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Treatment of avoidance behavior as an adjunct to exposure therapy: Insights from modern learning theory

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ABSTRACT

Pathological avoidance of benign stimuli is a hallmark of anxiety and related disorders, and exposure-based treatments have often encouraged the removal of avoidance, or safety behaviors, due to their negative effects on extinction learning. Unfortunately, empirical evidence suggests that avoidance behaviors can persist following treatment, and the mere availability of avoidance behavior can be sufficient to renew fear following successful extinction learning. The present paper critically examines the function of avoidance behavior through the lens of modern learning theory, and speculates on novel behavioral and pharmacological strategies for targeting avoidance as an adjunct to current evidence-based treatments.

Avoidance has long held a central role in theories regarding the genesis and maintenance of anxiety disorders. For example, Mowrer (1951) conceptualized avoidance as maintained through negative reinforcement resulting from anxiety reduction. More recently, avoidance has been conceptualized as being driven by expectation that a stimulus will lead to an aversive outcome (Declercq, De Houwer, & Baeyens, 2008). In both instances, avoidance becomes pathological when performed in response to relatively benign stimuli.

Although avoidance has been important in theories of anxiety, translational research has largely focused on other Pavlovian processes, such as extinction learning, as the principal mechanism of treatment for anxiety disorders (e.g., exposure therapy). The implicit assumption has been that avoidant behavior would decrease as individuals learned that a threatening stimulus (conditional stimulus or CS) no longer predicted an aversive outcome (unconditional stimulus or US). That is, following extinction training, and the repeated presentation of the CS in the absence of the US, there would no longer be any need to avoid the CS. However, empirical evidence suggests that avoidance behavior can persist following extinction (Rodriguez-Romaguera, Greenberg, Rasmussen, & Quirk, 2016; Solomon, Kamin, & Wynne, 1953), and the availability of avoidant behavior can renew fear even following successful extinction learning. For example, Vervliet and Indekeu (2015) conditioned avoidance behavior (a button press prevented a shock during a CS presentation) and then conducted extinction training where the avoidance behavior was not available. Self-reported fear and physiological arousal to the CS decreased during the extinction phase, however, simply making the avoidant response available at a later test phase when the CS was presented again caused fear to return to the CS. Similar results have been obtained in rodents (Bravo-Rivera, Roman-Ortiz, Montesinos-Cartagena, & Quirk, 2015).

This presents obstacles to evidence-based interventions based on extinction such as exposure therapy. In exposure-based treatment clients are often encouraged to refrain from avoidant behavior (e.g., use of anxiolytic medication, compulsive behaviors, having a “safe” person). However, the above evidence suggests that avoidance behavior may persist, and the mere availability of an avoidant response may be sufficient to renew fear following treatment. This may represent one reason patients relapse following exposure therapy (Ginsburg et al., 2014).

The reason for the deleterious impact of avoidant behavior availability following successful extinction or exposure remains unclear. One possibility is that removing avoidant behavior during extinction represents a context shift such that it differs from both

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the original context in which fear was acquired as well as other contexts that might be encountered after extinction/exposure. As such, when the avoidance response is available again, this represents another context shift from extinction, and fear is then renewed in the same way that it might if extinction had taken place in a different physical context/environment (Vansteenwegen et al., 2005; Vervliet & Indekeu, 2015). Regardless, these findings suggest that it may be important to examine the treatment of avoidance behavior as an adjunct to exposure-based procedures in order to mitigate renewal of symptoms (Vervliet & Indekeu, 2015).

The present paper critically examines the treatment of avoidant behavior through the lens of modern learning theory. Through examination of the various functions avoidant behavior may serve in associative learning processes, as well as its neurobiological substrates, we aim to highlight novel behavioral and pharmacological interventions that may serve as useful adjuncts to traditional evidence-based strategies for anxiety and related disorders. In addition, given the dearth of evidence elucidating the mechanisms responsible for the return of fear following treatment as a result of the availability of avoidance behavior, we conclude with concrete recommendations for future research.

1. Avoidant behavior as a conditional inhibitor

1.1. Extinction of conditional inhibition

Extinction learning is one of the presumed mechanisms of exposure therapy (Craske et al., 2008; Scheveneels, Boddez, Vervliet, & Hermans, 2016) and operates via error correction mechanisms, such that the associative strength of a CS is updated when the US does not occur. During learning, the greater discrepancy between what is predicted and what actually occurs, the larger the amount of associative change that takes place (Rescorla & Wagner, 1972). Conditional stimuli that predict the occurrence of a US are known as “conditional excitors” whereas stimuli that directly predict the absence of the US are “conditional inhibitors”. During extinction training, in which a conditional excitor is repeatedly presented in the absence of the US, the concurrent presence of conditional inhibitors decreases the expectation that a US will occur, resulting in less expectancy violation, and therefore negatively impacts extinction learning (Lovibond, Chen, Mitchell, & Weidemann, 2013; Lovibond, Mitchell, Minard, Brady, & Menzies, 2009; Rescorla, 1969).

