A new model for recovery-from-extinction effects in Pavlovian conditioning and exposure therapy

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Abstract

Exposure therapy is an effective intervention for anxiety-related problems. A mechanism of this intervention has been the extinction procedure in Pavlovian conditioning, and their findings have provided many effective intervention strategies that can promote the effect of and prevent relapse following exposure sessions. However, traditional associative theories that have explained Pavlovian conditioning cannot comprehensively explain their findings. In particular, it was difficult to explain the recovery-from-extinction effects, which is the reappearance of conditioned response following extinction. In this study, we propose a new associative model that can deal with procedures that promote an effect of extinction and many recovery-from-extinction effects. The cores of this model are that the asymptotic strength of the inhibitory association depends on the degree of excitatory association retrieved in a context in which CS is presented and that the retrieval is determined by the similarity between contexts during reinforcement and non-reinforcement and the present context. Moreover, this model assumes that these similarities change under specific conditions. By adding these assumptions to the traditional framework, many difficulties in explaining these phenomena can be resolved. Our model can provide not only a new perspective in associative learning, but also many implications for exposure therapy.

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Keywords: recovery-from-extinction effects, fear conditioning, associative learning, exposure therapy

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The development of procedures to reduce anxiety and fear and understanding of their mechanisms are important for clinical psychology. Anxiety-related disorders and problems are pervasive (Kessler et al. 2005) and have serious effects on the quality of life (e.g., Rapaport et al., 2005). One of the most effective interventions for these problems is exposure therapy, which is designed to eliminate anxiety and fear by deliberately continuing exposure to fear-evoking stimuli (Abramowitz et al., 2019). The effectiveness of exposure therapy on maladaptive anxiety and fear has been confirmed by many findings (e.g., Watts et al., 2013), and many packages in cognitive behavior therapy include the element of exposure therapy (Abramowitz et al., 2019). There are some approaches to exposure therapy (e.g., Foa & Kozak, 1986; Salkovskis et al., 2006; Craske et al., 2014). In particular, in recent years, the idea that exposure therapy is a clinical analogue for extinction procedures in Pavlovian fear conditioning has received a lot of attention from many researchers and therapists because it has proposed effective techniques based on the findings of Pavlovian extinction (e.g., Craske et al., 2014).

In fear conditioning, a subject acquires a fear response to a neutral stimulus (conditioned stimulus: CS) through pairings of the stimulus with an unconditioned

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stimulus (US). However, after fear conditioning, when CS is presented without US, the conditioned response (CR) to the CS is diminished, which refers to extinction (Pavlov, 1927). The explanation of the exposure therapy by fear conditioning and extinction assumes that maladaptive or pathological fear and anxiety are acquired by pairings of CS and US during traumatic events and can be attenuated by presentations of CS alone, that is, exposure to fear-provoking stimuli. This framework suggests that exposure therapy is an application of the extinction procedure in clinical settings, and the effect is due to response decrement to a CS by extinction procedure.

One advantage of this framework is that it can propose strategies to reduce fear by exposure sessions and relapse following the therapy based on the considerable experimental findings in Pavlovian conditioning. Given that exposure therapy is an extinction procedure to feared CS, the two aims can be achieved by promoting the effect of extinction procedure and preventing reappearance of the CR following the extinction. The reappearances are referred to as recovery-from-extinction effects (McConnell & Miller, 2014), and many experiments investigating these effects have been conducted to decrease relapse of anxious symptoms following intervention. For example, conducting extinction in multiple contexts is effective for decrement in the recovery-from-extinction effects in fear conditioning with rodents (e.g., Gunther et al., 1998) and humans

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(Bandarian-Balooch et al., 2012). This finding suggests that a relapse following exposure therapy can decrease by conducting interventions in many contexts or situations, which has been supported by some clinical research (e.g., Vansteenwegen et al., 2007). The effects of other procedures that can achieve the two aims in Pavlovian conditioning are also investigated in clinical situations and have been confirmed to be effective strategies in the exposure therapy (e.g., Shin & Newman, 2018).

Although the findings of Pavlovian conditioning have provided tremendous effective strategies in exposure therapy, the effects of these procedures cannot be accounted for in a consistent way. Traditionally, the effect of Pavlovian conditioning has been explained by associative learning theory, which is assumed to develop mental connections or associations between some events during conditioning. For example, the Rescorla-Wagner model (Rescorla & Wagner, 1972), which is the most influential associative model, can deal with fear reduction through extinction procedure by the error correction rule and propose some strategies to improve the effect of exposure therapy (e.g., Craske et al., 2008; Rescorla, 2006). However, this model cannot account for many recovery-from-extinction effects (e.g., Bouton & Bolles, 1979). Other traditional models cannot comprehensively explain these effects as well (McConnell & Miller, 2014).

So far, Bouton's model (Bouton, 1993) has been widely accepted as an account

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for the extinction and recovery-from-extinction effects. This model assumes that learning by extinction (inhibitory association) is more context-dependent than conditioning (excitatory association). Therefore, the effect of extinction is decreased when CS is presented outside the extinction context, resulting in the reappearance of the CR. This idea can explain many of the recovery-from-extinction effects that are difficult to explain using traditional models. However, some of the predictions from Bouton's model are also inconsistent with empirical findings as well as other models (McConnell & Miller, 2014). In addition, this model cannot propose strategies to improve the effects of extinction itself because it does not assume how to change the associative strengths on a trial-by-trial basis. These facts indicate that any associative theories are not sufficient to explain extinction. Thus, it is difficult to use traditional associative models as a mechanism of exposure therapy.

