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Emotion and Psychopathology

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One way we can learn about human behavior is to study what happens when things go wrong. Certainly a number of things "go wrong" in psychopathological disorders, and perhaps one of the more notable signs of things gone awry is some kind of emotional disturbance. One analysis has noted that as many as 85% of psychological disorders include disturbances in emotional processing of some kind (Thoits, 1985), whether they be "excesses" in emotion, "deficits" in emotion, or the lack of coherence among emotional components. Despite the widespread involvement of emotion dysfunction in psychopathology, systematic research into the nature, causes, and consequences of these disturbances has only recently been conducted. In this chapter, I briefly review the literature on emotion disturbance in three forms of psychopathology, with a particular emphasis on positioning these disturbances within the temporal course of the disorders. After first defining what I mean by emotion, emotion function, and emotion dysfunction, I then discuss the kinds of evidence that are needed to distinguish emotional features in psychopathology as either antecedents, concomitants, or consequences. I next review the available evidence on emotion disturbances in three different disorders: schizophrenia, unipolar depression, and social phobia. Finally, I consider the potential for basic emotion research paradigms to inform the study of the causes and manifestations of emotion disturbances in psychopathology, and conclude with suggestions for a research agenda in this area.

DEFINING AND MEASURING EMOTION

Emotion has been defined a number of different ways, and debate continues as to how to best define this somewhat elusive construct. Many emotion researchers agree, however, that emotions comprise a number of components, including (but not limited to) expressive, experiential, and physiological, which are typically coordinated within the individual. Moreover, the coordination of these components, under most circumstances, serves a number of important intra- and interpersonal functions (see, e.g., Ekman, 1994; Frijda, 1986; Keltner & Kring, 1998; Lang, Bradley, & Cuthbert, 1990; Levenson, 1992). More broadly, 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.

It is worth noting distinctions among various terms used in the emotion literature. Although the terms "affect" and "emotion" have been used interchangeably, a number of theorists and researchers have distinguished between the terms, both conceptually and empirically. Generally speaking, the term *affect* is most often used in reference to feeling states, whereas *emotions* comprise multiple components (only one of which is a feeling state) and are hypothesized to occur in response to some object, person, or situation, whether real or imagined (see, e.g., Feldman Barrett & Russell, 1999). Rosenberg (1998) considers affect a broad, organizing term under which emotions and moods can be subsumed. In addition, affect is hypothesized to be *trait-like*, whereas emotions and moods are (affective) *state-like*, given their transient nature. Adopting a somewhat different perspective, Russell and Feldman Barrett (1999; see also Feldman Barrett & Russell, 1999) argue that affect, or—as they put it—*core affect*, reflects feeling states that are ever present and are just one of many constituents of what they refer to as *prototypical emotion episodes*. These prototypical episodes are hypothesized to occur in response to something and comprise cognitive, behavioral, feeling, and physiological components. In their analysis, moods are core affects that endure for a longer period of time. Unfortunately, it is often difficult to make clear distinctions between affect, emotion, and mood based on the names of the measures employed in studies (Feldman Barrett & Russell, 1999). For example, although the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) assesses experienced *affect*, presumably so does the Differential Emotions Scale (DES; Izard, 1972). Using the framework of Russell and Feldman Barrett (1999), much of the research on emotion disturbance in psychopathology has been concerned with the various components of prototypical emotional episodes, one of which is core affect, although some research has been concerned with just one component, namely, the experience of feeling states or core affects. To ease interpretation, I will refer to feeling states in

response to some stimulus (e.g., film, person, situation) as experienced emotion. Feeling states that are assessed without explicit reference to an object will be referred to as affects. Of course, some measures used in various studies reviewed may not necessarily follow this rubric and thus will preclude this distinction. In those cases, however, I will note the component of emotion each measure refers to if it is not already obvious.

As my colleagues and I have argued elsewhere (Keltner & Kring, 1998; Kring & Bachorowski, 1999), the functions of emotion in persons with various psychopathological disorders are comparable to those for non-disordered individuals. In many different disorders, however, one or more components of emotional processing are impaired in some respect, thus interfering with the achievement of emotion-related functions. For example, nonverbal displays of fear and anxiety among individuals with social phobia may evoke complementary fear and anxiety in others (Dimberg & Ohman, 1996) or discourage others from interacting with them. Similarly, schizophrenia patients' absence of facial expressions may evoke negative responses from others (Krause, Steimer-Krause, & Hufnagel, 1992) and have a number of other consequences for social relationships and interactions (see, e.g., Hooley, Richters, Weintraub, & Neale, 1987).

