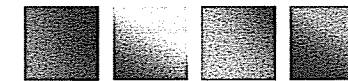


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CHAPTER 43



Emotion Disturbances as Transdiagnostic Processes in Psychopathology

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Emotion disturbances are ubiquitous in psychopathology. Even a cursory glance at the current version of the American diagnostic system—the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (DSM-IV-TR; American Psychiatric Association, 2000)—reveals that nearly all the diagnostic categories include symptoms that tap one type of emotion disturbance or another (see Table 43.1). These disturbances span both positive and negative emotions, and they include excesses of emotion (as in the case of specific and social phobias, with marked and persistent fear); deficits in emotion (as in the case of narcissistic personality disorder, with a lack of empathy); social emotional problems (as in autistic disorder, with a lack of emotional reciprocity); and regulation problems (as in borderline personality disorder, with difficulties in controlling anger). The pervasiveness of emotion disturbances in psychopathology suggests the potential for commonalities across disorders. Indeed, there may be emotional distur-

bances that are central to a number of different disorders; yet the manifestation of these disturbances may differ from disorder to disorder, thus helping to account for the different symptom constellations across disorders. In this chapter, I consider the utility of adopting a transdiagnostic approach to understanding emotion disturbances in psychopathology across several levels, including descriptive phenomenology, etiology, and treatment.

CONSTRAINING EMOTION AND AFFECT

Emotions have developed through the course of human evolutionary history to prepare organisms to act in response to a number of environmental stimuli and challenges. This account suggests that emotions, under most circumstances, serve a number of important intra- and interpersonal functions (e.g., Frijda, 1986; Keltner & Kring, 1998; Lang, Bradley, &

TABLE 43.1. Emotion-Based Symptoms in DSM-IV-TR Disorders

Disorders	Symptoms
<i>Schizophrenia and other psychotic disorders</i>	
Schizophrenia	
Schizoaffective disorder	
Schizophreniform disorder	Affective flattening, anhedonia
<i>Mood disorders</i>	
Major depressive episode	Depressed mood, anhedonia
Dysthymia	Depressed mood
Manic or hypomanic episode	Elevated, expansive, or irritable mood
<i>Anxiety disorders</i>	
Panic disorder	Intense fear or discomfort
Agoraphobia	Anxiety
Specific and social phobias	Marked and persistent fear, anxious anticipation
Obsessive-compulsive disorder	Marked anxiety or distress
Posttraumatic stress disorder	Irritability, anger, distress, anhedonia, restricted range of affect
Acute stress disorder	Anxiety or increased arousal
Generalized anxiety disorder	Excessive anxiety and worry, irritability
<i>Somatoform disorders</i>	
Hypochondriasis	Preoccupation with fears of having disease
<i>Eating disorders</i>	
Anorexia nervosa	Fear of gaining weight
<i>Sleep disorders</i>	
Sleep terror disorder	Intense fear and signs of autonomic arousal
Circadian rhythm sleep disorder, nightmare disorder	Clinically significant distress
<i>Impulse control disorders</i>	
Pathological gambling	Irritability, dysphoric mood
Trichotillomania	Tension; pleasure or relief after hair pulling
Intermittent explosive disorder	Rage, anger
Pyromania, kleptomania	Tension or excited mood
<i>Adjustment disorders</i>	
	Marked distress
<i>Personality disorders</i>	
Paranoid personality disorder	Quickness to react angrily
Schizoid personality disorder	Emotional coldness, detachment, flattened affect
Schizotypal personality disorder	Inappropriate or constricted affect, excessive social anxiety
Antisocial personality disorder	Lack of remorse, irritability
Borderline personality disorder	Affective instability due to marked reactivity of mood, inappropriate intense anger, or difficulty controlling anger
Histrionic personality disorder	Rapidly shifting and shallow expressions of emotion
Narcissistic personality disorder	Lack of empathy
Avoidant personality disorder	Fear of criticism, disapproval, or rejection
Dependent personality disorder	Fear of being unable to care for self, being left alone
<i>Substance-related disorders</i>	
Alcohol intoxication	Mood lability
Alcohol withdrawal	Anxiety
Amphetamine and cocaine intoxication	Euphoria or affective blunting; anxiety, tension, anger
Amphetamine and cocaine withdrawal	Dysphoric mood

(continued)

TABLE 43.1. (continued)

Disorders	Symptoms
<i>Substance-related disorders (cont.)</i>	
Caffeine intoxication	Nervousness, excitement
Cannabis intoxication	Euphoria, anxiety
<i>Dementias</i>	
Dementia due to Pick's disease	Emotional blunting
Dementia due to Huntington's disease	Depression, irritability, anxiety
<i>Childhood disorders</i>	
Autistic disorder, Asperger's disorder	Lack of emotional reciprocity; marked impairment in nonverbal behaviors, such as facial expression
Separation anxiety disorder	Distress, worry, fearfulness
Oppositional defiant disorder	Quickness to lose temper, get angry, be annoyed by others
Childhood disintegrative disorder	Impairment in nonverbal behaviors; lack of social or emotional reciprocity
<i>Other conditions</i>	
Bereavement	Guilt

