Behavioral Activation for Anxiety Disorders

Derek R. Hopko , Sarah M. C. Robertson and C. W. Lejuez

Abstract

Accumulating data suggest that behavioral activation interventions may be an effective approach to treating clinical depression. Given the high comorbidity and construct overlap between anxiety and mood disorders, a conceptually and methodologically integrated intervention that addresses symptoms of both conditions is indicated. As a means to this end, two questions are addressed. First, is the construct overlap and functional similarities of anxiety and mood disorders substantial enough to warrant an integrated intervention? Second, are behavioral activation treatments conceptually compatible with traditional behavioral interventions for anxiety disorders? To address these questions, behavioral activation interventions and their underlying principles briefly are described, followed by a functional analytic framework in which depressive and anxiety based symptom patterns are viewed as conceptually parallel in the context of a general negative affective syndrome. Finally, practical applications of behavioral activation for anxiety are discussed, and a case illustration is presented to highlight how behavioral activation may be used to treat a patient with coexistent anxiety and depressive pathology.
Key Words: Behavioral Activation, Anxiety, Depression, Emotional Disorders.

Behavioral activation may be defined as a therapeutic process that emphasizes structured attempts at engendering increases in overt behaviors that are likely to bring patients into contact with reinforcing environmental contingencies and produce corresponding improvements in thoughts, mood, and overall quality of life (Hopko, Lejuez, Ruggiero, & Eifert, 2003). Traditional behavior therapy for depressive disorders included an activation component whereby the objective was to increase access to pleasant events and positive reinforcers and simultaneously decrease the intensity and frequency of aversive events and consequences (Lewinsohn & Atwood, 1969; Lewinsohn & Graf, 1973; Zeiss, Lewinsohn, & Munoz, 1979). Empirical support for these interventions generally was favorable, with such approaches deemed as effective in attenuating depressive behaviors as cognitive and interpersonal skills training methods (Zeiss et al., 1979). Other researchers were less supportive of conventional behavior therapy, however, and it was demonstrated that mildly to moderately depressed college students did not become less depressed following participation in events and behaviors rated as highly pleasurable (Hammen & Glass, 1975). Given equivocal support for *pure* behavioral interventions, paradigmatic undercurrents associated with the cognitive revolution, and the contention that more multi-faceted interventions that included cognitive restructuring rendered superior outcomes to pure behavioral approaches (Blaney, 1981; McLean & Hakstian, 1979), these pioneering behavioral therapies for depression gradually were replaced by more integrative cognitive-behavioral therapies (Beck, Rush, Shaw, & Emery, 1979; Lewinsohn, Antonuccio, Breckenridge, & Teri, 1984; Lewinsohn, Munoz, Youngren, & Zeiss, 1986; Lewinsohn, Sullivan, & Grosscup, 1980; Rehm, 1977).

Following the publication of a component analysis study indicating that comprehensive cognitive-behavioral therapy was no more effective than a behavioral intervention in treating depression, interest in pure behavioral approaches was revitalized (Gortner, Gollan, Dobson, & Jacobson, 1998; Jacobson et al., 1996). Subsequent to this study, substantial preliminary data have accumulated that support the utility of two new interventions: *Behavioral Activation* (BA; Addis & Martell, 2004; Martell, Addis, & Jacobson, 2001) and the *Brief Behavioral Activation Treatment for Depression* (BATD; Lejuez, Hopko, & Hopko, 2001, 2002). These approaches have been effectively used with depressed patients in a community mental health center (Lejuez, Hopko, LePage, Hopko, & McNeil, 2001), an inpatient psychiatric facility (Hopko, Lejuez, LePage, Hopko, & McNeil, 2003), as a supplemental intervention for patients with co-existent Axis I (Hopko, Hopko, & Lejuez, 2004; Jakupak et al., in press; Mulick & Naugle, 2004) and Axis II disorders (Hopko, Sanchez, Hopko, Dvir, & Lejuez, 2003), in a group therapy format (Porter, Spates, & Smitham, 2004), and as a treatment for depressed cancer patients in primary care (Hopko, Bell, Armento, Hunt, & Lejuez, 2005). In perhaps the most compelling study to date,

outcome data indicate that behavioral activation (Martell et al., 2001) may be comparable to cognitive therapy and Paroxetine, with the psychosocial interventions associated with longer-term gains and reduced medical costs (Hollon, 2003).

Considering that behavioral activation interventions have almost exclusively been used in the context of treating depressive disorders and the consistent finding that approximately 50% of individuals with depression have a coexistent anxiety disorder (Kessler et al., 1996; Mineka, Watson, & Clark, 1998), further work is necessary to explore the adequacy of behavioral activation interventions in targeting anxiety-related behaviors. In line with this objective, at least three two questions need to be addressed. First, are the constructs of *anxiety* and *depression* functionally similar to a degree that would warrant the use of an integrated intervention? Second, if functional similarity is apparent, does this translate into the applied domain whereby behavioral activation treatments are conceptually compatible with efficacious interventions for anxiety disorders? Related to this issue, if theoretical and applied compatibility is evident, is it necessary to modify the behavioral activation process to better accommodate anxiety related symptoms? If so, how? To address these issues, behavioral activation interventions and their underlying principles briefly are described, followed by a functional analytic framework in which depressive and anxiety based symptom patterns are viewed as conceptually parallel in the context of a general negative affective syndrome (Barlow, Allen, & Choate, 2004; Barlow & Campbell, 2000). Finally, a case illustration is presented to highlight how behavioral activation may be used to simultaneously treat a patient with coexistent anxiety and depressive pathology.

Behavioral Activation Procedures and Principles. Behavioral activation (BA; Addis & Martell, 2004; Martell, Addis, & Jacobson, 2001) focuses on the functional aspects of depressive behavior, with the emphasis on evolving transactions between the person and environment over time and the identification of environmental triggers and ineffective coping responses involved in the etiology and maintenance of depressive affect (Martell, et al., 2001). The basic model highlights how the experience of being depressed involves bi-directional relationships among life events (e.g., loss, conflict, relationship distress, relocation, personnel change at work), modified reinforcement schedules (i.e., decreased response-contingent positive reinforcement, increased punishment, negative reinforcement of depressed behavior), and secondary problems that include avoidant coping, inactivity, and rumination. *Behavioral avoidance* is central to the BA model, with depressed behavior (e.g., inactivity, withdrawal) viewed as a coping strategy to avoid environmental circumstances that provide low levels of positive reinforcement or high levels of aversive control. Accordingly, the primary treatment objective is to decrease avoidant behaviors and increase response-contingent positive reinforcement, by first increasing a patient's awareness of how internal and/or external events (triggers) result in negative emotional responses that may effectively establish a recurrent avoidance pattern. Once this pattern is recognized, the principal goal is to assist the patient in reengaging in various healthy behaviors through the development of alternative coping strategies. To reduce escape and avoidance behaviors, patients learn to assess the function of their behavior, and thereby make an informed choice as to whether to continue escaping and avoiding or rather engage in more adaptive behaviors that may improve their mood and quality of life. Treatment components used to facilitate action and active coping include rating mastery and pleasure of activities, assigning activities to increase mastery and pleasure, mental rehearsal of assigned activities, role-playing behavioral assignments, therapist modeling, periodic distraction from problems or unpleasant events, mindfulness training or relaxation, self-reinforcement, and skills training methods (e.g., sleep hygiene, assertiveness, communication, problem solving).

In a more abbreviated behavioral activation treatment for depression (BATD; Lejuez, Hopko, & Hopko, 2001, 2002), depressed behavior is conceptualized within the framework of matching theory (Herrnstein, 1970; McDowell, 1982). In this model, the duration and frequency of depressed relative to nondepressed (or healthy) behavior is directly proportional to the relative value of reinforcement obtained for depressed versus nondepressed behavior. When the value (e.g., accessibility, duration, immediacy) of

reinforcers for depressed behavior increases through environmental change (e.g., increased access to social attention, increased opportunity to escape aversive tasks), the relative value of reinforcers for healthy behavior decreases, increasing the likelihood of depressive behavior. Similarly, when the value of reinforcers for healthy behavior is decreased through environmental change (e.g., decreased availability of peers), the relative value of reinforcers for depressed behavior simultaneously increases. Consistent with the BA model, the BATD paradigm focuses on modifying avoidance behaviors, and predicts that facilitating approach behaviors will increase contact with reinforcers for healthy behavior, decrease punishing environmental experiences, and thereby decrease depressed behavior. Based on this hypothesis, initial sessions of BATD consist of assessing the function of depressed behavior and efforts to weaken access to positive and negative reinforcement. A systematic activation approach then is initiated to increase the frequency and subsequent reinforcement of healthy behavior, that begins by identifying behavioral goals within major life areas that include relationships, education, employment, hobbies and recreational activities, physical/health issues, spirituality, and anxiety-eliciting situations (Hayes, Strosahl, & Wilson, 1999). Subsequent to goal selection, an activity hierarchy is constructed in which 15 activities are rated ranging from "easiest" to "most difficult" to accomplish. Using a master activity log (therapist) and weekly behavioral checkout (patient) to monitor progress, the patient progressively moves through the hierarchy. For each activity, the therapist and patient collaboratively determine what the *weekly* and *final* goals will be in terms of the frequency and duration of activity per week. Goals for each week are established as a function of patient success or difficulty with goals for the prior week.