Therefore, in this study, we propose a new associative model that can overcome these issues. In the first section, we review various procedures that improve the effect of extinction and decrease the recovery-from-extinction effects in Pavlovian conditioning and how these effects can be explained using Bouton's model (as explanations based on other influential models, see McConnell & Miller, 2014). This review will show that some of these phenomena cannot be explained by the traditional associative framework. Next,

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we propose a new model that can comprehensively explain such phenomena. The new model can explain how to improve the effects of and decrease the recovery-from-extinction effects, which can provide a valid model for exposure therapy based on the perspective of associative learning.

Summary of extinction and recovery from extinction effects and explanation of these effects

Bouton's model

In the associative perspective, a decrement in response caused by extinction and recovery-from-extinction effects have often been explained by Bouton's model. Bouton's model assumes that CS-US (i.e., excitatory) association is developed by pairing CS and US, while CS-no US (i.e., inhibitory) association is developed by CS presentation without US. CR is determined by the degree of retrievals of both associations, suggesting that CR occurs when retrieval of the excitatory association is stronger than that of the inhibitory association. One of the characteristic features of this model is that inhibitory association is context-dependent, indicating that when a subject goes outside the extinction context, the retrieval of inhibitory association is weakened. Thus, extinguished CR reappears when CS is presented outside the extinction context because the retrieval of inhibitory

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association is weaker than that of excitatory. The model can explain the effect of context changes on a CR in various interference preparations, such as latent inhibition, counter-conditioning, and reversal learning (Bouton, 1993). However, this model does not deal with the learning process of each association (McConnell & Miller, 2014). Thus, the model does not assume how both associations change with experience.

Procedures improving the effects of extinction

Many findings have indicated that the effect of extinction can be promoted by some procedures. One of the most famous procedures is to increase extinction trials, which decreases the CR at the end of the extinction phase (Pavlov, 1924). Another procedure is the deepened extinction (Rescorla, 2000; Rescorla, 2006). In this procedure, a CS and other stimuli producing CR (i.e., conditioned exciter) are simultaneously presented without US, resulting in a large decrement in the CR to the CS. However, if the CS and other stimuli inhibiting CR (i.e., conditioned inhibitor) are simultaneously presented without US in the extinction, the CR to the CS is larger than a CS after normal extinction, which refers to protection from extinction (Lovibond et al., 2000; Rescorla, 2003). These findings suggest that conducting many sessions of intervention and exposure to multiple fear stimuli and eliminating safety stimuli in a session can improve

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the effect of exposure therapy. Bouton's model cannot deal with these effects because this model does not assume how inhibitory association is formed.

Recovery-from-extinction effects

There are four types of recovery-from extinction effects: renewal, spontaneous recovery, reinstatement, and rapid reacquisition (Bouton, 2002). The renewal effect is defined as a reappearance of a CR by changes in the physical context after extinction (e.g., Bouton & Bolles, 1979) and can be categorized into three types. The most major and robust type is ABA renewal, which is the reappearance of extinguished CR when testing is conducted in the acquisition context after acquisition is conducted in one context and then extinction in another (e.g., Bouton & Bolles, 1979). The second type is ABC renewal (e.g., Bouton & Bolles, 1979), which is the reappearance of CR when acquisition, extinction, and test phases are conducted in all differential contexts. The third type is AAB renewal (e.g., Bouton & Ricker, 1994), which is a phenomenon in which CR reappears when acquisition and extinction are conducted in the same context and then tested in a differential context. These phenomena suggest that relapse occurs when an anxious individual goes outside the context in which the exposure therapy is conducted. In Bouton's model, renewal effects occur because inhibitory association is diminished by

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context change from the extinction context. However, this account has a weakness. Although this model predicts that the sizes of ABC and AAB renewal effects are the same because both effects are caused by changes in context from extinction context to different contexts (McConnell & Miller, 2014), many findings are inconsistent with this prediction, which have reported that the size of ABA renewal is larger than that of ABC and AAB renewal and ABC renewal is larger than AAB renewal (e.g., Thomas et al., 2003).

Spontaneous recovery is defined as a phenomenon in which CR reappears by passage of time following extinction, which was first reported by Pavlov (1927). This phenomenon suggests that passage of time following intervention can lead to a relapse of anxiety-related symptoms. Although this phenomenon is famous and robust in various preparations, many of the major models in associative theories cannot account for this phenomenon (McConnell & Miller, 2014). However, Bouton's model can account for this phenomenon by introducing an idea of change in the temporal context, that is, a variety of renewal effects. This explanation predicts that when the physical and temporal contexts are simultaneously manipulated, the return of CR is larger than individual manipulation because a huge change in context occurs, and this prediction is consistent with the results of many experiments (e.g., Rosas & Bouton, 1998).

