



Review

Anxiety and its treatment: Promoting science-based practice

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ABSTRACT

In this article we analyze ways that psychological science can inform the treatment of anxiety disorders. We focus on experimental psychopathology research to describe the structure of anxiety and the functions of danger, safety, predictability and controllability in contributing to disorder. We then address science-based practice in terms of principles of change and the benefits from the self-corrective nature of science, contrasting this form of practice with treatments that are not grounded in basic learning theory. Models for dissemination and implementation of science-based practices are described and related to practitioner attitudes regarding scientific evidence. Finally, we consider practice implications when treatments are, and are not, based on the informative role of clinical psychological science.

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A science-based approach to treat anxiety disorders rests on the proposition that methods should be grounded in: (a) well replicated basic findings derived from laboratory studies of anxiety and fear; and (b) controlled studies of the efficacy and effectiveness of treatment methods. Within this framework, when operating at their best, treatment methods (Nathan & Gorman, 2007) and hypothesized principles of behavior change (Rosen & Davison, 2003) are tied to our understanding of anxiety and the mechanisms that underlie psychiatric disorders. In this paper, we consider these issues; the

consequences of straying from a science-based model; and directions that can further the dissemination and implementation of empirically supported treatments.

1. Mechanisms underlying anxiety disorders

1.1. What anxiety is and is not

Fear or anxiety are not possessions that a person “has.” Nor are fear or anxiety part of one’s personhood – what one “is.” Rachman (1978) addressed the issue squarely when he declared that “Fear is not a lump (p. 7).” Rather, fear and anxiety are what one “does;” including the context and prospective time-frame in which they are experienced. Within this framework, fear and anxiety can be thought of as processes involving three different response domains

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(e.g., behavioral, physiologic, and cognitive) that bear temporal and causal relationships with each other (see *Sylvers, Lilienfeld, & LaPrairie, 2010*, for evidence that fear and anxiety are partly overlapping but different processes).

The behavioral domain for fear and anxiety comprises operant (instrumental) functions such as punishment and negative reinforcement, with the latter shaping the acquisition and maintenance of instrumental responses by terminating aversive events through escape, or by preventing the onset of aversive events through avoidance. The physiological domain of the fear/anxiety process includes respondent, reflex behaviors that are often unconditioned in nature, but which can also become acquired through the processes of direct exposure, vicarious exposure, and transfer of negative information (*Rachman, 1977*). The third domain involves verbal-symbolic (cognitive) responses. This domain provides for specification of the predictive relationship between contextual events and consequent behavioral responses. It also provides for the verbal-symbolic construction of causal/functional relationships between events and responses. Also involved are memorial processes that result in propositional networks (aka “schemata”) that instantiate relationships between physical and social contexts, and the behavioral consequences that may ensue (*Lang, 1979*).

Borkovec (1976) most clearly brought these matters to our attention in the opening moments of what some have called behavior therapy’s “cognitive revolution” (*Mahoney, 1974; Meichenbaum, 1977*; but see *O’Donohue, Ferguson, & Naugle, 2003*, for a dissenting view regarding whether this shift was genuinely a revolution). By considering the sequential and parallel relationships of three response domains, *Borkovec’s (1976)* model can account for fear and anxiety processes that go awry to produce psychopathology. This model has also stimulated a wide variety of behavioral and cognitive interventions that have gained substantial empirical support.

1.2. Prediction and control; danger and safety

Studies of danger and safety signals further illustrate how the development and implementation of effective treatments can be grounded in scientific theory. For example, research has demonstrated that aversive events that are unpredictable and uncontrollable exert a stronger negative impact on functioning than those that are predictable and controllable (*Baker & Stephenson, 2000; Mineka & Kihlstrom, 1978; Mineka & Zinbarg, 1996; Price & Geer, 1972*). *Zvolensky, Lejuez, and Eifert (2000)* noted that prediction or control may refer to either the specific eliciting stimulus (event and situation) or the individual’s responses to the stimulus. Of course, this ambiguity leaves open the important question: “Prediction and control of what?” The answer to that question appears to involve the psychological functions of danger and safety, and events that signal the occurrence of these conditions.

