

## Emotions and Psychopathology

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Emotional disturbances are central to diverse psychopathologies. In this article, we argue that the functions of emotion are comparable for persons with and without psychopathology. However, impairment in one or more components of emotional processing disrupts the achievement of adaptive emotion functions. Adopting a theoretical conceptualisation of emotional processes that stresses activity in centrally mediated approach and withdrawal systems, we discuss the role of emotion in several forms of psychopathology, including major depression, some of the anxiety disorders, psychopathy, and schizophrenia. In doing so, we highlight the nature of emotion disturbance and attendant behavioural and cognitive deficits. Finally, we discuss the merits of this approach for conceptualising emotional disturbance in psychopathology.

### INTRODUCTION

Most forms of psychopathology reflect disturbances in a number of areas, including emotional processing and emotional responding. Although emotions figure prominently in many disorders, the nature of emotional disturbance varies among disorders. For example, some of the anxiety disorders are marked by the experience of intense anxiety and/or fear, often occurring in the absence of an obvious precipitant (e.g. Barlow, 1991). By contrast, schizophrenia is often marked by diminished expression of emotion and, in some cases, diminished experience of emotion (e.g. Kring & Neale, 1996). These examples illustrate just two types of emotional disturbance in psychopathology: An excess of experienced emotion and a deficit in the expression of emotion. In this article, we will argue that

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many of these disturbances reflect a disruption in one or more components of emotional processing that, in turn, interfere with the achievement of adaptive emotion functions. Furthermore, not only are disturbed emotional processes salient features of these disorders, they are linked with hypothesised aetiological factors at both neurobiological and psychological levels.

Emotions are complex systems that developed through the course of human evolutionary history and that prepare an organism to act in response to environmental stimuli and challenges (cf. Keltner & Gross, this issue). We view these emotion systems as being fundamentally linked with two motivationally adaptive systems typically referred to as goal-directed approach and withdrawal systems. Although the specifics of theoretical accounts that describe these systems differ, the approach system has variously been referred to as the Behavioral Activation System (Fowles, 1980; also see Gray, 1987) and the Behavioral Facilitation System (Depue & Iacono, 1988; Depue, Krauss, & Spont, 1987), whereas the withdrawal system has been most often identified as the Behavioral Inhibition System (Fowles, 1980; Gray, 1976). A third system, which will be discussed in the section on anxiety disorders, is referred to by Gray as the Fight-Flight system (Gray, 1987), and responds to nonreward and unconditioned punishment.

We have adopted the approach and withdrawal system perspective to describe emotional processes within selected psychopathologies because, in our estimation, this approach provides a parsimonious account of emotional disturbance in these disorders. An alternative approach would be to adopt a strategy that emphasises a discrete emotions perspective, in which primacy would be given to an understanding of the disturbances within a particular emotion, such as sadness or anxiety. Although not clearly articulated by motivation system theorists, discrete emotions can be aligned with activity in either an approach or a withdrawal system. However, some emotions can also be meaningfully linked with activity in both motivation systems. For instance, anger is a negative emotion with behavioural components that can range from overt aggression to active avoidance. In a motivation system framework, it is possible for these "hot" and "cold" varieties of anger to be differentially mediated by activity in approach and withdrawal systems, respectively. A related point is that positive emotions are not invariably associated with approach system activity, nor are negative emotions always linked with withdrawal system activity. For instance, the experience of the negative emotion of sadness in depression has largely been attributed to disturbance in an approach motivation system.

Emotions have several components, including behavioural and expressive, subjective/experiential, physiological, and cognitive, and most func-

tional accounts of emotion assume that these components of emotion operate in relative synchrony in most situations. Indeed, in nondisturbed individuals, the co-ordinated engagement of the various emotion components subserves a number of adaptive organisational and motivational functions (Buck, 1994; Ekman, 1994a,b; Izard, 1993a,b; Lang, Bradley, & Cuthbert, 1990; MacLean, 1993; Nesse, 1990; Plutchik, 1993). As examples, some of these functions include stimulus perception and evaluation, organisation of motivated responding, behavioural regulation, and coping. Additionally, adaptive communicative functions include the modifiable production of expressive cues that signal motivational states and behavioural intentions (Fridlund, 1994; cf. Buck, 1994). In our estimation, theoretical conceptualisations of emotional processes that are linked to activity in centrally mediated approach and withdrawal systems (Davidson, 1992; Lang et al., 1990) are especially useful for integrating this multiplicity of functions with evident phylogenetic continuities in both neuroanatomy (MacLean, 1993) and behaviour (Davidson, 1992; Konorski, 1967; Lang et al., 1990).

We consider the *functions* of emotion in persons with psychopathology to be comparable to those for nondisordered individuals. However, in many psychopathologies, one or more *components* of emotional processing are impaired in some respect. Such deficits can occur, for instance, in the perception, experience, intensity, or display of emotions. Consequently, a disordered individual's ability to achieve one or more emotion functions in an adaptive fashion is impaired. Thus, we posit that many of the emotional disturbances in psychopathology can be construed as deficits in one or more components of emotional processing that disrupt the adaptive outcome of activity in approach and withdrawal systems. Although some theorists have speculated about the potentially adaptive functions of psychopathology specifically, and disordered emotional processes more generally (e.g. Nesse, 1990), the functional outcome of these disturbances will also necessarily involve the impaired achievement of motivationally significant goals.