Cuthbert, 1990; Levenson, 1994). As I have argued elsewhere, the functions of emotion in persons with psychopathology are comparable to those for persons without psychopathology (Keltner & Kring, 1998; Kring & Bachorowski, 1999). However, emotion disturbances in psychopathology interfere with the achievement of emotion-related functions. For example, the absence of facial expressions in a patient with schizophrenia may evoke negative responses from others (Krause, Steimer-Krause, & Hufnagel, 1992), thus negatively affecting his or her social relationships and interactions (e.g., Hooley, Richters, Weintraub, & Neale, 1987).

Emotions are typically considered to have multiple components, including expression, experience, and physiology. The extent to which these emotion components correspond with one another or cohere remains a topic of debate (e.g., Barrett, 2006a; Bradley & Lang, 2000; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Functionalist accounts of emotion suggest that coherence among components is adaptive (e.g., Levenson, 1994), but the empirical data supporting coherence are mixed (Barrett, 2006a). There are a number of reasons why particular emotion components may not cohere in any given study—including sample characteristics, emotion elicitation methods, emotion component measurements,

and data-analytic techniques, as well as whether even under ideal circumstances, emotion coherence is the exception rather than the norm. Nevertheless, the lack of coherence across multiple emotions, situations, and contexts has been observed in different psychological disorders (e.g., schizophrenia, psychopathy) and has been considered an emotional disturbance (e.g., Kring, 2001).

Although the terms "affect" and "emotion" are used interchangeably in the psychopathology literature, there are important conceptual and empirical distinctions between the terms. Generally speaking, "affect" is most often used in reference to feeling states, whereas "emotion" is used in reference to multiple components (only one of which is a feeling state). Barrett and colleagues have distinguished "core affect" from the more generic term "affect." Core affect reflects neurophysiological states that are an omnipresent indicator of a person's relationship to his or her environment at any given time (Barrett, 2006a; Barrett, Mesquita, Ochsner, & Gross, 2007; Russell, 2003; Barrett & Russell, 1999). Core affect is experienced as feelings of pleasure or displeasure, and to a lesser extent arousal or activation (Barrett, 2006a; Barrett et al., 2007). Although core affect is observed across cultures, there are nevertheless important individual and cultural differences (Barrett, 2006b; Mesquita &

Karasawa, 2002) that may be important for understanding disturbances in core affect in psychopathology.

THE TRANSDIAGNOSTIC APPROACH

In a recent and influential book, Harvey, Watkins, Mansell, and Shafran (2004) have reviewed the literature on cognitive and behavioral maintaining processes in psychopathology. Rather than organizing their book by disorder, they have instead adopted a transdiagnostic perspective, reviewing the evidence for common processes across different disorders. Their analysis points to an accumulating body of evidence of disruptions in a number of processes, such as attention, memory, reasoning, and avoidance, that are common across more than one adult disorder and that serve to maintain or exacerbate the symptoms of these disorders. Similar conclusions have been reached regarding common cognitive maintaining processes across the eating disorders (Fairburn, Cooper, & Shafran, 2003), and treatment approaches that target transdiagnostic processes across disorders have recently been developed for depression, anxiety, and eating disorders (e.g., Barlow, Allen, & Choate, 2004; Fairburn et al., 2003; Norton, Hayes, & Hope, 2004).

Harvey et al. (2004) have argued that there are a number of key advantages to adopting a transdiagnostic perspective in psychopathology. First, the transdiagnostic perspective may help to account for the high rates of comorbidity among the current DSM disorders. Indeed, comorbidity is the norm rather than the exception. In the National Comorbidity Survey (NCS), Kessler et al. (1994) found that nearly 80% of individuals with a lifetime diagnosis of one disorder had received at least one other lifetime diagnosis. In the NCS replication study, 45% of people who met criteria for one diagnosis in the prior 12 months met criteria for at least one more diagnosis (Kessler, Chiu, Demler, & Walters, 2005). Other studies have found similarly high rates of comorbidity. For example, nearly two-thirds of individuals with depression meet diagnostic criteria for an anxiety disorder (e.g., Mineka, Watson, & Clark, 1998), and as many as 50% of individuals who meet criteria for an anxiety disorder are depressed (e.g., Brown et al., 2001). The high level of comorbidity may well reflect problems

in the current diagnostic system (e.g., poor discriminant validity). However, the rampant comorbidity also suggests that there may be common symptoms or processes across disorders, including emotional processes.

