 produces more of a decrement in behavior reflecting the X-O1 pairings than does unpaired exposure to X and O2 or unpaired exposure to Y and O1? With sufficient phase 1 treatment, both latent inhibition and the US-preexposure effect are highly likely to occur. Teasing these contributing effects apart is not obvious and there remains plenty of room for creative minds to explore this issue. However, there are clear demonstrations that conditions exist in which two-phase associative proactive interference occurs even when these alternative sources of decrement in target responding are controlled for (e.g., Amundson, Escobar, & Miller, 2003). But these alternate sources of target response decrement (e.g., latent inhibition) can themselves be viewed in the framework of associative interference provided the X-alone presentations are regarded as generating the formation of a putative X-NoOutcome association, a viewpoint that we will maintain for the remainder of this paper.

3.2 Response competition

In the case of outcome interference (e.g., retroactive interference with memory of X-O1 pairings by subsequent X-O2 pairings), one might also ask whether the X-O2 training in phase 2 decreases responding to X indicative of an effective X-O1 association by interfering with X's retrieving the representation of O1, or does the decreased responding to X merely reflect response competition as opposed to any action on retrieval. That is, if O1 and O2 are stimuli that evoke different unconditioned responses, presentation of X at test elicits responding indicative of O1 but also responding appropriate for O2 such that the responding for O2 competes with and hence decreases responding appropriate for O1. Surely such response competition occurs in select situations, but the decrement in responding appropriate for O1 would arise from a process downstream of where memory retrieval presumably occurs. It is always possible that response competition may occur in the window following retrieval and prior to initiating a response, but that would still seem to require that the retrieved representations of O1 and O2 are associated to different terminal responses. Toward determining whether apparent outcome interference is anything more than response competition, one might embed both treatment phases of an outcome interference procedure within what is conventionally phase 1 of a sensory preconditioning procedure (see Table 1). Using this procedure, O2 never acquires a response potential so response competition as a result of X eliciting responding appropriate for O2 is precluded. This technique has been used on many occasions, with the result that outcome interference is evident even when response competition has been precluded (e.g., Escobar et al., 2001).

4. A taxonomy of associative interference

As previously stated, any given instance of two-phase associative interference can be assigned to one of four categories defined by whether the procedure is proactive or retroactive interference, and orthogonally, whether the situation is one of cue interference (two cues and one outcome) or outcome interference (one cue and two outcomes). Two additional axes on which different types of associative interference can be categorized are the nature of training in phase 1, and independently of the nature of training in phase 1 the nature of training in phase 2. Training during either phase may consist of excitatory X-O1 trials, explicitly inhibitory training (i.e., A-O1 trials interspersed with AX-NoO1 trials), or trials on which an element such as X (or O1) is presented by itself, which in most cases is simple nonreinforced exposure to X. Note that during inhibitory training, X is presented without O1 being presented *when O1 is explicitly expected* based on A being present. In contrast, in the case of mere nonreinforcement (i.e., X-NoOutcome trials), X is presented without O1 being presented *when O1 is not expected*. Thus, outcome interference does not necessarily require two explicitly presented outcomes as in counterconditioning (i.e., X-O1 followed by X-O2). The absence of an outcome may also interfere with subsequent formation of (or retrieval by) a later trained cue-outcome association (i.e., latent inhibition[2]; Lubow & Moore, 1959). One can also readily observe proactive interference with behavior