Although emotions play a role in most types of psychopathology, we will discuss the role of emotion in only a few psychopathologies: Unipolar depression, some of the anxiety disorders, psychopathy, and schizophrenia. We chose to include this particular group of disorders for several reasons. First, emotional disturbances are salient features in each of these conditions. Second, there is a fairly well-characterised body of theory and empirical research supporting the role of emotional disturbances in these disorders. Finally, by elaborating on the role of emotions in these four exemplars, we are able to extract principles about how emotions influence, interact with, and contribute to psychopathology more generally.

## UNIPOLAR DEPRESSION

The cardinal emotional symptoms of unipolar depression include sadness and anhedonia (i.e. a deficit in the capacity to experience pleasure). Both symptoms can be characterised as comparatively enduring mood states as well as phasic emotional reactions, and have been construed as outcomes of dysregulated activity (i.e. excessive variability) in an approach motivational system (Clark & Watson, 1991; Clark, Watson, & Mineka, 1994; Depue et al., 1987; Depue & Iacono, 1988; Fowles, 1994; Henriques, Glowacki, & Davidson, 1994; Tellegen, 1985). Other symptoms associated with depression, including comorbid anxiety and guilt, are more closely related to withdrawal system activity (Clark et al., 1994; Tellegen, 1985).

Some support for considering that the emotional features of depression reflect disturbances in both approach and withdrawal systems comes from the conceptual links that have been made between these two systems and levels of positive and negative affect (Clark & Watson, 1991; Clark et al., 1994; Watson, Clark, & Carey, 1988). When measured with the Positive and Negative Affect Schedule: General (Watson, Clark, & Tellegen, 1988), levels of positive affect and negative affect are construed as reflecting positions along two broad temperament dimensions. Thus, differences on each dimension are associated not only with characteristic moods and emotional responses, but also with particular cognitive styles and personality traits, such as extraversion and neuroticism. Persons with low levels of positive affect are apt to experience emotions such as sadness and dullness, whereas persons with high levels of negative affect frequently experience emotions such as anxiety, guilt, and hostility. Cumulative empirical evidence supports the position that this particular temperament combination is correlated with depression, with low positive affect showing some specificity to depression and the general distress characteristic of high negative affect related not only to depression, but to a variety of other emotional disorders, as well.

To our knowledge, no empirical investigations have been conducted that simultaneously examine the hypothesised associations among depression, levels of positive and negative affect, and approach and withdrawal system disturbance. However, Depue and his colleagues (Depue, Luciana, Arbisi, Collins, & Leon, 1994) described relations among several peripheral indices of central dopamine activity, a neurotransmitter known to mediate goal-directed approach behaviour (e.g. Le Moal & Simon, 1991), and positive emotionality (Tellegen & Waller, 1992), a trait variable that is thought to index sensitivity to signals of reward and that has strong theoretical links with an approach motivation system (i.e. the Behavioral Facilitation System; Depue et al., 1994). These empirical findings demonstrate that it may be feasible to broaden the range of experimental inquiry in depression and

systematically examine the conjoint influence of affective predispositions and motivation system disturbances.

The diverse behavioural, cognitive, psychophysiological, and neurochemical findings observed in depression have been variously related to depressive emotional symptoms, differences in dispositional affect, and disturbed activity in approach and withdrawal motivational systems. One replicated finding that serves as an exemplar of an integrated approach to understanding emotional deficits in depression involves asymmetrical patterns of electrocortical activation in brain frontal lobes. As examples, resting left frontal *hypoactivation* has been observed in both currently depressed (e.g. Henriques & Davidson, 1991) and previously but not currently depressed individuals (Henriques & Davidson, 1990). Furthermore, adolescents with depressed mothers, a group presumed to be at risk for depression by virtue of maternal clinical status, also manifest greater relative left frontal *hypoactivation* (Tomarken, Simien, & Garber, 1994). In contrast, greater relative left anterior *hyperactivation* has been observed in individuals thought not to be dispositionally prone to depression by virtue of reporting high levels of positive affectivity (Tomarken, Davidson, Wheeler, & Doss, 1992).

The activation asymmetries observed in depression are hypothesised to be one manifestation of disturbance in a reward-oriented approach system. Specifically, Davidson, Tomarken, and their colleagues (e.g. Davidson, 1992; Davidson & Tomarken, 1989; Henriques & Davidson, 1990; Tomarken & Keener, 1998) have proposed that stable, resting left frontal *hypoactivation* is a diathesis for depression that promotes vulnerability to the behavioural and emotional sequelae of approach system deficits, such as the inability to sustain goal-directed approach behaviour, the relative incapacity to respond to positive emotional stimuli, the prolonged maintenance of negative affect, and self-regulatory deficits in the capacity to use positive events to shift into positive emotional states. As described by Tomarken and Keener (1998), corroborating support for the hypothesised link between left frontal *hypoactivation* and approach system disturbance comes from the behavioural and psychological functions thought to be subserved by the frontal lobes (e.g. Fuster, 1990; Goldman-Rakic, 1987), along with evidence that the mesocorticolimbic dopaminergic system mediates approach behaviour (e.g. Stellar & Stellar, 1985).