Second, a transdiagnostic approach may be useful for developing and evaluating treatments. For example, recent theory and empirical data point to the notion that currently available treatments for anxiety and depression are changing common aspects of these disorders, rather than disorder-specific aspects (e.g., Barlow et al., 2004; Hayes, Strosahl, & Wilson, 1999; Persons, Roberts, & Zalecki, 2003). In addition, evidence from treatment outcome studies suggests that interventions for one disorder (e.g., depression) are also effective in treating other disorders (e.g., generalized anxiety disorder) (e.g., Brown & Barlow, 1992; Tsao, Mystkowski, Zucker, & Craske, 2002). A transdiagnostic perspective may illuminate the common mechanisms or processes across disorders, which may then be more directly targeted in treatment.

A transdiagnostic approach to emotion disturbances in psychopathology has been suggested, sometimes implicitly, by other theorists, researchers, and clinicians (e.g., Barlow et al., 2004; Patrick & Bernat, 2006; Thayer & Brosschot, 2005). However, the reach of such an approach has been fairly limited thus far, for at least two reasons. First, some disorders lend themselves more clearly to such an approach than others, given their comorbidities, comparable treatments, and treatment responses. For example, mood and anxiety disorders—two broad categories that are collectively referred to as the “emotional disorders” (e.g., Barlow, 2004; Watson, 2005)—have been discussed in transdiagnostic terms across many levels. Other disorders that may well benefit from a transdiagnostic approach (e.g., personality disorders) have not yet stimulated much transdiagnostic theory and research. Many of the examples of the transdiagnostic approach to emotion disturbances in psychopathology illustrated throughout the chapter will involve the mood and anxiety disorders.

A second reason why the transdiagnostic approach to emotion disturbances has been fairly limited is probably the fact that the prevailing paradigm in psychopathology research over the past several decades has been “disorder-centric.” That is, most investigations are designed to answer questions about the symp-

toms, causes, and treatments of individual disorders. In the realm of emotion and psychopathology, studies are typically designed to study a particular emotion disturbance in a particular disorder. For example, the literature is packed with studies of particular emotion disturbances (e.g., facial emotion perception deficits) in putatively distinct disorders (e.g., depression, borderline personality disorder, schizophrenia), with little consideration of the possibility that the emotion disturbance may cut across these disorders. Such disorder-centric research is perpetuated by the field's relatively greater emphasis on internal validity (e.g., tightly controlled study of one disorder) over external validity (e.g., naturalistic study of comorbid disorders), as reflected in editorial practices at top-flight journals and funding priorities at granting agencies. As such, the entry point for a majority of psychopathology studies is typically a single disorder rather than multiple disorders, with relatively less emphasis on mechanisms or processes (such as emotion disturbances) that may cut across traditional diagnostic boundaries. Unfortunately, this disorder-centric focus overlooks the fact that most disorders do not occur in “pure” form, and thus conclusions regarding specificity of emotion disturbances in X disorder may not be particularly informative with respect to understanding X disorder as it more commonly occurs in combination with Y disorder.

Despite these challenges, the promise of a transdiagnostic approach to emotion disturbances in psychopathology is evident at many levels. First, a transdiagnostic approach can inform the ways in which different disorders are classified. In other words, examining emotion-related commonalities at the symptom level may help to account for the high levels of comorbidity in the current diagnostic system, and in turn may provide guidance on refining diagnostic categories. Second, a transdiagnostic approach can be informative with respect to identifying common emotion-related causal and/or maintaining processes across disorders. Third, a transdiagnostic approach can be useful for treatment development, with an emphasis on changing emotion processes. In the remaining sections of this chapter, I consider the relative merits of adopting a transdiagnostic approach to emotion disturbances in psychopathology across these three levels: descriptive phenomenology, etiology, and treatment.

