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Anxiety as an Adaptive Emotion

2

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Penetrating the mysteries of anxiety is a task that has challenged scientists and philosophers for centuries (Barlow, 2000; Corr, 2011). For example, in his book *The Concept of Anxiety*, the 19th-century philosopher Søren Kierkegaard portrayed anxiety as the root of humanity, informing us of our options and being central to self-knowledge and individual responsibility (Kierkegaard, 1844/1981). In recent decades, the discovery that anxiety disorders are the most prevalent of all psychiatric illnesses, affecting approximately 14% of the population at any one time (Wittchen, Jacobi, Rehm, Gustavsson, Svensson, Jonsson, et al., 2011), has lent new urgency to efforts aimed at understanding its causes. Although anxiety seems to have many paradoxical features (e.g., it often impairs performance), if Kierkegaard is correct, it is also useful, perhaps even crucial to human life.

Building on Darwin's (1859) hypothesis that our psychological attributes are shaped by natural selection in the same way as our anatomical characteristics, modern theorists have come to favor a functional account of anxiety as a phenomenon that evolved to facilitate avoidance of threat and the assessment of risk (Deakin & Graeff, 1991; Blanchard, Blanchard, & Rodgers, 1991). The purpose of this chapter is to assess the validity of this functional view of anxiety by reviewing findings from three domains of the scientific literature. First, we describe studies from applied psychology that have explored the role of personality traits that reflect individual differences in anxiety proneness in influencing educational and occupational performance. Second, we describe the findings of studies that have attempted to explore a role for anxiety in human defensive reactions. Third, we evaluate a novel and emerging literature that investigates a possible role for anxiety in conscious awareness.

At the end of this chapter, based on our analysis of these three topic areas, we conclude that anxiety, once thought of as wholly negative and requiring "cure," instead evolved to serve useful adaptive, protective functions in humans, as in rodents. As a caveat, we caution that not all forms of human anxiety may be represented in rodents—there may well be truly human *angst*—but sufficient common ground is likely to exist between humans and rodents to make the study of basic, nonabstract, threat-related anxiety highly valuable in both species. Indeed, these more basic forms of anxiety probably form the necessary foundations of true human states of anxiety.

ANXIETY PRONENESS AND APPLIED PERFORMANCE

Study of the effects on applied performance of personality constructs that reflect individual differences in anxiety proneness has a long history in occupational and educational contexts. In correlational studies that examine the performance effects of anxiety proneness in isolation, results usually turn out to be inconclusive, showing either no effect or contradictory effects in which high levels of anxiety proneness appear to harm performance in some applied settings and benefit it in others (Barrick & Mount, 1991; Salgado, 1997). However, in studies that have examined the interaction of anxiety proneness and intelligence in determining performance, a clearer picture emerges. These studies show that a combination of high anxiety proneness and low intelligence typically leads to low performance but that high anxiety proneness combined with high intelligence usually allows performance to reach adequate levels, or even allows it to be boosted beyond the levels attained by equally intelligent but less anxiety-prone individuals. In this regard, there is an apparent strong complementarity between emotional and cognitive processes.

One of the first publications to examine systematically the possibility that anxiety proneness and intelligence combine to influence applied performance was Eliot Slater's (1943) article investigating the psychological makeup of 2,000 soldiers invalided out of the British Army for psychiatric reasons during the first 2 years of World War II. These soldiers were labeled as "neurotic," a personality profile characterized by Slater chiefly as representing proneness to experiencing negative emotions, such as anxiety, hysteria, depression, and hypochondria. In this seminal work, Slater concluded that soldiers who combined the neurotic personality profile with inadequate intelligence were at especially high risk of psychological breakdown compared with soldiers who were neurotic but with adequate intelligence or those with low intelligence but an emotionally stable, nonneurotic personality type. In his 1947 book *The Dimensions of Personality*, also based on research with psychiatric invalids from the British Army, Hans Eysenck extended this work by outlining a hypothetical mechanism for the interaction between anxiety proneness and intelligence: "army training imposes a considerable stress on the dull person, who may find difficulties in understanding and following instructions; this strain may lead to breakdown in persons constitutionally disposed towards neuroticism" (Eysenck, 1947, p. 112).

Understanding of personality has advanced considerably since the 1940s, chiefly by replacing ideas of personality types with the notion that personality is best described by a small number of continua or dimensions, on which each person possesses a score. Consequently, older ideas of the neurotic type have been replaced in most modern personality models with a dimension that reflects proneness to anxiety. Arguments persist about the labeling and precise content of this personality dimension, and it is variously dubbed trait anxiety, neuroticism, or (in reverse) emotional sta*bility*, but regardless of semantics it can be accepted that people scoring at the upper end of the dimension tend, among other manifestations, to ruminate a lot (specifically about negative events: worry) and people at the low end do not. This is not simply cognitive overactivity but defensively oriented cognition (Ormel et al., 2013). This broad consensus has allowed the accumulation of research findings relating this construct to applied performance. For example, Spielberger (1966) found that although students scoring high on anxiety proneness tended, on average, to show lower academic performance than those who scored low on anxiety proneness, performance was not correlated with anxiety proneness in the students in the top 20% in intelligence. Moreover, in the students above the 95th percentile in intelligence, the best performers were those with high scores on anxiety proneness. (For other examples, see Norem, Chapter 11, this volume.)