Reinstatement is defined as the reappearance of extinguished CR by

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presentations of US alone after extinction (Rescorla & Heth, 1975), suggesting that relapse occurs when a patient who received exposure therapy encounters a traumatic event again. According to Bouton's model, reinstatement is explained by the idea that the context-US association developed by the US alone presentations is a retrieval cue for the acquisition phase (Bouton & Nelson, 1998). However, Bouton's idea does not assume the effects of context-US association except reinstatement. Moreover, many findings have shown that this association affects CR after extinction only (e.g., Bouton & Nelson, 1998), which is needed to add an assumption that the context-US association functions as a retrieval cue only when CS is ambiguous. An alternative idea based on Bouton's model is that an interoceptive context that occurs by presentation of US functions to a retrieval cue for the acquisition phase (Bouton et al., 2006). This idea predicts that reinstatement occurs when the context in which the US is presented differs from the testing context because the interoceptive context is the same. However, empirical findings are inconsistent with this prediction (e.g., Bouton & Bolles, 1979).

Rapid reacquisition is a phenomenon in which a CR in the reacquisition phase following extinction develops more rapidly than in the initial acquisition phase or new stimulus (e.g., Bouton & Swartzentruber, 1989). This phenomenon suggests that re-experience in pairing of traumatic events and a stimulus rapidly induces the CR again.

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However, some studies have reported that reacquisition produces delayed development of CR (e.g., Bouton, 1986), and the cause of inconsistent findings has been mainly investigated from the perspective of context in the reacquisition phase (e.g., Bouton & Swartzentruber, 1989) and the number of extinction trials (e.g., Bouton, 1986). Thus, if reacquisition is conducted in the extinction context or many extinction trials are used, the rate of reacquisition is delayed (e.g., Bouton, 1986; Bouton & Swartzentruber, 1989). According to Bouton's model, it is predicted that when memory of the acquisition phase is strongly retrieved, rates of reacquisition are rapid and when the extinction phase is strongly retrieved, rates are slow, which has been confirmed by many findings (e.g., Bouton & Swartzentruber, 1989). However, the effect of the number of extinction trials cannot be accounted for in this model.

Prevention of recovery-from-extinction effects

Procedures that improve the effects of extinction can also decrease recovery-from-extinction effects. As mentioned above, massive extinction delays the rate of reacquisition (e.g., Bouton, 1986; Bouton & Swartzentruber, 1989). However, the literature investigating the effect of this procedure on other types of recovery-from-extinction effects showed inconsistent findings in the prevention effect (e.g., Tamai &

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Nakajima, 2000). Although many findings have indicated that the AAB renewal is reduced or eliminated in this procedure (e.g., Tamai & Nakajima, 2000; Rauhut et al., 2001; Rosas et al., 2007), the effect on the ABA or ABC renewal is mixed (e.g., Denniston et al., 2003; Tamai & Nakajima, 2000). In compound extinction, deepened extinction (i.e., compound extinction using another exciter) decreases subsequent renewal effects, spontaneous recovery, and reinstatement (e.g., Rescorla, 2006), although compound extinction using another inhibitor increases them (e.g., Thomas & Ayres, 2004). Thus, the promoting effect of extinction can produce a strong decrement not only in the CR during extinction but also in subsequent recovery-from-extinction effects. Bouton's model cannot explain the effect of these procedures.

Other procedures have been developed to decrease the recovery-from-extinction effects. For example, US presentations during the extinction procedure can decrease recovery-from-extinction effects. This strategy can be categorized into two procedures. First, CS-US presentations are occasionally conducted during extinction (e.g., Bouton et al., 2004; Gershman et al., 2013). This procedure is especially effective for delaying the rate of reacquisition (e.g., Bouton et al., 2004). The second procedure is the noncontingent presentation of CS and US, that is, both stimuli are presented in an explicitly unpaired manner. This procedure is effective for eliminating various recovery-from-extinction

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effects (e.g., Bouton et al., 2004; Rauhut et al., 2001), and the effect is larger than that of the former procedure (e.g., Bouton et al., 2004; Thompson et al., 2018). Bouton's model can deal with the effects of these procedures by assuming that US presentations in extinction produce habituation of the US (Bouton, 2002). However, the effect of this procedure is confirmed to be larger than that of the simple habituation procedure (Rauhut et al., 2001; Thomas et al., 2005). In addition, it is difficult to explain why a noncontingent procedure is more effective for preventing relapse than occasional reinforcement.

The presentation of an extinction cue during testing can also decrease recovery-from-extinction effects (e.g., Brooks & Bouton, 1993). Extinction cues refer to stimuli presented in the extinction phase and do not have associative strength. Contrary to the extinction cue, an acquisition cue, stimulus presented in acquisition and does not have any associative strength, strengthen subsequent CR when the cue is presented (e.g., Vansteenwegen et al., 2006). These effects can be explained by Bouton's model, assuming both cues increase retrieval of each memory of the phase presented in the cue.