Inherent to both activation interventions, depressive behavior is conceptualized from a contextual perspective that considers behavior as a function of the environmental contingencies that shape and maintain its occurrence, and encourages the identification of environment-behavior relations that may be measured objectively and reliably. From the behavioral activation perspective, therefore, patients and therapists target behavior that is within the realm of patient control and where the environmental context can be manipulated (Hayes et al., 1999). Private events (thoughts, feelings) do not fall into this category, are perceived as more difficult to observe and measure, and thus are not targeted in therapy. These private maladaptive behaviors are expected to attenuate following overt behavior change.

Relative to traditional treatments, newer activation protocols are more idiographic in that they target unique environmental contingencies maintaining depressed behavior. This emphasis has resulted in a movement from targeting pleasant events per se (Lewinsohn & Graf, 1973) to understanding the *functional* aspects of behavior change (Martell et al., 2001), which generally involves a detailed assessment of contingencies maintaining depressive behavior, an idiographic assessment of patients' short and long-term goals, and the subsequent targeting of value-based behaviors. Although this process may involve several strategies as outlined above, the critical mechanism of change is to decrease avoidance behavior and increase reward via principles of extinction, fading, shaping, and differential reinforcement of healthy behaviors (Hopko et al., 2003).

Functional Analytic Models of Depression and Anxiety. To illustrate the commonalities of anxiety and depressive symptom patterns and to make an argument for using an integrated behavior activation approach to independently or concurrently treat these symptoms, it is helpful to briefly review etiological models of anxiety and depressive disorders. Beginning with the work of C.B. Ferster (1973, 1981), several functional analytic models of clinical depression have been proposed. Ferster proposed that depression occurs as a result of sudden environmental changes in which opportunities for positive (social) reinforcement become limited, resulting in a response pattern of escape and avoidance behaviors (e.g., inactivity, rumination) that come to be associated with dysphoria, aversive experiences (e.g., negative social encounters, loneliness), and a narrowing of behavioral repertoires. Because healthy (non-depressed) behaviors are extinguished, depressed behaviors come to be reinforced on a stronger schedule, and avoidance behaviors (of both aversive and appetitive stimuli) limit opportunities to experience environmental reinforcement.

Peter Lewinsohn (1974) broadened the paradigm of Ferster and suggested the primary cause of depressive behaviors was low rates of response-contingent positive reinforcement (RCPR) for healthy behavior, a process believed to occur for three reasons. First, due to idiosyncratic learning histories and biological variables, lower rates of RCPR could be due to a limited number of events that were potentially rewarding to an individual (Lewinsohn & Graf, 1973; Lewinsohn & Libet, 1972). Second, limited RCPR may be a function of the reduced value of reinforcement in the environment, or a change in the frequency, magnitude, duration, immediacy, and certainty, of reward. Reinforcer value may be affected by a number of variables, including one's learning history, level of deprivation or satiation, experience with punitive contingencies, and interference created by psychological problems such as anxiety disorders (Barlow, 2002). Finally, limited RCPR may be influenced by restricted instrumental behaviors, or skills required to effectively obtain RCPR in the social environment (Segrin & Abramson, 1994).

In a third major model, the paradigmatic theory of depression (Eifert, Beach, & Wilson, 1998; Staats & Heiby, 1985) includes attention to biological risk factors, historical antecedent events, psychological vulnerability in the form of deficient and inappropriate basic behavioral repertoires, current antecedent or precipitating events, and the stimulus properties and consequences of depressive behaviors. Under conditions of inappropriate or deficient learning via interactions with the environment, repertoires may be formed that increase vulnerability to depression, such as negative self-evaluative feedback (emotional-motivational repertoire), depressive attributional style and distorted cognitive processing (language-cognitive repertoire), as well as deficient social skills and avoidance behavior (sensory-motor repertoire). When biological vulnerability (Malhi, Parker, & Greenwood, 2005) and historical antecedents such as early parental loss, insecure parental attachment, trauma, and non-contingent reward and punishment that affect repertoire development are taken into account, an individual may be predisposed toward developing depressive symptoms. Also contributing to depressive symptoms as manifested within behavioral repertoires, current environmental antecedents are highly functional and may include increased life stress, trauma, illness, decreased social support or reduction of reinforcement in another life domain, and associated changes in discriminative stimuli that decrease the likelihood of engaging in healthy (non-depressive) behavior and increase the likelihood of behaving in a depressed manner.

Several behavioral theories have also been outlined that independently highlight the acquisition and maintenance of anxiety symptoms and disorders. In the earliest of these theories, Mowrer (1939) presented the two-factor model of anxiety-related responding wherein anxiety is a conditioned response (CR) that is elicited in the presence of a conditioned stimulus (CS). According to Mowrer, this CR elicits escape and avoidance behaviors of the CS via operant conditioning principles whereby avoidance of the CS is negatively reinforced through fear reduction. Although extremely influential in the design and implementation of behavioral treatment methods, the comprehensiveness of the two-factor model has largely been questioned due its inability to explain the selectivity of phobias (Marks, 1969; Rachman, 1977), failures to extinguish phobic reactions despite non-reinforced presentations of the CS (Eysenck, 1979), and evidence that phobias often are acquired in one-trial learning (Marks, 1969). As a result of these shortcomings, a more explanatory model was adopted whereby anxiety and fear-based reactions were acquired not only through direct conditioning experiences, but also through vicarious conditioning and through transmission of information or instruction via the process of rule-governed behavior (Rachman, 1977). Through all of the three hypothesized pathways, the commonality is that regardless of the process by which fear and anxiety originates, avoidance behavior subsequently is negatively reinforced, allowing anxiety conditions to persist over time.

Developing from these initial conceptualizations, substantial theoretical and empirical work has been developed to better elucidate factors involved in the etiology and maintenance of anxiety disorders (cf. Barlow, 2002). In general, conditioning theory has now been expanded to better incorporate biological underpinnings of anxiety disorders, as well as certain cognitive processes that may be influential, such as the concepts of attentional narrowing and attentional shifts, probability learning,

information processing, perceived control, attributional style, and so forth (Abramson, Seligman, & Teasdale, 1978; Barlow, 2002; Barlow, Chorpita, & Turovsky; Martin & Levey, 1985; Mineka & Zinbarg, 1996; Rapee, Craske, Brown, & Barlow, 1996). Although these theories undoubtedly contribute to our understanding of anxiety disorders, they will not be elaborated upon in the present context. This decision is in part due to our central thesis that cognitive processes and adaptive cognitive modification may be just as likely to occur using pure environment-based (behavioral activation) strategies as compared to cognitive interventions (Hopko et al., 2003; Jacobson et al., 1996; Jacobson & Gortner, 2000; Zeiss, Lewinsohn, & Munoz, 1979). As such, the preference is to focus on overt behavior and associated contingencies of reinforcement as the primary unit of measurement, changes in which are causally related to the development and persistence of emotional disorders, with the associated premise that avoidance behavior is the fundamental underlying mechanism of emotional pathology. Accordingly, the discussion now focuses on the associations between anxious and depressive pathology, the objective being to highlight commonalities that would rationalize using behavioral activation to concurrently treat these symptom patterns.

Anxiety and Depression as a Negative Affect Syndrome: A Unified Theoretical Framework. The co-occurrence of anxiety and mood disorders is substantial, with depressed patients exhibiting a 12-month prevalence rate of 51% and a lifetime prevalence rate of 58% for any anxiety disorder (Clark, 1989; Kessler et al., 1996). Conversely, anxiety disorder patients generally exhibit similar prevalence rates for depressive disorders, with individuals experiencing panic disorder and generalized anxiety disorder (GAD) being particularly vulnerable to develop a depressive disorder (Brown et al., 2001; Clark, 1989; Kessler et al., 1998; Mineka, Watson, & Clark, 1998; Wittchen et al., 1994). Given this high degree of overlap, it is unsurprising that although there are symptom patterns that are unique to certain depressive and anxiety disorders (e.g., panic attacks, obsessions and compulsions, feelings of worthlessness), there also are several shared symptoms that include difficulties concentrating, psychomotor agitation or restlessness, fatigue, and sleep problems (Barbee, 1998). Barlow (2002) also has suggested that decreased control and predictability may be a common psychological vulnerability across patients with both types of disorders. There also are some data that suggest anxiety and mood disorders may be variable manifestations of similar neurobiological processes (cf. Barlow, 2002; Kendler, 1996) and it is common knowledge that certain medications, particularly the selective serotonin and norepinephrine reuptake inhibitors, are effective in treating both anxiety and mood disorder symptom clusters (Belzer & Schneier, 2004; Gorman & Papp, 2000). Indeed, the boundaries between anxiety and mood disorders have become increasingly indistinct over the last two decades, to a degree that many applied researchers and clinicians have adopted terms like *cothymia* and *mixed anxiety-depression* (MAD) to better capture shared etiological and syndromal patterns across disorders (Barlow & Campbell, 2000; Katon & Roy-Byrne, 1991; Rapaport, 2001; Rivas-Vazquez, Saffa-Biller, Ruiz, Blais, & Rivas-Vazquez, 2004). The significance of this movement is evident in that coexistent presentations have been proposed as more of a rule than an exception (Rapaport, 2001), and even more substantial, although the validity of a new diagnostic category has yet to be established, a recent multi-site filed trial confirmed the existence and impairment of a large number of patients with mixed subclinical anxious and depressive symptoms (Zinbarg et al., 1994).