[2]The latent inhibition effect (produced by repeated nonreinforced exposures prior to cue-outcome pairings) is usually viewed as disruption of subsequent formation of a later trained cue-outcome association resulting from a change in attention to the cue (e.g., Pearce & Hall, 1980). This proposed mechanism for latent inhibition would not be a form of associative interference in the sense that we are using the term here. But if cue preexposure disrupts retrieval of the outcome representation by the cue at test because of the

reflecting conditioned inhibition training in phase 2 as a result of mere nonreinforcement (i.e., latent inhibition treatment) in phase 1 (e.g., Friedman, Blaisdell, Escobar, & Miller, 1998; Rescorla, 1971). Conversely, there has been considerable difficulty in observing retroactive interference with conditioned inhibition trained in phase 1 by mere nonreinforcement (i.e., X-) in phase 2 (i.e., Zimmer-Hart & Rescorla, 1974; for a review, see Williams, Overmier, & LoLordo, 1992); however, some data suggest that such interference may be detected at least with some select parameters (Lotz & Lachnit, 2009; Melchers, Wolff, & Lachnit, 2006; Polack, Laborda, & Miller, 2012). Even then, the effects of mere nonreinforcement (i.e., extinction treatment) on previously acquired conditioned inhibitor appears to have less influence on behavioral control by a conditioned inhibitor than when nonreinforcement precedes conditioned inhibition training. Thus, we see here that first-experienced mere nonreinforcement has more impact on conditioned inhibition training than does second-experienced mere nonreinforcement. Although one can observe a reduction in conditioned inhibition as a result of mere exposure to a conditioned inhibitor, the difference in the robustness and parameter dependence of the decrement constrains the view that conditioned inhibition and conditioned excitation are symmetrical processes. In summary, beyond designating four broad types of associative interference in the forms of retroactive cue, retroactive outcome, proactive cue, and proactive outcome interference, there are additional subtypes that we outline below.

4.1 Specific instances of associative interference

We now consider the possibilities presented by a particular outcome being present, merely absent, or explicitly absent despite being expected during phase 1. Similarly, the same three possibilities exist during phase 2, and the possibilities here are independent of those during phase 1. Thus, these three by three set of possibilities crossed with the previously mentioned four different types of associative interference suggest a total of 36 potentially different subtypes of two-phase associative interference. However, not all 36 subtypes are equally interesting and several do not logically qualify as interference. For example, outcome interference when treatment is mere nonreinforcement in both phases 1 and 2 is meaningless because there cannot be different outcomes when there are no outcomes. Given both proactive and retroactive instances of this situation produce the same problem, we must reduce the number of potential subtypes to 34. Moreover, what cue interference means in the case of target training consisting of mere nonreinforcement is ambiguous, but it is not necessarily meaningless. Thus, although these designations are helpful for comparing across interference paradigms, one must still be cautious when looking at associative interference in terms of these subtypes.

4.2 Extinction as simply one instance of associative interference

Extinction is only a one instance out of the many different types of associative interference; specifically, it is retroactive outcome interference with excitatory conditioning in phase 1 and nonreinforcement in phase 2. Importantly, nonreinforcement in phase 1 (i.e., latent

interaction of the two distinct types of training, it would be an instance of proactive interference. Evidence of recovery from latent inhibition favors an interference view of latent inhibition (e.g., Kasprow, Catterson, Schachtman, & Miller, 1984; Grahame, Barnet, Gunther, & Miller, 1994), but in fact latent inhibition is probably caused by multiple factors (Hall, 1991).

inhibition treatment) is unambiguously a case of the cue appearing when there is no expectation of the outcome. However, the same nonreinforcement treatment in phase 2, given prior excitatory training of that cue in phase 1, is an instance of the cue occurring alone when the subject does have some expectation concerning the occurrence of the outcome. This explicit violation of the expectation of an outcome in phase 2 is what challenges categorizing extinction treatment as a form of pure nonreinforced exposure to the cue (i.e., with no expectation concerning any outcome) as opposed to actually being a form of inhibition training as was initially proposed by Pavlov (1927). That is, the cue might be expected to acquire an inhibitory association with the US in phase 2 due to its occurrence when the US is expected based on the same cue having previously been trained as a conditioned excitor during phase 1. From this perspective, the cue may simultaneously have an excitatory association with the US from phase 1 and an inhibitory association from phase 2 that interferes with expression of the first-learned excitatory association at test (e.g., Bouton, 1997). Thus, when comparing the prospective and retrospective forms of the same 'type' of interference (e.g., nonreinforced presentations of the target cue by itself), there is reason to anticipate asymmetries. Nevertheless, given the large variety of associative interference interactions that in principle can be generated after controlling for potential nonassociative sources of decrement in behavior indicative of the target association, one might ask whether there are common rules that apply to them all, or at least an appreciable proportion of them. It would be particularly helpful to investigate and identify whether there are clear parallels between what is observed in extinction and what might be observed across other types of associative interference. These sorts of questions are largely unanswered in the literature, so below we consider what limited answers the literature does provide.