This pattern of findings has since been replicated with varying degrees of fidelity in other applied settings. For example, Corr and Gray (1995) studied the effects of intelligence and personality on performance in 196 financial services salespeople, revealing that sales success correlated positively with a negative attributional style (an aspect of neuroticism linked to a pessimistic, depressive, self-critical attitude), but only in the more intelligent salespeople—presumably, they were better able to make sense of and use the critical feedback they received from potential customers (e.g., using superior sales strategies to overcome objections). Similarly, Perkins and Corr (2005) investigated the effects of anxiety proneness and intelligence on job performance in 68 managers from a range of functional areas in a global securities company within a larger U.K. financial institution. This study revealed that, in the more intelligent managers, anxiety proneness was positively correlated with performance but that, as intelligence declined across the sample, this relationship disappeared. Mellanby and Zimdars (2011) obtained a partial replication of this interaction in an educational context, finding that, in 383 students, scores on anxiety proneness were higher in students obtaining the highest level of university degree compared with those obtaining the second highest level. However, this result reached statistical significance only in female undergraduates.

Applied studies outside an academic or office environment have further supported the notion that anxiety proneness interacts with intelligence to influence performance but have suggested a slightly different pattern for the interaction under these circumstances. For example, Perkins and Corr (2006a) studied the effects of personality and intelligence on the performance of 669 British military officer candidates as they underwent a 3-day officer-selection assessment. This assessment process is deliberately designed to be highly stressful and physically demanding in an effort to reveal those who could (or could not) cope with the demands of combat leadership. This investigation found that performance ratings were negatively correlated with neuroticism scores in the less intelligent officer candidates, whereas in the more intelligent individuals, neuroticism scores were uncorrelated with performance. Because the officer candidates were volunteers, not conscripts, and required relatively good high school grades in order to attend the assessment, a degree of range restriction with regard to both intelligence and personality would be expected to reduce the effects of this interaction.

In an effort to explore the combined effects of anxiety proneness and intelligence on military performance without problems of range restriction, Leikas, Mäkinen, Lönnqvist, and Verkasalo (2009) conducted a longitudinal study of 152 Finnish Army conscripts during a 1-year-long basic training period. Because these participants had no choice but to join the army, they could be viewed as providing a more representative sample of the human population than that used by Perkins and Corr (2006a). Despite the differences in nationality and psychological makeup of their sample compared with the earlier British study, the analysis by Leikas et al. (2009) revealed a similar pattern of results: An interaction of intelligence and neuroticism predicted adjustment among the conscripts. This interaction showed that lower levels of neuroticism were related to better adjustment to military life (as measured by self-evaluations, superior evaluations, military passport evaluations, and number of sick days), but only among individuals with low intelligence scores. In contrast, in conscripts with high scores on intelligence, neuroticism was unrelated to adjustment.

Viewed together, these latter studies suggest that, at least in military settings, the performance boosting effects of anxiety proneness in highly intelligent individuals that were seen in academic or office jobs have vanished. This pattern of results raises the possibility that the more physically hazardous a job is, the more anxiety free the personalities of good performers must be, regardless of their intelligence. Some evidence exists to support this idea, as people employed in hazardous occupations (police officers, firefighters, electrical engineers, airline pilots, and flight attendants) have been found to be less apprehensive, less tense, less imaginative, and more emotionally stable than people employed in five nonhazardous occupations (janitors, nuns, priests, forepersons, and artists; Cattell, Eber, & Tatsuoka, 1970). In a study of 101 British Army officer cadets, it was found that fear proneness was negatively correlated with combat judgment in simulations of battle situations (Perkins, Kemp & Corr, 2007). Bomb disposal operators are significantly less neurotic than the general population, and the most successful operators are significantly less neurotic than their less successful colleagues (Hallam & Rachman, 1980). Moreover, military pilot applicants as a group score significantly lower on neuroticism than the general population, and those pilot cadets that pass training to become fully fledged combat aviators are even less neurotic than their peers already low in neuroticism (Bartram & Dale, 1982). This latter finding seems to be a product of the additional hazardousness specific to military aviation rather than of flying per se, as civilian amateur pilots are much closer to the general population norms in terms of average neuroticism scores than their military counterparts (Bartram, 1995).

In conclusion, these studies suggest that high levels of anxiety proneness do boost applied performance in intellectually demanding, desk-based activities, but only in relatively intelligent individuals. The more physically hazardous the situation, the more detrimental high levels of anxiety proneness become to applied performance, until a point of extreme dangerousness is reached (as with bomb disposal officer or military pilot roles) at which even high levels of intelligence cannot buffer the detrimental effects of high levels of anxiety proneness. Although the precise causal reason for these divergent patterns of effects has not been tested, one obvious possibility is that in cerebral, desk-based roles, with no element of physical hazard and in which employees have the luxury of making decisions over a period of hours, days, or even weeks, the high levels of rumination that are displayed by highly anxiety-prone individuals may increase the quality of decision making when combined with high intelligence. Additionally, in less frantic and/or dangerous roles, intelligent but highly anxiety-prone individuals may have the time to invent and apply anxiety management strategies similar to those taught in the cognitive-behavioral therapy clinic. This do-it-yourself anxiety management process has been documented in famously successful, intelligent but anxiety-prone individuals such as Sir Winston Churchill (Wilson, 1966; see Perkins & Corr, 2006a).