In large part due to high rates of comorbidity and shared symptom patterns, the contention has been made that heterogeneity of anxiety and depressive symptom patterns is but an inconsequential variant of what is more importantly a broader *general neurotic* or *negative affect syndrome* (Andrews, 1990; Barlow et al., 2004; Tyrer, 1989). In addition to overlap across disorders, other data have supported this perspective (cf. Barlow et al., 2004). For example, it has become increasingly apparent that the provision of cognitive-behavioral therapy for a given anxiety or depressive disorder often results in the attenuation of symptoms associated with the treated condition, but also involves transfer effects to the "non-targeted" disorder (Borkevec, Abel, & Newman, 1995; Brown, Antony, & Barlow, 1995; Schulberg et al., 1996; Stanley et al., 2001). Furthermore, through utilization of multivariate structural modeling

studies, there are substantial data to support the notion that mood disorders are highly related to anxiety disorders, particularly GAD (Barlow et al., 2004; Brown, Chorpita, & Barlow, 1998; Chorpita, Albano, & Barlow, 1998). In general, these data indicate that two higher-order factors emerge, negative and positive affect, that have significant paths (.31-.74) to each of five diagnostic factors (depression, GAD, panic disorder, OCD, and social phobia). Barlow and colleagues (Barlow, 2002, Barlow et al., 2004) proceed to postulate that a negative affect syndrome is a product of biological vulnerabilities, early conditioning experiences, a psychological vulnerabilities (e.g., uncontrollability), and more proximal life stressors, the latter of which is a function of environmentally-based contingencies that contribute to anxious and depressive behaviors and mood states (as outlined in functional models outlined above).

Theoretical Compatibility of a Negative Affect Syndrome with Behaviorism. It is precisely this type of conceptualization that is appealing for behavior analysts and behavior therapists. Although behaviorists have supported utilization of a categorical system such as the Diagnostic and Statistical Manual for Mental Disorders (DSM, American Psychiatric Association, 2001) in that communication, financial compensation, and treatment design can be enhanced (Nelson-Gray & Paulson, 2004; Scotti, Morris, McNeil, & Hawkins, 1996), it generally is understood that reference to DSM nomenclature should represent a starting point for the clear specification of problem behaviors and their causal factors necessary for an accurate case conceptualization (Nelson-Gray, 2003). Indeed, the notion of a negative affect syndrome involves a de-emphasis on the form, structure, and categorization of abnormal behavior. Instead, there is increased focus on the common underling functions of different emotional disorders (i.e., avoidance), and more emphasis on identifying important, controllable, and causal environmental factors that are related to the etiology and maintenance of depressive and anxiety symptoms. In Barlow's model (2002, 2004), the etiology of emotional disorders involves a biological vulnerability and early life experiences that elicit a "sense of uncontrollability that seems to be at the core of negative affect and derivative states of anxiety and depression" (Barlow et al., 2004, p.213). When environmental factors that create "distress" are experienced in the context of this diathesis, the result is an anxiety or depressive disorder. So the development of emotional disorders (and avoidance behavior) ultimately is a function of biological and psychological (or learned) predispositions, in combination with more proximal environmental changes that are non-rewarding and elicit a negative emotional response. With reference to this conceptualization that is common to anxiety and depressive disorders, Barlow and colleagues (2004) postulate that the essential targets for change are action tendencies, perceived uncontrollability, and self-focused attention. These intervention targets are addressed using treatment components that include cognitive reappraisal and the reduction of emotional and behavioral avoidance (e.g., modifying action tendencies).

In the context of encouraging data on behavior activation strategies and the premise that overt behavior modification may be sufficient to not only diminish avoidance behavior, but also enhance one's sense of control and predictability over the environment and decrease self-focused attention (e.g., ruminative behavior), we offer a reformulated model of emotional disorders that is presented in Figure 1. Similar to the Barlow (2002) paradigm, certain biological vulnerabilities that include genetic and temperament variables contribute to emotional pathology. However, in contrast to the earlier model, the process of learning experiences toward understanding the etiology of emotional disorders is expanded upon, and the concept of environmental stress is further developed. In addition, we propose three somewhat divergent pathways by which emotional disorders may evolve, all of which are based on a diathesis-stress model of abnormal behavior. In the first pathway, biological vulnerabilities combine with maladaptive learning processes in which the earlier highlighted functional analytic models of anxiety and depression are central toward understanding the development of emotional disturbance. Through processes of direct conditioning and vicarious conditioning, information transmission and processing, and environmental change that may involve decreased RCPR, consequences may follow that set the stage for a clinical anxiety or depressive disorder.

Although not specifically developed to apply to anxiety or depression, Kanfer and Grimm's (1977) model represents an organizational structure of behaviors that are classified on the basis of function as opposed to problem content, and highlights how maladaptive learning might translate into negative affect. Within their model that is incorporated into Figure 1, behavioral and emotional problems are formulated with reference to one or a combination of four consequences of *maladaptive learning*, this concept expanding upon Barlow's (2002) concept of generalized psychological vulnerability. First, *behavior deficits* are highlighted as an important antecedent to psychological problems. Behavior deficits generally are presumed to occur as a result of an inadequate knowledge base for guiding behavior, skills deficits, inadequate self-directing responses, inability to alter responses in conflict situations, and deficits in self-monitoring and self-reinforcement [note the similarities to Lynn Rehm's (1977) self-control model of depression]. These behavior deficits and associated consequences could presumably contribute to avoidance behavior, perceived as a defining feature of emotional disorders. Second, *behavior excesses* such as inappropriately conditioned anxiety to objects or events and excessive self-observational activity may be problematic in that these behavioral patterns might be associated with increased depressive and anxious affect as well as inappropriate standards of reinforcement. Third, *problems in environmental stimulus control* may contribute to anxious and depressive affect. This functional class would include stimuli that elicit inappropriate emotional reactions, exposure to restrictive environments that do not provide opportunities for reinforcement, [similar to Lewinsohn's (1974) formulation], and the inefficient arrangement of controlling stimuli for daily activities (e.g., poor time management). This process might also include inappropriate self-generated stimulus control that would involve tendencies to inaccurately describe one's abilities or behaviors as they pertain to certain contexts as well as inappropriate covert (or verbal) behaviors. Finally, *inappropriate contingency arrangement* may result in negative affect. This might result from a history of limited response-contingent positive reinforcement, environmental maintenance of undesirable behavior (through reinforcement strategies), response noncontingent reinforcement, and even excessive use of reinforcement for desirable behaviors that might result in satiation. When these maladaptive learning processes are of significance in that environmental contingencies of reinforcement are sufficiently altered to produce aversive consequences and decreased reward, an emotional disorder could evolve.