Humans and other species learn a great deal about the nature and function of danger through contiguity and contingency learning, or what *Mowrer (1960a,b)* referred to, respectively, as sign learning and solution learning. During initial contiguity learning of danger, individuals associate unconditioned aversive events with prior events that provide predictive information about when and where the aversive event will begin. The accompanying affective response of fear can then be transferred to the predictive stimulus, thereby turning that stimulus into a danger signal. Danger signals can serve discriminative functions for instrumental behavior in terms of approach or avoidance. Danger signals also acquire what *Mowrer* called “type-I” secondary reinforcement, referring to behaviors that are learned because they terminate the signals and accompanying distress. *Staats (1968)* extended

the analysis of contiguity and contingency learning to incorporate language processes that provide for the acquisition of signal functions in humans. Within this framework, the connotative meaning (negative valence) acquired through evaluative conditioning is transferred to the danger signal (*Bradley, Moulder, & Lang, 2005*). When evaluative meaning is transferred, the stimulus can also function as a discriminative cue for instrumental avoidance behavior. If the instrumental behavior eliminates the danger signal, it can also reduce the negative valence elicited by the signal.

One kind of safety signal is defined as that which predicts the offset of an aversive event, and consequently the learning of “relief.” This process was referred to by *Mowrer (1960a, pp. 101, 129)* as “type-2” secondary reinforcement. However, there exists the potential for learning a second form of safety signal: a stimulus that predicts the absence of onset of an aversive event. *Gray (1971)* characterized the mechanism thusly: “[the] omission of an anticipated punishment is a reinforcing event...and confers rewarding properties on stimuli (safety signals) which occur in association with it” (p. 170). Notice that this type of safety signal predicts the absence of (expected) onset of an aversive event, which might be considered “respite” rather than relief. Moreover, if respite signals were also appetitive, it would follow that the search for such signals would increase in probability, and should they be found, the search behavior would be strengthened. This too appears to have reinforcement value, if only on a partial or intermittent schedule.

White and Barlow (2002) described safety behaviors as actions that promote a sense of security. For instance, the home environment, hospitals, and cellular phones serve as powerful safety signals among those prone to panic and agoraphobia (*Rachman, 1984*). Similarly, such individuals may be hyper-vigilant for the exact location of exits or escape routes; these are stimuli that reliably predict the termination of the aversive environment. Agoraphobic individuals experience less fear if they are accompanied by a trusted companion when leaving home or venturing into an unfamiliar situation (*Carter, Hollon, Carson, & Shelton, 1995; Rachman, 1984*). By the same token, cues for safety in the internal environment may be somatic in nature, such as the perceived “deceleration” of the sympathetic nervous system. “Reassurance-seeking,” as commonly observed in generalized anxiety disorder (GAD), or ritualistic behaviors seen in obsessive-compulsive disorder (OCD) may also be conceptualized as attempts to attain safety, if only temporarily (*Woody & Rachman, 1994*).

Although danger signals often involve external and visible environment cues, those arising from the internal environment may be the most salient or poignant. For instance, detection of an acute increase in sympathetic nervous system activity can become a signal for danger among those individuals prone to panic attacks (*Clark, 1986; Craske, Lang, Tsao, Mystkowski, & Rowe, 2001; Schmidt, Forsyth, Santiago, & Trukowski, 2002; White & Barlow, 2002*) or those identified as “anxiety sensitive” (*Eifert, Zvolensky, Sorrell, Hopko, & Lejuez, 1999*). Intrusive, worrisome thoughts that occur in OCD and GAD take the form and function of warning the individual that impending harmful events are likely to occur in the future (*Jones & Menzies, 1998; Wells & Carter, 2001*). Similarly, *Arntz, Rauner, and van den Hout (1995)* demonstrated that patients with a variety of anxiety disorders inferred danger on the basis of their anxiety responses, whereas non-anxious controls inferred danger only on the basis of more objective information. Likewise, expectancies, such as anticipatory anxiety or the over-prediction of danger, also play functional roles in escape and avoidance responding (*Cox, Swinson, Norton, & Kuch, 1991*).

Preparedness theory posits that humans are equipped by natural selection to acquire phobic behaviors relative to specific stimuli, namely, those that posed threats to our primate ancestors.

That is, there is a general predisposition to fear some signals (e.g., predators, unfamiliar dark places) and not others (e.g., guns and electric sockets), even when these signals are roughly equated in their potential for danger. Studies have provided at least some support for this theory (Barlow, 2002, p. 238; but see McNally, 1987). For example, Ohman (1986) demonstrated that electrodermal responses to phobic-relevant stimuli (e.g., snakes and spiders) are acquired more quickly, and are more resistant to extinction, than similar responses to phobic-irrelevant stimuli (e.g., flowers and mushrooms). Despite these differences, the interaction of danger and safety reveals a complementary functional relationship. As Jacobs and LoLordo (1977) noted, to learn what is safe, the learning of danger must occur first. Only through this contingent process can the individual learn about those signals that predict the termination of danger, or its absence. Moreover, the more reliable the prediction of danger onset, the more rapidly safety signals can be learned (Gray, 1971; Mackintosh, 1975). Unreliable cues that signal danger will still trigger the search for safety, sometimes at a frantic pace, but the organism will eventually become exhausted from its unsuccessful and futile efforts, often retiring to a state of learned helplessness (Maier & Seligman, 1976; Woody & Rachman, 1994).