In actively hazardous jobs, such as piloting a jet fighter plane, in which high-stakes decisions must be made under extreme second-by-second time pressure (e.g., whether to destroy a target that may be friend or foe), the same rumination process is unlikely to be advantageous, as it would slow decision making to the point at which the job cannot be performed adequately. In addition, it is plausible that the time required to apply cognitive strategies that can reduce anxiety is not available when in a cockpit of a fighter jet on active duty.

ANXIETY AND HUMAN DEFENSIVE REACTIONS

The difficulty that highly anxiety-prone individuals, however intelligent they may be, have in performing adequately in highly hazardous jobs, such as bomb disposal or combat flying, suggests circumstantially that anxiety is elicited primarily by threat. Given that personality constructs reflecting individual differences in anxiety proneness have a substantial genetic basis (Bouchard, 1994; Plomin, DeFries, McClearn, & McGuffin, 2008), it would appear that the brain systems mediating anxiety are the products of natural selection and, therefore, that anxiety evolved as a threat response. This idea is widely supported by rodent findings (Blanchard & Blanchard, 2008) but has only begun to be explored scientifically in humans recently using paradigms that index responses to threat.

The origins of these human defense studies lie in rodent experiments that show that drugs with clinical effectiveness against anxiety disorders systematically alter the innate defensive reactions of rodents (e.g., Blanchard, Blanchard, Tom, & Rodgers 1990). The use of threat-naive rodent subjects in these experiments verifies the idea that anxiety is an evolved reaction to threat that serves as a psychological prompt to avoid harm, as it suggests that behavior is innate and not learned (Blanchard & Blanchard, 2008). In the case of humans, this adaptive defensive explanation for anxiety has considerable heuristic promise, as it allows anxietyrelated illness to be viewed as reflecting alterations in defensive brain systems. Similarly, personality traits associated with anxiety proneness can be readily explained as reflecting individual differences in the reactivity of these defensive brain systems. However, there are long-standing concerns that rodent models of psychological processes are too simple to apply in humans (Matthews, 2008): For example, there is no evidence that rodents experience anxiety of an abstract type related to existential issues, whereas historical and literary accounts abundantly point to the existence of such angst in humans. Concerns of this type have raised a need for studies of human defense that can test the validity of the defensive explanation for anxiety in humans.

The first step toward the experimental study of human defense was made using a threat scenario questionnaire (Blanchard, Hynd, Minke, Minemoto, & Blanchard, 2001) in which participants were presented with 12 situations containing different types of threat (modeled on typical animal paradigms) and were asked to choose a response from a list of 10 plausible options (modeled on typical animal defensive reactions). This study showed that human responses to threat are patterned in a similar manner to those of rodents, with ambiguously threatening stimuli, such as suspicious noises, eliciting risk assessment and clear threats, such as the presence of a predator, eliciting more explosive or intense responses (e.g., fight and flight). This finding was replicated and extended by a study examining the effect of human personality on threat responses (Perkins & Corr, 2006b), which revealed that the tendency to select a flight response was positively correlated with scores on the Fear Survey Schedule (FSS; Wolpe & Lang, 1977).

This latter questionnaire was originally created to measure phobic change under therapy but has been recognized subsequently as a useful measure of trait individual differences in sensitivity to aversive or threatening stimuli (e.g., Cook, Davis, Hawk, Spence, & Gautier, 1992). The value of this questionnaire has been demonstrated in a human defense context by a recent study using the threat scenario questionnaire, in which participants were asked to rate each scenario for threat intensity before then selecting which defensive reaction they would deploy in real life (Perkins, Cooper, Abdelall, Smillie, & Corr, 2010). It was found that perceptions of threat intensity mediated the association between FSS scores and the tendency to select flight responses. This result indicates that the reason that high scorers on the FSS tend to select flight responses is that they perceive the threat scenarios as more intensely threatening than do average participants. Because scores on this same questionnaire have already been found to be correlated positively with neuroticism (e.g., Abdel-Khalek, 1988) and negatively with combat judgment in British Army officer cadets (Perkins et al., 2007), it appears that the FSS captures individual differences in responsivity to threat that plausibly make up one facet of human neuroticism.