Figure 1, Next Page

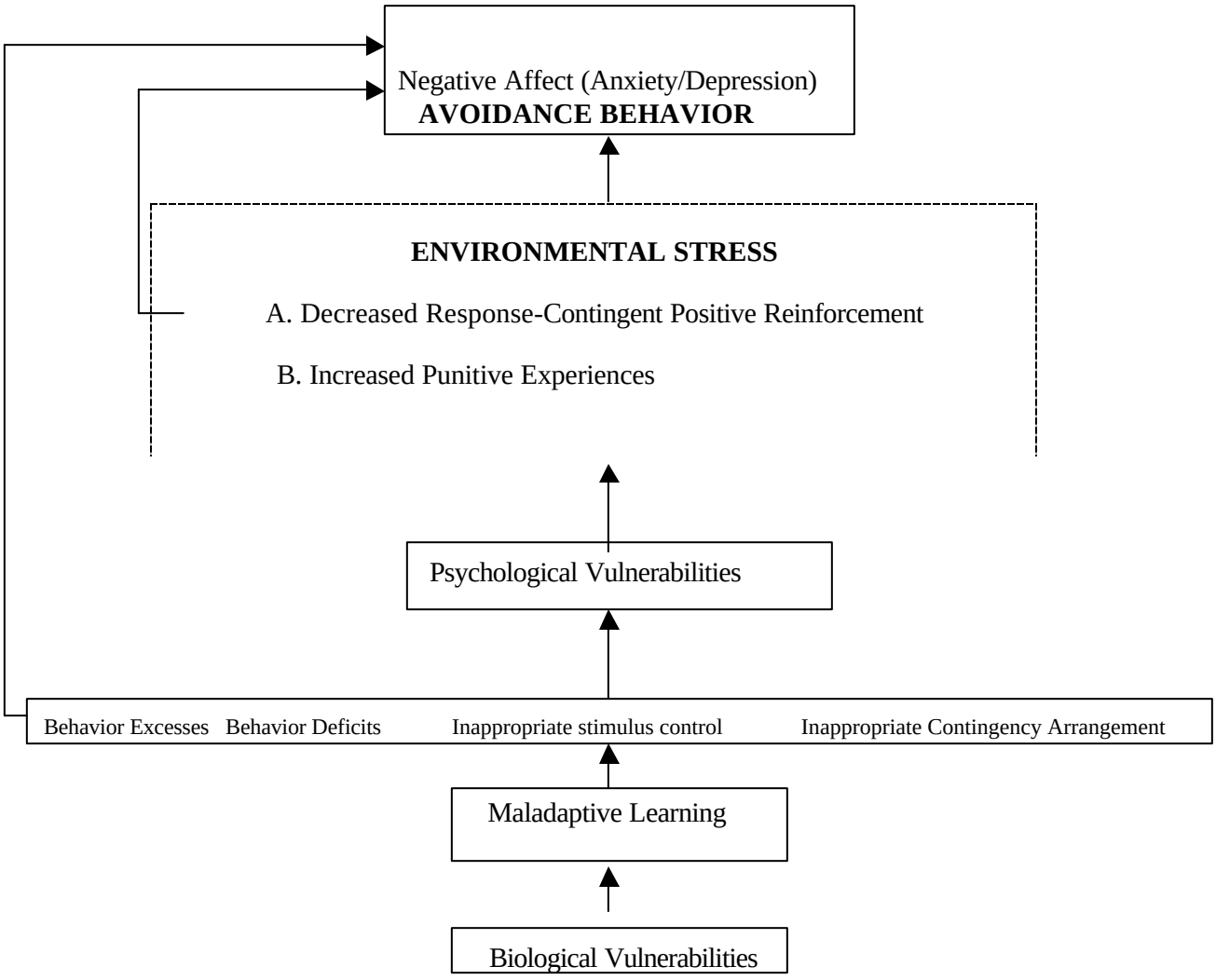

Figure 1. Reformulated model of the etiology of emotional disorders.

In a second pathway, biological and maladaptive learning variables may provide establishing operations for an emotional disorder, but may not be sufficient to elicit the development of a clinical syndrome. In such cases, the experience of further environmental "stress" (indicated by the dotted line) might be necessary for clinical symptoms of anxiety and depressive disorders to manifest, including clinically significant avoidant behavior. Environmental stress, in the form of physiological, cognitive, and/or behavioral dispositions, would be experienced as a result of an environmental change in which an individual experienced decreased response-contingent positive reinforcement and/or increased punishment. This environmental change might include limitations insofar as the quantitative aspects of reinforcement are concerned, such as the number of reinforcers available, and the duration, frequency, and magnitude of reinforcement (Lewinsohn, 1974). In addition, qualitative aspects of potentially reinforcing events may be inadequate, such as the type (e.g., social, intellectual) and or function of reinforcers (e.g., achievement, consumatory, stimulation seeking). The availability of reinforcement in the environment also would be affected by life changes that might include death of loved ones, relocation, occupational circumstances, financial hardship, social isolation, and so forth. Additionally, the experience of stress may be more directly related to increased environmental punishment, for example, when social, educational, or occupational behaviors are punished and subsequently decrease in frequency. When decreased reinforcement or increased punishment occurs, and individuals are biologically and

psychologically predisposed to negative affect, an anxiety or depressive disorder, or both, may develop. As illustrated in Figure 1, psychological vulnerabilities may also contribute to this process (see Barlow, 2002 for a discussion of specific psychological vulnerabilities by clinical disorder). These psychological vulnerabilities would be the product of early learning experiences in which individuals encounter uncontrollable or unpredictable events that could result in perceptions of diminished control, heightened anxiety sensitivity (Reiss, Peterson, Gursky, & McNally, 1986), increased helplessness and hopelessness and a negative attributional response pattern (Abramson, Metalsky, & Alloy, 1989; Seligman, 1975), as well as increased neurobiological activity, perhaps through activation of the behavioral inhibition system (Barlow, 2002).

To summarize, when biological vulnerabilities combine with maladaptive learning experiences and possible psychological vulnerabilities, environmental contingencies may occur such that individuals experience reduced environmental reward and possibly increased punishment. It is noteworthy that the extent of maladaptive learning will be quite variable across individuals who exhibit negative affect, and feasible that environmental stress may directly elicit negative affect in the absence of any substantial maladaptive learning history (the third pathway). For example, the sudden death of a family member may result in a tremendous diminution of reinforcement and elicit a clinically significant emotional problem, without prior maladaptive learning experiences. Regardless of the pathway, altered contingencies of reinforcement are hypothesized to create problems with anxiety and depression. Whether an anxiety, depressive disorder, or both develop, will largely be a function of idiosyncratic biological variables, differential maladaptive learning experiences, and divergent psychological vulnerabilities. Although it is clear that the negative affect syndrome that is created may include physiological and cognitive components (Lang, 1968), in our view the defining feature of anxiety and depressive disorders is the avoidance behavior associated with these problems. For example, there are ample data to support the prevalence of avoidance behaviors in individuals with anxiety and depressive disorders (Barlow, 2002; Dickson & MacLeod, 2004). As illustrated by Barlow and colleagues (2004), avoidance tendencies may manifest in different ways as a function of primary symptom cluster. Social phobia is associated with avoidance of performance-interactions, PTSD with avoidance of strong affect and trauma cues, specific phobias with avoidance of objects or situations, depression with general withdrawal, and so forth. In line with the focus of avoidance as the hallmark feature of affective disorders, facilitating approach behaviors to facilitate the extinction process (anxiety) and increase RCPR (depressive disorders) have been highly effective means of treatment (Barlow, 2002; Hollon, 2003; Hopko, Lejuez, LePage et al., 2003; Jacobson et al., 1996). Importantly, there also are some data to suggest cognitive symptoms of depression may be equally attenuated following pure behavioral approaches relative to traditional cognitive therapy (Jacobson et al., 1996; Zeiss, Lewinsohn, & Munoz, 1979).

So within this behavioral philosophy, the contention is that anxiety and depressive disorders, or the negative affect syndrome (including cognitive symptoms), can best be ameliorated through interventions that incorporate strategies to modify approach behaviors and eliminate the negative reinforcement associated with avoidance behaviors. In doing so, the depressed individual can reduce withdrawal behaviors and thereby work toward increasing response contingent positive reinforcement. The anxious individual can approach anxiety-eliciting situations or objects, extinguish fear through behavioral exposure, and subsequently also increase RCPR. So the end result is the same, although a slightly different process is involved. For depressed behavior, functional analytic strategies are used to identify positive and negative reinforcers that maintain or strengthen depressive behavior. These reinforcers subsequently are targeted for reduction or outright elimination using the principle of extinction (Ferster, 1973; Lewinsohn, 1974). Escape and avoidance patterns are extinguished by first examining the consequences and function of depressed behavior. Patients are then led to recognize that depressed behavior (i.e., lethargy, passivity) may be a function of trying to avoid aversive situations. Through differential reinforcement of incompatible (healthy) behavior, depressed behavior is extinguished by providing alternate sources of environmental reinforcement via the facilitation of approach behavior.

Over time, this extinction process increases the value of reinforcers for approach relative to avoidance behavior. In contrast, for anxious avoidance patterns a more direct extinction process is at work. Exposing individuals to aversive conditioned stimuli while preventing an avoidance response is an application of the principle of extinction within a classical conditioning framework. Without experiencing the anticipated traumatic event, over time anxious responding in the presence of the conditioned stimuli is likely to extinguish. Although exposure strategies are not fundamental to contemporary behavioral activation interventions for depression, avoidance behaviors characteristic of depressed individuals may partially be a function of the aversive nature of situations or individuals. To the extent that avoidance behavior occurs to minimize anxiety elicited by these contexts, the therapeutic effects of guided activity (or activation) and graduated systematic exposure might be functionally similar. In line with this position, the central thesis of this article is that behavior activation processes may be used and adapted to fulfill treatment objectives associated with both anxiety and depressive symptom patterns, and a general affect syndrome, the applied nature of which is illustrated in the following section.