The synthesis of danger and safety signals can inform us as to when the world is anxiety-eliciting for any given individual and perhaps even for a given disorder of anxiety. At the same time, not all safety signals are created equal, and safe people, places, and things do not always result in relief or respite. For example, a single, unpredictable, and uncontrollable traumatic event that occurs in a previous safe place may undermine the individual's general sense of safety, as in the development of posttraumatic stress disorder (PTSD; Foa, Zinbarg, & Rothbaum, 1992). In a sample of 212 Pentagon staff members 13 months after the September 11, 2001, terrorist attack, those with PTSD reported a lower perception of safety at home, at work, and in usual activities and travel (Grieger, Fullerton, & Ursano, 2004). Irregular or unreliable safety signals are problematic, as their inconsistency maintains fear and may lead to continued and erratic searches for safety (Woody & Rachman, 1994). Over-reliance on safety signals may produce the effect of maintaining catastrophic beliefs in the face of repeated panics during which the feared outcome does not occur (Salkovskis, Clark, & Gelder, 1996). Some safety signals may also interfere with the habituation process during exposure to feared objects or situations, as when a trusted companion inadvertently accommodates maladaptive escape in response to signs of anxiety from the fearful individual (Rachman, 1984). Safety signals may also interfere with or prevent the learning of corrective information, thereby functioning to maintain anxiety (Salkovskis, 1991). Reassurance-seeking and ritualistic behavior may provide acute relief, but they leave untested the true likelihood of threat overestimation.

1.3. Implications for treatment of anxiety disorders

Pathways that lead to the acquisition of fear and anxiety are: (1) direct exposure to danger signals and dangerous stimuli through classical contiguity learning; (2) direct negative reinforcement of instrumental escape and avoidance from dangerous stimuli and their signals; (3) observational learning of what is dangerous and what to do about it (Rachman, 1977); and (4) information concerning potentially dangerous stimuli. The modification of fear can follow the same pathways when the strategic goals are focused on improvement in the predictability of dangerous events, the improvement in the predictability of safety, and the instrumental control of those events. Thus, the basic task of the clinician treating anxiety disorders is to elucidate the content and organization of

the subjective experience of the client. One way to do so is to adopt and implement Lang's (1977, 1979) characterization of the propositional network, which serves as the functional basis for fear imagery and its relationship to physiological activation and somato-motoric adaptation. In a classic paper, Foa and Kozak (1986) suggested that effective treatment of anxiety disorders "disintegrates" the fear structure by changing the propositional content and connective structure of the network. Grayson and Borkovec (1978) showed that modifying the response propositional features of fear narratives in systematic desensitization (e.g., hierarchy items), from avoidance and non-coping to that of mastery, results in less intense subjective fear.

In cognitive-behavioral treatment, such components as relaxation training and exposure can be implemented to assist anxious individuals to gain improved predictability and control over cues that signal danger and safety and to improve the adaptive function of responses to danger and safety. Additionally, the implementation of self-instructional thoughts or coping statements can function as readily available, internally driven sources of safety information. Indeed, several empirically supported interventions for anxiety disorders are designed to improve onset and offset of prediction and control (Barlow & Craske, 2001; Craske, Barlow, & O'Leary, 1992). For instance, self-monitoring and record keeping of panic attacks clarify learned expectations, identify precipitating events, and specify the consequences of those events. In addition, planned exposure trials are designed to provide predictable and unambiguous onset of the feared stimuli with equally predictable termination of those stimuli.

Despite the utility of applying a danger signal analysis, the empirical treatment literature is relatively sparse in the validation of treatments that are designed for improved prediction and control of safety. Available studies are primarily limited to the examination of safety signal function in panic disorder (PD). For instance, White and Barlow (2002) argued that the most important aspect of a behavioral assessment of PD is to understand the interrelationships between avoidance (safety seeking behaviors) of internal (physiological arousal) and external (signals) cues. Treatment should then be designed to improve self-generated, instrumental abilities to control external stressors and the signals that predict them. Treatment should also be designed to exercise self-control over psychophysiological processes (internal cues) that have become signals for danger, as in the phenomenon of anxiety sensitivity (Taylor, 1995; Taylor & Cox, 1998).