These findings also touch on an interesting issue in the study of anxiety, namely, how it relates to fear. Correlational studies show that scores on questionnaire measures of fear proneness are typically only modestly correlated with scores on questionnaires measuring anxiety proneness, suggesting that, psychometrically at least, fear proneness and anxiety proneness may be separable (Gray & McNaughton, 2000; Perkins et al., 2007). A distinction between fear and anxiety has also been found in facial expressions: Using an actor-observer paradigm, a facial expression posed in response to ambiguously threatening scenarios (Figure 2.1, image 1) was preferentially labeled by naive observers as representing anxiety, whereas a facial expression posed in response to clear threat scenarios (Figure 2.1, image 6) was preferentially labeled as representing fear (Perkins, Inchley-Mort, Pickering, Corr, & Burgess, 2012). This split of fear and anxiety depending on the clarity of the threat stimulus closely echoes rodent findings and the results of threat scenario studies (Blanchard et al., 2001), suggesting that fear and anxiety are functionally separable phenomena in both rodents and humans.

The nature of these functional differences between facial expressions for anxiety and fear plausibly relates to the difference in information gathering required by a situation that *might* contain a threat versus a situation in which the threat is already apparent. Thus the facial expression of anxiety contains environmental scanning behavior that is likely to aid the localization of an ambiguous threat. Based on this analysis, it is plausible that the anxious facial expression initially evolved by natural selection because it conferred a survival advantage in situations that contained ambiguous or potential threats and only subsequently became a social signal of anxiety. In contrast, it seems plausible that the fixed-gaze facial expression for fear might have evolved by natural selection as a response to situations containing a clear threat because it conferred a survival advantage by allowing the individual displaying it to gather a maximal amount of information about the threat so that an appropriate counterattack or other defensive maneuver can be launched. The fixed pattern of defensive reactions in relation to clear versus ambiguous threats is detailed by Gray and McNaughton (2000).

However, actual threats activate whole-body reactions, not just facial expressions, creating a need for human defense studies that investigate associations between anxiety and avoidance behavior as expressed by integrated bodily action-in this important regard, defensive reactions are "embodied." The systematic measurement of human defensive behavior is acknowledged to be ethically and practically difficult (e.g., Blanchard & Blanchard, 2008); however, some recent studies suggest that these problems can be substantially overcome by the use of computer-based measures of active avoidance behavior that use relatively innocuous, yet unpleasant, threat stimuli. The hand movements used to operate these computer tasks differ physically from archetypal mammalian avoidance behaviors, such as running, but appear to be mediated by the same defensive brain systems owing to their functional equivalence (i.e., both types of behavior serve to reduce threat). For example, Mobbs et al. (2007) examined the effects of threat proximity on brain activity by means of a task in which a cursor was pursued around an onscreen maze by a computer-controlled threat stimulus



FIGURE 2.1. Facial expressions posed in response to emotive scenarios. Images 1 and 6 were posed in response to scenarios describing ambiguous threat and clear threat, respectively. Image 3 was intended to be an expressionless control stimulus. The remaining images were posed in response to scenarios intended to convey happiness (image 2), interest (image 4), surprise (image 5), anger (image 7), sadness (image 8), and disgust (image 9). Image 1 was preferentially labeled as representing anxiety, not fear or any other major emotion. Image 6 was preferentially labeled as representing fear, not anxiety or any other major emotion. From Perkins et al. (2012). Copyright 2012 by the American Psychological Association. Reprinted by permission.

that inflicted a mild but unpleasant electric shock to the participant if it caught up. The participants controlled the cursor by tapping direction keys with their fingers while their brains were scanned by functional magnetic resonance imaging (fMRI). Mobbs et al. (2007) found that, as the threat stimulus neared the cursor (i.e., threat intensity increased), brain activity shifted from the ventromedial prefrontal cortex to the periaqueductal gray. Because this pattern of change in brain activity was predicted on the basis of studies in nonhuman mammals (e.g., Fanselow, 1994), this finding suggests that, in humans, computerized active avoidance tasks engage the same brain systems that govern mammalian defensive behavior.

In order to conduct pharmacological tests in humans of associations

between anxiety and threat avoidance behavior, the joystick-operated runway task (JORT) was created, a computer-based translation of the mouse defense test battery (MDTB; Griebel, Sanger, & Perrault, 1997). The MDTB allows the systematic on-demand elicitation and measurement of defensive behaviors in mice and consists of two straight sections of runway each 2 m long, joined at each end by curved sections and surrounded by walls 0.30 m high (Figure 2.2A). A mouse is placed in the runway and exposed to an anesthetized rat held in the experimenter's hand, with its behavior being video recorded for subsequent scoring (in the wild rats are predators of mice, and so mice respond to the anesthetized rat as if it were a lethal threat; Nikulina, 1991). By fitting or removing a pair of temporary doors, the MDTB can be configured so that the mouse is either trapped in a closed section of straight runway or free to move along an endless runway.

In the closed-runway configuration of the MDTB, forward-backward oscillations are part of risk assessment and are conceptualized as indicating anxiety, and flight behavior in the endless runway configuration is conceptualized as indicating fear (Blanchard, Griebel, & Blanchard, 2003). These hypothetical behavior-emotion associations have been validated by drug studies: Forward-backward oscillations in the closed-runway configuration are preferentially altered by drugs that are clinically effective against generalized anxiety disorder (e.g., Stemmelin et al., 2008). In the endless-runway configuration, flight intensity (i.e., running speed) is preferentially reduced by drugs that are clinically effective against panic disorder, suggesting that this behavior indicates fear (Griebel, Blanchard, Agnes, & Blanchard, 1995). The JORT was specifically designed to retain the dual configuration of the MDTB (Figure 2B, 2C), so that the intensity of responses to threats of different threat situations could be separately measured, rendering it a plausible means of conducting a pharmacological dissection of human defense.