Behavioral Activation as an Integrative Treatment. The primary treatment objective of behavioral activation is to reduce avoidance patterns of behavior. In the case of depressed individuals, depressive behaviors, such as passivity, lethargy, hypersomnia, suicidal ideation, and even cognitive (ruminative) behavior are perceived as having an avoidance function. Depressed individuals develop this escape and avoidance response pattern to reduce the aversive nature of internal or external environments (Ferster, 1973). In this conceptualization, when depression develops due to decreased RCPR and or increased environmental punishment, avoidance behavior occurs more frequently because it is (negatively) reinforced on a richer schedule. For example, passivity allows a depressed individual to avoid anxiety-eliciting situations or aversive contexts that elicit discomfort, and subsequently allows for the immediate and predictable reduction of any negative consequences that might occur as the result of approach behavior, such as unwanted physiological reactions, cognitive experiences, negative social interactions, and so forth. This immediate and certain outcome is a more "valued" reinforcement schedule than that associated with the potentially delayed and unpredictable qualities of reinforcement connected with approach behavior. This same pattern generally occurs in individuals with anxiety conditions, where anxious behaviors such as removal from aversive stimuli, pacing, restlessness, and even substance abuse are negatively reinforced. Unfortunately for both anxious and depressed individuals, this process also has the effect of narrowing behavioral repertoires and limiting potential opportunities for environmental reinforcement that may positively impact mood. In a similar way, the ruminative behavior that is characteristic of depressed and anxious individuals (Barlow, 2002; Beck et al., 1979) also may serve an avoidance function. For example, rumination may allow individuals to avoid experiencing and or participating in events or activities and the associated emotional consequences that might follow. In other words, worry might function to avoid greater arousal and distress (Borkovec & Roemer, 1995). This process has been referred to as *experiential avoidance*, or the attempt to alter the form or frequency of private experiences or the contexts in which they occur by being unwilling to be mindful of bodily sensations, thoughts, memories, images, or behavioral predispositions (Hayes et al., 1999). As speculated upon, although rumination may be aversive in itself, it is conceivable that it is maintained by the avoidance of even more aversive conditions (Martell et al., 2001).

It is acknowledged that anxiety and depressive disorders have multiple symptoms, some of which are shared, and some that are inherent to specific disorders. Across individuals and disorders, aversive stimuli that elicit emotional problems and specific avoidance patterns also are quite ideographic. Recognizing these issues, the multitude of factors that may be involved in the etiology and maintenance of anxiety and depressive disorders, and the many theories that highlight differential causes and correlates, a common and unifying basis for understanding and treating emotional disorders is the functional avoidance tendencies that characterize these conditions. Behavioral activation is an intervention that has shown promise as a reasonably uncomplicated and ideographic treatment to address the avoidance tendencies of depressed individuals, and is a treatment we assert may assist individuals

with anxiety disorders. First, consistent with the overarching theme of avoidance and the concept of a general negative affect syndrome, it is conceivable that the homogeneity across conditions and shared functional characteristics are similar enough such that contemporary behavioral activation interventions could be used in their present form, without modification (Hopko et al., 2003). Although very preliminary, there are some data that support this contention. For example, the severity of post-traumatic stress disorder symptoms has been shown to decline following behavioral activation treatment, even in the absence of direct exposure-based strategies (Jakupak et al., in press; Mulick & Naugle, 2004). A briefer behavioral activation intervention also showed some success in treating a patient with depression and co-existent panic disorder with agoraphobia (Hopko et al., 2004). In contrast to these data, however, although cancer patients diagnosed with depression had significant pre-post treatment gains on measures of depressive symptoms and quality of life following 9-sessions of behavioral activation, somatic anxiety did not decrease significantly as measured by the Beck Anxiety Inventory (Hopko et al., 2005). More systematic work clearly is necessary to explore whether behavioral activation in its current form is adequate to promote reductions in anxiety-related responding.

As an alternative, and taking more of the perspective that depressive and anxiety disorders are significantly heterogeneous to warrant different approaches, it may be reasonable to supplement behavioral activation protocols with efficacious interventions for anxiety disorders (Barlow, 2002; DeRubeis & Crits-Christoph, 1998). For example, as depicted in the case illustration in the following section, exposure-based strategies such as systematic desensitization and flooding could easily be incorporated into the progressive structure of behavioral activation. As applied relaxation is an effective intervention for generalized anxiety disorder, and also is systematically taught and practiced in the context of progressive muscle relaxation, this strategy also could easily be included in the behavioral activation process. In a similar manner, the response prevention strategies used for obsessive-compulsive disorder, interoceptive exposure techniques for panic disorder treatment, and the exposure methods of cognitive-behavioral treatments for social phobia, specific phobia, and PTSD also would be compatible. For clinicians who feel uncomfortable when not more directly addressing the cognitive components of anxiety and depressive disorders, the rumination cued activation strategies of behavioral activation might also be an appealing alternative (Addis & Martell, 2004; Martell et al., 2001). Indeed, all of these strategies have been incorporated into an ongoing treatment outcome study of behavioral activation for cancer patients in which we have demonstrated clinically significant pre-post treatment gains on measures of anxiety, depression, and quality of life (Hopko et al., 2005b). Continued work will assist in further exploring the efficacy of behavioral activation interventions for anxiety and also help to calibrate interventions insofar as practical applications are concerned. With reference to the reformulated model of emotional disorders highlighted earlier, the following case illustration provides an idea of how behavior activation may be used as an intervention for anxiety disorders.

Case Illustration. "Sharon" is a 31-year old married Caucasian female with three sons (ages 4, 8 and 13 years). Her 8-year old son was diagnosed with autism, and her 13-year old son currently is receiving treatment for bipolar disorder. Sharon completed two years of college, but dropped out of school due to financial stress. Sharon met DSM-IV-TR criteria for major depression, social anxiety disorder, and a specific (worm) phobia. Her symptoms of depression included depressed mood, loss of interest, hypersomnia, psychomotor retardation, fatigue, guilt, and impaired concentration. Sharon also reported significant symptoms of social anxiety, including an avoidance of parties and social gatherings, difficulty being assertive, and strong discomfort around strangers. In addition to social anxiety, Sharon reported an extreme phobia of worms. Her phobia was so debilitating that she was unable to speak the word worm, exhibited intense physiological and cognitive arousal upon seeing or thinking about worms, and did all she could to escape and avoid worms. She was particularly fearful about making physical contact with worms. This specific phobia caused significant functional impairment in that Sharon did not let her children play outside because she was fearful that they would inadvertently bring a worm into the house. She also would not allow her children to have a pet dog because she was fearful the dog would

come into contact with worms. Sharon's physiological symptoms of anxiety include increased heart rate, derealization, hot flashes, and nausea. Cognitive symptoms included patterns of catastrophic thinking, decreased self-esteem, and thoughts of worthlessness. Behavioral symptoms included escape and avoidance of social situations and contact with worms.

History of the Disorders.

Sharon had a family history of anxiety and depression and reported several experiences with psychotherapy beginning when she was 8 years old, following the kidnapping and murder of her cousin. After a year of treatment, her mood improved and her anxiety decreased. Sharon also indicated several aversive experiences with worms when she was in grade school. Most prominently, she reported that her peers frequently teased her about being fearful of worms and often threw worms in her direction. On at least one occasion a worm fell down her shirt, and there were other instances where she would find worms in unexpected places such as her school desk. Such events resulted in strong autonomic reactions. She also reported receiving psychotherapy after a divorce from her first husband, as she again experienced an episode of depression. Since she re-married, she has been psychologically healthy and reported that she was recently feeling terrific, until recently diagnosed with colon cancer. Sharon had a family history of cancer, so she was hypervigilant about her physical health. She reportedly exercised six days a week, was physically fit, and abstained from drinking alcohol in order to take care of her body and prevent an occurrence of cancer. In fact, Sharon reported that doctors were initially skeptical of her test results because she appeared so healthy. However, her diagnosis of colon cancer was confirmed and she had a colon resection. A month after surgery, she began chemotherapy, which she currently receives bimonthly. Sharon's diagnosis of colon cancer has deeply affected her and has undoubtedly contributed to her symptoms of depression and anxiety.

Psychological Assessment.

The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Brown, DiNardo, & Barlow, 1994) was administered and Sharon coded positive for major depression, social anxiety disorder, and a specific phobia. Sharon also was administered the *Hamilton Rating Scale for Depression* (HRSD; Hamilton, 1960), *Beck Depression Inventory-II* (BDI-II; Beck, Steer, & Brown, 1996), *Beck Anxiety Inventory* (BAI; Beck & Steer, 1993), *Quality of Life Inventory* (QOLI; Frisch, 1994), and the *Medical Outcomes Study Short Form* (SF-36; Ware & Sherbourne, 1992). Sharon's scores of 28 on the HRSD and 40 on the BDI-II indicated that her depression was severe and her score of 30 on the BAI indicated a moderate-severe level of anxiety. In addition, Sharon's quality of life was poor as assessed via the QOLI (-2 on a scale of –6 to +6) and the SF-36 (mental health = 24, social functioning = 25, physical functioning = 40, and general health =20).