Many findings indicate that conducting extinction in multiple contexts decreases recovery-from-extinction effects (e.g., Gunther et al., 1998; Dunsmoor et al., 2014), while some studies could not replicate this effect (e.g., Bouton et al., 2006). Recently, some authors reported that this procedure is only effective when it is combined with massive

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extinction (e.g., Thomas et al., 2009), suggesting that the number of nonreinforcement trials in each context is crucial. According to Bouton's model, this procedure generates generalization across various contexts because many of the common elements in various contexts receive extinction in this procedure (Bouton, 2000). This can also explain why extinction of relapse is effective for decreasing subsequent relapse. Empirical findings have confirmed that extinction of relapse can also eliminate subsequent relapse, which is confirmed in renewal, spontaneous recovery, and reinstatement (e.g., Rescorla, 2004; Quirk, 2002; Holmes & Westbrook, 2013). This procedure is effective when the type of extinguished relapse is different from subsequent relapse (Holmes & Westbrook, 2013). Bouton's model can also explain that conducting extinction in the context in which relapse occurs again can strengthen the retrieval of memory of extinction in the context and promote generalization of the memory to various contexts. However, Bouton's model cannot explain the interaction effect between extinction in multiple contexts and the number of trials.

Extending inter-trial intervals (ITI) can also decrease relapse after extinction (e.g., Urcelay et al., 2009). Bouton's model assumes that this procedure is an extinction in context with long interval between CS presentation and subsequent CS presentation. Thus, this strategy can reduce spontaneous recovery because it is caused by extinction in

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various temporal contexts. A finding that if ITI and delay after extinction are the same, spontaneous recovery does not occur (Bouton & García-Gutiérrez, 2006) supports this idea.

Summary

In this section, we describe Bouton's model that has been widely accepted as an explanation of extinction, recovery-from-extinction effects, and strategies for their prevention. However, as noted above, these phenomena cannot be perfectly accounted for in Bouton's model, although this model can explain these phenomena better than other models. Since Bouton's model does not assume how an inhibitory association is formed through extinction, it is a serious problem for explaining the mechanism of exposure therapy. This issue indicates that the application of the exposure therapy in the model is limited to decreasing relapses after an intervention. To overcome this problem and provide a valid model of exposure therapy based on the findings of Pavlovian extinction, an associative model that can comprehensively explain these phenomena is necessary. In the next section, we propose a new model to resolve these problems and suggest some clinical implications for exposure therapy.

New model

The new model is based on the assumptions of many traditional models (Bouton, 1993; Rescorla & Wagner, 1974; Pearce, 1987; Pearce & Hall, 1980; Laborda & Miller, 2012; Capaldi, 1994) In this model, a CR is determined by the following formula:

$$CR = Ve * S^e + Vi * S^i \quad (1)$$

In equation 1, Ve is an excitatory associative strength and Vi is inhibitory. CR is determined by the sum of their strengths. However, their strengths are affected by similarities, S , between a context in which CS is presented and contexts in which each association is developed. S^e represents the similarity between the acquisition and present context, and S^i represents the similarity between the extinction and present context. Similarities refer to the extent to which a subject recognizes that a context is the same as another context. Thus, S^e means the extent to which a subject recognizes that the present context is the same for acquisition context, and S^i means the extent to which a subject recognizes that the present context is the same for extinction context. These similarities determine how much each association is retrieved in a context in which the CS is presented. If the similarity is high, the association is strongly retrieved in the present context, while low similarity leads to weak retrieval. That is, the CR is determined by two factors: strengths in both associations and the similarities between the present context and

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the contexts in which each association is developed.

This model assumes the following formulae to explain how both associations are developed on trial t .

$$\Delta Ve_t = r_t \alpha^e (\lambda - \Sigma(Ve_{t-1} * S^e) + \Sigma(Vi_{t-1} * S^i)) \quad (2)$$

$$\Delta Vi_t = (1 - r_t) \alpha^i (0 - (\Sigma(Ve_{t-1} * S^e) + \Sigma(Vi_{t-1} * S^i))) \quad (3)$$

Ve and Vi are developed by simple error correction rules (Eq. 2 and 3). If a CS is paired with US (i.e., r is 1), ΔVe_t , the change in Ve on the trial t increases. This change is determined by the difference between λ indicating the intensity of the US and the strength of Ve on trial $t - 1$ and the rate parameter (α^e). If the CS is presented without US (i.e., r is 0), ΔVi_t , the change in Vi on trial t is diminished. This change is determined by the difference between λ during extinction (i.e., 0) and the total strength of Ve and Vi retrieved in the context in which the CS is presented (i.e., $\Sigma(Ve_{t-1} * S^e) + \Sigma(Vi_{t-1} * S^i)$) and a rate parameter (α^i). This assumption indicates that the upper limit of the absolute value of strength in the inhibitory association (i.e., Vi) is the strength of Ve retrieved during a non-reinforcement trial (i.e., $Ve * S^e$), suggesting that the extent to which the subject retrieves the excitatory association in the extinction context is important for developing inhibitory association. This assumption is based on an idea of traditional models (e.g., Pearce & Hall, 1980), in which an inhibitory association is

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developed by pairings of CS and inhibitory reinforcer (e.g., relief) occurred due to the omission of US. This assumption suggests that because in this model, V_e retrieved in a context determines the CR in the context, V_i produced by the absence of US in the context is also determined by the retrieved V_e .