Pharmacological tests of the JORT have not supported the same clean distinction between anxiety and fear that appears to exist in rodents: Contrary to rodent results, the anti-panic drug citalopram exerted no significant effect on flight intensity in 30 human males (Perkins et al., 2009), but in line with rodent results, the same study found that 1 mg of the anti-anxiety drug lorazepam altered forward–backward oscillation during conflict. However, in a more detailed, multidose follow-up study, a higher dose of lorazepam progressively altered flight intensity, as well as oscillation, in a dose-dependent manner during goal conflict (Perkins et al., 2013). The capacity of lorazepam to alter human defensive behavior in two separate studies nevertheless suggests that anxiety and defensiveness are linked in humans and that activity in the brain systems that control perceptions of threat intensity are damped by this anti-anxiety drug. A genetic study of JORT responses provides additional support for a link between anxiety disorders and defense, as a candidate genetic risk factor for panic



FIGURE 2.2. (A) The mouse defense test battery (MDTB). (B, C) The human translation of the MDTB, the joystick-operated runway task (JORT). A force-sensing joystick apparatus (PH-JS1; Psyal, London) controls the speed of a cursor (green dot) in an onscreen runway; the harder the joystick is pushed, the faster the cursor travels. In the one-way active-avoidance phase, this cursor is pursued by a single threat stimulus (red dot; B). Participants receive an unpleasant but harmless 115-dB white noise burst if the red dot collides with the green dot. The two-way active-avoidance phase (C) is identical, except that a second red dot travels ahead of the green dot at a constant velocity, causing a goal conflict whereby the participant has to travel fast enough to avoid the pursuing threat, but not so fast that he or she collides with the leading threat stimulus. From Perkins et al. (2013). Copyright 2013 by Macmillian Publishing Limited. Reprinted by permission.

disorder was found to intensify flight behavior in 200 healthy adult volunteers (Perkins et al., 2011).

The reason for the broader responsivity of human defensive behavior to this anxiety-reducing drug may have to do with the greater cognitive elaboration in human experimental volunteers: In this specific context,

participants know they will receive punishment at some point, and this may be enough to elicit mild anxiety that floods the whole testing session, regardless of the specific stage of the task (Davis, Walker, Miles, & Grillon, 2010). Thus the human defense findings, so far, suggest that the JORT may be viewed as a general measure of threat sensitivity rather than a tool for clean behavioral dissection of fear and anxiety-indeed, in the absence of a high intensity of fear elicited by an unambiguous threat, anxiety may always prevail in typical human experiments. Interestingly, the defense state of rodents modulates the effects of anti-anxiety drugs: When the animal is in a state of mild defensiveness and is already showing risk assessment behavior (such as when exposed to the odor of a predator), antianxiety drugs reduce risk assessment behavior, but when the animal is in a state of severe threat (such as when exposed to a predator), anti-anxiety drugs increase risk assessment behavior (Blanchard et al., 1991). As risk assessment is typically deployed at lower levels of perceived threat intensity than other defensive behaviors, such as flight, freezing, or defensive attack (Blanchard et al., 2003), this pattern of drug effects has been interpreted as suggesting that anti-anxiety drugs cause threats, in general, to appear less intense, moving the animal down the perceived threat intensity gradient that has prethreat behaviors at the bottom, risk assessment in the middle, and more intensely defensive behaviors, such as freezing, fleeing, or fighting, at the top (Blanchard et al., 2003). This analysis gives rise to the construct of perceived "defensive distance" (Blanchard et al., 1990). Different states of threat in rodents have been likened to personality differences in humans—a drugged rat being portrayed as analogous to a human with an anxiety-resistant personality (McNaughton & Corr, 2004).

ANXIETY IN CONSCIOUS AWARENESS

We can learn much about the adaptive value of anxiety by examining its pathological expression. This is especially true if we assume that anxiety has an evolutionary function. However, one of the main, but by no means exclusive, features of anxiety is its subjective nature—its experiential angst as represented in conscious awareness. Indeed, we could not say that someone was anxious unless he or she made a verbal complaint of it. In an attempt to explore the subjective experience of anxiety, Corr (2011) proposed a conceptual model of the different neural–behavioral levels of control seen in anxiety. This model sought to answer two questions: (1) What is the content of subjective awareness? (2) What might be its functions? It was noted that people with anxiety report ruminating about, specifically, bad events; their focus of attention is on possible bad outcomes, and they find themselves easily distracted. These features are often said to characterize the person with anxiety as someone who is hypervigilant for threat with a negative cognitive bias. Though the target for treatment in patients with anxiety, these features highlight some of the positive aspects of anxiety, which, especially in nonpathological states, may be adaptive.