DESCRIPTIVE PSYCHOPATHOLOGY AND DIAGNOSIS

In an effort to spur the field toward greater attention to emotion and psychopathology, Berenbaum, Raghavan, Le, Vernon, and Gomez (2003) have proposed a separate taxonomy of emotional disturbances as a companion to the current diagnostic system. Their taxonomy parses specific emotion (e.g., shame, guilt, happiness, fear) disturbances into three broad areas of disruption: “valence,” “intensity/regulation,” and “disconnections.” Each of the broad categories is further subdivided to achieve greater specificity of particular emotion disturbances. Emotional valence disturbances can involve pleasant or unpleasant emotions, as well as too much or too little of these emotions. For example, the limited experience of pleasure (i.e., anhedonia) that characterizes depression and schizophrenia, as well as the excess of fear in panic disorder, would constitute valence disturbances. Emotional intensity/regulation disturbances are defined as over- or underregulation of both pleasant and unpleasant emotions. For example, mania, which is characterized by excesses in both pleasant (joy, euphoria) and unpleasant (irritability) emotions, would be construed as an emotional intensity/regulation disturbance. Disconnection disturbances reflect disconnections between the expressive component of emotion and other components, as in schizophrenia, where patients experience strong feelings yet do not express them outwardly (e.g., Berenbaum & Oltmanns, 1992; Kring & Neale, 1996; Kring & Earnst, 1999). Disconnection disturbances also reflect a lack of conscious awareness of one's own emotional responses. Berenbaum et al. (2003) conclude their paper with nine recommendations for future research, but these do not explicitly include the possibility of examining these disturbances across disorders. Instead, the recommendations are geared toward amplifying our understanding of specific disorders; examining emotion disturbances independently from diagnosed disorders; modifying treatments for specific disorders by targeting emotion disturbances; and examining similarities and differences in the disturbances across gender, culture, and the lifespan. Although these are certainly important goals for future research, the transdiagnostic implications of this taxonomy are also ripe for further investigation.

In a commentary on Berenbaum et al.'s article, Watson (2003) has argued that including the taxonomy as a companion to the current diagnostic system would add unnecessary complexity and serve to further reify already problematic diagnostic categories. Although he does not suggest a transdiagnostic approach, Watson has nonetheless argued for "scrapping these heterogeneous diagnostic categories and focusing instead on homogeneous symptom clusters" (p. 237). In this view, focusing on the current DSM symptoms, many of which reflect emotion disturbances, may be a more fruitful way of classifying psychopathology.

More recently, Watson (2005) has expanded upon this idea, arguing that the current configuration of mood disorders and anxiety disorders ought to be replaced with a different configuration. Specifically, he proposes a quantitative hierarchical model to account for the high levels of comorbidity between mood and anxiety disorders, drawing upon structural analyses of the diagnostic symptoms at both the phenotypic and genotypic levels. His model includes an overarching domain referred to as "emotional disorders." This would consist of three subdomains, including what Watson calls "distress disorders" (generalized anxiety disorder, posttraumatic stress disorder, major depressive disorder, dysthymia), "bipolar disorders" (bipolar I and II disorders, cyclothymia), and "fear disorders" (specific and social phobias, panic disorder, agoraphobia). Watson's proposal is more than a simple reshuffling of diagnostic categories, and this approach is certainly a more rational and empirically supported approach to situating the mood and anxiety disorders in the diagnostic system than is the current, purely phenomenological approach. The model also points to the promise of a transdiagnostic approach to the descriptive phenomenology of mental disorders.

Indeed, part of the impetus for Watson's (2005) proposed model has come from the influential theory and research on common and distinct emotion disturbances in the mood and anxiety disorders. Clark and Watson (1991) originally proposed the tripartite model to account for the relationship between anxiety and depression (see also Watson, Clark, et al., 1995; Watson, Weber, et al., 1995; Watson, Weise, Vaidya, & Tellegen, 1999). In this model, a general distress factor characterized by high levels of negative activation (NA) is common to both anxiety and depression; a sec-

ond factor, characterized by low levels of positive activation (PA) or pleasurable engagement with the environment, is specific to depression; and a third factor, variously referred to as "anxious arousal" (AA) or "somatic arousal," is specific to anxiety. A revision to the model was later proposed, to account better for the heterogeneity among the anxiety disorders (Mineka et al., 1998). The revised model, termed the integrative hierarchical model, followed from additional data suggesting that high levels of AA are more characteristic of panic disorder in particular than of the anxiety disorders in general (Brown, Chorpita, & Barlow, 1998; Zinbarg & Barlow, 1996).

Watson (2005) has argued that data generated from the integrative hierarchical model thus far are not sufficient to account fully for the comorbidity among the mood and anxiety disorders. Thus he has proposed the new quantitative model to account better for the extensive comorbidities, while at the same time retaining the current diagnostic categories (i.e., a disorder-centric approach). An alternative to Watson's model is the dissection of mood and anxiety disorders into categories based on emotion-related difficulties. Thus, instead of separate categories for the different mood and anxiety disorders, there might be an overarching category of general distress disorders with subdomains of low-PA disorders and high-AA disorders. Brown et al. (1998) proposed such a structural model, showing that many of the anxiety disorders and depression were better accounted for by the emotion-based factors of NA, PA, and AA, though they did not explicitly call for the scrapping of the specific mood and anxiety disorder categories. More recently, Barlow and colleagues have pushed this idea further, noting that "DSM-IV emotional disorder categories do not qualify in any sense as real entities . . . but do seem to be useful concepts or constructs that emerge as 'blips' on a general background of NAS [negative affect syndrome]" (Barlow et al., 2004, p. 212), and suggesting that future revisions of the DSM may do well to eliminate many of the current categories of disorders (Moses & Barlow, 2006). This is a fairly radical proposal, but perhaps one whose time has (nearly) come.