Our model assumes that the similarities are determined by the objective distance between two contexts (testing context and acquisition or extinction context), such as hue or passage of time and subjective bias in the distance. This idea is based on the findings in stimulus generalization that there are two types of similarities that determine stimulus generalization: physical and conceptual similarities (e.g., Dunsmoor et al., 2011). An important assumption about the similarity in our model is the initial value, which can be determined by various factors. In particular, the initial value of S^e should differ from S^i because context change has little effect on CR before extinction (e.g., Bouton & Bolles, 1979), suggesting that regardless of the type of or distance between contexts, the initial value of S^e is approximately 1.0 and larger than that of S^i . That is, the context in which acquisition takes place is recognized to be similar for other contexts than the context in which extinction occurs.

Another assumption regarding the similarities is that they change through trials under specific conditions. For example, when acquisition is conducted in context A and

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then extinction treatment takes place in context B, the S^e (i.e., similarity that affects retrieval of Ve) between contexts A and B is diminished through extinction trials. Thus, when one type of association is developed in a context and subsequently another type is developed in another context, the similarity between both contexts corresponding to the former type is reduced. However, if the same type is developed in different contexts, the similarity between their contexts corresponding to the type increases. This rule is based on the findings of acquired equivalence and distinctiveness effect (e.g., Honey & Hall, 1989). In this effect, if two stimuli (CS_A and CS_B) receive the same outcome, a generalization between them is promoted (Honey & Hall, 1989). Moreover, if a subject receives the same type of trials about CS and US (i.e., reinforcement or non-reinforcement) in two contexts, a generalization between these contextual stimuli is also promoted (Honey & Watt, 1999). This finding indicates that the acquired equivalence and distinctiveness effect occur in contextual stimuli as occasion setters (Ross & Holland, 1981). Our model extends this effect to the contextual stimuli as retrieval cues. Thus, in this model, when the same type of trials is conducted in multiple contexts, the similarity between their contextual stimuli increases. In contrast, different types of trials are conducted in multiple contexts, and the similarity is diminished.

This change in similarity also follows an error correction rule as well as the

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associative strengths. For example, when the acquisition phase takes place in context A and then the extinction phase takes place in context B, S^e during extinction is changed according to the following formulae:

$$\Delta S_{t,A,B}^e = \alpha^S(0 - S_{t-1,A,B}^e) \quad (4)$$

In formula 4, $\Delta S_{t,A,B}^e$ represents the change in S^e between contexts A and B ($S_{A,B}^e$) on trial t . α^S is a rate parameter regarding the similarities. After these two phases, when the CS is conditioned again in context C, $S_{A,C}^e$ increases and $S_{B,C}^i$ decreases in the same manner.

$$\Delta S_{t,A,C}^e = \alpha^S(1 - \Delta S_{t-1,A,C}^e) \quad (5)$$

$$\Delta S_{t,B,C}^i = \alpha^S(0 - \Delta S_{t-1,B,C}^i) \quad (6)$$

Explanation of acquisition, extinction, and recovery-from-extinction effects by the new model

According to our model, an increment in CR during the acquisition phase is explained by the increment in excitatory strength and extinction by inhibitory. Many findings have indicated that whether the extinction context is the same as the acquisition context does not affect the decrement in CR during the extinction phase (e.g., Bouton & Bolles, 1979). Our model assumes that although the CR on an initial trial in a different

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context (context B) other than acquisition (context A) is diminished by similarity between acquisition and extinction context (e.g., $Ve * S_{A,B}^e$), this effect is fairly small because S^e is almost 1.0 regardless the type of context. Thus, it is predicted that there would be no significant difference in the CR if extinction is conducted in either context (left panel of Figure 1).

Our model can deal with various phenomena that cannot be explained by traditional models. For example, this model can account for a difference in the size of the three renewal effects. In the ABA renewal, the similarity between the acquisition and testing context (i.e., $S_{A,A}^e$) is almost 1.0 in testing, while the similarity between the extinction and testing context (i.e., $S_{B,A}^i$) is smaller than $S_{A,A}^e$. Therefore, in testing, the retrieved inhibitory association is diminished ($Vi * S_{B,A}^i$), although excitatory association remains intact ($Ve * S_{A,A}^e$). Moreover, because the strength of the inhibitory association in the extinction phase is determined by the strength of the excitatory association retrieved in the extinction phase ($Ve * S_{A,B}^i$), the strength of the inhibitory association is less than the absolute value of excitatory association. By combining these two effects, the CR reappears during testing in the ABA renewal design. In ABC renewal, by the same mechanisms as that of ABA renewal, the size of inhibitory association acquired in the extinction phase is smaller than that of the excitatory association. However,

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in the test phase, unlike the ABA renewal design, excitatory association is also slightly diminished (i.e., $V_e * S_{A,C}^e$). Therefore, although CR increases during testing, the size is smaller than that of the ABA renewal. In the AAB renewal design, S^e and S^i are basically the same as those in the ABC design during testing because testing is conducted in a context that differs from both acquisition and extinction contexts. However, the strength of the inhibitory association in the ABC design is smaller than that of the AAB design because the excitatory association retrieved during the extinction phase is $V_e * S_{A,B}^e$ in the ABC design, while it is $V_e * S_{A,A}^e$ (i.e., almost $V_e * 1.0$) in the AAB design. Thus, when acquisition and extinction are conducted in different contexts, the strength of the inhibitory association is smaller than when conducted in the same context, resulting in smaller recovery in the AAB renewal than that of the ABC renewal. The right panel of Figure 1 shows the quantitative simulation of the three renewal effects using this model. These simulations indicate that there are some differences in the CR in the test phase of the three renewal designs. These predictions are consistent with empirical evidence (e.g., Bouton & Bolles, 1979; Thomas et al., 2003).