In the present framework, extinction is an example of retroactive outcome interference in which excitatory training of the target cue occurs in phase 1 (i.e., X-Outcome), followed by nonreinforcement of the cue in phase 2 (i.e., X-NoOutcome). Compared to counterconditioning (i.e., X-Outcome1 followed by X-Outcome2), extinction is a less straight forward example of outcome interference in that there is only one explicit outcome. However, the X-NoOutcome extinction trials in phase 2 serve as new conflicting learning that appears to be encoded and available to influence later test performance. Admittedly, the concept of an association with an absent event (i.e., NoOutcome) requires a bit of abstraction, but that is precisely what a recalled, but absent event is, abstract (i.e., a mental representation, but not physically present). One of the questions considered here is whether the rules governing extinction are unique, or do they also apply to other types of associative interference phenomena. If extinction is an atypical instance example of associative interference (or not an instance of associative interference at all), then the recent emphasis on extinction over more typical examples of interference represents neglect of a major source of forgetting. Alternatively, if extinction is a form of associative interference with X-NoOutcome being the interfering association, a better understanding of associative interference will improve our understanding of extinction.

5. Comparing interference produced by nonreinforcement, inhibition training, and excitatory training

Each type of associative interference decrements target responding, and extinction is no exception. The question is whether each other type of associative interference changes in the same manner as the well-researched phenomenon of extinction, as a function of variables such as retention interval, the effects of context shifts, the spacing of trials within sessions, and the amount of time between phases.

5.1 Trial spacing and retention intervals

It is well documented that spaced trials promote strong, enduring memory for learned material (e.g., Barela, 1999). Relatively little work has focused specifically on the spacing effect across the varied types of interference. Enhancement of the interfering association (if we may take the liberty of calling an X-NoOutcome memory an association between two events) through increased trial spacing generally produces more interference. In contrast, increased trial spacing during phase 1 target training ordinarily makes the target association more resistant to interference. The enhanced extinction observed when extinction trials are widely spaced occurs readily under most circumstances and also reduces spontaneous recovery and renewal (Urcelay, Wheeler, & Miller, 2009; Westbrook, Smith, & Charnock, 1985). Of course, this depends to a degree on when one assesses learning, considering that massed trials do provide faster emergence of, but less persistent, behavior reflecting the training (Barela, 1999, Teichner, 1952), which appears to hold for extinction treatment as well as excitatory conditioning. Thus, extinction rates recorded during extinction training are faster when massed, but the amount of recovery of phase 1 acquisition after relatively long phase 2-test intervals is more pronounced. The temporary effects of massed extinction trials indicate that the reduced responding is not solely the result of enhanced retrieval of the interfering NoOutcome memory. Presumably, the cumulative build-up of activation of the recent nonreinforced events also contributes; however, the latter is likely not engaging classic retrieval mechanisms. These two factors jointly contribute to this recency effect.