A distinct but similarly influential line of research has provided evidence that particular cognitive or attributional styles may function as diatheses for depression. Like the hemispheric activation asymmetry work, available cognitive models are generally compatible with accounts of depression that emphasise disturbances in both approach and withdrawal motivation systems (Fowles, 1994). As one example, the hopelessness model (Abramson, Metalsky, & Alloy, 1989) posits that the tendency

to make stable, global attributions for important negative life events is a cognitive diathesis for a hopelessness subtype of depression. Studies reviewed by Clark and her colleagues (1994) indicate that this hopelessness attributional style is linked to negative but not positive affect, suggesting that this cognitive style is not specific to depression and may be more closely associated with generalised emotional distress and corresponding disturbances in a withdrawal motivational system. In contrast, other empirical findings (Jolly, Dyck, Kramer, & Wherry, 1994; cf. Alloy, 1991) indicate that the tripartite influence of positive affect, negative affect, and the hopelessness attributional style are more predictive of depressive symptoms than either emotional or cognitive features alone. These results indicate that individual differences in dispositional affect that have putative links to approach and withdrawal systems are also associated with a cognitive style that has been postulated as a diathesis for at least some forms of depression. Thus, further conceptual and empirical work is necessary to tease apart the manner in which depressed cognitive styles are aligned with positive and negative affect dimensions and, in turn, with motivation system disturbances.

Unipolar depression has been linked with several components of emotional responding, such as biases in the perception of and response to emotionally significant stimuli. For instance, depression is associated with selective impairments in the ability to identify facial expressions of emotion as well as a generalised negative bias in affect discrimination (Gur et al., 1992). Furthermore, deficits in the recognition of facial cues that signal emotional states have been associated with less adaptive behavioural and emotional responses among depressed individuals, such as greater avoidance of and less tolerance to those facial cues (Persad & Polivy, 1993). Accumulated evidence supports the contention that depression is also associated with cognitive biases for the processing of emotional stimuli (for reviews, see MacLeod & Mathews, 1991; Mineka & Sutton, 1992). One well-established finding is that depression is related to memory biases for mood-congruent stimuli. These biases have been reported for both automatic and strategic memory processes and, importantly, appear to be specific to depression (Bradley, Mogg, & Williams, 1995). Mood-congruent memory biases, along with prolonged self-focused rumination (Nolen-Hoeksema & Morrow, 1993; Tomarken & Keener, 1998) and other cognitive vulnerability factors, such as discrepancies between actual and ideal self-representations (e.g. Strauman, 1992), might contribute to the maintenance of depressed states. Evidence that memory biases dissipate on remission (MacLeod & Mathews, 1991) suggests that such biases are state but not trait markers of the disorder.

Although descriptive accounts of depression frequently mention diminished facial expressivity, empirical investigations have not systematically

examined this component of emotion in depression. However, interest in vocal expression, generally, and the impact of maternal depression on child development, in particular, have prompted some investigators to specify the nature of expressive vocal deficits in depression. The speech of depressed adults has been qualitatively described as “flat”, “dull”, and slow in tempo (Buck, 1984; Hargreaves, Starkweather, & Blacker, 1965; Levin, Hall, Knight, & Alpert, 1985; Murray & Arnott, 1993; Scherer, 1986). Several comparatively quantitative acoustic parameters, such as those derived through the analysis of digitised waveform representations of speech, have also proven useful in distinguishing between the speech of depressed and nondepressed individuals. For example, Bettes (1988) reported that mothers with self-reported symptoms of depression produced infant-directed speech with narrower pitch contours than were observed in the infant-directed speech of control mothers. Similarly, Kaplan, Bachorowski, and Zarlengo-Strouse (in press) also noted different patterns of pitch modulation and variability in the infant-directed speech of mothers with symptoms of depression. Importantly, they found that this expressive deficit was related to impairments in infant associative learning (see also Hoff, Kaplan, Zarlengo-Strouse, & Bachorowski, 1999), suggesting that the infant-directed speech produced by depressed caregivers may be one variable that mediates the relationship between maternal depression and cognitive-emotional disturbances in their children (e.g. Cohn & Campbell, 1992; Murray, 1992).

Although speculative, the expressive speech deficits frequently observed in depression seem to be consistent with an approach motivation system disturbance. Many of these production deficits, such as difficulty in speech initiation, longer and more frequent pauses, and an overall slower rate, can be included in the cluster of symptoms referred to as psychomotor retardation. Furthermore, from a speech production perspective, these symptoms are consistent with descriptions of the behavioural characteristics associated with approach system disturbance, such as difficulty in the initiation of behaviour. Although the specific pathways and mechanisms that underlie speech and other motor deficits in depression are not known, one likely system of involvement includes the basal ganglia, with its remarkably high concentration of dopamine (Côté & Crutcher, 1991) and its role in some of the motor aspects of speech production (Borden, Harris, & Raphael, 1994).