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Figure 1. A simulation of acquisition, extinction, and three renewal effects when $\lambda = 1$,

$$\alpha^e = 0.5, \alpha^i = 0.1, S_{A,A}^e = 1, S_{A,B}^e \text{ and } S_{A,C}^e = 0.9, S_{B,A}^i \text{ and } S_{B,C}^i = 0.8.$$

The left panel indicates the total strength of V_e and V_i during acquisition (1–10 trial) and extinction (11–50 trial) phases in the two groups. Group AA is assumed to receive extinction in the acquisition context and Group AB in the new context. The right panel indicates the strengths during the test phase in each group. Group NE does not receive extinction (i.e., acquisition only), whereas other groups receive acquisition and extinction. The sequence of letters in their groups represents the contexts of acquisition, extinction, and testing. All simulation codes in the study are available for download in

“https://osf.io/mfzd8/?view_only=fe5a8efcf3774e8f8719d336ee6e40f3”

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This model can also explain spontaneous recovery by introducing the idea of temporal context based on Bouton (1993). Thus, spontaneous recovery is a variety of renewal effects, which occur through changes in the temporal context with passage of time following the extinction phase. When a CS is presented following an interval that differs from that in the previous extinction phase, the temporal context in which CS is presented is changed from extinction, and then, the retrieval of V_i is interfered by a decay of S^i (Figure 2).

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Figure 2. A simulation of spontaneous recovery when $S^e = 0.9$, $S^i = 0.8$ in Group Short and $S^e = 0.855$, $S^i = 0.64$ in Group Long.

Group Immediate indicates the total strength in the test phase immediately following extinction. Groups Short and Long represent the total strength during the test phase after extinction and then passing time. The duration between extinction and testing of Group Long is assumed to be longer than that of Group Short. In the simulation, we assumed that this additive duration in Group Long induces a large change in context than that of Group short, which is represented as a change in similarities (S^e was multiplied by 0.9 and S^i was multiplied by 0.80 in Group Long). Other parameters were identical to those in the simulation of Figure 1.

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In our model, reinstatement is explained in a different way from that of traditional models. This model assumes that US alone presentations following extinction increase the similarity between acquisition and test context (i.e., S^e) and decrease the similarity between extinction and test context (i.e., S^i) because US itself acts as a type of context. Thus, US presentations manipulate the distance between these contexts. This idea is mainly based on a reinforcer context in instrumental conditioning (Trask & Bouton, 2016), which indicates that the delivery of reinforcer is a type of context and has been supported by considerable instrumental renewal and resurgence literature (e.g., Trask & Bouton, 2016). According to our model, reinstatement occurs because a subject recognizes that the testing context is like the acquisition context (i.e., increment in S^e) and dissimilar to the extinction context (i.e., decrement in S^i) due to the existence of the US. This can also explain the context dependency of reinstatement. Previous studies have reported that reinstatement occurs only when testing context and the US alone presentation context is the same (Bouton & Bolles, 1979). Since this model explains reinstatement as a result of manipulation of the distance between the US presentation context and acquisition and extinction contexts, reinstatement occurs only in the context in which US presentation is conducted.

The rate of reacquisition can be explained by the change in similarities between

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reacquisition context and acquisition and extinction contexts. For example, acquisition and then extinction are conducted in context A, $S_{A,A}^e$ is reduced compared to the initial value according to the rule of similarity. Therefore, if reacquisition is conducted in context A, the rate of reacquisition is delayed compared to the initial conditioning, while if reacquisition is conducted in the acquisition or a neutral context, it is rapid (Figure 3). This prediction is consistent with the experimental findings that investigate the rate of reacquisition using AAA, ABB, ABA, and AAB procedures (Bouton & Swartzentruber, 1989).

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Figure 3. A simulation of the reacquisition (10 trials) following extinction.

The sequence of letters in the five groups represents the contexts of acquisition, extinction, and reacquisition. The parameters were identical to the simulation results shown in Figure 1.

**Explanation of the procedure that improves the effects of extinction and prevents
recovery-from-extinction effects in the new model**

Our model can also comprehensively explain the effects of increasing extinction trials and compound extinction on promoting extinction and major procedures for preventing recovery-from-extinction effects. Simply, an increment in the number of extinction trials strengthens V_i , which decreases not only CR at the end of the extinction phase but also recovery-from-extinction effects. However, this effect is partial and does not eliminate relapse because the upper limit of V_i is the absolute value of $V_e * S^e$. Therefore, although the AAB renewal can only be reduced by this procedure because V_e and V_i are almost the same in absolute value by massive extinction, other types are difficult to eliminate. Second, massive extinction decreases S^e between acquisition and extinction contexts because subjects receive incompatible information with acquisition during extinction. Thus, the number of extinction trials also affects the decrement in S^e , resulting in delayed reacquisition during the subsequent reacquisition phase in the extinction context.