One might also consider the interval between the target and two interfering treatments. Systematic variation of the time between phase 1 and phase 2 treatments has received considerable attention recently but the findings have been inconsistent. The different results appear to depend, at least in part, on the length of the retention interval between phase 2 and the final test. The effect of retention interval would seem to scale to the interval between phase 1 and phase 2 (e.g., Johnson, Escobar, & Kimble, 2010). Generally, as the retention interval between phase 2 learning and the critical test increases, retroactive interference wanes and proactive interference becomes more evident, which is consistent with Devenport's temporal weighting rule (Devenport, Hill, Wilson, & Ogden, 1997). One exception is that interference from mere nonreinforcement (X-) tends to weaken over time not only if it is retroactively interfering (i.e., extinction; Pavlov, 1927), but also if it is proactively interfering (i.e., latent inhibition; Aguado, Symonds, & Hall, 1994; Killcross, Kiernan, Dwyer, & Westbrook, 1998; but see De La Casa & Lubow, 2002).

5.2 Context shifts

Considerable research has been directed on the effects of changes in context on various types of interference. Much of this work has emphasized ‘renewal,’ which [narrowly speaking] is the recovery from retroactive interference induced by nonreinforcement of a previously reinforced cue (i.e., extinction) as a result of testing outside of the context in which extinction occurred. Considering that renewal effects are so well documented, we here consider accounts of it. Bouton (1993) proposed two rather different accounts of renewal. Both are based on the extinction context becoming an occasion setter for the memory of extinction training as seems to be the case based on the strong renewal that is observed after training in context A, extinction in context B and testing in context C (i.e., ABC renewal; Bouton & Bolles, 1979). But the rule determining why extinction learning becomes context specific remained unclear. One possibility suggested by Bouton is that nonreinforced learning itself is prone to be context specific. The second possibility suggested by Bouton is that second-learned information (whether it involves excitatory, inhibitory, or nonreinforcement training) is prone to be context specific.

Renewal designs involving extinction fail to distinguish between nonreinforcement and second-learned information being context specific because the two factors are confounded in a typical extinction/renewal design. One must reverse the order of excitatory training with respect to nonreinforcement training to begin to untangle the influence on renewal of nonreinforcement from information that is second learned. Thus, we must look at proactive outcome interference between nonreinforcement in phase 1 (i.e., latent inhibition treatment) and reinforcement in phase 2 rather than the retroactive outcome interference that constitutes an extinction paradigm. Miller, Laborda, Polack, and Miguez (2015) observed similar degrees of context dependence of nonreinforcement treatment independent of whether that nonreinforcement was proactive (latent inhibition) or retroactive (extinction) interference. This suggests that nonreinforcement treatment (i.e., X-) is prone to be context dependent whether or not it is second learned. However, additional research is required to determine the extent to which similar processes underlie the observed similar decreases in conditioned excitatory responding produced by latent inhibition treatment and by extinction treatment. The possibility of different underlying processes is a concern because clearly no outcome is expected during latent inhibition treatment whereas presentation of the target cue alone during extinction provides an expectation of the outcome.

When Bouton (1993) spoke about ‘nonreinforcement,’ he grouped within it not only X-NoOutcome treatment but also conditioned inhibition training (e.g., A-Outcome / AX-NoOutcome). Conditioned inhibition training in phase 1, like extinction treatment, is susceptible to ABA renewal-like recovery effects where the first letter represents the context of phase 1 treatment (conditioned inhibition training in this case), the second letter is the context of phase 2 treatment (conditioned excitation training in this case), and the third letter is the context of inhibition testing (e.g., Fiori, Barnet, & Miller, 1994). However, an ABA relative to ABB design does not adequately assess whether the behavioral change induced by testing on Context A is the result of a failure of phase 1 inhibition training to transfer to Context B or the failure of phase 2 excitatory training to transfer to Context A. In this case, context specificity may be the result of either inhibition training or second-learned

information being context specific. An ABC compared to ABA context shift design avoids such concerns because the second-learned excitatory training must transfer to a different context in both cases. The only difference between the ABA and ABC designs is the degree to which the first-learned nonreinforcement transfers to the new context (C). Nelson (2002, 2009) found that first-learned conditioned inhibition transfers well to Context C. This leads to the conclusion that nonreinforcement (i.e., X-NoOutcome treatment) is context specific regardless of whether it is trained first or second, whereas conditioned inhibition training is context specific primarily when it is trained second (and not even then if the first-trained relationship is simple nonreinforcement i.e., X-). Alternatively stated, omission of an expected outcome (i.e., as in conditioned inhibition training) appears to be symmetrical with conditioned excitation training with respect to renewal, whereas omission of an outcome that is not expected (i.e., X-NoOutcome treatment) is context specific regardless of whether it is first or second learned. Thus, we see that all types of absence of an outcome are not equivalent with respect to renewal; in this respect, mere nonreinforcement is unique relative to both excitatory and inhibitory conditioning.