## ANXIETY DISORDERS

The anxiety disorders are a heterogenous group of disorders that typically involve a number of negative emotions, the most prominent being anxiety, fear, and disgust. Although the different anxiety disorders likely vary in the

extent to which various emotional processes are disturbed, there is a good deal of evidence that most, if not all, of the anxiety disorders are characterised by heightened negative affect (e.g. Chorpita & Barlow, 1998; Clark & Watson, 1991; Watson et al., 1995; Zinbarg & Barlow, 1996). Additionally, several of the anxiety disorders are also associated with behavioural avoidance of the situations or stimuli in which anxiety is elicited. Such behavioural avoidance is often used to minimise subjective feelings of anxiety or fear, as in the case of an individual with a phobia of flying who studiously avoids airplane travel no matter how inconvenient.

Although heightened negative affect may be shared by most of the anxiety disorders, specific emotions, such as fear, figure more prominently in some disorders, such as specific phobias and panic disorder, more than in others. Although the terms "anxiety" and "fear" are often used interchangeably, these two emotions can be distinguished conceptually, empirically, and clinically (Öhman, 1993). For example, Barlow (1988) described anxiety, which he refers to as anxious apprehension, as a mood state characterised by both negative affect and somatic tension that is associated with the anticipation of future danger or misfortune. By contrast, fear is an immediate alarm reaction to a perceived threat or danger (Barlow, 1988). Furthermore, recent evidence points to distinct neurophysiological pathways for fear (LeDoux, 1995a) and anxiety (Gray, 1995). It is important to note that among individuals with anxiety disorders, the presence of negative emotions is not dysfunctional *per se*. For example, the fear response that characterises a panic attack is an otherwise normal or functionally adaptive response that occurs at an inappropriate time (Barlow, 1988, 1991). To account for these responses occurring in the absence of significant threat, many theorists have argued that some of the anxiety disorders, such as panic disorder and specific phobias, reflect inappropriate activation of and/or disturbance in the motivation systems underlying these negative emotions. In our analysis, we will consider three of the anxiety disorders that have been conceptually linked to motivation systems: Panic disorder, specific phobias, and generalised anxiety disorder.

Although a number of different etiological theories for these three anxiety disorders have emerged, these theories are markedly similar in that they either directly or indirectly implicate disruptions in withdrawal motivational systems as a key etiological contributor (e.g. Barlow, 1988; Chorpita & Barlow, 1998; Gray, 1976, 1982; Lang, 1995; Lange et al., 1990). Gray has postulated that the neurobiologically based Behavioral Inhibition System (BIS) is an emotion system that serves to inhibit ongoing behaviour in response to conditioned stimuli associated with punishment and frustrative nonreward (e.g. Gray, 1976, 1979, 1982; cf. Wallace, Bachorowski, & Newman, 1991). In addition to inhibiting behaviour, activity in the BIS is also related to increased arousal and increased



vigilance to environmental stimuli. Anxiety, according to Gray, reflects activity in the BIS. One primary source of evidence linking this system to anxiety disorders comes from studies showing that anxiolytic medications affect behaviour and physiology (e.g. electrodermal activity) believed to be associated with inhibition system activity in both animals (e.g. Gray, 1979) and humans (e.g. Landon, Sher, & Shah, 1993). Panic, according to Gray, reflects activity in his proposed Fight-Flight System. Notably, Gray's Fight-Flight system is similar to Cannon's description of the emergency (fight or flight) reaction (e.g. Cannon, 1929).

Barlow's (1988, 1991) model specifies that anxious apprehension is marked by heightened arousal, high negative affect, perceptions of unpredictability and uncontrollability, and worry. Anxious apprehension is considered by Barlow to be the primary characteristic of generalised anxiety disorder. Moreover, anxious apprehension is often adequately assessed using measures of negative affect or neuroticism (Barlow, 1988). Panic, according to Barlow, is the clinical manifestation of fear. More specifically, panic attacks are viewed as alarm reactions elicited *without* exposure to a particular stimulus. By contrast, the alarm reaction seen in specific phobias is typically elicited following exposure to the feared object or situation (see also Mineka, 1985; Mineka & Cook, 1993). Key cognitive components of Barlow's theory are the concepts of predictability and controllability (see also Garber, Miller, & Abramson, 1980), such that individuals with various anxiety disorders often perceive that negative events and panic reactions are neither predictable nor controllable. For example, the likelihood of a panic disorder patient having a panic attack following pharmacological provocation is dramatically reduced if the patient is told about the type and time course of the effects the drug will produce (Barlow, 1988). It is informative to note that Barlow's concept of anxious apprehension shares a number of similar features with Gray's Behavioral Inhibition System, such as heightened arousal, preparation for stress and challenge, and anticipation of aversive outcomes. Similarly, Barlow's alarm reaction resembles Gray's description of the Fight-Flight System (Fowles, 1994).