Adopting a multicomponential and functional definition of emotion has several implications for the study of emotion and psychopathology. First, researchers interested in specifying the nature of emotion disturbances in psychopathology ought to measure more than one component of emotion. Unfortunately, much of psychopathology research has relied upon the use of clinical rating scales that typically assess only one component of emotion. For example, a commonly used rating scale in schizophrenia called the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1983) contains a subscale to measure the symptom called flat affect. Flat affect refers to a lack of outward expression of emotion (facially, vocally, and gesturally). To make these ratings, trained clinicians interview a patient and then rate the extent to which the patient was expressive during the interview. Although informative with respect to understanding emotional expression, using just this scale does not provide information on the experiential or physiological components of emotion. As will be discussed below, a complete understanding of emotion disturbance in schizophrenia requires assessment of the other emotion components. Second, a number of laboratory-based paradigms have been developed in the basic emotion literature that explicitly include methods for measuring and analyzing multiple components of emotion. Recently, psychopathology researchers have taken advantage of the methodological advancements made by basic emotion researchers and have incorporated these measures and paradigms into the study of different psychopathological disorders. For example, reliable self-report measures of the broad dimensions of affect (i.e., feeling states) have

been developed (see, e.g., PANAS; Watson, Clark, & Tellegen, 1988) in nonpatient populations and have been successfully applied to the study of depression (see, e.g., Watson, Clark, & Carey, 1988) and social anxiety (see, e.g., Wallace & Alden, 1997). Third, understanding both the intra- and interpersonal functions of emotion informs the study of emotion disturbance in psychopathology much the same way, for example, that understanding brain function informs the study of the behavioral sequelae of brain damage.

ANTECEDENTS, CONCOMITANTS, AND CONSEQUENCES

In their seminal review of cognitive theories of depression, Barnett and Gotlib (1988) distinguished between the concepts of antecedents, concomitants, and consequences. Briefly, for a variable to be considered an antecedent of a psychological disorder, it must be shown to precede the onset of the disorder. Those features that are observed during an episode of a psychological disorder may be more accurately construed as concomitants, and those features that persist after the episode has abated might be considered consequences. Building upon Zubin and Spring's (1977) diathesis-stress model of schizophrenia, Dawson and colleagues (Dawson & Nuechterlein, 1984; Dawson, Nuechterlein, Schell, Gitlin, & Ventura, 1994; Nuechterlein & Dawson, 1984) described related concepts referred to as episode and vulnerability indicators. *Episode indicators* are those features that are present during a psychotic episode but not during remission; *stable vulnerability indicators* are features that are present both during remission and psychosis; and *mediating vulnerability factors* are features that are abnormal during remission but are markedly more deficient during psychosis.

In order to understand the status of emotional disturbances in different psychological disorders, it is necessary to review the evidence for their occurrence in the context of the temporal course of the disorder. Specifically, is there evidence showing that emotion disturbances precede the onset of a given disorder? Such evidence would support the role of an observed emotion disturbance as antecedent to the disorder or as a vulnerability indicator. Moreover, knowing that an emotion disturbance predates the onset of a particular disorder allows for more unambiguous claims about the causal status of that disturbance. A prospective, longitudinal study is the best design to determine whether or not emotional disturbances precede the onset of a disorder. Unfortunately, few such studies have been conducted. However, other forms of indirect evidence can be garnered to support the role of emotional disturbances as antecedent to the disorder, including follow-back and retrospective studies. These methods are not without their limitations, however, which I discuss below.

Is there evidence that emotional disturbances are present only during an active symptomatic state? Such evidence suggests that the disturbances are better construed as concomitants or episode indicators. Indeed, most of the research on emotional disturbances in psychopathology has employed cross-sectional designs that are ideally suited to evaluate whether particular emotional features can be construed as concomitants. Finally, if the evidence indicates that emotion disturbances persist after symptomatic recovery or predate a relapse, the disturbances can be construed as consequences of the disorder. Prospective, longitudinal designs are again the best method for ascertaining whether emotional features can be considered to be a consequence of the disorder. To examine whether emotion disturbances predict relapse, it is important to distinguish between relapse and recurrence of symptoms (Hollon & Cobb, 1993), and to distinguish symptoms from broader emotional dysfunction.