Bearing on the distinction between outcome omission when the outcome was and was not expected, Miguez, Soares, and Miller (2016; Experiment 2) examined the context dependency of outcome interference with conditioned inhibition training relative to latent inhibition treatment during phase 1, when excitatory training occurred in phase 2. Phase 1 and phase 2 training were provided sequentially in physically distinctive contexts, followed by testing in the context of phase 1 training, phase 2 training, or a context that was familiar but in which no training had previously occurred. Miguez et al. observed that phase 1 conditioned inhibition training readily transferred to the neutral context, whereas proactive interference with the excitatory training in phase 2 produced by phase 1 latent inhibition training was appreciably attenuated in the neutral context. These observations lend support to the conclusion that nonreinforcement is always relatively context specific, whereas excitatory or inhibitory training is only context specific when it is second learned.

The greater lability of mere nonreinforcement relative to both conditioned inhibition and conditioned excitation can also be assessed by varying the retention interval following phase 2 training. With latent inhibition treatment in phase 1 and excitatory training in phase 2, little spontaneous recovery of latent inhibition (i.e., nonresponding) is ordinarily observed (Aguado et al., 1994; Killcross et al., 1998; but see De La Casa & Lubow, 2002). In contrast, with inhibitory training in phase 1 and excitatory training in phase 2, spontaneous recovery of behavior consistent with the phase 1 inhibitory training is observed (Sissons & Miller, 2009).

Likely, the high context dependency of latent inhibition arises from strong associations between the cue and the context in which latent inhibition treatment occurs, as hypothesized by both Wagner's (1981) Sometimes Opponent Process (SOP) theory and Miller and Matzel's (1988) Comparator Hypothesis accounts of the latent inhibition effect; however, these two models differ appreciably with respect to the critical action of the hypothesized cue-context association.

6. Fate of interfered memories

In section 4, we delineated numerous possible types of associative interference. But what is the fate (or fates) of these associations that have been subject to interference? If we only document decreased behavioral control by the target cue, we would not know whether the decrement reflects impaired retrieval of the target association or a loss of the target association (to be more precise, an erasure of the phase 1 target association in the case of retroactive interference and a failure to learn the phase 2 target association in the proactive case). The only way to differentiate between a retrieval failure and an irreversible loss of information is to induce recovery of the target association through some manipulation that precludes the possibility of relevant new learning about the target association.

6.1 Recovery from retroactive interference

Empirically, we see that interference effects are often reversible, and this is particularly true for retroactive interference, that is, when the target information is presented in phase 1. Testing outside of the context of phase 2 treatment or long retention intervals tend to result in the recovery of first-learned associations as has been demonstrated with numerous reports of renewal, spontaneous recovery, and resurgence effects (e.g., Miguez, Cham, & Miller, 2012; Bouton & Schepers, 2014). The relative ease with which retroactive interference can be attenuated following assorted interference treatments indicates that most retroactive interference is a performance deficit (i.e., a retrieval or expression failure). This is a particularly salient point when it is merely the test context that determines the degree of responding, at least when the interfering association was trained in a different context than the target association. AAB renewal also suggests retroactive interference is a performance failure even when both the target and interfering phases occur in the same context; however, AAB renewal is often weak, likely because of both the excitatory status and the ambiguous discriminative status of the training context (Laborda, Witnauer, & Miller, 2011; Polack, Laborda, & Miller, 2013; Tamai & Nakajima, 2000; Thomas, Larson, & Ayres 2003).