It is true that the field's disorder-centric approach, which has been central at least since the development of DSM-III (Wilson, 1993), has proven to be advantageous in the diagnosis, assessment, and treatment of various disor-

ders. Furthermore, reconfiguring the current diagnostic categories based only on emotion-related disturbances might leave out much clinically relevant information. However, a transdiagnostic approach would not necessarily have to supplant the current diagnostic categories for it to inform our understanding of the symptoms of various disorders. Indeed, mental disorders do not just consist of emotion-related symptoms. Different symptoms reflect other processes, including cognitive (e.g., inattention, thought disorder), behavioral (e.g., avoidance), and interpersonal (e.g., no close friends or confidants) processes. However, understanding the emotion-related symptoms that cut across disorders may help to refine the current diagnostic categories "without necessarily reconfiguring them, as Watson (2005) has suggested. For example, knowing that individuals with depression and panic disorder share the heightened experience of NA, even though panic is also characterized by AA and depression is characterized by low PA, provides key information about similarities and differences between these two disorders that is not readily acknowledged in the current diagnostic system.

CAUSAL AND MAINTAINING PROCESSES

There have been several recent reviews of emotion disturbances in psychopathology (e.g., Kring & Werner, 2004; Rottenberg & Johnson, 2007). Instead of duplicating these efforts, I focus here on candidate transdiagnostic emotion disturbances that may reflect causal or maintaining processes. There are many points along the temporal course of disorders at which a transdiagnostic perspective may be informative (Barnett & Gotlib, 1988; Harvey et al., 2004). Specifically, transdiagnostic emotion disturbances may be antecedents (i.e., predisposing or vulnerability factors), concomitants, or consequences (i.e., perpetuating or maintaining factors). For an emotion disturbance to be considered an antecedent transdiagnostic process, it must be shown to precede the onset of a disorder. Disturbances that are observed during active episodes of disorders may be more accurately construed as concomitant transdiagnostic processes, and disturbances that persist after active episodes have abated might be considered consequences or maintaining processes. Much of the evidence to date regarding specific

emotion disturbances in particular disorders is most readily interpreted as evidence for maintaining processes. There has been less theoretical, conceptual, and empirical work on possible transdiagnostic causal or maintaining emotion processes than in the domains of phenomenology and treatment. Indeed, the disorder-centric focus of research has been particularly dominant in studies of possible etiological emotion disturbances, making integration across disorders more of a challenge. Although space constraints preclude me from considering all possible transdiagnostic emotion disturbances, I briefly consider four promising candidates here: core affect, emotion awareness, emotion regulation, and emotion disconnections.

Core Affect

As discussed earlier, Barrett and colleagues define "core affect" as neurophysiological states that are experienced as pleasant or unpleasant and are ever-present indicators of a person's relationship to his or her environment (Barrett, 2006a; Barrett et al., 2007; Russell, 2003; Barrett & Russell, 1999). Findings from several studies that have measured reports of feeling states have indicated that the experience of excessive unpleasant affect (although it is not necessarily conceived of as "core affect") is common across many different disorders, including depression (for a review, see Mineka et al., 1998), the anxiety disorders (Mineka et al., 1998), eating disorders (e.g., Stice, 2001), schizophrenia (for a review, see Kring, 2001), substance-related disorders (e.g., Kassel, Stroud, & Paronis, 2003), and a number of personality disorders (e.g., Berenbaum et al., 2006; Huperich, 2005; Putnam & Silk, 2005). Integrating findings across behavioral and brain imaging studies, Barrett et al. (2007) have suggested that disturbances in core affect may reflect an important emotion-related transdiagnostic process in psychopathology. Indeed, the conceptual, theoretical, and empirical advances regarding core affect among healthy individuals are ripe for translation into the realm of psychopathology.

Emotion Awareness

Barrett and Gross (2001) have argued that knowledge and awareness of one's emotions are necessary prerequisites to effective emotion

regulation. However, simply having knowledge about emotion is not sufficient; rather, greater accessibility of that emotion knowledge is believed to promote effective emotion regulation. Individuals who describe their feelings in a more differentiated manner (e.g., "sad," "confused," "elated") rather than more globally (e.g., "good," "bad") have greater accessibility to and awareness of emotion knowledge and use this knowledge when the regulation of emotion may be necessary (Barrett, Gross, Christensen, & Benvenuto, 2001).