EMOTION DISTURBANCES IN SPECIFIC PSYCHOLOGICAL DISORDERS

As noted at the outset of this chapter, emotional disturbances figure prominently in many different psychological disorders. Space constraints prohibits consideration of many disorders, and thus I have chosen to focus on three disorders: schizophrenia, unipolar depression, and social phobia. My reason for choosing these three also stems from the fact that there is a growing body of theory and empirical research on the nature of emotional disturbances in these disorders. Although not typically considered an "emotional" disorder, there has been more research conducted on the emotional features of schizophrenia than on emotional disturbances in the mood or anxiety disorders. Nonetheless, a number of recent findings point to the importance of additional systematic study of emotion disturbances in the more traditional "emotional" disorders, which include unipolar depression and social phobia. I will first review the evidence for emotional disturbances in each disorder and then consider whether the disturbances can be construed as an antecedent, concomitant, or consequence (or some combination of these three) to the disorder. Where possible, the review is limited to studies that have assessed multiple components of emotions. These studies include laboratory-based as well as more naturalistic studies employing measures of facial expression, vocal expression, and self-report of emotional experience. Although recent studies of schizophrenia have also incorporated physiological indices of emotional responding heart rate, and skin conductance (see, e.g., Kring & Earnst, in press; Kring & Neale, 1996), there are not yet many published studies in depression or social phobia that have included these measures. Studies of this sort are now underway and will likely further illuminate the nature of emotional disturbances in many forms of psychopathology.

Schizophrenia

Emotion Disturbances

During the 1990s, a number of empirical studies of emotion disturbance in schizophrenia have found that schizophrenia patients show fewer observable facial expressions in response to emotional stimuli (see, e.g., Berenbaum & Oltmanns, 1992; Dworkin, Clark, Amador, & Gorman, 1996; Krause et al., 1992; Kring & Earnst, 1999; Kring, Kerr, Smith, & Neale, 1993; Kring & Neale, 1996) and during social interaction (see, e.g., Borod et al., 1989; Krause, Steimer, Sanger-Alt, & Wagner, 1989; Kring, Alpert, Neale, & Harvey, 1994; Martin, Borod, Alpert, Brozgold, & Welkowitz, 1990; Mattes, Schneider, Heimann, & Birbaumer, 1995) than do nonpatients. Interestingly, however, these laboratory studies also find that schizophrenia patients report experiencing similar or even greater amounts of emotion compared with nonpatients. Thus, schizophrenia patients' outward displays of emotion are not often an accurate reflection of their experienced emotion. Stated differently, schizophrenia patients report experiencing strong emotions in response to a variety of emotionally evocative stimuli, yet they do not often display these feelings outwardly. Clinically, dampened expressive behavior most closely resembles the symptom of schizophrenia referred to as flat affect. By all outward appearances, patients with flat affect appear to experience no emotion. Yet, as demonstrated by studies that assess multiple components of emotion, the outward display belies the internal emotional experience.

A number of studies have found that schizophrenia patients' and nonpatient controls' reports of emotional experience are similar using a wide variety of self-report measures of emotion. Nonetheless, it has been suggested that schizophrenia patients cannot report how they actually feel but instead are reporting based on how they think the investigators might want them to respond (i.e., response bias). However, additional evidence of emotional responding among schizophrenia patients renders the possibility of response bias less plausible. Although patients displayed fewer observable facial expressions in response to emotionally stimuli, recent studies have shown that patients display very subtle, microexpressive displays in a manner consistent with the valence of the stimuli (Earnst et al., 1996; Kring, Kerr, & Earnst, 1999; Mattes et al., 1995). For example, in response to positive stimuli schizophrenic patients exhibit more zygomatic (cheek) muscle activity, which is typically associated with positive emotion, than corrugator (brow) muscle activity, which is typically associated with negative emotion, in response to positive stimuli. By contrast, patients exhibit more corrugator activity than zygomatic activity in response to negative stimuli (Kring & Earnst, in press; Kring, Kerr, & Earnst, 1999). Thus, these

findings of more subtle facial muscle activity in response to emotional stimuli bolster the conclusion that schizophrenic patients are responding emotionally.

Despite the well-replicated pattern of diminished expression and intact emotional experience among schizophrenia patients, the findings from laboratory studies are seemingly inconsistent with other evidence demonstrating schizophrenia patients to have the symptom of anhedonia, or the diminished capacity to experience pleasure (see, e.g., Blanchard, Bellack, & Mueser, 1994). Indeed, anhedonia appears to be fairly prevalent in schizophrenia, with one study finding as many as three-fourths of patients to have at least moderate levels of anhedonia and one-fourth of patients to have severe anhedonia (Fenton & McGlashan, 1991). More recent studies have examined the relationship between anhedonia and reports of emotional experience in response to emotional stimuli. For example, Blanchard and colleagues (1994) found that schizophrenic patients' scores on a measure of anhedonia were negatively correlated with their reports of positive emotion after both positive and negative emotion-eliciting film clips. That is, the higher the patients scored on the anhedonia measure, the less positive emotion they reported feeling after viewing emotionally evocative stimuli. Unfortunately, this study did not include a nonpatient comparison group with which the patients' reports of emotional experience could be compared. However, Berenbaum and Oltmanns (1992) found that schizophrenic patients with flat affect did *not* differ from nonpatients in their reported emotional experience to emotional film clips even though they scored higher on measures of anhedonia.

