Exposure therapy for obsessive–compulsive disorder: An optimizing inhibitory learning approach

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ABSTRACT

This paper is part of a series that explores the application of different therapies to a case of obsessive–compulsive disorder (OCD). It describes the application of an optimizing inhibitory learning approach to exposure with response prevention for OCD. This paper covers the basic theory and applications of the optimizing inhibitory learning approach for OCD and provides a detailed case conceptualization. Specific examples illustrate how to conduct exposure in a manner that enhances inhibitory learning with the goal of enhancing long-term exposure therapy outcomes. The goal of this paper is to familiarize clinicians with the function that inhibitory learning serves in exposure treatment and how to apply the research findings to maximize inhibitory learning in the context of treating OCD.

1. Introduction

Despite substantial research demonstrating the efficacy of cognitive–behavioral therapy incorporating exposure with response prevention (ERP) in the treatment of obsessive compulsive disorder (OCD), quantitative reviews have concluded that some patients remain symptomatic after this intervention (Abramowitz, 1998). In this paper, we present a promising approach to improving OCD treatment outcomes. Specifically, we show how to apply developments in basic learning, memory, and extinction research to the implementation of exposure therapy for OCD. We draw on Monica’s case to demonstrate concretely how to apply this methodology to optimize inhibitory learning – the type of learning that occurs during exposure therapy (Craske, Kircanski, et al., 2008; Craske, Liao, Brown, & Vervliet, 2012).

1.1. Description of the Theoretical Model

The use of ERP in the treatment of OCD cases such as Monica’s is derived from the learning theory view of (a) obsessional thoughts (and their external triggering stimuli) as conditioned stimuli that provoke fear and anxiety as conditioned responses; and (b) avoidance, compulsive rituals, and other safety cues, as strategies for managing obsessional fear that become negatively reinforced by the reduction in distress they engender. For many years, the efficacy of ERP was understood in terms of the highly influential emotional processing theory (Foa, Huppert, & Cahill, 2006; Foa & Kozak, 1986), which proposes that long-term outcomes in exposure therapy result from initially activating fear and then sustaining the exposure until fear reduction (habituation) occurs both within and between exposure sessions. From the perspective of emotional processing theory, then, effective treatment would require activating Monica’s fear of unnatural foods and products by having her confront these stimuli and use them in the ways that most people do on a daily basis. Monica would also be instructed to allow herself to imagine her feared consequences of doing so. Then, working our way up her fear hierarchy, a therapist following emotional processing theory would focus on the reduction of fear within and between exposure sessions. This would result in extinction of her conditioned fear and anxiety responses to obsessional stimuli, at least over the short-term. Monica would also be helped to resist performing compulsive rituals and to cease engaging in other anxiety-reduction or avoidance behavior, all of which would interfere with extinction. Once her fear had habituated in the presence of each item on her hierarchy, and in the absence of rituals, her treatment would be considered a success according to emotional processing theory.

Assessing the scientific validity of emotional processing theory, however, reveals that its basic assumptions are not well supported by the research evidence (Craske, Kircanski, et al., 2008; Craske et al., 2012). Successful habituation during exposure therapy for non-OCD anxiety disorders often fails to predict long-term outcomes. Conversely, successful long-term outcomes can occur in the absence of habituation during exposure therapy. How can this
be? As we will see, emotional processing theory fails to incorporate numerous developments in basic learning and memory processes strongly relevant to exposure and extinction. These developments point toward inhibitory learning as the core mechanism of extinction (Bouton, 1993; Bouton, Woods, Moody, Sunsay, & Garcia-Gutierrez, 2006; Vervliet, Craske, & Hermans, 2013) – the process driving long-term exposure therapy outcomes for OCD and other anxiety disorders (Craske, Kircanski, et al., 2008; Craske et al., 2012).

From the inhibitory learning perspective, the original danger-based association between the conditioned and unconditioned stimulus remains intact. In OCD, such associations concern not only external triggers of obsessions, but also the obsessional thoughts and doubts themselves (Abramowitz & Arch, 2014). For Monica, these associations would be “unnatural foods cause cancer,” and “uncertainty about becoming ill in the future is intolerable”. Through exposure therapy, competing non-danger-based associations between the conditioned and unconditioned stimulus are formed (“Unnatural foods are generally safe,” “Uncertainty is tolerable”). From an inhibitory learning perspective, the goal of exposure therapy for OCD is to optimize the likelihood that the non-danger associations successfully inhibit access to and retrieval of the threat associations over the long term. In other words, the goal is to maximize the strength, durability, and generalization of the learning that takes place during exposure. The degree to which threat-based versus non-threat-based associations are expressed at retest (after finishing exposure therapy) depends on the strength of inhibitory learning across time and context – rather than, as put forth by emotional processing theory, the level of fear experienced during exposure (Craske, Kircanski, et al., 2008; Craske et al., 2012). People with anxiety disorders, as well as those at high risk for developing them, show deficits in inhibitory learning (e.g., Craske, Waters, et al., 2008; Lissek et al., 2010). Thus, optimizing inhibitory learning during exposure therapy offers the potential to enhance treatment efficacy as well as to compensate for the deficits that are likely present within anxious individuals prior to the start of treatment (see Craske et al., 2012)).

