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Mechanisms of exposure therapy: How neuroscience can improve psychological treatments for anxiety disorders

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Abstract

Exposure therapy for anxiety disorders has been one of success stories of clinical psychology and psychiatry. Nevertheless, a significant minority of patients fail to benefit from extant treatments. This clinical impasse is prompting renewed attempts to understand fear and its reduction at neural, cellular, and molecular as well as behavioral levels of analysis. The purpose of this article is to provide a review of theories of exposure therapy, including recent developments in emotional processing theory, and to discuss insights from neuroscience that promise to improve psychological treatments for reducing pathological fears.
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Cognitive-behavior therapy (CBT) for anxiety disorders is among the indisputable success stories of our field (Barlow, 2002; Craske, 1999). Syndromes once deemed nearly intractable during the heyday of psychoanalysis have yielded to science-based efforts to overcome them. Individuals suffering from an anxiety disorder today have far better prospects for relief than did their counterparts several decades ago. Embodying the principle of exposure, today's treatments affirm that the conquest of our fears requires confrontation with the things we fear the most.

These notable achievements notwithstanding, many patients fail to benefit fully from extant CBT interventions (Barlow, Allen, & Choate, 2004), and many who do benefit fail to maintain their gains (Brown & Barlow, 1995). We have now reached a therapeutic impasse, and further advances will require a deeper understanding of the mechanisms of fear and its reduction. The purpose of this article is to review insights emerging from the field of neuroscience that promise to improve our psychological treatments for anxiety disorders. Prior to addressing these breakthroughs, I review theories of exposure therapy.

1. Historical background

Contemporary exposure treatments for anxiety disorders have their roots in Wolpe's (1958) systematic desensitization. Unusual among the founders of behavior therapy, Wolpe was a physician (but not a psychiatrist) who had done experimental research on the learning and unlearning of fears in cats. Although he did very little laboratory work thereafter, his seminal experiments provided the conceptual and procedural foundations for his revolutionary clinical work. In the canonical version of desensitization, the patient was first taught progressive, deep

muscle relaxation. The therapist then had the patient imagine a hierarchy of brief, anxiety-provoking scenes. Each imagined scene was designed to provoke a dose of anxiety that could be overridden by the competing state of relaxation. Viewing neurotic fears as akin to Pavlovian conditioned responses (CRs), Wolpe conceptualized the imagined scene as the functional equivalent of a conditioned stimulus (CS). He believed that the competing relaxation response reciprocally inhibited the weaker anxiety response, thereby replacing the associative bond between stimulus (imagined fear scene) and response (neurotic anxiety) with one between the stimulus and relaxation response.

In retrospect, it is odd that Wolpe described his method as *behavior* therapy. After all, desensitization did not require much behavior of the patient, other than to become profoundly relaxed before generating a series of mental images. Indeed, for a therapy inspired by neo-Hullian S-R learning theory, a naive observer would have been at a loss to identify any stimuli or any responses. The stimuli were imaginary, and the responses were covert and unobservable.

Nevertheless, systematic desensitization had the undeniable virtue of being the first psychotherapy whose ingredients and procedures were so explicit as to permit controlled laboratory evaluation of its efficacy. Capitalizing on Wolpe's procedural lucidity, Lang and his students launched studies testing hypotheses about desensitization's mechanisms of action (Lang & Lazovik, 1963; Lang, Lazovik, & Reynolds, 1965). Inspired by Lang's pioneering work, psychologists conducted dozens of experiments designed to dismantle the components of desensitization and isolate its active ingredients (McGlynn, Mealiea, & Landau, 1981). Many of these analogue studies concerned fears of snakes in college students, prompting one wag to quip years later about the Great Snake Phobia Epidemic that had swept through American psychology departments during the 1960s and 1970s.

In parallel to work on systematic desensitization, other clinical innovators developed and tested implosive therapy (Stampfl & Levis, 1967) and its close cousins, imaginal (e.g., Foa, Blau, Prout, & Latimer, 1977; Watson & Marks, 1971) and *in vivo* (e.g., Watson, Mullett, & Pillay, 1973) flooding. Ironically, although advocates of both systematic desensitization and flooding appealed to animal learning and conditioning theory, each made opposing recommendations on how to treat pathological fear. Desensitizaton required the therapist to go easy, initiating only small amounts of distress in the patient, whereas flooding required the therapist to maximize anxiety for a sufficiently long duration to enable extinction of phobic fear.

The conclusions arising from this wave of theory-driven research on the mediating mechanisms of desensitization and flooding were oddly atheoretical. The trappings deemed essential for each treatment turned out to be not all that important. Progressive muscle relaxation, hierarchal presentation of imagined fear scenes, and so forth were not critical to desensitization, and maximal anxiety was not critical for flooding. Following Marks (1978), many clinicians concluded that the key element in fear reduction was simply sufficient exposure – preferably *in vivo* – to evocative cues until distress diminished. For pragmatic therapists, *why* exposure worked mattered little; *that* it worked was the key point.

2. Emotional processing theory

Theoretical agnosticism about mediating mechanisms is acceptable only when treatment works with flawless fidelity. But when patients fail to benefit, we need to know why. Dissatisfied with this atheoretical state of affairs, Foa and Kozak (1986) endeavored to elucidate the mechanisms of exposure therapy. Their classic article – "Emotional Processing of Fear: Exposure to Corrective Information" – has exerted a tremendous influence on clinical thinking and practice. As I document elsewhere (McNally, 2006), Foa and Kozak's article has earned remarkably high citation counts in the *Social Sciences Citation Index* year after year. Their emotional processing theory has been updated twice: ten years later (Foa & McNally, 1996), and 20 years later (Foa, Huppert, & Cahill, 2006).

Building on the work of Rachman (1980) and Lang (1977, 1979), Foa and Kozak proposed that fear is represented in memory as a network comprising stimulus propositions that express information about feared cues, response propositions that express information about behavioral and physiologic responses to these cues, and meaning propositions that elaborate on the significance of other elements in the fear structure. Applying a computer metaphor, Foa and Kozak characterized the fear network as a program for avoiding threat. Pathological fear, they said, amounted to errors in this program, characterized by excessive response elements, resistance to change, and impairments in processing certain types of information about danger and safety. Exposure therapy amounted to reprogramming the computational code to diminish clinical pathology. Opening the program, they argued, can be indexed by self-reports of increased fear and by heightened physiologic activity (e.g., heart rate). The job of the therapist is to provide information incompatible with pathological aspects of the program.