Corr's (2011) model, which builds on a more general model of consciousness by Corr (2010), argues that all behaviors (and thoughts, feelings, etc.) are automatically organized and executed without the immediate control of consciousness, which simply takes too long to be generated by the brain to have control over the events it represents. When everything is "going to plan," then we are not generally aware of events; it is only at critical junctures that psychological events enter conscious awareness, and these events tend to be ones that involve error, usually in the form of actual states of the world departing from expected states. For example, while driving a car we may brake suddenly and only then realize why this happened—that is, we are conscious of the error only after it has occurred and only after the brain has automatically organized the appropriate response. Corr's (2011) model assumes that stimuli associated with error signals enter conscious awareness and that they are replayed there for detailed analysis; and, after this analysis, the automatic neural-behavioral machinery that controls behavior at any given moment can be adjusted so that future behavior is more appropriate when the same set of stimuli, which led to the error signal, are encountered again. By this means, we learn from experience. This model is shown in Figure 2.3.

The model is built on an elaboration of the behavioral inhibition system (BIS; Gray & McNaughton, 2000). The BIS is responsible for the resolution of goal conflict in general (e.g., between approach and avoidance, as in foraging situations). Once activated, it generates the "watch out for danger" emotion of anxiety, which entails the inhibition of prepotent conflicting behaviors, the engagement of risk assessment processes, and the scanning of memory and the environment to help resolve concurrent goal conflict. The BIS resolves conflicts by increasing, by recursive loops, the negative valence of stimuli until behavioral resolution occurs in favor of approach or avoidance. Subjectively, this state is experienced as worry and rumination. The person with the associated personality factor is worry prone and anxious constantly on the lookout for possible signs of danger, a state that clinically maps onto such conditions as generalized anxiety, depression, and obsessive-compulsive disorder (OCD). In everyday life, we are comparing the actual state of the world against the predicted state, in which we are crossing a busy road, preparing to speak to someone, or simply walking down the street; thus the opportunities for detecting conflicts between goals, and thus the generation of related anxiety, are numerous.

This extended BIS model assumes that anxiety results from the



FIGURE 2.3. Information-processing diagram of the functioning of the behavioral inhibition system (BIS) in automatic and controlled modes, containing basic reward–approach and punishment–avoidance factors. Behavioral plans (Plans) lead to predictions (Prediction Generator; path 1) of future states of the world; the Prediction Generator receives input from path 2a and sends output to path 2b, stored previous experience (Memory). The BIS (Goal-State Comparator) receives input from the Prediction Generator (path 3) and then compares the response-reinforcement outcomes (World: Actual State) with predictions (path 4). Then one of two things happens: (1) "everything is going to plan," and the BIS Goal-State Comparator sends input to the Prediction Generator to continue the motor program ("just checking" mode; path 5); or (2) the BIS Goal-State Generator detects a mismatch between prediction and outcome and generates an error signal (path 6), which leads to activation of the BIS and controlled processes.

Once the BIS is activated, there is inhibition of the reward–approach system (path 7a) and the punishment–avoidance system (path 7b); and at this time the BIS initiates cautious behavior and risk assessment, which then inform Plans (path 8), which simultaneously receives input about current states from the Reward–Approach and Punishment–Avoidance systems (paths 9a, 9b), as well as input about the nature of the conflict from the BIS Goal-State Comparator (path 10). Plans initiate appropriate behavior, and the cycle is repeated until behavioral resolution is achieved in the form of avoidance or approach. To illustrate in relation to BIS and anxiety, bold lines highlight possible sources of dysfunction seen in anxiety states/ conditions, which may include: (1) inappropriate plans (path 1); (2) inadequate prediction generation (path 3); (3) inappropriate retrieval/storage of information from/to memory (paths 2a, 2b); or (4) overactive BIS comparator (path 6).

detection of goal conflict, often involving aversive stimuli, which attract conscious, attentional, and controlled cognitive processing. In everyday life, this is highly valuable, but when extreme it leads to clinical anxiety. In the normal course of the day, the BIS works effortlessly and resolves conflicts without engaging conscious awareness; but where tried-and-tested strategies do not work, the full toolkit of cognitive analysis is brought to bear on the problem, and anxiety-related outputs are experienced. These processes might account for the interaction of anxiety and cognitive ability: A low capacity to analyze the stimuli associated with the generation of error signals will be less likely to resolve them, and future goal conflicts may result. In contrast, the more cognitively able individual has a larger cognitive toolkit and, therefore, is better equipped to resolve the goal-conflict problem.

Corr's (2011) model draws attention to the functional nature of anxiety processes, which allow for immediate and fast defensive responses, as well as delayed and slow deliberative processes designed to allow learning to occur that affects the next iteration of defensive behavior. In everyday life, the effects of these processes go largely unnoticed; however, when they are amplified by either external threat and/or heightened sensitivity of the system, then they are labeled problematic and, often, pathological.

CONCLUSION

Although it has long been suspected that anxiety is more than just a symptom of illness (Kierkegaard, 1844/1981), it is only relatively recently that substantial amounts of empirical data have accrued to show that anxiety has a positive, adaptive side, to do with keeping one safe. In this chapter, we have reviewed relevant data on three themes: the occupational value of anxiety, the defensive value of anxiety, and the value of anxiety as part of conscious awareness. All of these themes are unified by a further theme that runs through all of biology, namely evolution by natural selection (Darwin, 1859). By viewing anxiety in the context of natural selection, it can be seen that the subjective unpleasantness of anxiety is no barrier to its preservation, which acts on any attribute, however unpleasant, that aids survival and reproduction.