6.2 Recovery from proactive interference

Recovery of behavior reflecting target acquisition following proactive interference (i.e., target training in phase 2) is somewhat more elusive than recovery from retroactive interference. At least with latent inhibition treatment, some recovery is often observed as one increases the retention interval between phase 2 and test (i.e., the interfering effect of the phase 1 latent inhibition treatment wanes; e.g., Bakner, Strohen, Marvin, & Riccio, 1991). Latent inhibition is also observed to produce less proactive interference when testing occurs outside the context in which both latent inhibition and excitatory training occurred (i.e., an AAB context shift at test produces a recovery from the latent inhibition deficit in responding; Miller et al. 2015; but the recovery is ordinarily not complete; Westbrook, Jones, Bailey, & Harris, 2000). Additionally, outcome reinstatement (i.e., presentation of the outcome between phase 2 target excitatory training and testing) reveals the second-learned excitatory association following latent inhibition treatment (Kasprow, Catterson, Schachtman, & Miller, 1984). Additionally, proactive interference by latent inhibition treatment can be reduced after phase 2 excitatory training by extinction of the context in which latent inhibition treatment had been administered (Grahame et al., 1994). This is

consistent with part or all of the effects on nonreinforcement treatment depending on context-cue associations.

6.3 Reinstatement

Notably, outcome reinstatement induces recovery from both retroactive and proactive outcome interference at least when the target memory is of excitatory training (e.g., Heth & Rescorla, 1975). Similarly, presentation of the cue alone (i.e., cue reinstatement) sometimes also recovers responding following extinction treatments, provided the amount of cue exposure is limited to avoid [further] extinction (e.g., the increase in conditioned responding that is often seen after the first extinction trial; e.g., Pavlov, 1927).

6.4 Retrospective revaluation

To the limited extent examined, deflating the interfering association also induces recovery of an excitatory target association from both retroactive cue interference (i.e., retrospective revaluation; Miguez, Laborda, & Miller, 2014) and proactive cue interference (Amundson et al., 2003). Based on the data available to date, recovery from associative interference through one or another means suggests that most interference is a retrieval failure rather than an irreversible loss of memory. Thus, the observed recovery from essentially all types of associative interference examined to date suggests important commonalities across the numerous types of interference.

6.5 Better controlled comparisons between different types of interference are needed

Despite the commonalities and differences across the various types of associative interference described above, few papers have compared different types of associative interference within well controlled experiments. Even some prior papers that highlighted parallels across different types of associative interference have relied heavily on comparisons across multiple experiments. Holmes, Leung, and Westbrook (2016) provide an excellent exception to this by contrasting the effects of ABA renewal on counterconditioning and extinction within a single experiment. Additionally, they went on to compare these two types of retroactive outcome interference with respect to ABC renewal, with the neutral test context being matched to the two other contexts in terms of their reinforcement histories. Throughout their various experiments, Holmes et al. observed reliably less responding to the target cue trained during phase 1 following counterconditioning than following extinction in phase 2 when testing occurred in the same context in which the phase 2 treatment had occurred. Although counterconditioning was more effective at initially reducing expression of phase 1 learning during the phase 2 interference treatment, larger amounts of renewal (i.e., recovery) were observed in the neutral test context (C) following counterconditioning. One might have suspected that this was due to an easier discrimination between phase 1 and phase 2 in counterconditioning than extinction. That is, the presence of a new outcome during phase 2 may have made the context of phase 2 more distinctive for the counterconditioning group than for the extinction group. With the added outcome missing on the test trials that followed phase 2 for the counterconditioning group, testing less closely resembled the stimulus conditions of phase 2. However, the use of a within-subject design by Holmes et al. in their Experiment 2 circumvented these potential differences between extinction and counterconditioning because extinction and counterconditioning occurred

interspersed within the same training sessions, but greater renewal following counterconditioning treatments than extinction was still observed. Holmes et al. provide a good example of what needs to be done more generally to identify similarities and differences between extinction effects and interference effects more broadly. Considerably more research focusing on comparing and contrasting the various types of interference is needed to begin to map commonalities and differences.