A similar analysis of signal functions in other anxiety disorders considers the relative importance of the perception of danger and safety in the implementation of specific treatment procedures for specific features of these disorders. For example, PD and PTSD patients are less physiologically responsive to fear memory imagery than are specific phobics and socially anxious individuals. In addition, people with PD and PTSD report the most anxiety and mood symptoms, and exhibit the most frequent comorbidity with depression (Cuthbert et al., 2003). Thus, the balance between the perception of danger and safety may differ between patients with focal fear of specific objects or events and those with intense and generalized negative affect.

A final point to consider in treatment is that all things threatening and safe cannot be completely and unequivocally predictable and controllable. Anxious patients maintaining unrealistic expectations of complete predictability and controllability may not fare well over the course of treatment and may be prone to relapse over the longer term. Although behaviorally-oriented interventions are designed to improve one's sense of prediction and control of danger and safety, a goal of treatment should also be to improve one's tolerance and acceptance of objects, situations, and events that are objectively and factually unpredictable, uncontrollable, or both.

2. Science-based practice

2.1. General considerations

In the best of all scientific worlds, there should be a correspondence between the mechanisms that account for disorder (treatment targets) and the mechanisms that underlie therapeutic change (active treatment ingredients). Mechanisms that account for disorder are empirically addressed through psychometric and experimental psychopathology research. Mechanisms underlying treatment are addressed by experimental analysis of treatment content (or components) for the efficacy of the treatments in question. Elucidation of these active treatment components is achieved through the experimental analysis of treatment efficacy. This type of analysis refers to a systematic approach in which (1) a treatment's efficacy is tested against a logical progression of comparison groups, and (2) the treatment package is dismantled into its constituent components that are independently tested to determine which are the active ingredients of the treatment (Borkovec & Costonguay, 1998; Lohr, DeMaio, & McGlynn, 2003; Lohr, Olatunji, Parker, & DeMaio, 2005). Strength of evidence for treatment efficacy increases monotonically when demonstrated against the following comparison conditions: wait-list control, attention control, nonspecific factor control, alternative credible treatment, and therapeutic alliance (Wampold, 2001). This type of experimental control demonstrates that the procedure contains an active ingredient that adds incremental efficacy to the treatment beyond that attributable to nonspecific factors, thereby providing strong evidence of therapeutic efficacy (Lohr, DeMaio, et al., 2003; Lohr, Hooke, Gist, & Tolin, 2003b; Lohr et al., 2005).

2.2. Empirically supported principles of behavior change

Numerous interventions, including systematic desensitization, flooding, virtual reality therapies, and imaginal rehearsal, are based on a principle of change whereby properly managed exposure to an aversive stimulus leads to anxiety reduction. Within this framework, the question becomes one of how to structure and manage exposure to maximize therapeutic benefit.

Consultation room exposure is most formally implemented by means of imaginal rehearsal, as in progression through a systematic desensitization hierarchy, or through flooding. "Exposure" can also occur in the process of mapping the propositional fear network. One or the other of these processes can be followed by structured exposure to the feared events outside the consultation room. For example, exposure tactics can be re-designed to encourage travel through dangerous situations toward safe places or people (Rachman, 1984). Placing a trusted companion on the far side of a bridge may encourage approach behavior in specific phobias, thereby facilitating habituation, fear-inhibiting mastery behavior, or both (e.g., Sartory, Master, & Rachman, 1989).

Active clinician participation can be the vehicle for participant modeling, coaching, and the acquisition of non-fearful behavior. It may facilitate the inhibition of fear responses, the acquisition of fear-incompatible response, or both, such as those involved in active coping (Grayson & Borkovec, 1978) and mastery skills (Hodges, McCauley, Ryan, & Stroschal, 1979). Moreover, clinicians may gain the satisfaction of directly witnessing the clinical gains that they otherwise would only hear about in the consultation room.

Empirically supported principles of change should be based upon theoretical principles that are consistent with those change processes. For example, emotional processing theory (EPT: Foa & Kozak, 1986) is based on Lang's (1977, 1979) bioinformational analysis of fear which posits propositional networks involving stimuli, responses, and meaning features that provide for relational

characteristics among them. Stimulus propositions specify what things and events elicit fear, such as the spider-phobic's reactions to webs, locations, and locomotor features of spiders. Response propositions refer to the way in which the individual spider phobic would respond in the presence of spider threat, including change in heart rate, escape, and avoidance. Meaning propositions refer to the subjective interpretations about stimuli that elicit fear or their responses during the state of fear. For example, a spider-phobic individual may interpret the spider as "scary" or their escape as "life-saving." Interrelations between these propositional components comprise fear structures that are activated when information is presented that is consistent with the networks.