What is the evidence for emotion awareness difficulties in psychopathology? Although being aware of how one feels is at the heart of most types of psychotherapy, surprisingly little research has explicitly examined emotion awareness in psychopathology and how it may wax or wane with the exacerbation and remission of symptoms. There is some evidence suggesting that patients with schizophrenia do not differ from individuals without schizophrenia in terms of their emotion knowledge. However, patients with schizophrenia differentiate less among emotional states, and thus may be less effective at emotion regulation (Kring, Barrett, & Gard, 2003). Clinical conceptualizations of borderline personality disorder include the notion that patients have difficulty distinguishing among different emotional states (e.g., Westen, 1991), but the empirical confirmation of this notion is needed. A recent investigation of schizotypal personality disorder symptoms found that such symptoms were associated with poor emotion clarity, but more attention to emotions (Berenbaum et al., 2006).

Related to emotion awareness, the construct of "alexithymia" refers to difficulties in verbalizing feelings. The most widely used measure of this construct, the Toronto Alexithymia Scale (Bagby, Parker, & Taylor, 1994), includes two subscales that appear important to emotion awareness: difficulty in identifying feelings and difficulty in describing feelings. This measure has been widely used in correlational studies of psychiatric symptoms (e.g., Grabe, Spitzer, & Freyberger, 2004; Parker, Bagby, & Taylor, 1991), anxiety disorders (e.g., Frewin, Pain, Dozois, & Lanius, 2006; Parker, Taylor, Bagby, & Acklin, 1993; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005), eating disorders (e.g., Cochrane et al., 1993), personality disorders (e.g., Berenbaum, 1996), substance-related disorders (e.g., Speranza et al., 2004), and insomnia (e.g., Lundh & Broman, 2006).

However, the linkages between this construct and the various disorders are not always replicated across studies. Additional work is thus needed to clarify alexithymia as a transdiagnostic process, beyond correlations between the one measure and symptoms within disorders.

Emotion Regulation

Emotion regulation problems have been at the forefront of discussions about emotion disturbances in psychopathology for at least the last 10 years (e.g., Barlow et al., 2004; Gross & Muñoz, 1995; Kring & Werner, 2004; Linehan, 1993). Broadly, emotion regulation refers to processes that serve to modify what we feel, when we feel it, and how we use that feeling to guide behavior (e.g., Gross, 1998). Many current diagnostic criteria explicitly refer to emotion regulation difficulties. For example, the criteria "difficulty controlling anger" in borderline personality disorder; "efforts to avoid feelings" in posttraumatic stress disorder (PTSD); "difficulty controlling worry" in generalized anxiety disorder; and "rapidly shifting expressions of emotion" in histrionic personality disorder all point to difficulties in regulating emotions.

Despite the perceived importance of emotion regulation deficits in psychopathology, it is difficult to integrate the literatures across disorders, because of the myriad approaches to constraining the concept of emotion regulation across studies. For example, some researchers do not distinguish emotional responding from regulation, following from theory that suggests the processes are indistinguishable (e.g., Campos, Frankel, & Camras, 2004; Davidson, 2000). Other studies examine emotion regulation within the individual, whereas still other studies examine emotion regulation from the outside (e.g., having others provide soothing to down-regulate negative emotion). Disentangling emotion from emotion regulation remains a critical challenge for the field (Rottenberg & Gross, 2003). Greater conceptual clarity will advance our understanding of how emotion regulation difficulties may be linked across different disorders.

A close cousin to emotion regulation is the time course, or chronometry, of emotional responses (e.g., Davidson, 1998). Emotional responses are not wholly temporally constrained by the presence of an eliciting stimulus, but in-

stead vary in their peak and duration in ways that may hold important information about the nature of emotion disturbances in psychopathology. Two elements of the time course of an emotional response that have been studied in healthy populations are (1) the time from the onset to the peak intensity of the response, and (2) the recovery time, or the time it takes for the emotional response to resolve. This latter process, recovery time, is a probable transdiagnostic emotion disturbance that remains a topic for future research. For example, the prolonged experience of NA associated with depression, generalized anxiety disorder, and eating disorders, or the prolonged experience of PA associated with bipolar disorder, may reflect a difficulty in the recovery time of emotional responding (e.g., Tomarken & Keener, 1998).

Emotion Disconnections

A good deal of evidence indicates that patients with schizophrenia report experiencing strong emotions in response to a variety of emotionally evocative stimuli (films, pictures, social interactions); yet they do not often display these feelings outwardly (for reviews, see Kring, 2001; Kring & Werner, 2004). In other words, these patients' outward displays of emotion are not often accurate reflections of their experienced emotion, indicating a disconnection between emotion response components. There is some evidence to suggest that this disconnection may be present prior to the onset of the illness. Walker, Grimes, Davis, and Smith (1993) analyzed home movies of adults with schizophrenia that were made before the adults developed schizophrenia. They found that girls displayed fewer joy expressions, and that both boys and girls displayed more negative facial expressions, compared to their healthy siblings.