An apparent discrepancy emerges in the studies reviewed thus far. With few exceptions, studies that present emotionally evocative stimuli to schizophrenic patients find that patients report experiencing the same amount of positive emotion as nonpatients. Yet, other studies find that schizophrenic patients score higher on measures of anhedonia, indicating that they experience less positive emotion, particularly pleasure, than nonpatients. How can these findings be reconciled? This pattern of findings suggests that the nature of hedonic deficit in schizophrenia may be more circumscribed. As we have suggested elsewhere (Kring, 1999; Germans & Kring, 2000), although schizophrenic patients may not report a pleasure deficit when positive stimuli are presented to them, they may manifest an impaired ability to anticipate the hedonic value of forthcoming pleasurable experiences. It has been hypothesized that hedonic experience includes both appetitive (anticipatory) and consummatory components (see, e.g., Klein, 1987). In other words, the pleasure one derives from the imagining or the expectancy of a rewarding or pleasurable experience (appetitive pleasure) leads to the pursuit and engagement in the pleasurable activity, which results in consummatory pleasure. The research described above suggests that

when presented with emotional material, patients can and do experience positive emotion. However, when asked more generally about whether they find circumstances pleasurable, they are likely to report experiencing less positive emotion.

Other evidence supports this claim. For example, Myin-Germeys, Delespaul, and deVries (in press) had schizophrenic patients complete self-reports of emotional experience at random, daily time intervals over a 2-week period and found that they reported experiencing less positive emotion and more negative emotion than did nonpatient controls. Myin-Germeys and colleagues concluded that the hedonic deficit evident in these patients' self-reports might be linked to the decreased frequency with which these patients participated in pleasurable activities and social interactions, perhaps because they could not anticipate that such activities would be pleasurable. Indeed, Delespaul (1995) found that when asked to report their daily activities, schizophrenic patients described themselves as "doing nothing" (versus engaging in hobbies, sports, social activities, or watching television) five times more frequently than did nonpatient controls. Thus, on a daily basis, schizophrenic patients may not report experiencing pleasure, particularly pleasure linked with social interaction, because they are not participating in pleasurable activities. Although these findings support the notion that anhedonia is linked to a failure to engage in pleasurable activities, it is difficult to determine whether patients' diminished engagement in rewarding pastimes is a cause or a consequence of hedonic deficit.

Other research has indicated that schizophrenia patients may have deficits in emotion perception (see, e.g., Kerr & Neale, 1993; Mueser et al., 1996; Salem, Kring, & Kerr, 1996). However, it is important to note that this deficit does not appear to be specific to emotion perception. Rather, the schizophrenic patients in these studies manifested a more generalized deficit in perceiving faces and voices. Moreover, one study did not find evidence for emotion perception deficits among acutely ill schizophrenia patients (see, e.g., Bellack, Blanchard, & Mueser, 1996), and this result has led some investigators to speculate that antipsychotic medications may be better able to ameliorate perception deficits (including emotion perception) among acutely ill patients but not more chronically disturbed patients (Mueser et al., 1996).

Antecedent, Concomitant, or Consequence?

Unfortunately, few prospective studies on emotion disturbances in schizophrenia have been conducted. Nonetheless, indirect evidence suggests that at least some of the observed emotion disturbances may predate the onset of schizophrenia. More specifically, findings from several studies suggest that schizophrenia patients may display fewer facial expressions (particu-

larly positive expressions) and report experiencing less positive emotion and more negative emotion prior to the onset of the illness. For example, Walker, Grimes, Davis, and Smith (1993) obtained home movies of adults with schizophrenia that were made before these adults developed schizophrenia. They coded facial expressions from the home movies of preschizophrenic boys and girls and found that the girls displayed fewer joy expressions and that both the boys and the girls displayed *more* negative facial expressions compared to their healthy siblings (Walker et al., 1993). It could be the case, however, that the preschizophrenic children showed different types of behaviors or engaged in different types of social interactions captured by the home movies. Thus, these differences could have resulted in different (but appropriate) facial expressions.