EPT theory then posits that the effect of the exposure component in treatment will be proportional to the amount and content of information provided during exposure once the structure is activated by threat. Fear reduction occurs because of the weakening or dis-integration of the relations between the network of propositions. Reduction in fear responding may come from the disconfirmation of the meaning propositions ("If a spider is near, it will bite me") during exposure. It may also involve decreases in physiological responding, which then weakens the response propositions ("If I am near as spider, my heart will pound until it bursts"). The weakening of the fear structure during the habituation process is proposed at the substantive mechanism in the exposure process.

Another theoretical mechanism accounting for fear reduction is the optimization of inhibitory learning. Craske and Mystkowski (2006) and Craske et al. (2008) argue that what is learned in exposure is the inhibition of previously learned fear associations (spider-bite). That is, rather than the "erasure" of previous associations, it is learning that inhibits those associations (Bouton, 2004; Bouton, Woods, Moody, Sunsay, & Garcia-Gutierrez, 2006). Craske et al. (2008) has identified a number of clinical strategies that bolster inhibitory learning during exposure, one of which is the maintenance of arousal during exposure that may lead to better toleration of the sensations of fear that may facilitate inhibitory learning. One such procedure for the enhancement of inhibitory learning might involve the prevention of safety signals or behaviors during exposure. For example, exposing panic disorder patients to panic cues (e.g., physiologic arousal, specific contexts) without access to escape behaviors may enhance such learning. Another procedure might use multiple, and different stimuli during exposure may also enhance inhibitory learning (Vansteenwegen, Dirikx, Hermans, Vervliet, & Eelen, 2006; Vansteenwegen et al., 2007). For example, Rowe and Craske (1998) showed that when multiple stimulus examples were used during exposure for spider fear, there was less fear experienced to a novel spider than when the same spider was used during exposure.

Procedural modification of the treatment of some anxiety disorders is informed by recent psychopathology research is the function that that the emotion of disgust exercises in some specific phobias and in contamination-related OCD (Olatunji & McKay, 2009). Moreover, experimental psychopathology research has shown that disgust is a more refractory emotion than fear when applying exposure and response prevention procedures (Cisler et al., 2011; Olatunji, Forsyth, & Cherian, 2007). It follows that treatments for disgust and fear-mediated disorders may differ for the modification of these emotions (Adams, Badour, Lohr, & Feldner, in press; Meunier & Tolin, 2009). For example, McKay (2006) tested the effects of five sessions of in vivo exposure and response prevention on the extinction of subjective and anxiety in people meeting the criteria for OCD. Treatment was composed of 60 min sessions that were divided between 30 min of disgust-focused and 30 min of anxiety-focused exposure exercises. The results showed that both disgust and anxiety were reduced by exposure, but that those individuals with contamination aversion showed slower reductions in subjective disgust reactions over the course of treatment

relative to their non-contamination counterparts. Thus, treatment of disgust-related disorders may require more extensive exposure, or additional components of treatment, as compared with usual treatment for specific phobias (e.g., snakes and heights). We should note that the facilitation of inhibitory learning by using multiple and different stimuli during exposure for the treatment of fear (Rowe & Craske, 1998; Vansteenwegen et al., 2007) is also beneficial in the reduction of disgust responding (Viar-Paxton & Olatunji, *in press*).

Lastly, the manner in which treatment components are presented or framed to a patient may also influence treatment outcome. For example, Goldfried and Trier (1974) demonstrated that when relaxation training is learned as an active coping skill, its effects on anxiety symptoms are superior to standard progressive relaxation training. We propose that the phrases “self-control” and “active coping skill” are labels that further an individual’s perception of increased ability to control safety and danger when circumstances (signals) call for such skills.

2.3. Self-correcting benefits of science based practice

Empirically supported practices build upon scientific theory and ideally state the terms under which data can falsify the theory on which those practices are based (Lakatos, 1970; Lilienfeld, 1998; Popper, 1959). This adherence to empirically sound methods, and provisions for falsifiability, makes the self-correction of science possible. The absence of falsifiability renders evidentiary claims scientifically superfluous, often “pseudoscientific” (Lilienfeld, Lynn, & Lohr, 2003). Another feature of pseudoscientific theories is the repeated invocation of “ad hoc” hypotheses, to discount evidence that is inconsistent or contradictory, especially when such hypotheses fail to enhance those theories’ content, predictive power, or both.