A third recent and related theoretical contribution is the tripartite model of anxiety and depression proposed by Clark and Watson (1991). Although originally intended as a means to distinguish anxious and depressed mood and anxiety and depressive syndromes, the model has implications for specific anxiety disorders. Briefly, their model holds that the symptom overlap in the anxiety and mood disorders is accounted for by a general marker of heightened dispositional negative affect. However, each of these "distress" disorders can be distinguished by characteristics that are believed to be relatively unique to each. Specifically, heightened somatic arousal and tension characterises anxiety. Watson et al. (1995) provided support for this tripartite distinction in factor analytic studies across five

different samples. Furthermore, Watson et al. (1988) demonstrated that heightened negative affect characterised both patients with anxiety and with depression, whereas lowered positive affect was only characteristic of the depressed patients. The tripartite model is consistent with Gray's Behavioral Inhibition System insofar as heightened negative affect and somatic arousal have been linked to activity in the inhibition system. And as noted earlier, Barlow's concept of anxious apprehension is consistent with elevated negative affect or neuroticism.

It is important to note that in Clark and Watson's model, negative affect is construed as the "temperamental core" of neuroticism (Clark et al., 1994), which suggests that negative affect reflects a stable, emotional vulnerability factor for anxiety (and depression). However, the extant data on this important supposition are lacking. Prospective, longitudinal studies are necessary to disentangle the state and trait contributions of negative affect to anxiety. Moreover, additional research (e.g. Zinbarg & Barlow, 1996) to further elucidate emotional factors that distinguish the specific anxiety disorders is needed.

Similar to the literature reviewed for unipolar depression, a number of the anxiety disorders are also characterised by biases in the perception of emotionally significant stimuli. For example, a number of studies have found that panic disorder patients misperceive harmless events or objects in the environment as threatening (Barlow, 1988; Clark, 1988; McNally, 1990). Moreover, cumulative evidence suggests that anxious patients are more likely to attend to threatening stimuli and make biased judgements about the likelihood of negative outcomes as well as the covariation between these outcomes and feared stimuli (Mathews & MacLeod, 1994; Mineka & Sutton, 1992; Tomarken, Mineka, & Cook, 1989; Tomarken, Sutton, & Mineka, 1995). The directional influences of these cognitive biases and emotional processing are not well understood and should be a focus of future investigations on the linkage between cognitive and emotional factors in anxiety disorders (Öhman, 1993).

Surprisingly, little is known about the expressive component of emotion among patients with anxiety disorders, although there is reason to believe that this component might be desynchronous from other components, at least in specific phobias. In a study of spider and snake phobics, participants reported equivalently high levels of fear and disgust, yet facial expressions of disgust were far more common than facial expressions of fear (A.J. Tomarken, personal communication, August 1994). This finding is consistent with other research showing that small animals such as spiders also elicit strong reports of disgust and that these reports of disgust are positively correlated with fear intensity among phobics (e.g. Ware, Jain, Burges, & Davey, 1994). Moreover, spider phobics have been found to have a higher sensitivity to disgust than nonphobics (Merckelbach, de Jong,

Arntz, & Schouten, 1993; Mulkens, de Jong, & Merckelbach, 1996). These findings highlight the importance of considering discrete emotions in addition to broad emotion dimensions when considering the role of emotion in the anxiety disorders, specifically, and psychopathology in general.

## PSYCHOPATHY

Cleckley's (1941) authoritative description of psychopathy emphasised deficient emotional processes, including impoverished emotional reactions, lack of anxiety, and a disjunction between the lexical and experiential components of emotion in his delineation of the core features of the disorder. Although attention to these cardinal emotional features is notably absent from recent DSM conceptualisations of Antisocial Personality Disorder (Hare, Hart, & Harpur, 1991; Sutker, 1994; Widiger et al., 1996), contemporary theoretical accounts, supported by a growing corpus of empirical findings, continue to posit that emotional disturbances are both salient and fundamental aspects of psychopathy.

Following from the manifest difficulties that psychopaths have in learning from punishment, several investigators have examined psychophysiological disturbances in negative emotions, such as fear and anxiety, whereas others have focused on attendant cognitive-behavioural deficits, such as poor passive-avoidance learning. Using variants of the startle-probe paradigm, compelling evidence that psychopaths respond to aversive stimuli in an anomalous fashion has been provided by Patrick and his colleagues (e.g. Patrick, 1994; Patrick, Bradley, & Lang, 1993). In these experiments, the amplitude of eyeblink startle response to an aversive, task-irrelevant stimulus, such as a 95-decibel white noise acoustic startle, is used as an index of defensive emotional reactions. Typically, startle responses are potentiated during induced fear and other aversive states but are diminished during positive emotional states. In comparison to controls, psychopaths show comparable startle inhibition during exposure to pleasant stimuli but importantly fail to show startle potentiation during exposure to aversive stimuli. Additionally, associations between startle reactions and factor scores on the Revised Psychopathy Checklist (PCL-R; Hare, 1992) suggest that deviant responding in the probe paradigm is linked to a core deficiency in emotional processes but not to antisocial behaviour more generally.