In adherence with the behavioral activation treatment protocol, Sharon completed daily diaries where she recorded her primary activities throughout the day. These diaries revealed that the majority of her time was spent sleeping and watching TV, with these activities not eliciting very much pleasure or satisfaction. She did find enjoyment, however, when she and her husband went out to dinner and when she spent time with her children. After the daily dairies were reviewed, a life goals and values assessment was completed. Sharon's life goals included fostering a closer relationship with her children, developing new friendships, spending more time with her husband in mutually rewarding activities, trying new hobbies, finding time to exercise, attending church services, and confronting her phobia of worms and becoming less socially anxious.

Case Conceptualization.

The case formulation was conceptualized according to the reformulated negative affect model presented previously. Sharon was perceived as having substantial biological vulnerabilities in the form of medical and psychiatric problems (i.e., family history of cancer, anxiety and depression). Sharon had also undergone some maladaptive learning processes whereby she had a direct conditioning experience with worms that resulted in inappropriate stimulus control. Although her relationship to her first husband was in many ways unsatisfactory, as a couple they engaged in a substantial number of activities that she found quite pleasurable. Thus, although several aversive experiences and consequences were removed following her divorce, there also was a decrease in response contingent positive reinforcement, particularly associated with the social network that Sharon and her ex-husband had shared prior to the divorce, but one in which she no longer belonged. In addition to these more distal experiences, Sharon experienced substantial and more proximal environmental stressors. For example, her diagnosis of colon cancer and subsequent chemotherapy had altered her lifestyle substantially, affecting her perceived ability engage in many activities that she used to find rewarding. Although she deeply loved her children, she also appeared overburdened by having to care for them, particularly given their disabilities, and especially considering the increased fatigue associated with cancer treatment. It was apparent that her maladaptive learning experiences and present experience of being diagnosed and treated for cancer also could have elicited a psychological vulnerability to negative affect. In particular, an initial sense of "uncontrollability" associated with the disintegration of her first marriage was compounded by the experience of being diagnosed with cancer, especially when she had put forth substantial effort to achieve a healthy lifestyle. A negative attributional style also had apparently evolved over the course of time, with Sharon frequently taking responsibility for negative events and experiencing a significant level of self-blame, particularly related to the restrictions she was imposing on her children (e.g., limited outdoor activity). As a consequence of these factors, Sharon was experiencing physiological, cognitive, and behavioral symptoms of depression, including a rather extensive avoidance of social situations and outdoor activities. In addition to escape and avoidance strategies associated with anxiety-eliciting situations, lethargy and anhedonia were evident in that Sharon had ceased to expose herself to previously rewarding behaviors that included exercising, church activities, and social behaviors. This combination of escape and avoidance of aversive situations and withdrawal from contingencies of reinforcement were conceptualized as critical factors in maintaining anxious and depressive affect. The long-term, negative consequences of such responses included inadequate exposure to anxiety eliciting situations that inhibited the extinction process, lack of response-contingent reinforcement, and the sacrifice of highly valued behaviors. Balanced against these negative consequences were positive consequences of avoidance behavior that included the removal (or lessening) of physiological and cognitive symptoms of anxiety following instances of both active avoidance/escape (leaving an anxiety-provoking situation) and passive avoidance/escape behavior (staying in bed, watching television).

Targets Selected For Treatment.

Based on this conceptualization, a primary goal of treatment included increasing response contingent reinforcement for Sharon's behaviors. In order to alleviate depressive symptoms, she was asked to complete different activities related to each specific life goal. These specific behaviors were measured in terms of quantity and duration. For example, she was asked to spend 30 minutes each day playing with her children, 40 minutes completing her exercise video at least three times per week, and 20 minutes each day practicing relaxation techniques (both breathing and posture exercises). Among the other activities targeted for intervention were attending church, increasing prayer time, going out for dinner with her husband, small trips with her husband, and writing letters and calling friends with whom she had restricted contact. Sharon reported throughout treatment that systematically increasing pleasurable activities helped to attenuate depression symptoms.

Another goal of treatment involved teaching Sharon to recognize and resist avoidant behavioral patterns. Sharon had developed a clear pattern of experiential avoidance in her life. She stated that she didn't like interacting with people because of experienced physiological anxiety and her ultimate fear of social rejection. For example, when she was in the grocery store, she rarely made eye contact with cashiers. Rather, she presented herself as distant and disinterested so that she wouldn't elicit an interaction. In trying to reduce anxiety in her life by avoiding social interactions, Sharon was unknowingly also forfeiting opportunities to extinguish her fear and to learn that interacting with some individuals could be pleasant and rewarding. Within the behavioral activation protocol, social approach behaviors were encouraged to increase the likelihood of Sharon experiencing response contingent reinforcement. Specific activities were selected for Sharon to engage in, including attending a cancer support group, being more active in church groups, spontaneous interactions with strangers, talking to her husband about her feelings regarding their relationship, and establishing play dates with other moms and children in her neighborhood. This latter activity also served the purpose of providing Sharon with more free time to reduce the burden of being the primary caregiver. The successful completion of these approach behaviors contributed to improving Sharon's mood as well as increasing her self-efficacy.

Another goal of treatment was to help Sharon confront her fears around her specific phobia of worms. It became clear during assessment that Sharon utilized experiential avoidance to cope with her anxiety about worms. Rather than approach a worm and learn that she was capable of coping with the situation, she would schedule her days so that she minimized the amount of time she spent outside. In order to treat this phobia, we developed a hierarchy of anxiety-provoking scenarios with worms and proceeded through the hierarchy in a systematic fashion. Hierarchical goals were outlined on the master activity log of the behavioral activation protocol. The first goal was for Sharon to write the word "worm" ten times and then speak the word "worm" ten times. This was initially very difficult for Sharon, as it took her several minutes to write the word "worm" one time. However, consistent with behavioral theory, this process became easier over time. Once Sharon demonstrated proficiency in reading and writing the word, she then was given the assignment to purchase and read a book about worms. She completed this task successfully. She brought her husband along with her on this task, who undoubtedly served as a safety signal for her. However, she was able to ask the salesperson where the books on worms were located without the help of her husband. After she completed this task, several imaginal exercises were completed that induced significant anxiety. These imaginal exercises involved Sharon approaching a worm, picking it up, and allowing it to crawl on her hand. In order to combat physiological symptoms of anxiety, she utilized relaxation techniques that she learned in the context of behavioral activation. The next step on the hierarchy involved Sharon interacting with an artificial worm in session. She was able to manipulate this worm, although she was very tentative in the initial stages. She also took the artificial worm home and practiced interacting with it daily. Finally, she was given the task of approaching a real worm and staying within a five-foot radius of the worm for five minutes. Although therapy ended at this time, as Sharon was seen in the context of a treatment outcome study involving a 9-week behavior activation intervention, she was instructed to continue to systematically expose herself to feared stimuli, both social and worm-related.

Treatment Outcome. Throughout the treatment protocol, Sharon demonstrated improvement in her symptoms of depression and anxiety. At the termination of treatment, Sharon's scores on the HRSD and the BDI-II reduced to 13 and 18, respectively. Her score on the BAI also was reduced to a 12, indicating mild-moderate levels of anxiety. Sharon's quality of life also improved (1), as did her scores on the SF-36 (mental health = 50, social functioning = 62, physical functioning = 60, and general health = 65). She also demonstrated substantial improvement in her relationships with her children. By the end of treatment, she was spending 10 minutes with each child discussing their day, and 30 minutes playing with them outside. Perhaps the most robust effects of treatment involved progress with her worm phobia. At the end of treatment, she was able to manipulate a fake worm, and reported that the phobia was not interfering with her life in a significant way.

References

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological review, 96,* 358-372.

Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87,* 49-74.

Addis, M. E., & Martell, C. R. (2004). *Overcoming depression one step at a time: The new behavioral activation approach for getting your life back.* Oakland, CA: New Harbinger Publications.

Andrews, G. (1990). Classification of neurotic disorders. *Journal of the Royal Society of Medicine, 83,* 606-607.

Barbee, J. G. (1998). Mixed symptoms and syndromes of anxiety and depression: Diagnostic, prognostic, and etiologic issues. *Annals of Clinical Psychiatry, 10,* 15-29.

Barlow, D. H. (2002). *Anxiety and its Disorders: The Nature and Treatment of Anxiety and Panic* (2nd Ed.). New York: Guilford.

Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified treatment of emotional disorders. *Behavior Therapy, 35,* 205-230.