As in all areas of health care, the treatment of anxiety disorders has seen its share of pseudoscientific thinking, ill-founded methods, and exaggerated claims. Consider, for example, what came to be known as “power therapies” for the treatment of posttraumatic stress disorder (Figley, 1997). Power therapies included a variety of trademarked procedures, widely promoted with claims of near miraculous cures (Rosen, Lohr, McNally, & Herbert, 1998). Among these therapies were thought field therapy (TFT: Callahan, 1985); eye movement desensitization and reprocessing (EMDR: Shapiro, 1995); trauma incident reduction (TIR: Gerbode, 1995); and emotional freedom techniques (EFT: Craig, 1997). In the context of these methods, it is interesting to consider an observation made by Walsh (1912), a full century ago, that faddish techniques of the day often parallel recent trends in science. True to Walsh’s argument, power therapies use computer terminology to speak of “accelerated information processing,” neural networks, and storage of cellular memory. Despite attempts to evoke the aura of science, there has been a striking absence of support for energy based power therapies (Gaudio & Herbert, 2000; Pignotti & Thyer, 2009). At the same time, large numbers of clinicians continue to employ these methods. Pignotti and Thyer (2012) surveyed 400 licensed social workers across the United States and found that fully 75% of them used a novel and unsupported method, often including one of the power therapies.

Poole, de Jongh, and Spector (1999) argued that EMDR should not be linked to other power therapies such as TFT, TIR, and EFT. In several respects there may be some merit to their argument: EMDR has been empirically assessed in dozens of studies, shown to be effective relative to wait-list controls, and listed as “probably efficacious” for the treatment of civilian PTSD (Chambless et al., 1998). At the same time, similarities between EMDR and other power therapies (see Rosen et al., 1998), as well as with other historically flawed treatments (McNally, 1999a) are striking. For example,

efficacy studies by independent researchers have shown that EMDR, although better than no treatment, is not clearly better than a credible placebo intervention (Goldstein, DeBeurs, Chambless, & Wilson, 2000) or usual methods that employ traditional exposure based methods (Devilley & Spence, 1999; Muris, Merckelbach, Holdrinet, & Sijenaar, 1998; Rothbaum, Astin, & Marsteller, 2005; Taylor, Thodarson, Maxfield, Fedoroff, & Lovell, 2003). Moreover, both experimental (Devilley, Spence, & Rapee, 1998; Devilly, 2002) and meta-analytic (Davidson & Parker, 2001) research has convincingly shown that eye movements, or other forms of bilateral stimulation purported to be the active ingredient of EMDR, have no measurable effect on clinical symptoms. In sum, “what is effective in EMDR is not new, and what is new is not effective” (McNally, 1999b, p. 619).

Despite these findings, the promotion of EMDR as a distinctively efficacious treatment continues unabated (www.emdria.org), with claims that the method works faster than traditional cognitive-behavioral therapies. Consider, for example, claims advanced by the founder of EMDR, wherein the method was portrayed as “extraordinary,” a “miracle,” and “the most revolutionary, important method to emerge in psychotherapy in decades” (Shapiro & Forrest, 1997). Illustrating Walsh’s observation (Walsh, 1912) that fad therapies adopt the language of recent scientific innovations, one reviewer who was quoted in Shapiro’s and Forrest’s book referred to EMDR as the “silicon chip of psychotherapy” (M. Elkin in Shapiro & Forrest, 1997). Consistent with other power therapies, many proponents of EMDR have provided a series of ad hoc hypotheses as a means to discount inconsistent or contradictory evidence (see Rosen et al., 1998). Thus, when studies yielded data that questioned the efficacy of EMDR, it was suggested that higher levels of training were needed to properly implement the procedures. When studies employed this higher level of training, and still found eye movements superfluous to treatment effects, it was suggested that alternative forms of bilateral stimulation were equally effective. These changing standards and hypotheses led DeBell and Jones (1997) to observe, “With so many sanctioned variations, one begins to wonder whether EMDR is standardizable” (p. 161).