Follow-up studies of adults who were seen in mental health clinics as children suggest that schizophrenia patients, particularly males, were shy, withdrawn, anxious, depressed, and socially isolated as children (Fleming & Ricks, 1970; Robbins, 1966). Follow-back studies that rely on archival data have found that preschizophrenic boys were emotionally unstable, depressed, nervous, and impulsive and that preschizophrenic girls were withdrawn and passive (Watt, 1972). Using both follow-up and follow-back method, Knight and Roff (1983, 1985) found evidence that affective disturbances appeared in childhood and persisted into adulthood. Although the findings from these studies are interesting, it remains unclear whether this pattern of social and emotional behavior was actually related to the onset of schizophrenia and whether the patterns were specific to schizophrenia due to the retrospective nature of the design and the fairly limited sample (i.e., patients seen in clinics as children are not necessarily representative of all schizophrenia patients). However, findings from prospective high-risk studies have reported similar findings. High-risk studies identify a group of children at risk for developing schizophrenia (typically defined as having a biological parent with schizophrenia) and then follow them from childhood through the period of risk (Neale & Oltmanns, 1980). Teacher ratings from the Copenhagen High-Risk Study indicated that boys and girls who were later diagnosed with schizophrenia were more emotionally labile, socially withdrawn, socially anxious, and relatively unexpressive than children who did not develop schizophrenia (Olin, John, & Mednick, 1995; Olin & Mednick, 1996). Findings from the New York High-Risk Project indicated that anhedonia was significantly associated with having a biological parent with schizophrenia but not affective disorders (Erlenmeyer-Kimling et al., 1993) and that flat affect was greater among adolescents at risk for developing schizophrenia than among adolescents at risk for developing affective disorders (Dworkin et al., 1991).

Although there is suggestive evidence that at least some type of emotion disturbance in schizophrenia may predate the onset of the disorder,

many of the observed emotion disturbances in schizophrenia can perhaps be most accurately construed as concomitants of the disorder. Indeed, most of the research on emotion in schizophrenia has been cross-sectional and has included patients with at least some degree of residual symptoms. This is not to say, however, that the findings from this cross-sectional research are uninformative. Indeed, findings from these studies have further elucidated the nature of emotion disturbances in schizophrenia and have set the stage for further prospective, longitudinal studies.

With respect to whether emotion disturbances in schizophrenia can be construed as consequences of the disorder, there is some evidence that emotional disturbances in schizophrenia are fairly stable. Both the symptoms of flat affect and anhedonia are chronic (Knight, Roff, Barnett, & Moss, 1979) and stable across time (Blanchard, Mueser, & Bellack, 1998; Keefe et al., 1991; Kring & Earnst, 1999; Lewine, 1991; Pfohl & Winokur, 1982). In a longitudinal study conducted by my laboratory (Kring & Earnst, 1999), diminished emotional expression was stable across 5 months. However, our study did not specifically include assessments during and following a symptomatic episode.

Unipolar Depression

Emotion Disturbances

Two of the current diagnostic criteria (DSM-IV; American Psychiatric Association, 1994) for unipolar depression reflect emotion disturbances: sad mood and loss of interest or pleasure (anhedonia). Although the specific emotion of sadness would seem to be an ideal emotion for further study in depression, most of the research into the nature of emotion disturbances in depression has focused on broader feeling states or core affects (see Russell & Feldman Barrett, 1999). More specifically, evidence indicates that depression can be broadly characterized by low levels of positive affect and heightened levels of negative affect (see, e.g., Watson, Clark, & Carey, 1988). Positive and negative affect are associated with emotional responses, cognitive styles, and personality traits, such as extraversion and neuroticism (Watson, Clark, & Tellegen, 1988). Persons with low levels of positive affect are likely to experience emotions such as sadness and to be interpersonally disengaged. By contrast, persons with high levels of negative affect frequently experience emotions such as anxiety, guilt, and anger. Related to findings of heightened negative affect among individuals with depression, comorbid anxiety is also associated with unipolar depression (see, e.g., DiNardo & Barlow, 1990). Thus, although sadness is certainly central to emotion, it is not uncommon for individuals with depression to experience a number of different negative emotions.

Davidson, Tomarken, and colleagues have found that a particular pattern of resting brain activity is related to positive and negative affect as well as with depression. More specifically, resting left frontal *hypoactivation* has been observed in both currently depressed individuals (see, e.g., Allen, Iacono, Depue, & Arbisi, 1993; Henriques & Davidson, 1991) and previously depressed individuals (Henriques & Davidson, 1990), whereas greater relative left frontal *hyperactivation* has been observed in individuals who report generally experiencing high levels of positive affect and low levels of negative affect (Tomarken, Davidson, Wheeler, & Doss, 1992). Tomarken and Keener (1998) have suggested that relative left frontal hypoactivation may be a marker of risk for depression that is reflected by a number of behavioral and emotional deficits, including the relative incapacity to respond to positive emotional stimuli and self-regulatory deficits in the capacity to use positive events to shift into positive emotional states.

Additional evidence from studies that assess components of emotion episodes indicates that individuals with unipolar depression may also exhibit dampened facial, vocal, and gestural expressive behavior (Berenbaum & Oltmanns, 1992; Ekman & Friesen, 1974; Gotlib & Robinson, 1982; Hargreaves, Starkweather, & Blacker, 1965; Jones & Pansa, 1979; Kaplan, Bachorowski, & Zarlengo-Strouse, 1999; Murray & Arnott, 1993; Scherer, 1986; Ulrich & Harms, 1985; Waxer, 1974). Moreover, dampened expressive behavior among individuals with depression may be specific to positive expressions. For example, Berenbaum and Oltmanns (1992) found that depressed individuals showed fewer facial expressions in response to positive stimuli (but not to negative stimuli) than did nonpatients and schizophrenic patients with flat affect. As noted in the discussion of schizophrenia, diminished expression and experience in response to positive emotional stimuli is broadly consistent with the clinical symptom of anhedonia.