7. Conclusions

As traditionally defined, *Persistence* and *Relapse* are applicable only to retroactive interference. But proactive interference is the flip side of retroactive interference, and, operationally at least, the difference depends only on whether one tests on the content of phase 1 or phase 2 training. The current emphasis on the mechanisms that produce persistence and relapse in extinction paradigms should encourage researchers to examine other instances of retroactive interference. The processes responsible for retroactive interference may well apply across many or all two-phase associative interference situations. However, exceptions may exist, as appears to be the case with latent inhibition, which as a source of interference appears to be substantially more labile than either conditioned inhibition or conditioned excitation treatments. It remains unclear whether the peculiarities concerning latent inhibition undermine the argument in favor of commonalities across different types of interference or whether the lack of an explicit outcome during latent inhibition treatment makes it inappropriate to view it as an example of associative interference.

Although there is still much to be learned about the similarities and difference across the various types of two-phase associative interference, a few tentative conclusions are now possible. One is that the behavioral effects of X-NoOutcome experiences, extinction as well as latent inhibition treatments, appear to be more labile than excitatory X-Outcome associations and inhibitory X-Outcome associations. Another is that there seem to be three critical determinants of test performance following two-phase associative interference. *First*, there is the relative degree of similarity of the context of target training and context of interference treatment with respect to the context of testing. If the context of testing more closely resembles that of one of the two phases of treatment, one sees stronger expression of that memory at the expense of the alternative memory. *Second*, the intervals from phase 1 treatment to testing and from Phase 2 treatment to testing appear to strongly influence interference, with relative recency favoring behavioral control by phase 2 treatment when the ratio of these two intervals is large. *Third*, the relative strengths of the two associations strongly influence the observed behavior. As one might expect, stronger associations are favored for expression (e.g., Denniston, Chang, & Miller, 2003). Thus, differences in the number of training trials in each phase can shape the amount of interference observed, as can modifying the effective strengths of the associations after the conventional two phases of interference training has taken place (e.g., posttraining associative deflation of the interfering association; Miguez, Laborda, & Miller, 2014). These three factors conjointly influence the degree that associative interference will be observed.

Researchers' usual focus on only one type of associative interference, typically extinction, has left large gaps in our understanding about interference in general. Above we have described some of the particularly cloudy areas that need to be examined and hope that these remarks will draw attention to those areas and encourage future research comparing the different types of associative interference. Much effort has been placed on identifying the features of one particular type of associative interference, namely extinction. We suggest that more attention to carefully controlled comparisons across different types of interference would be illuminating with respect to both theory and application.

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Highlights

1. Associative interference is a detrimental interaction between associations sharing some but not all features
2. Extinction is an example of retroactive outcome interference between an outcome and its absence
3. Most recent investigations of associative interference have focused on extinction and recovery
4. Similar mechanisms appear to apply to a wide range, but not all forms of associative interference

Table 1

A design to assess two-phase outcome associative interference circumventing potential response competition.

Group	Phase 1	Phase 2	Phase 3	Test	Expected responding
Interference	X-O1	X-O2	O1-US	X→R(US)?	cr
Control	X-O1	X / O2	O1-US	X→R(US)?	CR

Note: This table depicts retroactive interference. Simply reversing the events between phases 1 and 2 would convert it into a proactive interference design. X is a cue; O1 and O2 are affectively neutral stimuli that evoke no unconditioned response. The unconditioned stimulus (US) is a stimulus that evokes an unconditioned response. A slash separates events that are explicitly unpaired. cr (CR) indicates a weak (strong) response (R) appropriate for the US (and hence appropriate for O1 which is paired with O1 in phase 3.