Clinicians who apply science-based practices to their patients will appreciate the real mechanisms of change that underlie planned exposure-based practice, and forego the waving of fingers in front of their patients’ eyes for the purpose of producing bi-lateral stimulation. Science-based practices similarly inform clinicians that tapping “pressure points” to re-align energy fields without prior empirical support. Instead, in both cases, we can more parsimoniously attribute any improvement from treatments such as EMDR and TFT results from to exposure and other well established learning principles we have described, or from placebo and expectational processes (Rosen et al., 1998).

Other fads and fancies further illustrate the need for clinicians to employ science-based practices. Consider, for example, the history of critical incident stress debriefing (CISD; Mitchell, 1983, 1988; Mitchell & Everly, 1993, 1998), a method predicated on the assumption that early intervention in the aftermath of trauma can significantly reduce risk of psychiatric morbidity. As the procedure became more widely promoted, claims regarding its breadth of applicability, efficacy, and purported empirical grounding grew more expansive. Some of its proponents asserted that CISD was a “tested and proven” approach that was the only way to deliver “the right kind of help,” and that alternative approaches might even engender harm (Mitchell, 1992). Yet published studies largely failed to support CISD methods and at least some evidence suggests that CISD can sometimes increase the risk of posttraumatic stress symptoms following trauma (Carlier, Lamberts, van Uchelen, & Gersons, 1998; Gist, Lubin, & Redburn, 1998; Kenardy et al., 1996; Lee, Slade, & Lygo, 1996; Lohr, Hooke, et al., 2003b). A meta-analysis

by Van Emmerick, Kampuis, Hulsbosch, and Emmelkamp (2002) of 7 outcome studies concluded that the effect size of CISD was not different than zero (including a range of negative values within its 95% confidence interval) and was less effective than either non-intervention control conditions or alternative interventions against which it has been compared. This body of research was reviewed by McNally, Bryan, and Ehlers (2003) who concluded: “Although the majority of debriefed survivors describe the experience as helpful, there is no convincing evidence that debriefing reduces the incidence of PTSD, and some controlled studies suggest that it may impede natural recovery from trauma. Most studies show that individuals who receive debriefing fare no better than those who do not receive debriefing (p. 45).”

As a result of these analyses, a widening range of guidelines for evidence-based practice, including the Cochrane Reviews (Rose, Bisson, & Wessely, 2004), the United Kingdom’s National Institute for Clinical Excellence (NICE, 2005), the World Health Organization (WHO, 2006), and the Australian Centre for Posttraumatic Mental Health (2007), have offered specific recommendations contraindicating routine debriefing. Applying our understanding of exposure-based treatments to these findings, it would appear that CISD may have provided incidental and uncontrolled exposure, absent any attempt to ensure that anxiety was habituating within the debriefing experience. This failure to titrate exposure idiosyncratically for each individual, conjoined with the failure to assess habituation before terminating the session, may help to explain why CISD appears to be harmful for at least some participants.

3. Disseminating science-based practices

3.1. Changing practitioners’ attitudes

Research exists to guide our efforts in the process of promoting science-based practices (Young, Connolly, & Lohr, 2008). Diffusion research, for example, is focused on identifying the processes by which people adopt innovations (e.g., technologies, ideas, and health practices). Particularly pertinent is the ubiquitous finding that possession and communication of empirically derived scientific data are not significant factors in persuading people to adopt innovations (Coleman, Katz, & Menzel, 1959; Menzel & Katz, 1955). The diffusion model predicts that dissemination efforts that primarily target receptive people (supporters) will actually strengthen non-receptive practitioners’ beliefs against empirically based treatment. Diffusion research also indicates that influence of potential adopter’s attitudes is related to many factors, not least of which is the identity and affiliation of the person delivering innovation information (Rogers, 2003). Diffusion research suggests that until an innovation influences opinion leaders among end-users, it is unlikely to exert a broad effect on a system (Rogers, 2003). Thus, one task is to seek out opinion leaders and educate them about empirically based treatment such that they diffuse this knowledge to others in their social environment.

3.2. Models for improved implementation and dissemination

In a series of papers on “implementation science,” Glasgow and his colleagues observed that current approaches to studying treatment outcomes have generally failed to produce rapid, robust and sustainable real world health care programs and policies (Glasgow & Chambers, 2012; Glasgow et al., 2012; Glasgow & Steiner, 2012; Kessler & Glasgow, 2011). These authors argued that it is time to think and act differently: moving beyond tightly controlled RCTs and fixed protocols, toward research designs that are flexible, use mixed methods, and display external validity. New models for demonstrating treatment efficacy in actual clinic

settings may greatly contribute to the readiness of practitioners to adopt empirically supported methods.