According to our model, compound extinction using additional excitors results in an ample increase in inhibitory association than normal extinction by the error correction rule as well as many models, such as the Rescorla-Wagner model, predicting

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that recovery-from-extinction effects can be diminished because the inhibitory association retrieved in testing is larger than normal extinction. This model also predicts an increment in the protection-from-extinction effect if extinction is conducted with a conditioned inhibitor. Thus, the effect of extinction increases when $\Sigma(Ve_t * S^e) + \Sigma(Vi_{t-1} * S^i)$ is large in the extinction phase and decreases when it is small, and this effect changes the intensity of the recovery-from-extinction effects.

The effect of US presentations in the extinction phase is explained by introducing the US context hypothesis as well as reinstatement. If a US is presented during extinction, Ve retrieved during extinction increases because the distance between the acquisition and extinction context becomes shorter than the initial value, and as a result, a strong inhibitory association is developed in extinction. Additionally, our model can predict the difference between occasional reinforcement and unpair presentation in extinction. In our model, occasional reinforcement in extinction is expected to increase Ve because of reinforcement, and the effects on preventing spontaneous recovery and renewal effect is smaller than that of the unpair procedure or relapse are possible to be larger than normal extinction (Figure 4). On the contrary, this procedure induces delayed reacquisition because US presentations during reacquisition are part of the extinction context; therefore, S^i in the reacquisition phase after occasional reinforcement is larger than that of in

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normal extinction.

A similar account can also be used in the effects of extinction cue and changing the similarity of physical, internal, and temporal contexts between extinction and test context for preventing recovery-from-extinction effects (e.g., Bandarian-Balooch & Neumann, 2011). Both procedures increase the retrieval of inhibitory association in testing by causing an increment in S^i between extinction and testing context, resulting in a decrement in the recovery-from-extinction effects. Contrary to these procedures, the effect of acquisition cue can be explained by an increment in S^e during testing.

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Figure 4. A simulation of total associative strength in the normal extinction procedure (Group Ext), occasional reinforcement (Group OR), and unpairing with CS and US (Group UP) procedure in extinction on the ABA renewal effect.

In Group PR and UP, reinforcement or US presentation is assumed to be conducted in 5, 15, and 25 trials. The parameters used are identical to the simulation of Group ABA in Figure 1. The left panel represents the extinction phase, and the right panel represents the test phase.

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In extinction in multiple contexts, our model assumes that this procedure extinguishes the ABC renewal every time a subject moves in a neutral context. As described above, the ABC renewal occurs by changing the strengths of retrieval, and extinction of this renewal increases Vi because $\Sigma(Ve * S_{A,C}^e) + \Sigma(Vi * S_{B,C}^i)$ is larger during the test phase than in the ordinal extinction (i.e., AAA procedure). Thus, re-extinction of recovery-from-extinction effects increases Vi , resulting in reducing subsequent recovery (Figure 5). This explanation is consistent with the findings that when recovery-from-extinction effects are extinguished again, subsequent relapse is decreased than in the first time (e.g., Rescorla, 2004). Our model can also explain the effect of extending inter-trial or inter-session intervals in the same way. Thus, these procedures can be accounted for by extinction in multiple temporal contexts, indicating that Vi increases during testing. Additionally, our model predicts that the effect of extinction in multiple contexts is diminished when extinction trials are limited because the effect of extinction itself is reduced.

However, this prediction does not fit with the finding that extinction in multiple physical contexts does not affect spontaneous recovery (Dunsmoor et al., 2014), because an increment in Vi predicts a decrease in all recovery-from-extinction effects. Although whether this phenomenon is robust should be investigated in future studies, this finding

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might indicate that this procedure promotes generalization between contexts, which is the same idea as Bouton's model. This assumption suggests that extinction in multiple contexts using one contextual dimension, such as physical context, provides little generalization to another contextual dimension, such as temporal context, because both dimensions have few common elements. Moreover, this assumption can be applied to explain the interaction between extinction in multiple contexts and the number of extinction trials, which suggests that many trials in each context might produce many generalization to new contexts because the elements in each context receive many extinction trials. In sum, our model assumes that the effect of extinction in multiple contexts is achieved by both strengthening V_i and promoting contextual generalization.

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Figure 5. A simulation of total associative strength in the extinction in multiple contexts

(Group M) and a single context (Group S) on the ABA and ABC renewal effect.

The parameters used are identical to the simulation of Group ABC in Figure 1, except that the number of extinction trials is changed to 30. Group M is assumed to move to the neutral context in 10, 20, and 30 trials.

Clinical implication of this model

This model can provide many clinical implications for improving the effect of and preventing relapse following exposure therapy. First, the number of exposures to the feared stimulus is important for both aims. As the number of exposures to the feared CS increases, the effect of therapy improves and relapse decreases. However, this effect gradually diminishes as the number of exposures increases because prediction errors gradually become small. Therefore, in such cases, some procedures that enhance fear within the sessions, such as simultaneous exposure to the stimulus and other feared stimuli, or verbal instructions that increase the expectation of the outcome (i.e., the expectancy violation strategy) are effective. This model also suggests that eliminating safety cues results in an improvement in the effect because the prediction error increases, suggesting that conducting exposure sessions without safety cues or behaviors, such as a therapist, partner, or family with a patient, is effective.