In addition to disturbances in emotion expression and experience, other studies have found that individuals with depression have deficits in emotion perception and biases in the processing of emotional material. For example, Gur and colleagues (1992) found that depressed individuals demonstrated a general negative bias in an emotion discrimination task as well as an impairment in facial expression recognition. Furthermore, some studies have shown that depression is linked to memory biases for mood-congruent stimuli (Bradley, Mogg, & Williams, 1995; Mathews & MacLeod, 1994).

Antecedent, Concomitant, or Consequence?

Similar to the research on emotional features of schizophrenia, most of the studies have been designed to more fully characterize the emotional symptoms of depression. Thus, most of the evidence reviewed above suggests

that the emotional disturbances associated with unipolar depression can be construed as concomitants or episode indicators. For example, with respect to emotion-related cognitive biases, the evidence suggests that memory biases dissipate upon remission (MacLeod & Mathews, 1991), indicating that such biases are state but not trait markers of depression.

Unlike the literature on cognitive aspects of depression, very few studies have examined whether these emotion disturbances predate the onset of the first episode of depression or persist after other symptoms abate. Although a number of high-risk studies have been conducted whereby children with at least one depressed parent are compared with children of nondepressed parents (and, in some instances, children of parents with other psychological disorders), few of these studies have systematically assessed emotion disturbances among these children. Rather, high-risk studies have typically sought to determine the children's risk for developing depression and possible behavioral and psychosocial risk factors that predict the onset of depression. Thus, there is evidence that children of depressed parents are more likely to experience sad mood, anxiety, fears, and anhedonia than children of nondepressed parents (see, e.g., Billings & Moos, 1983; Welner, Welner, McCrory, & Leonard, 1977), although it remains unclear whether this pattern is specific to children of depressed parents (see, e.g., Downey & Coyne, 1990; Lee & Gotlib, 1989a, 1989b). Other recent evidence suggests that some of the emotion disturbances associated with depression may predate the onset of the first episode. Recall that relative decreased activation of left frontal brain areas has been associated with depression. This same pattern of left frontal hypoactivation has been observed in infants of depressed mothers (see, e.g., Field, Fox, Pickens, & Nawrocki, 1995). In addition, greater left frontal hypoactivation has been found among adolescents at risk for developing depression (defined by having a mother who was currently or previously depressed) even though the adolescents did not differ from a comparable control group in current depressed mood (Tomarken, Simien, & Garber, 1994). Insofar as this pattern of brain activity is linked with positive affect (Tomarken et al., 1992), these findings suggest that electroencephalographic measures of frontal brain activity may be emotion-related vulnerability indicators for depression (Tomarken & Keener, 1998).

It seems somewhat paradoxical to think that emotion disturbances in depression would persist after symptomatic recovery, since the symptoms that are alleviated comprise the observed emotion disturbances (i.e., sad mood and anhedonia). However, recent evidence suggests that certain treatments for depression may be more effective in targeting both positive and negative affect. For instance, Tomarken, Elkins, Anderson, Shelton, and Hitt (2000) have found that the combined treatment of sleep deprivation and antidepressant medication was associated with an increase in positive

affect and a decrease in negative affect. By contrast, patients who received only antidepressant medication experienced a decrease in negative affect but no corresponding increase in positive affect. Thus, these findings indirectly suggest that, depending upon the treatment, low positive affect may persist after the episode of depression has gone into remission. Clearly, more research is needed to understand the changes in positive and negative affect that occur both during and after treatment.

Social Phobia

Emotion Disturbances

Social phobia is characterized by anxiety, fear, and avoidance of social situations, performance, and evaluations. Indeed, individuals with social phobia do not experience such anxiety when alone, but rather experience extreme anxiety when confronted with a social situation that involves interaction or presumed evaluation (Barlow, 1988). Theories of social anxiety hold that social anxiety is an extreme manifestation of an otherwise useful response that has evolved to promote an individual's sensitivity to others' disapproval (Barlow, 1988), to facilitate integration into a social group (Baumeister & Leary, 1995; Miller & Leary, 1992), or to negotiate power and status differences (Gilbert & Trower, 1990). When trait social anxiety reaches extreme levels, social phobia may result (Leary & Kowalski, 1995).