Anomalous startle potentiation in psychopaths has been observed despite verbal reports that corroborated the aversive nature of the stimuli and that did not differ from controls' self-reports (Patrick et al., 1993). Additional evidence for this "semantic dementia" (Cleckley, 1941), or discordance between the linguistic and experiential components of emotion, has been found with both fear imaging (Patrick, Cuthbert, & Lang, 1994) and incidental memory paradigms (Christianson, et al., 1996).

Similarly, Williamson, Harpur, and Hare (1991) found that although the rated emotional pleasantness of positive, negative, and neutral words did not differ for psychopaths and nonpsychopaths, psychopaths demonstrated a relative failure to differentiate emotional and neutral words at both behavioural and electrocortical levels.

Deviant peripheral physiological responding to both actual and anticipated aversive events has been routinely observed with psychopathic samples. For example, differences in several indices of electrodermal activity during classical aversive conditioning paradigms and in the anticipation of noxious stimuli have been demonstrated (e.g. Hare, 1978; Siddle & Trasler, 1981; for a review, see 1994). Such differences are consistent with the well-recognised behavioural difficulty of psychopaths to learn to withhold responding in order to avoid punishment (Fowles, 1994; Patterson & Newman, 1993). Beginning with the classic work of Lykken (1957), investigators have consistently demonstrated this passive-avoidance deficit in a variety of behavioural contexts. Research conducted by Newman and his colleagues (e.g. Arnett, Smith, & Newman, 1997; Newman, 1987; Newman & Kosson, 1986; Newman, Patterson, Howland, & Nichols, 1990) has elucidated the contexts that are especially likely to engender passive-avoidance deficits in psychopaths. In particular, avoidance learning deficits are most readily apparent when the behavioural paradigm also promotes appetitive responding for reward. In such joint reward and punishment contexts, psychopaths fail to modulate behavioural responding in an adaptive fashion in that they fail to learn to withhold responding in order to avoid punishment.

The theoretical models advanced by Fowles (1980, 1994) and by Newman and his colleagues (Gorenstein & Newman, 1980; Newman, 1998; Patterson & Newman, 1993) attempt to account for the diverse behavioural and psychophysiological findings associated with psychopathy. Although both theorists generally adhere to an approach-withdrawal motivation system conceptualisation, with specific recourse to Gray's (1978, 1982, 1987) neuropsychological theory, the differences between their perspectives have potentially important implications for understanding the emotional and behavioural features observed in psychopathy.

Fowles' (1980, 1994; cf. Quay, 1993) perspective is that psychopathic deficits are largely attributable to a weak or deficient Behavioral Inhibition System (BIS). Normally, cues for punishment act as inputs to the BIS, and various behavioral and emotional consequences ensue. In contrast, a weak BIS means that cues for punishment are less likely to activate the BIS. In the absence of BIS engagement, the adaptive sequelae of BIS activity, including the inhibition of ongoing behaviour, reflection, and the experience of anxiety, will not occur. Thus, the behavioural and psychophysiological anomalies associated with psychopathy are thought to arise because

the psychological processes that would normally function to motivate adaptive behavioural and emotional responding in response to cues for punishment do not occur. Although not as pivotal to his framework, Fowles (1994) has speculated that psychopathy may also involve a strong or overactive Behavioral Activation System (BAS).

As described by Arnett, Smith, and Newman (1997), two general models have been proposed by Newman and his colleagues. The first model (Gorenstein & Newman, 1980), which stresses septal-hippocampal system disturbances as underlying the disinhibited behaviour associated with psychopathy, is consistent with a strong BAS. The second model (Newman, 1998; Patterson & Newman, 1993; cf. Newman & Wallace, 1993a,b), which has been the primary focus of empirical investigations conducted by this group, proposes that a fundamental disturbance in psychopathy is a deficit in response modulation. Specifically, psychopaths are thought to have difficulty suspending reward-oriented approach behaviour in order to accommodate feedback from the environment. That is, rather than emphasising impairment in either activation or inhibition systems, this model predicts that a primary disturbance in psychopathy is a deficit in the reciprocal inhibition of activation and inhibition systems. Although Newman emphasises information-processing characteristics, such as the allocation of attention to motivationally significant stimuli, this model can also account for some of the notable emotional features of psychopathy, including low fear and low anxiety (see Newman, 1998, for a detailed examination of models of psychopathy).

The aforementioned theoretical approaches readily account for many of the diverse features associated with psychopathy, although a detailed application of each model to the various behavioural and psychophysiological findings associated with psychopathy leads to distinctions that can only be resolved through further empirical work. To date, few empirical investigations have specifically tested these models as competing hypotheses. One exception are the studies conducted by Arnett et al. (1997), who tested the weak-BIS, strong-BAS, and modulation deficit models with both passive- and active-avoidance paradigms. Although there was some evidence in support of all three interpretations, the findings were best explained with recourse to the strong-BAS and poor response modulation approaches: Psychopaths demonstrated exaggerated responding to reward, and this approach activation subsequently interfered with their ability to inhibit responding to punishment cues. As these investigators note, it will be important to more explicitly attend to emotional characteristics, such as deficits in empathy and anxiety (e.g. Levenson, Kiehl, & Fitzpatrick, 1995), in a comprehensive account of psychopathy.