In addition to innovative research strategies that promote real world applications, psychologists are considering alternative models of delivery (Gunter & Whittal, 2010). Kazdin and Blase (2011a) proposed that self-help materials, and other innovative delivery options, provided feasible alternatives to traditional one-on-one therapies. Their suggestion that psychotherapy research and practice needed to be “rebooted” set off a robust debate (Kazdin & Blase, 2011b). Whatever may be debated, there is no question that alternative delivery models can improve patient access and further the implementation of empirically supported therapies. In this context, the emerging field of computer-assisted therapies is receiving increased attention (Kiluk et al., 2011). Roy-Byrne et al., 2010 recently demonstrated that the computerized delivery of evidence-based treatments for multiple anxiety disorders achieved better results in a primary care center than usual care (Roy-Byrne et al., 2010; Craske et al., 2011).

Finally, promoting the adoption of science-based practices may require the establishment of national, private, for-profit business ventures focused on dissemination of training and provision of empirically supported services. Although some in our field may feel that “profit” is a dirty word, financial gain does typically serve as a powerful reinforcer for human behavior. Should this straightforward behavioral principle be ethically integrated into dissemination strategies, it could substantially advance the adoption of science-based practices. Some in our own field have adapted this model to great effect, such as Marsha Linehan’s promotion of empirically supported treatments for severe personality disorder and suicidal behavior (www.behavioraltech.com).

4. Practice implications

A desire to help others is assumed to be the most important consideration that motivates those who provide mental health services. Yet, few practitioners appreciate how difficult it can be to determine whether treatment efforts are effective. Most specifically, practitioners often do not recognize the formidable obstacles to determining whether a treatment is more effective than doing nothing, or whether the beneficial effects of a specific treatment exceed those of nonspecific factors (“just getting help”) that are common to many different therapies (Chambless & Ollendick, 2001). Moreover, they frequently provide treatment while holding two implicit misconceptions. First is the misconception that “doing something is better than doing nothing.” A growing body of research, some of which we have already reviewed, questions this assumption (see also Lilienfeld, 2007; Lilienfeld et al., 2003).

Second, many therapists assume that they can rely on self-evidential knowledge, such that controlled research is not necessary to assess treatment effectiveness. These therapists believe it is self-evident that their preferred therapy “works” because their clients tell them so and they see improvement across the process of treatment. Self-evidential knowledge, however, is subject to the fallacy of naive realism, the belief that the world operates exactly as we see it, that “seeing is believing” (Ross & Ward, 1996). Therapists operating under naive realism may not realize that their assumptions, beliefs, and expectations may influence their perceptions of the world (“believing is seeing”), and that crucial unknown or unmeasured variables may account for causal perceptions (Segall, Campbell, & Herskovits, 1966). Thus, students may assume incorrectly that they can rely on the raw data of their direct experience to gauge treatment effectiveness and fail to appreciate that change may only be apparent, or if real, may be due a variety of unknown and often non-intuitive variables (e.g., regression to the mean).

Also, self-evidential knowledge is prone to confirmation bias, which is the tendency to ignore or minimize evidence that disconfirms assumptions, beliefs, and expectations and to focus on evidence that supports them (Nickerson, 1998). Specifically, therapists may overestimate the effectiveness of therapy because of the propensity to attend to and remember examples of positive change, while forgetting or discounting evidence that change has not occurred (see Grove & Meehl, 1996).

When considering the need for science-based practices, it may be helpful to keep in mind the long history of failed medical treatments. Doing so may engender a degree of humility that naive realism may preclude. Prior to 1890, most treatments (e.g., bleeding, blistering, and patent medicines) were believed by doctors to be effective, but were actually ineffective or harmful (Grove & Meehl, 1996). In psychiatry, early reports of the effectiveness of prefrontal lobotomy were based on surgeons' informal and clinical observations. Subsequent controlled observations showed quite a different story (Dawes, 1994). These historical examples demonstrate that exclusive reliance on clinical judgment, rather than controlled and well replicated data, can lead practitioners to fall victim to the promotion of pseudoscientific techniques (e.g., Dawes, 1994; Herbert et al., 2000; Lilienfeld, 2007; Lilienfeld et al., 2005). To avoid such outcomes, the evaluation of all psychosocial treatments must rest upon the substantive aspects of procedures rather than on their superficial appearance. The practice and research communities will jointly benefit only when psychosocial interventions are marketed and accepted on the basis of sound theories of anxiety and its disorders, sound theories of specific treatments of those disorders, and probative evidence of treatment efficacy and effectiveness.

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