

Invited Essay

Optimizing inhibitory learning during exposure therapy

Michelle G. Craske*, Katharina Kircanski, Moriel Zelikowsky,
Jayson Mystkowski, Najwa Chowdhury, Aaron Baker

Department of Psychology, 1285 Franz Hall, Box 951563, Los Angeles, CA 90095-1563, USA

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Abstract

Prevailing models of exposure therapy for phobias and anxiety disorders construe level of fear throughout exposure trials as an index of corrective learning. However, the evidence, reviewed herein, indicates that neither the degree by which fear reduces nor the ending fear level predict therapeutic outcome. Developments in the theory and science of fear extinction, and learning and memory, indicate that ‘performance during training’ is not commensurate with learning at the process level. Inhibitory learning is recognized as being central to extinction and access to secondary inhibitory associations is subject to influences such as context and time, rather than fear during extinction training. Strategies for enhancing inhibitory learning, and its retrieval over time and context, are reviewed along with their clinical implications for exposure therapy and directions for future research.

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Introduction

The primary goal of this paper is to address the gap between advances in the basic science of extinction learning and memory on the one hand, and models and methods of exposure therapy for phobias and anxiety disorders on the other hand. The argument to be made is that the customary reductions in reported fear and physiological arousal throughout exposure therapy are not evidence for corrective learning. Therapeutic efforts are better directed towards toleration of distress within a structure that enhances the consolidation and retrievability of exposure-based inhibitory learning over context and time. The goal is not to question the necessity of exposure therapy for phobias and anxiety disorders; it is already well established that phobias and anxiety disorders respond positively to approaches such as cognitive therapy (e.g., Norton & Price, 2007) and medications (e.g., Roy-Byrne & Cowley, 2002) as well as exposure-based therapies. Rather, the goal is to update conceptualizations of the mechanisms underlying exposure therapy.

The first of two lines of basic science research from which we draw is extinction learning, since the extinction of conditioned fear can be viewed as a laboratory analogue for exposure therapy (Bouton, Mineka, & Barlow,

*Corresponding author. Department of Psychology, 405 Hilgard Avenue, Los Angeles, CA 90095-1563, USA. Tel.: +1 310 825 8403; fax: +1 310 206 5895.

E-mail address: craske@psych.ucla.edu (M.G. Craske).

2001; Davey, 1997; Eelen, Hermans & Baeyens, 2001; Mineka, 1985).¹ Knowledge of the mechanisms underlying extinction learning, and the resultant conditions that facilitate or hamper extinction learning, may help to sharpen exposure treatments and maximize outcomes in both the short and long run (i.e., relapse prevention). Indeed, extinction learning has served as the explicit model of behavior therapy for phobias for many years (see Eelen & Vervliet, 2006), and extinction-like processes continue to be emphasized, albeit in ways that lag behind recent advances.

The second line of basic science research pertains to learning and memory, since what is learned throughout exposure therapy is intended to be remembered in different places and at later points in time once exposure therapy is over. The evidence pertaining to the retrieval strength of learning, presented cogently in the ‘new theory of disuse’ (Bjork & Bjork, 1992, 2006), has relevance to the long-term outcomes from exposure therapy. Naturally, this line of research overlaps with the science of extinction learning.

Before discussing these advances, we overview the prevailing model of exposure therapy for phobias and anxiety disorders, which purports that fear levels throughout exposure therapy are reflective of learning and are critical to overall therapeutic outcome.

Emotional processing theory

The concept of habituation (e.g., Groves & Thompson, 1970) was combined with the concept of ‘corrective learning’ to explain the effects of exposure therapy in the widely known ‘emotional processing’ theory (EPT), initiated by Rachman (1980), extensively expanded by Foa and Kozak (1986) and subsequently revised to take into account developments in context specificity of extinction (Foa & McNally, 1996). EPT purports that the effects of exposure therapy derive from activation of a ‘fear structure’ and integration of information that is incompatible with it, resulting in the development of a non-fear structure that replaces (Foa & Kozak, 1986) or competes with (Foa & McNally, 1996) the original one. A ‘fear structure’, as first put forth by Lang (1971), is a set of propositions about a stimulus (e.g., spider), response (e.g., racing heart) and their meaning (e.g., ‘I will be poisoned’) that are stored in memory. The fear structure is posited to be activated by inputs that match part of the structure (such as a spider, a racing heart or a thought about poisoning), which generalizes to activate other parts of the structure. The index of activation is fear, measured subjectively and physiologically.

Once activated, corrective learning occurs through integration of information that is incompatible with the structure. Incompatible information derives from two primary sources. The first is within-session habituation (WSH) of the physiological and/or verbalized fear response, that disassociates the stimulus from response propositions (i.e., the stimulus is no longer connected with fear responding). WSH is considered a necessary pre-requisite for the second piece of incompatible information, which derives from between-session habituation (BSH) over repeated occasions of exposure. BSH is purported to form the basis for long-term learning, and to be mediated by changes in the meaning proposition, in the form of lowered probability of harm (i.e., risk) and lessened negativity (i.e., valence) of the stimulus.

Hence, successful learning is indexed by initial fear activation (IFA), WSH and BSH habituation of the fear response. EPT clearly guided the focus of exposure therapy upon initial elevation followed by within- and between-session reductions in reported fear and physiological arousal, as continuation of those responses was presumed to represent erroneous evaluation of the probability of risk and negative valence. The evidence for these premises is reviewed in the following section.

Fear activation, within-session and between-session habituation as indices of learning?

IFA is operationalized as the peak response during the first exposure trial, or the first part thereof, where peak is defined as maximum fear levels (self-report or physiology) minus baseline levels (e.g., Kozak, Foa, & Steketee, 1988).² WSH is measured as the difference between the peak response and the end response of an exposure trial. BSH is measured either as the difference in peak responses from the first to the last exposure

¹Note, however, this is not to imply that extinction is the only mechanism accountable for the effects of exposure therapy.

²Subtraction of baseline values, however, may result in underestimation of peak fear, and whether peak responding is best defined by the highest value at the start of exposure or throughout the exposure trial is unclear.

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