REFERENCES

- Abdel-Khalek, A. M. (1988). The Fear Survey Schedule III and its correlation with personality in Egyptian samples. *Journal of Behavior Therapy and Experimental Psychiatry*, 19, 113–118.
- Barlow, D. H. (2000). Unraveling the mysteries of anxiety and its disorders from the perspective of emotion theory. *American Psychologist*, 55, 1247–1263.

- Barrick, M. R., & Mount, M. K. (1991). The Big Five personality dimensions and job performance: A meta-analysis. *Personnel Psychology*, 44, 1–26.
- Bartram, D. (1995). The predictive validity of the EPI and 16PF for military flying training. Journal of Occupational and Organizational Psychology, 68, 229–236.
- Bartram, D., & Dale, H. C. A. (1982). The Eysenck Personality Inventory as a selection test for military pilots. *Journal of Occupational Psychology*, 55, 287–296.
- Blanchard, D. C., & Blanchard, R. J. (2008). Defensive behaviors, fear and anxiety. In R. J. Blanchard, D. C. Blanchard, G. Griebel, & D. Nutt (Eds.), *Hand*book of anxiety and fear (Vol. 17, pp. 63–79). Amsterdam: Academic Press.
- Blanchard, D. C., Blanchard, R. J., & Rodgers, R. J. (1991). Risk assessment and animal models of anxiety. In B. Olivier, J. Mos, & J. L. Slangen (Eds.), Animal models in psychopharmacology (pp. 117–134). Basel: Birkhauser.
- Blanchard, D. C., Blanchard, R. J., Tom, P., & Rodgers, R. J. (1990). Diazepam changes risk assessment in an anxiety/defense test battery. *Psychopharmacol*ogy, 101, 511-518.
- Blanchard, D. C., Griebel, G., & Blanchard, R. J. (2003). The mouse defense test battery: Pharmacological and behavioral assays for anxiety and panic. *European Journal of Pharmacology*, 463, 97–116.
- Blanchard, D. C., Hynd, A. L., Minke, K. A., Minemoto, T., & Blanchard, R. J. (2001). Human defensive behaviors to threat scenarios show parallels to fearand anxiety-related defense patterns of non-human mammals. *Neuroscience* and Biobehavioral Reviews, 25, 761–770.
- Bouchard, T. J. (1994). Genes, environment, and personality. *Science*, 264, 1700–1701.
- Cattell, R. B., Eber, H. W., & Tatsuoka, M. M. (1970). Handbook for the Sixteen Personality Factor Questionnaire (16PF). Champaign, IL: Institute for Personality and Ability Testing.
- Cook, E. W., Davis, T. L., Hawk, L. W., Spence, E. L., & Gautier, C. H. (1992). Fearfulness and startle potentiation during aversive visual stimuli. *Psychophysiology*, 29, 633-645.
- Corr, P. J. (2010). Automatic and controlled processes in behavioral control: Implications for personality psychology. *European Journal of Personality*, 24, 376–403.
- Corr, P. J. (2011). Anxiety: Splitting the phenomenological atom. *Personality and Individual Differences*, 50, 889–897.
- Corr, P. J., & Gray, J. A. (1995). Attributional style, socialization and cognitive ability as predictors of sales success: A predictive validity study. *Personality and Individual Differences*, 18, 241–252.
- Darwin, C. (1859). The origin of species by means of natural selection. London: Murray.
- Davis, M., Walker, D. L., Miles, L., & Grillon, C. (2010). Phasic vs. sustained fear in rats and humans: Role of the extended amygdala in fear vs. anxiety. *Neuropsychopharmacology*, 35, 105–135
- Deakin, J. F. W., & Graeff, F. G. (1991). 5HT and mechanisms of defense. *Journal* of *Psychopharmacology*, *5*, 305–315.