Social phobia's most apparent emotional disturbance is the heightened experience of anxiety, fear, and other negative emotions in the context of a social situation. Recent studies have begun to assess the nature and specificity of this disturbance in the experience component of emotion. For example, Wallace and Alden (1997) assessed reports of positive and negative affect in individuals with social phobia and nonclinical controls following successful and unsuccessful social interactions. Individuals both with and without social phobia reported experiencing more negative affect following the unsuccessful social interaction than the successful one. However, only the individuals with social phobia reported significantly greater negative affect and less positive affect than controls following both kinds of interactions.

Although a handful of studies have assessed a broad array of nonverbal behaviors associated with social phobia, no study has systematically examined facial expressions of emotion. Nevertheless, there appear to be nonverbal cues that are reliably associated with social anxiety. For example, Marcus and Wilson (1996) studied social anxiety among college women during an observed speaking task. Observers' ratings of anxiety were significantly related to speakers' reports of anxiety even though speakers rated

themselves as more anxious than observers rated them. These findings indirectly suggest that social anxiety is composed of relatively easily recognizable nonverbal behaviors and cues. In our laboratory, we have examined how social anxiety influences social interactions between men and women meeting for the first time. Dyads who were more socially anxious smiled and nodded less often, exhibited greater speech dysfluencies such as stuttering, leaned to the side more often, and were generally less socially engaging and inviting than dyads who were less socially anxious (Kring, Wissman, & Schmid, 1999).

Research on emotion-related cognitive processes and social phobia indicates that socially anxious individuals may have a bias in attending to negative emotional stimuli, including threat words, facial expressions, and evaluation-related nonverbal cues (Asmundson & Stein, 1994; Hope, Rapee, Heimberg, & Dombeck, 1990; Pozo, Carver, Wellens, & Scheier, 1991; Veljaca & Rapee, 1998; Winton, Clark, & Edelmann, 1995). For example, Winton and colleagues (1995) presented slides of different facial expressions of emotion to individuals scoring high and low on a measure of social anxiety. Socially anxious participants were more likely to identify neutral facial expressions as negative and rated all the expressions more negatively than did participants who were not socially anxious.

Antecedent, Concomitant, or Consequence?

Theorists have suggested that a biological and psychological propensity to experience anxiety in combination with stressful life events involving social interaction set the stage for the development of social phobia (see, e.g., Barlow, 1988). Unfortunately, measurement and definition of this predisposition to experience heightened anxiety remain to be elucidated. One recent study, however, suggests that a particular temperamental style may be uniquely linked to social phobia. Mick and Telch (1998) found that retrospective reports of behavioral inhibition, a temperamental style that involves avoidance, isolation, excessive sympathetic arousal, and withdrawal in the face of novel situations, were related to symptoms of social phobia but not generalized anxiety disorder.

Nonetheless, that social anxiety manifests itself only in the context of a social situation suggests that the emotional disturbance is perhaps best construed as a concomitant rather than as an antecedent or a consequence. This is not to say, however, that repeated exposure to anxiety provoking social interactions will not exacerbate or at least contribute to the maintenance of social phobia over time. In addition, there are likely to be a number of interpersonal consequences of the experience and display of heightened anxiety. For example, individuals with social phobia nonverbally communicate to others the inordinate risk of embarrassment in social inter-

action, thus precluding the desire for interacting with those individuals. In addition, it seems likely that interactions with socially phobic individuals will be more frustrating and distressing and perhaps eventually avoided (Keltner & Kring, 1998).

CONCLUSION, INTEGRATION, AND DIRECTIONS

Emotion disturbances are common among many different forms of psychopathology. Research in the past 15 years has sought to more fully characterize the nature of these emotion disturbances. Although this chapter has focused on the disturbances that characterize just three disorders, a number of other disorders include widespread emotion disturbances. For example, many of the personality disorders include problems in emotion regulation, such as borderline personality disorder (see, e.g., Farchaus-Stein, 1996; Linehan, 1987; Shearin & Linehan, 1994) and psychopathy (Arnett, Smith, & Newman, 1997; Patrick, Bradley, & Lang, 1993; Patrick, Cuthbert, & Lang, 1994). Nearly all of the anxiety disorders are characterized by heightened negative affect (see, e.g., Chorpita & Barlow, 1998; Clark & Watson, 1991; Watson et al., 1995), yet recent research has attempted to further elucidate emotional disturbances that distinguish the specific anxiety disorders (see, e.g., Mulken, de Jong, & Merkelbach, 1996; Zinbarg & Barlow, 1996). Challenges for the next generation of research include isolating the specificity of emotion disturbances within different disorders, more clearly locating the disturbances within the temporal course of the disorder, examining both specific, discrete emotions as well as broader emotion dimensions, linking emotion disturbances with other disorder-related disturbances, and developing and implementing interventions that target emotion disturbances.