Barlow, D. H., & Campbell, L. A. (2000). Mixed anxiety-depression and its implications for models of anxiety and mood disorders. *Comprehensive Psychology, 41,* 55-60.

Barlow, D. H., Chorpita, B. F., & Turovsky, J. (1996). Fear, panic, anxiety, and disorders of emotion. In D. A. Hope (Ed.), *Nebraska Symposium on Motivation: Vol. 43. Perspectives on Anxiety, panic, and fear* (pp.251-328). Lincoln: University of Nebraska Press.

Beck, A. T., Rush, A. J., Shaw, B. J., & Emery, G. (1979). *Cognitive Therapy of Depression*. New York: Guilford Press.

Beck, A. T., & Steer, R. A. (1993). *Beck Anxiety Inventory: Manual* (2nd edition). San Antonio, TX: The Psychiatric Corporation.

Beck, A. T., Steer, R. A. & Brown, G. K. (1996). *Manual for Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.

Belzer, K., & Schneier, F. R. (2004) Comorbidity of anxiety and depressive disorders: Issues in conceptualization, assessment, and treatment. *Journal of Psychiatric Practice, 10,* 296-306.

Blaney, P. H. (1981). The effectiveness of cognitive and behavioral therapies. In L. P. Rehm (Ed.), *Behavior Therapy for Depression: Present Status and Future Directions* (pp.1-32). New York: Academic Press.

Borkovec, T. D., Abel, J. L., & Newman, H. (1995). Effects of psychotherapy on comorbid conditions in generalized anxiety disorder. *Journal of Consulting and Clinical Psychology, 63,* 479-483.

Borkovec T. D., & Roemer, L. (1995). Perceived functions of worry among generalized anxiety disorder

subjects: Distraction from more emotionally distressing topics? *Journal of Behavior Therapy and Experimental Psychiatry, 26,* 25-30.

Brown, T. A., Antony, M. M., & Barlow, D. H. (1995). Diagnostic comorbidity in panic disorder: Effect on treatment outcome and course of comorbid diagnoses following treatment. *Journal of Consulting and Clinical Psychology, 63,* 408-418.

Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology, 110,* 49-58.

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM- IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology, 107,* 179-192.

Brown, T. A., DiNardo, P. A., & Barlow, D. H. (1994). *The Anxiety Disorder Interview Schedule for DSM-IV.* Center for Stress and Anxiety Disorders. Albany: State University of New York.

Chorpita, B. F., Albano, A. M., & Barlow, D. H. (1998). The structure of negative emotions in a clinical sample of children and adolescents. *Journal of Abnormal Child Psychology, 107,* 74-85.

Clark, L. A. (1989). The anxiety and depressive disorders: Descriptive psychopathology and differential diagnosis. In P. C. Kendall and D. Watson (Eds.), *Anxiety and depression: Distinctive and overlapping features.* (pp. 83-129). San Diego: Academic Press.

DeRubeis, R. J., & Crits-Christoph, P. (1998). Empirically supported individual and group psychological treatments for adult mental disorders. *Journal of Consulting and Clinical Psychology, 66,* 37-52.

Dickson, J. M., & MacLeod, A. K. (2004). Approach and avoidance goals and plans: Their relationship to anxiety and depression. *Cognitive Therapy and Research, 28,* 415-432.

Eifert, G. H., Beach, B. K., & Wilson, P. H. (1998). Depression: Behavioral principles and implications for treatment and relapse prevention. In J. J. Plaud and G. H. Eifert (Eds.), *From Behavior Research to Behavior Therapy (pp. 68-97).* Needham Heights, MA: Allyn & Bacon.

Eysenck, H. J. (1979). The conditioning model of neurosis. *Behavioral and Brain Sciences, 2,* 155-199.

Ferster, C. B. (1973). A functional analysis of depression. *American Psychologist, 28,* 857-870.

Ferster, C. B. (1981). A functional analysis of behavior therapy. In L. P. Rehm (Ed.), *Behavior Therapy for Depression: Present status and future directions* (pp. 181-196). New York: Academic Press.

Frisch, M. B. (1994). *Manual and Treatment Guide for the Quality of Life Inventory.* Minneapolis, MN: National Computer Systems, Inc.

Gorman, J. M., & Papp, L. A. (2000). Efficacy of venlafaxine in mixed depression-anxiety states. *Depression and Anxiety, 12,* 77-80.

Gortner, E. T., Gollan, J. K., Dobson, K. S., & Jacobson, N. S. (1998). Cognitive-behavioral treatment for depression: Relapse prevention. *Journal of Consulting and Clinical Psychology, 66,* 377-384.

Hamilton, M. (1960). A rating scale for depression. *Neurology, Neurosurgery and Psychiatry, 23,* 56-61.

Hammen, C. L., & Glass, D. R. (1975). Expression, activity, and evaluation of reinforcement. *Journal of Abnormal Psychology, 84,* 718-721.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and Commitment therapy: An Experiential Approach to Behavior Change.* New York: Guilford Press.

Herrnstein, R. J. (1970). On the law of effect. *Journal of the Experimental Analysis of Behavior, 13,* 243-266. Hollon, S. D. (2003). *Behavioral activation, cognitive therapy, and antidepressant medication in the treatment of major depression.* Symposium presented at the 37th Annual convention of the Association for the Advancement of Behavior Therapy, Boston, MA, 2003.

Hopko, D. R., Bell, J. L., Armento, M. E. A., Hunt, M. K., & Lejuez, C. W. (2005). Behavior therapy for depressed cancer patients in primary care. *Psychotherapy: Theory, Research, Practice, Training, 42,* 236-243.

Hopko, D. R., Bell, J. L., Armento, M. E. A., Wolf, N., Hunt, M. K., & Lejuez, C. W. (2005b). A Brief Behavioral Activation Treatment for Depression (BATD): Preliminary Outcomes with Depressed Cancer Patients. Symposium presented at the 39th Annual meeting for the Association for Behavior and Cognitive Therapies, Washington, D.C. [Behavioral Activation Therapy for Complex Psychiatric and Medical Conditions].

Hopko, D. R., Hopko, S. D., & Lejuez, C. W. (2004). Behavioral activation as an intervention for co-existent depressive and anxiety symptoms. *Clinical Case Studies, 3,* 37-48.

Hopko, D. R., Lejuez, C. W., LePage, J., Hopko, S. D., & McNeil, D. W. (2003). A brief behavioral activation treatment for depression: A randomized trial within an inpatient psychiatric hospital. *Behavior Modification, 27,* 458-469.

Hopko, D. R., Lejuez, C. W., Ruggiero, K. J., & Eifert, G. H. (2003). Contemporary behavioral activation treatments for depression: Procedures, principles, and progress. *Clinical Psychology Review, 23,* 699-717.

Hopko, D. R., Sanchez, L., Hopko, S. D., Dvir, S., & Lejuez, C. W. (2003). Behavioral activation and the prevention of suicide in patients with borderline personality disorder. *Journal of Personality Disorders, 17,* 460-478.

Jacobson, N. S., Dobson, K. S., Truax, P. A., Addis, M. E., Koerner, K., Gollan, J. K., Gortner, E., & Prince, S. E. (1996). A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology, 64,* 295-304.

Jacobson, N. S., & Gortner, E. T. (2000). Can depression be de-medicalized in the 21st century: Scientific revolutions, counter-revolutions and the magnetic field of normal science. *Behaviour Research and Therapy, 38,* 103-117.

Jakupak, M., Roberts, L., Martell, C., Mulick, P., Michael, S., Reed, R., Balsam, K. F., Yoshimoto, D., & McFall, M. (in press). A pilot study of behavioral activation for veterans with posttraumatic stress disorder. *Journal of Traumatic Stress.*

Kanfer, F. H., & Grimm, L. G. (1977). Behavioral analysis: Selecting target behaviors in the interview. *Behavior Modification, 1,* 7-28.

Katon, W., & Roy-Byrne, P. P. (1991). Mixed anxiety and depression. *Journal of Abnormal Psychology, 100,* 337-345.

Kendler, K. S. (1996). Major depression and generalized anxiety disorder: Same genes (partly), different environments – Revisited. *British Journal of Psychiatry, 168,* 68-75.

Kessler, R. C., Stang, P. E., Wittchen, H. U., Ustan, T. B., Roy-Byrne, P. P., & Walters, E. E. (1998). Lifetime panic-depression comorbidity in the National Comorbidity Survey. *Archives of General Psychiatry, 55,* 801-808.

Kessler, R. C., Nelson, C. B., McGonagle, K. A., Liu, J., Swartz, M., & Blazer, D. G. (1996). Comorbidity of DSM-III-R major depressive disorder in the general population: Results from the National Comorbidity Survey. *British Journal of Psychiatry, 168,* 17-30.