## SCHIZOPHRENIA

Although not typically thought of as an “emotional” disorder *per se*, more recent empirical research in schizophrenia has identified a number of emotional disturbances in persons diagnosed with this disorder. Unlike the disorders reviewed so far, however, the emotional disturbances in schizophrenia have not often been construed within the approach-withdrawal framework. On a more general level, however, Fowles (1992, 1994) has hypothesised that activity in both the Behavioral Inhibition and Behavioral Activation Systems is related to the nonspecific genetic liability for the disorder. Furthermore, Fowles has suggested that activation (and perhaps overactivity) in the BAS may be associated with the positive symptoms of schizophrenia (e.g. hallucinations, delusions), whereas activity in the BIS in conjunction with underactivation of the BAS may be associated with some of the negative symptoms (e.g. withdrawal, lack of spontaneity). Unfortunately, the linkage between these systems and specific emotional disturbances in schizophrenia are not yet well understood.

One of the most salient emotional disturbances among schizophrenic patients appears to be their diminished expression of emotion. Recent studies of the linkage between expressive behaviour and experienced emotion in schizophrenia have confirmed the historical writings of Bleuler (1911/1950) and Kraepelin (1904), who noted that schizophrenic patients seemed to experience a wide range of emotions yet did not often display them outwardly. That is, compared to nonpatients, schizophrenic patients show less observable facial expression despite reporting equally intense amounts of experienced emotion (e.g. Berenbaum & Oltmanns, 1992; Dworkin, Clark, Amador, & Gorman, 1996; Dworkin et al., 1993; Krause, Steimer-Krause, & Hufnagel, 1992; Kring & Earnst, in press; Kring, Kerr, Smith, & Neale, 1993; Kring & Neale, 1996). Importantly, this diminished expressivity does not appear to be a function of neuroleptic medication side-effects (Kring & Earnst, in press), and it is not limited to specific emotions.

Although schizophrenic patients are less expressive than nonpatients, their emotional response profile is not devoid of facial expression. Indeed, compared to nonpatients, schizophrenic patients demonstrate similar or greater micro-expressive facial behaviour (assessed via electromyography; EMG) in response to emotional films (Mattes, Schneider, Heimann, & Birbaumer, 1995), pictures of facial expressions (Kring, Kerr, & Earnst, 1999), and while discussing pleasant and unpleasant events (Mattes et al., 1995). Thus, schizophrenic patients’ overt expressive behaviour is dampened, yet their reports of experienced emotion as well as their EMG responding are similar to nonpatients. This raises the interesting

possibility that schizophrenic patients have a different threshold for producing observable displays. Such a threshold model would predict that patients' expressive behaviour would be observable in response to stimuli of sufficient intensity (Ekman, 1992).

There is reason to believe that at least some schizophrenic patients might also have reductions in experienced emotion, particularly pleasant emotions. Indeed, anhedonia has been posited to be a central feature of schizophrenia by some theorists (e.g. Meehl, 1962; Rado, 1962). Similar to the discussion on depression earlier, Fowles (1992, 1994) has postulated that anhedonia in schizophrenia reflects a disturbance in the Behavioral Activation System. To the extent that positive affect reflects activity in the activation system, schizophrenic patients with anhedonia should manifest decreased positive affect. Consistent with this reasoning, Blanchard, Mueser, and Bellack (1998) found that schizophrenic patients reported greater trait negative affect but less trait positive affect than nonpatients. Moreover, anhedonia was associated with low positive affect and high negative affect in this sample. These findings are also consistent with a recent meta-analysis indicating that schizophrenic patients report increased neuroticism and decreased extraversion relative to controls (Berenbaum & Fujita, 1994).

Another line of investigation on emotion in schizophrenia has found that chronically ill schizophrenic patients do less well on tasks of emotion perception (facial and vocal) than nonpatients (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 generalised deficit in perceiving faces and voices. One recent study of acutely ill schizophrenic patients did not find evidence for emotion perception deficits (e.g. Bellack, Blanchard, & Mueser, 1996), and this result has led some to speculate that antipsychotic medications may be better able to ameliorate perception deficits (including emotion perception) among acutely ill but not more chronically disturbed patients (Mueser et al., 1996). Alternatively, and perhaps more likely, it may be the case that emotion perception deficits (and emotional disturbance more generally) are present in only a subset of patients with schizophrenia. Indeed, the heterogeneity of schizophrenia has led many researchers to hypothesise that schizophrenia is not one disease entity, but rather a compilation of multiple disease entities, each with different aetiologies (e.g. Carpenter, Buchanan, Kirkpatrick, Tamminga, & Wood, 1993).