Studies using the emotion-modulated startle paradigm (Lang et al., 1990) have observed a different disconnection among individuals with psychopathy. Compared to healthy controls, these individuals showed comparable startle inhibition during exposure to pleasant stimuli, but they did not show startle potentiation during exposure to aversive stimuli (e.g., Patrick, 1994; Patrick, Bradley, & Lang, 1993). However, the individuals with psychopathy did not differ from controls in their reported emotional experience to the aversive stimuli. Additional evidence for this disconnection has been found

in imagery studies (Patrick, Cuthbert, & Lang, 1994) and incidental memory paradigms (Christianson et al., 1996).

In both of the examples above, patients' reports of emotional experience were indistinguishable from healthy controls, but their behavioral or psychophysiological responses differed. It may well be that such emotion disturbances are better construed as reflecting a relatively intact core affect system with corresponding behavioral system disturbances.

TREATMENT DEVELOPMENT AND EVALUATION

Although medication is a common form of treatment for many different disorders, very few investigations have explicitly adopted a transdiagnostic approach to evaluating pharmacological treatment. Nevertheless, the evidence that particular medications may be effective for multiple disorders is hiding in plain sight. For example, studies have found that antidepressant medications are effective at reducing the symptoms of several other disorders, including specific and social phobias (e.g., Stein et al., 1998; Van Ameringen et al., 2001), panic disorder (White & Barlow, 2002), generalized anxiety disorder (Lydiard & Monnier, 2004), obsessive-compulsive disorder (Steketee & Barlow, 2002), posttraumatic stress disorder (Brady et al., 2000), some of the personality disorders (e.g., Rinne, van den Brink, Wouters, & van Dyck, 2002), and eating disorders (e.g., Walsh et al., 2000). Following from such evidence, medications that were originally approved by the U.S. Food and Drug Administration (FDA) for the treatment of depression have since received approval (or an "indication," in FDA terminology) for the treatment of other disorders. For example, paroxetine (Paxil) was later approved for the treatment of obsessive-compulsive disorder, panic disorder, generalized anxiety disorder, and social anxiety; fluoxetine (Prozac) was later approved for the treatment of obsessive-compulsive disorder and bulimia nervosa; sertraline (Zoloft) was later approved for the treatment of obsessive-compulsive disorder, panic disorder, social anxiety, and PTSD.

Although it may be the case that antidepressant medications are rather blunt instruments for targeting the general distress that is common across disorders, little research has di-

rectly examined the emotion-related mechanisms by which the medications might exert their transdiagnostic effects. However, we know that the selective serotonin reuptake inhibitors work on the neurotransmitter serotonin (as well as others, including dopamine; e.g., Svenningsson et al., 2002), functionally leaving more serotonin in the synapse, and that disruptions in serotonin have been implicated in depression (e.g., Thase, Jindal, & Howland, 2002), anxiety disorders (e.g., Stein, 1998), and eating disorders (e.g., Carrasco, Dyaz-Marsa, Hollander, Cesar, & Saiz-Ruiz, 2000; Kaye et al., 1998). We also know a good deal about how serotonin works throughout the brain, and perhaps not surprisingly, this neurotransmitter is heavily concentrated in areas of the brain linked with emotion (e.g., Barrett et al., 2007; Wrase et al., 2006). Finally, research has indicated that serotonin levels are associated with PA among healthy individuals (e.g., Duffy et al., 2006; Zald & Depue, 2001). The building blocks are thus available for constructing a transdiagnostic approach to medication treatment that explicitly links pharmacology, neuroscience, and emotion. Much of this integrative work remains to be done, but it is certainly a fruitful avenue for future research.

Historically, different forms of psychotherapy were conceived of as treatments that could be applied across disorders or clinical problems (e.g., psychoanalysis). Furthermore, despite the distinctly different theoretical traditions underpinning various types of psychotherapy (e.g., psychodynamic, interpersonal, gestalt, client-centered, behavioral), each of these traditions has included some consideration of emotion (for reviews, see Greenberg, 2002b; Greenberg & Safran, 1987). One form of psychotherapy that is relevant to the focus of this chapter is emotion-focused therapy (EFT), which was developed by Leslie Greenberg (e.g., Greenberg, 2002a). Boiled down to its essence, EFT is based on the idea that some emotions are adaptive, whereas others are maladaptive. Maladaptive emotions are based on an underlying loneliness, abandonment, worthlessness, anger, or inadequacy, and they can interfere with a person's relationships and overall functioning. The primary therapeutic goal is for a client to become more aware of these maladaptive emotions, to understand the source of these feelings, and to learn emotion regulation skills. According to Greenberg (2002a), EFT is better

suiited to particular types of clinical conditions (including depression and generalized anxiety disorder) and less well suited to others (such as panic disorder). Unfortunately, data regarding the efficacy of this treatment are limited. No randomized controlled clinical trials have been conducted, although smaller studies examining the process of change within EFT indicate that the treatment is effective for some clinical problems (Greenberg, 2002a). The theoretical foundations of this treatment continue to be enhanced by research in emotion, emotion and psychopathology, and affective neuroscience (e.g., Greenberg, 2002b, 2004, and Chapter 6 of this volume); a worthwhile endeavor for future research would be to examine whether this treatment is effective in targeting transdiagnostic emotion-related disturbances.