Lang, P. J. (1968). Fear reduction and fear behavior: Problems in treating a construct. In J. M. Schlien (Ed.), *Research in psychotherapy* (Vol. III). Washington, DC: APA.

Lejuez, C. W., Hopko, D. R., & Hopko, S. D. (2001). A brief behavioral activation treatment for depression: Treatment manual. *Behavior Modification, 25,* 255-286.

Lejuez, C. W., Hopko, D. R., & Hopko, S. D. (2002). *The Brief Behavioral Activation Treatment for Depression (BATD): A Comprehensive Patient Guide.* Boston, MA: Pearson Custom Publishing.

Lejuez, C. W., Hopko, D. R., LePage, J., Hopko, S. D., & McNeil, D. W. (2001). A brief behavioral activation treatment for depression. *Cognitive and Behavioral Practice, 8,* 164-175.

Lewinsohn, P. M. (1974). A behavioral approach to depression. In R. M. Friedman and M. M. Katz (Eds.). *The psychology of depression: Contemporary theory and research.* New York: Wiley.

Lewinsohn, P. M., Antonuccio, D. O., Breckenridge, J. S., & Teri, L. (1984). *The "Coping with Depression" Course*. Eugene, OR: Castalia.

Lewinsohn, P. M., & Atwood, G. E. (1969). Depression: A clinical-research approach. *Psychotherapy: Theory, Research, & Practice, 6,* 166-171.

Lewinsohn, P. M., & Graf, M. (1973). Pleasant events and depression. *Journal of Consulting and Clinical Psychology, 2,* 261-268.

Lewinsohn, P. M., & Libet, J. (1972). Pleasant events, activity schedules, and depressions. *Journal of Abnormal Psychology, 3,* 291-295.

Lewinsohn, P. M., Munoz, R. F., Youngren, M. A., & Zeiss, A. M. (1986). *Control Your Depression.* New York: Prentice Hall Press.

Lewinsohn, P. M., Sullivan, J. M., & Grosscup, S. J. (1980). Changing reinforcing events: An approach to the treatment of depression. *Psychotherapy: Theory, Research, and Practice, 47,* 322-334.

Malhi, G. S., Parker, G. B., & Greenwood, J. (2005). Structural and functional models of depression: From sub-types to substrates. *Acta Psychiatrica Scandinavica, 111,* 94-105.

Marks, I. M. (1969). *Fears and phobias.* Heineman, London.

Martell, C. R., Addis, M. E., & Jacobson, N. S. (2001). *Depression in Context: Strategies for Guided Action.* New York: W. W. Norton.

Martin, I., & Levey, A. B. (1985). Conditioning, evaluation, and cognitions: An axis of integration. *Behaviour Research and Therapy, 23,* 167-175.

McDowell, J. J (1982). The importance of Herrnstein's mathematical statement of the law of effect for behavior therapy. *American Psychologist, 37,* 771-779.

McLean, P. D., & Hakstian, A. R., (1979). Clinical depression: Comparative efficacy of outpatient treatments. *Journal of Consulting and Clinical Psychology, 47,* 818-836.

Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology, 49,* 377-412.

Mineka, S., & Zinbarg, R. (1996). Conditioning and etiological models of anxiety disorders: Stress in dynamic-context anxiety models. In D. A. Hope (Ed.), *Nebraska Symposium on Motivation: Vol. 43. Perspectives on Anxiety, panic, and fear* (pp.135-210). Lincoln: University of Nebraska Press.

Mowrer, O. H. (1939). Stimulus response theory of anxiety. *Psychological Review, 46,* 553-565.

Mulick, P. S., & Naugle, A. E. (2004). Behavioral Activation for comorbid PTSD and major depression: A case study. *Cognitive and Behavioral Practice, 11,* 378-387.

Nelson-Gray, R. O. (2003). Treatment utility of psychological assessment. *Psychological Assessment, 15,* 521-531.

Nelson-Gray, R. O., & Paulson, J. F. (2004). Behavioral Assessment and the DSM system. In S. N. Haynes & E. M. Heiby (Eds.), *Comprehensive handbook of psychological assessment (Vol.3)* (pp. 470-486). Hoboken, NJ: John Wiley.

Porter, J. F., Spates, C., & Smitham, S. S. (2004). Behavioral activation group therapy in public mental health settings: A pilot investigation. *Professional Psychology: Research & Practice, 35,* 297-301.

Rachman, S. (1977). The conditioning theory of fear-acquisition: A critical examination. *Behaviour Research and Therapy, 15,* 375-387.

Rapaport, M. H. (2001). Prevalence, recognition, and treatment of comorbid depression and anxiety. *Journal of Clinical Psychiatry, 62,* 6-10.

Rapee, R. M., Craske, M. G., Brown, T. A., & Barlow, D. H. (1996). Measurement of perceived control over anxiety-related events. *Behavior Therapy, 27,* 279-293.

Rehm, L. P. (1977). A self-control model of depression. *Behavior Therapy, 8,* 787-804.

Reiss, S., Peterson, R. A., Gursky, D. M., & McNally, R. J. (1986). Anxiety sensitivity, anxiety frequency, and the prediction of fearfulness. *Behaviour Research and Therapy, 24,* 1-8. Rivas-Vazquez, R. A., Saffa-Biller, D., Ruiz, I., Blais, M. A., & Rivas-Vazquez, A. (2004). Current isues in anxiety and depression: Comorbid, mixed, and subthreshold disorders. *Professional Psychology: Research and Practice, 35,* 74-83.

Schulberg, H. C., Block, M.R., Madonia, M.J., Scott, C.P., Rodriguez, E., Imber, S.D., Perel, J., Lave, J.R., Houck, P.R., & Coulehman, J.L. (1996). Treating major depression in primary care: eight-month clinical outcomes. *Archives of General Psychiatry, 53,* 913-919.

Scotti, J. R., Morris, C. B., McNeil, C. B., & Hawkins, R. P. (1996). DSM-IV and disorders of childhood and adolescence: Can structural criteria be functional? *Journal of Consulting and Clinical Psychology, 64,* 1177-1191.

Segrin, C., & Abramson, L. Y. (1994). Negative reactions to depressive behaviors: A communications theories analysis. *Journal of Abnormal Psychology, 103,* 655-668.

Seligman, M. E. P. (1975). *Helplessness: On depression, development, and death*. San Francisco: Freeman.

Staats, A. W., & Heiby, E. M. (1985). Paradigmatic behaviorism's theory of depression: Unified, explanatory, and heuristic. In S. Reiss and R. R. Bootzin (Eds.), *Theoretical Issues in Behavior Therapy* (pp. 279-330). New York: Academic Press.

Stanley, M. A., Beck, J. G., Novy, D. M., Averill, P. M., Swann, A. C., Diefenbach, G., & Hopko, D. R. (2003). Cognitive-behavioral treatment of late-life generalized anxiety disorder. *Journal of Consulting and Clinical Psychology, 71,* 309-319.

Tyrer, P. J. (1989). *Classification of neurosis.* Chichester, England: Wiley.

Ware, J. E., & Sherbourne, C. D. (1992). The MOS 36-Item Short-Form Health Survey (SF-36): I. Conceptual framework and item selection. *Medical Care, 30,* 473-483.

Wittchen, H. U., Zhao, S., Kessler, R. C., & Eaton, W. W. (1994). DSM-III-R generalized anxiety disorder in the National Comorbidity Survey. *Archives of General Psychiatry, 51,* 355-364.

Zeiss, A. M., Lewinsohn, P. M., & Munoz, R. F. (1979). Nonspecific improvement effects in depression using interpersonal cognitive, and pleasant events focused treatments. *Journal of Consulting and Clinical Psychology, 47,* 427-439.

Zinbarg, R. E., Barlow, D. H., Liebowitz, M., Street, L., Broadhead, E., Katon, W., Roy-Byrne, P., Levine, J. P., Teheiani, M., & Richards, J. (1994). The DSM-IV field trial for mixed anxiety-depression. *American Journal of Psychiatry, 151,* 1153-1162.

Address correspondence to:

Derek R. Hopko
The University of Tennessee - Knoxville
Department of Psychology, Room 301D
Austin Peay Building

Knoxville, Tennessee 37996-0900
Phone: (865) 974-3368
Fax: (865) 974-3330
E-Mail: dhopko@utk.edu

Author Contact information:

1. Sarah M. C. Robertson, M. A.
The University of Tennessee
Department of Psychology
307 Austin Peay Building
Knoxville, TN 37996-0900
Phone: (865) 974-3368
Email: srober37@utk.edu

2. Carl W. Lejuez, Ph.D.
Associate Professor and Director,
Center for Addictions, Personality, and Emotion Research (CAPER)
Department of Psychology
1123c Biology/Psychology Building
University of Maryland
College Park, MD 20742
Office: (301) 405-5932
Cell: (240) 535-0467
Fax: (301) 314-9566
Email: clejuez@psyc.umd.edu