- Eysenck, H. J. (1947). *The dimensions of personality*. London: Kegan Paul, Trench & Trubner.
- Fanselow, M. S. (1994). Neural organization of the defensive behavior system responsible for fear. *Psychonomic Bulletin and Review*, 1, 429-438.
- Gray, J. A., & McNaughton, N. (2000). The neuropsychology of anxiety: An enquiry into the functions of the septohippocampal system (2nd ed.). Oxford, UK: Oxford University Press.
- Griebel, G., Blanchard, D. C., Agnes, R., & Blanchard, R. J. (1995). Differential modulation of antipredator defensive behavior in Swiss–Webster mice following acute and chronic treatment with imipramine and fluoxetine. *Psychopharmacology*, 120, 57–66.
- Griebel, G., Sanger, D. J., & Perrault, G. (1997). Genetic differences in the mouse defense test battery. *Aggressive Behavior*, 23, 19–31.
- Hallam, R. S., & Rachman, S. J. (1980). Courageous acts or courageous actors? *Personality and Individual Differences*, 1, 341–346.
- Kierkegaard, S. (1981). *The concept of anxiety*. Princeton, NJ: Princeton University Press. (Original work published 1844)
- Leikas, S. S., Mäkinen, S., Lönnqvist, J.-E., & Verkasalo, M. (2009). Cognitive ability × emotional stability interactions on adjustment. *European Journal of Personality*, 23, 329–342.
- Matthews, G. (2008). Reinforcement sensitivity theory: A critique from cognitive science. In P. J. Corr (Ed.), *The reinforcement sensitivity theory of personality* (pp. 482–507). Cambridge, UK: Cambridge University Press.
- McNaughton, N., & Corr, P. J. (2004). A two-dimensional neuropsychology of defense: Fear/anxiety and defensive distance. *Neuroscience and Biobehavioral Reviews*, 28, 285–305.
- Mellanby, J., & Zimdars, A. (2011). Trait anxiety and degree performance. *Higher Education*, 61, 357–370.
- Mobbs, D., Petrovic, P., Marchant, J., Hassabis, D., Weiskopf, N., Seymour, B., et al. (2007). When fear is near: Threat imminence elicits prefrontal–periaqueductal gray shifts in humans. *Science*, *317*, 1079–1083.
- Nikulina, E. M. (1991). Neural control of predatory aggression in wild and domesticated animals. *Neuroscience and Biobehavioral Reviews*, 15, 545–547.
- Ormel, J., Bastiaansen, A., Riese, H., Bos, E. H., Servaas, M., Ellenbogen, M., et al. (2013). The biological and psychological basis of neuroticism: Current status and future directions. *Neuroscience and Biobehavioral Reviews*, 37, 59–72.
- Perkins, A. M., Cooper, A., Abdelall, M., Smillie, L. D., & Corr, P. J. (2010). Personality and defensive reactions: Fear, trait anxiety and threat magnification. *Journal of Personality*, 78, 1071–1090.
- Perkins, A. M., & Corr, P. J. (2005). Can worriers be winners?: The association between worrying and job performance. *Personality and Individual Differences*, 38, 25–31.
- Perkins, A. M., & Corr, P. J. (2006). Cognitive ability as a buffer to neuroticism: Churchill's secret weapon? *Personality and Individual Differences*, 40, 39–51.
- Perkins, A. M., Ettinger, U., Davis, R., Foster, R., Williams, S. C. R., & Corr, P. J. (2009). Effects of lorazepam and citalopram on human defensive reactions:

Ethopharmacological differentiation of fear and anxiety. Journal of Neuroscience, 29, 12617–12624.

- Perkins, A. M., Ettinger, U., Weaver, K., Schmechtig, A., Schrantee, A., Morrison, P. D., et al. (2013, April 16). Advancing the defensive explanation for anxiety disorders: Lorazepam effects on human defense are systematically modulated by personality and threat-type. *Translational Psychiatry*, *3*, e246 (online).
- Perkins, A. M., Ettinger, U., Williams, S. C. R., Reuter, M., Hennig, J., & Corr, P. J. (2011). Flight behavior in humans is intensified by a candidate genetic risk factor for panic disorder: Evidence from a translational model of fear and anxiety. *Molecular Psychiatry*, 16, 242–244.
- Perkins, A. M., Inchley-Mort, S. L., Pickering, A. D., Corr, P. J., & Burgess, A. P. (2012). A facial expression for anxiety. *Journal of Personality and Social Psychology*, 102, 910–924.
- Perkins, A. M., Kemp, S. E., & Corr, P. J. (2007). Fear and anxiety as separable emotions: An investigation of the revised reinforcement sensitivity theory of personality. *Emotion*, 7, 252–261.
- Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral* genetics (5th ed.). New York: Worth.
- Salgado, J. F. (1997). The five-factor model of personality and job performance in the European community. *Journal of Applied Psychology*, 82, 30–43.
- Slater, E. (1943). The neurotic constitution: A statistical study of two thousand neurotic soldiers. *Journal of Neurology and Psychiatry*, 6, 1–16.
- Spielberger, C. D. (1966). Theory and research on anxiety. In C. D. Spielberger (Ed.), *Anxiety and behavior* (pp. 3–19). New York: Academic Press.
- Stemmelin, J., Cohen, C., Terranova, J. P., Lopez-Grancha, M., Pichat, P., Bergis, O., et al. (2008). Stimulation of the b3-adrenoceptor as a novel treatment strategy for anxiety and depressive disorders. *Neuropsychopharmacology*, 33, 574-587.
- Wilson, C. M. (Lord Moran). (1966). Winston Churchill: The struggle for survival 1940–1965. London: Constable.
- Wittchen H. U., Jacobi, F., Rehm, J., Gustavsson, A., Svensson, M., Jonsson, B., et al (2011). The size and burden of mental disorders and other disorders of the brain in Europe 2010. European Neuropsychopharmacology, 21, 655–679.
- Wolpe, J., & Lang, P. J. (1977). *Manual for the Fear Survey Schedule*. San Diego, CA: Educational and Industrial Testing Service.

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