It seems that one fruitful approach to meeting these challenges is to continue to incorporate basic emotion methods and paradigms into the study of psychopathology. For instance, researchers interested in studying emotion disturbances in psychological disorders ought to include measures of multiple emotion components and frame hypotheses about emotion disturbances with an empirically validated theoretical approach to emotion (Keltner & Kring, 1998; Kring & Bachorowski, 1999; Tomarken & Keener, 1998). Moreover, a combination of both laboratory and naturalistic research will likely yield the most complete picture of emotion disturbances in psychopathology. Thus, laboratory findings of heightened negative emotional experience among schizophrenia patients has been confirmed in studies assessing patients' emotional experience in daily life. Other approaches to the study of emotion will also generate relevant information about the nature of emotion disturbances in psychopathology. For exam-

ple, narrative approaches have been successfully employed in the study of agoraphobia and bereavement (Capps & Bonanno, in press; Capps & Ochs, 1995). Personal narratives are rich sources of information about current, past, and future life events (see, e.g., McAdams, 1996; Ochs & Capps, 1996). This approach can provide information about *how* individuals use language to describe their emotions and make sense of the illness experience. Combining this approach with more traditional clinical interviews will undoubtedly help to elucidate how emotion figures prominently in various forms of psychopathology.

Although there are a number of important directions for future research, I note two here in some detail. First, the specificity of emotion disturbances in psychopathology needs to be clarified. For example, although some evidence indicates that heightened negative affect in combination with lowered positive affect is specific to unipolar depression (Watson, Clark, & Carey, 1988), other studies have found the same pattern in social phobia (Wallace & Alden, 1997) and schizophrenia (Blanchard, Mueser, & Bellack, 1998). Notably, studies finding this pattern in other disorders did not include a comparison group of individuals with unipolar depression, so it is difficult to know if these patient groups demonstrate a similar pattern of reported positive and negative affect. Indeed, it is necessary to include other psychopathological comparison groups to get at the specificity of emotion disturbances. Nonetheless, these findings suggest that lowered positive affect may not be specific to unipolar depression. Is it important to identify specific emotion disturbances for different disorders? It may be the case that broad characterizations of emotion disturbances identify a number of different disorders and that specificity is the exception rather than the rule. Indeed, heightened negative affect is a prime example of a general distress factor common to many different psychological disturbances. However, some specificity may nonetheless be detected among different disorders. For example, schizophrenia patients are less expressive of both positive and negative emotions (Kring & Earnst, 1999), whereas depressed patients may only exhibit fewer positive expressions (Berenbaum & Oltmanns, 1992). Identifying a specific emotion disturbance will aid in linking that disturbance to other known biological, cognitive, and behavioral features of a particular disorder. A second direction for research is to more clearly locate emotion disturbances within the temporal course of the disorder. A major focus of this chapter has been to consider whether the observed emotion disturbances in various disorders can be considered antecedents, concomitants, or consequences. The starting place for much of the research on emotion and psychopathology has been to more fully characterize the emotional symptoms. In essence, these studies have generated construct validity evidence for emotional symptoms and have thus pro-

vided support for the role of emotional features as concomitants of different disorders. However, in some cases, this research has gone beyond validating symptoms. For example, laboratory research has confirmed that schizophrenia patients with the symptom of flat affect are less expressive across a variety of situations. Moreover, this research has shown that the emotion disturbance in schizophrenia involves not only the expressive component of emotion; rather, it is best characterized by a pattern of expressive, experiential, and psychophysiological emotion components (Kring & Neale, 1996). Continued research that seeks to fully delineate the emotional symptoms of various psychological disorders is still an important direction for research.

This research strategy that has begun with emotional symptoms or features of a particular disorder is in contrast to, for example, research on cognitive accounts of depression. In that research, the focus is not on symptoms per se, but rather on broader cognitive styles that are hypothesized to account for the symptoms. However, psychopathology researchers interested in emotion are now well equipped to further investigate questions about mechanism. The search for mechanisms leads to questions about antecedents and consequences of emotional disturbances. As reviewed above, indirect evidence supports the role of emotion disturbances in schizophrenia and unipolar depression as antecedents and consequences of the respective disorders. Further prospective studies that assess the emotional responding in the same patients across episodes are sorely needed. Moreover, new relevant findings from ongoing high-risk studies will continue to be generated as the samples pass through the period of risk. Although these studies were not necessarily designed to study emotional risk factors, they nonetheless contain a number of measures relevant to answering questions about whether emotion disturbances predict the onset and/or maintenance of different disorders.

In summary, emotional disturbances figure prominently in psychopathology. Additional research is needed, however, to illuminate the manner in which disturbed components of emotional processes contribute to the onset, maintenance, and long-term consequences of the disorders. Research that encompasses a wide variety of methods and multiple levels of analysis is the most promising approach to further our understanding and treatment of emotion dysfunction.

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