The shift to more disorder-specific psychotherapeutic approaches perhaps began in the late 1950s, with the pioneering work of Joseph Wolpe (1958), who developed systematic desensitization for the treatment of specific phobias. Additional disorder-specific psychotherapies were developed in the 1960s, as cognitive-behavioral therapies became more prominent. A number of other influences in the field since the 1970s have converged to solidify a disorder-specific approach to treatment development. These have included the greater demand to show that psychotherapy is effective; the development of the DSM-III, which was heavily influenced by the medical model (Wilson, 1993); the sophistication of research methods to evaluate treatment outcomes (e.g., Barlow, 2004; Westen, Novotny, & Thompson-Brenner, 2004); the emphasis on evidence-based practice (e.g., Barlow, 1996; Chambless & Hollon, 1998; Kendall, 1998); the efforts to position psychology as a health care profession and thus in the larger health care context (e.g., Johnson, 2001); and the observation that psychological treatments tailored to specific disorders are as effective as, or more effective than, other types of interventions. Indeed, as Barlow (2004) has noted, "few would argue that diversity in procedures to address specific aspects of pathology is not necessary" (p. 873).

The disorder-specific approach to treatment development, particularly in the context of empirically supported treatments (Chambless & Hollon, 1998), has spawned a large number of individual treatment protocols and manuals. This proliferation of different treatment proto-

cols has undoubtedly benefited countless individuals who have received these treatments, as they have been shown to be effective (Nathan & Gorman, 2002). However, the sheer magnitude of treatment protocols has become a bit overwhelming to treatment professionals, with respect to both learning the varied protocols and disseminating them to a broader range of treatment providers (Barlow et al., 2004; Persons, 2005). Furthermore, the reality in clinical practice is that providers often select bits and pieces from a number of different protocols, in order to provide the best possible treatment for a given individual patient (Persons, 2005). Partly in reaction to this overwhelming number of treatment protocols, there has recently been a call for more unified treatments across disorders. This conversation has been situated primarily within the mood and anxiety disorders, and it has been informed by research on emotion-related disturbances that are common across these disorders.

Following from research on the structural configuration of descriptive phenomenology and shared etiologies across mood and anxiety disorders, Barlow and colleagues have proposed a unified treatment for these disorders (Barlow et al., 2004; Moses & Barlow, 2006). The focus of this intervention is on putative emotion-related mechanisms that may be driving the emotion disturbances that cut across mood and anxiety disorders. The treatment has three main components: (1) altering cognitive reappraisals, a key component in emotion regulation processes; (2) preventing emotional avoidance; and (3) changing emotion action tendencies, or replacing emotion behaviors associated with fear and anxiety with behaviors related to positive emotions. These key components of the intervention have their origin in basic science in emotion, in both healthy and disordered individuals. Time will tell whether the intervention is as effective as other available treatments. Because the intervention is designed to target emotion-related mechanisms that cut across disorders, it is transdiagnostic at heart, and it seems likely that it will pay off. Indeed, there has been a call for more treatments to target mechanisms rather than disorders per se. For example, Rosen and Davison (2003) have argued that we should be defining empirically supported principles of change rather than empirically supported treatments.

Other recent treatments have been designed to target emotional disturbances, such as

Mennin and colleagues' emotion regulation treatment for generalized anxiety disorder (e.g., Mennin, 2004). Though this intervention was designed around the emotion regulation problems associated with generalized anxiety disorder, it seems probable that it would be useful for a number of disorders with similar difficulties in emotion regulation, such as an inability to down-regulate intense negative emotions and a lack of awareness of negative emotions.

SUMMARY AND CONCLUSIONS

Advances in affective neuroscience and basic behavioral research in emotion have greatly contributed to our understanding of emotion disturbances in psychopathology. Indeed, methods, theories, and measures developed in these domains have allowed us to achieve greater clarity regarding the reach of emotion disturbances across many different disorders. With this clarity has come the realization that many of the observed emotion disturbances may be common across disorders. Progress in understanding the reach of transdiagnostic emotion disturbances has begun to be achieved at the levels of descriptive phenomenology and treatment. Although transdiagnostic treatment approaches targeting emotion disturbances are grounded in theory regarding emotion-based mechanisms, more work is needed to unpack the nature of transdiagnostic etiological processes that are emotion-based.

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