Moreover, if exposure to feared stimuli is conducted when a patient strongly retrieves memory of a traumatic event, the patient learns that the stimulus is safe strongly. Since the degree of retrieval is determined by the similarity between contexts in which a traumatic event occurs and exposure therapy is conducted, making these contexts similar is recommended for maximizing learning in therapy. It is also predicted that when relapse

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is reduced in exposure therapy, future relapse is unlikely to occur. Therefore, with the consent of the patient, it is useful to intentionally produce a relapse by relatively safe procedures, such as context change or passing time, and then extinguish the fear by exposure sessions. For this aim, although it is also effective to extinguish reinstatement or conduct US presentations during the exposure session, it is possible that these procedures are unethical and dangerous in some cases. It is also important that experiences of exposure sessions generalize to many situations. Conducting exposure sessions in multiple contexts (e.g., therapy room, outdoors, patient's home) is more appropriate than conducting it in one context. In particular, if relapse can occur only in specific situations, it is effective to create the context in which exposure therapy is conducted as similar as possible.

Moreover, our model can provide a new account for traditional exposure techniques. For example, imaginary exposure in session and in vivo exposure as homework has been used to induce habituation within and across sessions in emotional processing theory (Foa & Kozak, 1986). Although the prolonged exposure using these two strategies are effective interventions for pathological problems (Powers et al., 2010), this account itself has not been supported by empirical evidence (Craske et al., 2008). In our model, these strategies can be explained by different ideas based on associative

learning perspectives. Thus, imaginary exposure in a session induces strong inhibitory learning (i.e., V_i) to intensely feared stimuli that cannot be presented in vivo exposure. However, in vivo exposure, homework can promote inhibitory learning by re-extinction of relapse and generalization across many contexts. These restructuring of traditional strategies might suggest some ways to increase this effectiveness.

Summary and future implications

We described that the new model proposed in this study can comprehensively explain many procedures that promote the effects of extinction and recovery-from-extinction effects. Traditional models cannot provide sufficient explanations for these phenomena, especially the recovery-from-extinction effects. Thus, our model is the most appropriate among the associative learning models as an explanation of recovery-from-extinction effects. From a clinical perspective, this model can help understand the mechanisms of exposure therapy and provide many implications for the therapy.

However, there are some limitations to this model. First, our model cannot account for all phenomena in extinction and recovery-from-extinction effects. For example, recovery-from-extinction effects can be diminished if the interval between acquisition and extinction is short (e.g., Myers et al., 2006) or if CS is presented before

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extinction (e.g., Schiller et al., 2010). These phenomena cannot be predicted by our model as well as by other associative models. Second, the factors determining the similarity are unclear. As described above, the similarity might be determined by the distance between two contexts, which is composed of various dimensions (e.g., physical, temporal, or internal dimensions) and subjective bias or psychological similarity (e.g., Dunsmoor & Murphy, 2015). However, this relationship between them might be very complex. For example, Rescorla (2004) reported that although spontaneous recovery is larger with increment in interval following extinction, these effects are negatively accelerated. Thus, the relationship between them is not linear. Investigation of this relationship is critical for uncovering the fundamental nature of similarity. Lastly, our model does not distinguish CS- no US association from conditioned inhibition. Thus, our model considered conditioned inhibitors as a variety of CS-no US association in compound extinction. However, the conditioned inhibitor is context-free, unlike the ordinal association acquired during extinction (Bouton & Nelson, 1994). This distinction might indicate that there are different mechanisms in both. One method of resolving this difficulty is to add an assumption about the order of learning in our model. Since our model is mainly focused on extinction procedures, reinforcement trials are followed by non-reinforcement trials in many phenomena. If a CS becomes a conditioned inhibition by feature negative

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discrimination using another exciter before the acquisition phase, and then the CS is paired with the US, the context dependency in S^e and S^i can reverse, as reported in a previous study (Fiori et al., 1994). Thus, it is possible that S^e and S^i may be dependent on the order of learning (first or second), and not on the type of association (V^e or V).

From a clinical perspective, although our model can provide many clinical implications, it is unclear whether this model can predict the actual decrement of fear response by exposure therapy. The validity of this model in exposure therapy must be confirmed in clinical research. However, traditionally, exposure therapy usually includes complex and various procedures based on the findings of research investigating the effect of interventions. As a result, the principles of their techniques seem to be inconsistent. Our model might resolve this issue.

Conclusion

In this study, we proposed a new associative model to explain many procedures that improve the effect of extinction and prevent recovery-from-extinction effects in Pavlovian conditioning. Since many of these phenomena cannot be comprehensively explained by traditional models, our model can provide a new perspective for explaining these phenomena. Moreover, these phenomena are critical in exposure therapy because

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exposure therapy is thought to be an analogue of extinction in Pavlovian conditioning. Thus, when this model is used as a mechanism of exposure therapy, many clinical implications, especially promoting the effect of exposure and prevention of relapse after intervention, can be provided.

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