Avoidant behaviors, or “safety behaviors”, have often been discussed in terms of conditional inhibition (Krypotos, Effting, Kindt, & Beckers, 2015). For example, the use of benzodiazepines in panic disorder, washing one's hands in obsessive-compulsive disorder, and a combat veteran sitting with his back to a wall in a restaurant are all examples of avoidant behaviors that may function as conditional inhibitors as they are directly associated with the decreased likelihood of the US occurring (See Fig. 1 for a graphical representation of the relationship between a CS+, conditional inhibitor, and a US). Importantly, despite functioning as a conditional inhibitor, the availability of avoidance behavior may still become a contextual feature and lead to context renewal (Vervliet & Indekeu, 2015). That is, when the avoidance behavior is available following treatment, this may represent a context shift from exposure procedures when avoidance was prohibited, and results in a return of fear. The implication would be that allowing some avoidance behavior during exposure may be beneficial to reduce subsequent context renewal, although the deleterious impact of avoidance behavior (e.g., conditional inhibitors) on extinction learning represents a significant problem. Thus, conditional inhibition has to be reduced, or the negative effects of conditional inhibitors on extinction learning needs to mitigated, prior to allowing avoidance behaviors that function as inhibitors during exposure therapy. Below, we discuss specific treatment approaches for targeting conditional inhibitors as an adjunct to exposure therapy.

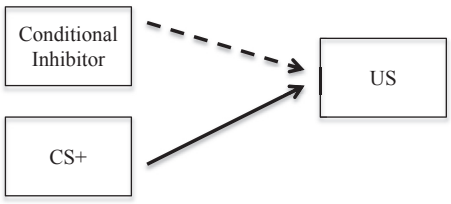

Fig. 1. Relationship of a conditional inhibitor to a US. Dashed lines represent direct inhibitor associations, whereas solid lines represent direct excitatory associations.

The traditional paradigm for developing conditional inhibition is to pair a neutral stimulus (B) with an excitatory stimulus (A) without reinforcement (e.g., A+ then AB-). The resulting decrease in associative strength gradually transforms the previously neutral stimulus into an inhibitor. For example, engaging in compulsive behavior (neutral stimulus) when one has obsessive thoughts (conditional stimulus) gradually transforms the compulsive behavior into a conditional inhibitor when the US doesn't occur. However, dominant learning models suggest that presenting a conditional inhibitor by itself should result in a gradual loss of inhibition, and may offer one potential strategy for targeting avoidance behavior. Rescorla and Wagner (1972) conceptualized change in associative strength as a function of the total amount of learning a US can support (λ) minus the sum of the associative strength of all the stimuli present on a given trial (ΣV). Let us assume a negative associative strength of a conditional inhibitor of -.5. Presenting it alone, in the absence of another CS or US should result in a net positive amount of associative change (λ - ΣV becomes 0 − [-0.5]) that will gradually eliminate inhibition (Zimmer-Hart & Rescorla, 1974). For example, an individual with obsessive-compulsive disorder may be asked to wash her hands compulsively in the absence of touching a contaminated surface while someone with panic disorder may be asked to take a benzodiazepine at times when he is not anxious. Although this is consistent with dominant learning models, numerous animal studies have failed to find any loss of inhibition after repeatedly presenting a conditional inhibitor in isolation (e.g., DeVito & Fowler, 1987).

However, in a study of human contingency learning, Melchers, Wolff and Lachnit (2006) argued that one *could* produce extinction of conditional inhibition depending on the nature of the US. The authors argue that traditional Pavlovian procedures use unconditional stimuli that only vary unidirectionally. For example, one is either shocked or not shocked in conditioning and extinction experiments. However, the Rescorla-Wagner model's assumption that inhibition is the opposite of excitation would necessitate that the US can take on values less than zero. When the US can only vary in one direction, a conditional inhibitor predicts the non-occurrence of the US and there is no discrepancy, or extinction learning, when it is presented alone without the US. However, when the US can take on both positive and negative values, then presenting a conditional inhibitor in isolation can still lead to expectancy violation.

In the Melchers et al. (2006) study, participants were divided into two groups tasked with determining whether a fictional individual's hormone levels would rise (US) based upon consumption of certain foods (CS). In one group, hormone levels could only rise or remain the same (unidirectional group), whereas in the other group hormone levels could rise, remain the same, or decrease (bidirectional group). Using standard paradigms for developing inhibition the authors demonstrated that you could reduce