Consistent with this multiple disease model, Carpenter and colleagues (e.g. Carpenter et al., 1993; Carpenter, Heinrichs, & Wagman, 1988) have argued that schizophrenic patients who have enduring and primary negative symptoms (i.e. deficit symptoms) represent a distinct aetiologic

subtype. Furthermore, these investigators hypothesised that dysfunction involving frontal cortex and limbic structures might account for deficit symptomatology (e.g. Buchanan et al., 1994; Tamminga et al., 1992). Interestingly, the essence of deficit symptoms is based on Kraepelin's (1919/1971, p. 74) notion of an avolitional process, described as "... emotional dullness, failure of mental activities, loss of mastery over volition, of endeavor, and of ability for independent action", and thus not surprisingly a number of the deficit symptoms involve emotional features (e.g. diminished emotional range, curbing of interests, and restricted affect). The presence of these features suggests that these patients might not only exhibit diminished emotional expression, but also diminished emotional experience (Earnst & Kring, 1999). Moreover, the behaviours comprising deficit symptoms also suggest underactivation of the Behavioral Activation System.

Although cognitive deficits are also prominent features of schizophrenia, the linkage between cognitive and emotional factors is not well understood and is an important direction for future research. This linkage can be empirically tested using the startle-probe paradigm which is a relatively nonverbal method for measuring activation of emotion and motivation systems (Lang et al., 1990; Neale, Blanchard, Kerr, Kring, & Smith, 1998).

## CONCLUSION

We have considered the nature of emotional disturbance in diverse psychopathologies, and we have argued that the functions of emotions are comparable in persons with and without psychopathology. However, these functions are not readily achieved among individuals with psychopathology due to impairments in one or more components of emotional processing. For instance, the inability of depressed and schizophrenic patients to accurately perceive facial expressions of emotion in others can be expected to interfere with their capacity to effectively respond to those socioemotional cues.

Recourse to the approach-withdrawal motivation heuristic has several important advantages. One indication of the explanatory breadth of this framework lies in its ability to account for the deficits that are associated with different components of emotional processes within a particular disorder. The utility of the framework is also demonstrated by its ability to account for the emotional and behavioural impairments typical of disorders that have markedly different symptom pictures. Furthermore, this perspective readily incorporates diverse methodologies and levels of analysis, including symptom descriptions, cognitive styles, and psychophysiological responding.



Advancements in both neuroimaging techniques and the ability to alter selectively regulatory activity in particular neurophysiological systems are having an immediate impact on our understanding of various mental disorders. For investigators of psychopathology, some of what is particularly exciting about these advancements is the promise that they hold for our ability to specify the linkages among neuropathology, neuroregulation, emotional processes, and maladaptive behaviour. In our progress towards this goal, the approach-withdrawal framework continues to be a practical unifying tool. Moreover, in part because of its consideration of phylogenetic continuities in motivation-behaviour systems, the motivation system perspective makes it possible to derive testable predictions about disruptions in the achievement of adaptive, motivationally significant goals that occur as a consequence of primary impairments in one or more emotion components. For example, a depression-related disturbance in the prosodic, expressive components of speech can consequently be expected to produce impairments in the ability to provide vocal signals about motivational state to others.

Although we contend that activity in approach and withdrawal systems underlies motivated behaviour, and that disturbances in these systems can produce serious emotional dysfunction, we acknowledge that this framework does not fully account for the range of emotion disturbances in psychopathology. For example, the disjunction among emotion response components in schizophrenia is not readily accounted for by this framework. It is also the case that the pathological experience and expression of particular discrete emotions gives rise to some of the unique features observed within various disorders. Furthermore, the exact ways in which the discrete emotions are derived from or "fall out of" broad motivational systems are not known. This criticism is particularly relevant for anxiety disorders, which involve a number of different negative emotions (Tomarken & Hollon, 1991). However, recent neurophysiological and behavioural data may be the starting place for some definitive answers in this regard. The research conducted by LeDoux (e.g. 1995a,b), who has made significant contributions to an understanding of the neurophysiological pathways involved in fear, and the work of Gray (e.g. 1987, 1995), who has provided similarly influential evidence regarding pathways for anxiety, are exemplars of such research. Perhaps the most productive approach to studying emotional disturbance in psychopathology is one that explicitly considers specific emotions within the motivation system framework.

Another shortcoming of the application of the motivation system framework is that it does not readily accommodate the dynamics of emotion expression in social interaction, which can arguably be considered the "emotional signature" of psychopathology. For example,

interacting with a nonexpressive schizophrenic patient is bound to evoke a wide variety of responses from an interaction partner. Similarly, the failure of a depressed individual to recognise readily positive emotional cues in others further constrains social communication. In short, many of the emotional disturbances evident in psychopathology will evoke emotional responses in others (e.g. Hooley & Teasdale, 1989; Joiner, Alfano, & Metalsky, 1992). These responses, whether they be positive or negative, likely mediate the development and maintenance of social relationships in disordered individuals, and they may also serve to maintain emotional disturbances in psychopathology (Keltner, Moffitt, & Stouthamer-Loeber, 1995).

In summary, emotional processes figure prominently in psychopathology. Yet, surprisingly little empirical research has systematically examined the manner in which disturbed components of emotional processes interfere with adaptive behaviour in individuals with these disorders. Our review of this literature suggests that one foundation for such systematic examinations, the approach and withdrawal motivation system framework, provides a means by which the linkages among emotion and other manifestations of psychopathology can be elucidated.

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