In addition, we believe that increasing tolerance of fear, disgust, and uncertainty has important clinical value in treating OCD and complements the goal of inhibitory learning. Forsyth, Eifert, and Barrios (2006) argue that anxiety disorders are caused by rigid attempts to avoid and control the internal experiences of anxiety and fear rather than by the presence of anxiety and fear in themselves. From this perspective, exposure therapy for OCD should aim to increase tolerance of fear, disgust, and uncertainty, for at least two reasons. First, the goal of increasing tolerance for these experiences complements the goal of increasing inhibitory learning. To the degree that distress is tolerated (“fear of unnatural food is tolerable, so I don’t have to rid myself of this fear,” “Uncertainty about future illness is a fact of life, so I should learn to accept not knowing for sure”), inhibitory associations can be more robustly acquired (“if fear is tolerable, then I can push myself to eat more unnatural food and teach myself that I can manage this normal risk and uncertainty”) (see Arch and Craske, 2011)).

Second, previously extinguished fears can remain vulnerable to reinstatement – the return of fear following an encounter with a previously feared stimulus – following shifts in time and context. The clinical implication of reinstatement means that inhibitory learning may occasionally fail, e.g., becomes weakened to the point that the original fear-based association reemerges (see Vervliet et al., 2013)). Increasing tolerance for uncomfortable OCD-related experiences such as fear, disgust, and uncertainty during exposure therapy is thus theorized to reduce the likelihood that a lapse in inhibitory learning (a reemergence of the original fear association) will lead to a full-blown relapse of OCD (see Abramowitz and Arch, 2014). Thus, promoting greater tolerance of fear, disgust, and uncertainty may function to present relapse.

1.2. Selecting and setting up exposure tasks

Drawing upon Monica’s case of OCD, we illustrate how to conduct exposure therapy with the goal of optimizing inhibitory learning (see also Abramowitz and Arch, 2014)). This approach would aim to enhance Monica’s inhibitory learning, inhibitory regulation (approaches that engage the prefrontal cortex during exposure in a manner that enhances inhibitory learning; Craske, Kircanski, et al., 2008; Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014) and retrieval of non-danger associations (approaches that promote remembering new learning in diverse contexts and over time), using methods derived from the basic science of learning and memory. Extant evidence from clinical and analog studies yields stronger support for some inhibitory optimizing methods than for others (Vervliet et al., 2013). To treat Monica’s OCD, we present approaches to optimizing inhibitory learning for which some (human) clinical research evidence exists and that are implementable in a typical clinic context (e.g., we will omit recommendations to use pharmacological aids that may enhance inhibitory learning but are not available in most clinics or practice settings). We also note that components of the optimizing inhibitory learning approach have been successfully demonstrated within contamination anxiety samples (Kircanski, Mortazavi, et al., 2012), but not yet within diagnosed OCD samples. However, OCD along with most other anxiety disorders can be explained by fear conditioning within diagnosed OCD samples. However, OCD along with most other anxiety disorders can be explained by fear conditioning models (see Craske et al., 2014)). Thus, although future work is needed to test this directly, it is reasonable to expect that similar optimizing inhibitory learning principles will apply to OCD.

2. Enhancing inhibitory learning during exposure

Treatment would aim to enhance the inhibition of Monica’s fear-based associations with unnatural foods and products (and reduce her behavioral avoidance) by designing exposure practices that violate her expectancies about threat and danger, use variable exposure practice and deepened extinction, and remove safety signals and behaviors. Before beginning exposure, the following rationale would be presented:

The main goal of exposure therapy is to learn something new. In this case, we want you to learn two important things – first, that unnatural foods and products are generally safe enough to use. Second, we want you to learn that you can tolerate the fear and uncertainty that is provoked when you use these sorts of foods and products. In other words, there is no way to know exactly how something you eat today might affect you in the future. Most health-conscious people do their best to avoid harmful foods and products, but are able to manage the inevitable uncertainties of life. And plenty of health-conscious people eat and use the sorts of foods and products that make you fearful. By repeatedly confronting these fears, including the fear of uncertainty about your health, you will learn that you are able to manage these thoughts and feelings without the need to avoid or do compulsive rituals.

Once Monica understands this rationale for exposure, the therapist would work with her to construct a list of feared situations and stimuli, and rate how much anxiety each item on the list provokes – just like constructing a traditional fear hierarchy. Traditional exposure moves steadily from lower item on the hierarchy to the highest, with each item repeated until anxiety diminishes or disappears (i.e., habituates). In contrast,
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