
A Contemporary Learning Theory Perspective on the Etiology of Anxiety Disorders

It's Not What You Thought It Was

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The authors describe how contemporary learning theory and research provide the basis for perspectives on the etiology and maintenance of anxiety disorders that capture the complexity associated with individual differences in the development and course of these disorders. These insights from modern research on learning overcome the shortcomings of earlier overly simplistic behavioral approaches, which sometimes have been justifiably criticized. The authors show how considerations of early learning histories and temperamental vulnerabilities affect the short- and long-term outcomes of experiences with stressful events. They also demonstrate how contextual variables during and following stressful learning events affect the course of anxiety disorder symptoms once they develop. This range of variables can lead to a rich and nuanced understanding of the etiology and course of anxiety disorders.

Keywords: anxiety disorders, etiology, maintenance, learning theory

Approximately 29% of the U.S. population is estimated to have or to have had one or more diagnosable anxiety disorders at some point in their lives, making anxiety disorders the most common category of diagnoses in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV;* American Psychiatric Association, 1994; see Kessler, Berglund, Demler, Jin, & Walters, 2005). Most of us might intuitively guess that anxiety disorders would typically develop during or following a frightening or traumatic event or during a period of significant stress when many of us experience some anxiety. Yet it is also obvious that not everyone undergoing traumas or highly stressful periods develops an anxiety disorder. Any good etiological theory must be able to account for this and many other apparent mysteries involved in who does and who does not develop an anxiety disorder. Consider the following examples, which illustrate the kinds of issues we address in this article:

Emily and Marian both had had traumatic experiences with dogs. Emily was hiking with her own dog when another dog attacked her and bit her on the wrist. She was terrified. The wound became badly infected and very painful, requiring medical treatment. Marian was walking in the fields when she became terrified by

three large, growling dogs that chased her to a fence. One began tearing at her pant legs, but their owner fortunately intervened before she was physically injured. Why did Marian but not Emily go on to develop dog phobia when only Emily was actually bitten by a dog?¹

Ahmet and Hasan were both male Turkish citizens in their 30s who were arrested, imprisoned, and tortured. Ahmet was imprisoned for several years and experienced a great deal of torture; Hasan was imprisoned for several weeks and experienced far less torture (M. Başoğlu, personal communication, 1997). Why did Hasan but not Ahmet develop posttraumatic stress disorder? What accounts for such individual differences?

Although behavioral/learning approaches were the dominant empirical perspective on anxiety disorders from the 1920s until the 1970s, these approaches have been widely criticized since the early 1970s (for reviews, see Mineka, 1985; Rachman, 1978, 1990). Many of these criticisms stemmed from the inability of early learning approaches to account for the diverse factors involved in the origins of people's anxieties, such as the individual differences illustrated in the cases cited above. More recently, however, a resurgence of interest in learning approaches has occurred, as these approaches have incorporated some of the complexity predicted by contemporary learning theory and research (e.g., Barlow, 1988, 2002; Craske, 1999).

In this article we discuss how prior criticisms of earlier learning approaches are addressed by contemporary approaches grounded in modern research on learning (e.g., see Rescorla, 1988, for one classic review).² Unfortunately, however, these advances have not been adequately com-

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¹ Unless otherwise noted, all cases presented in this article are slight adaptations of cases that the authors either have been acquainted with personally or have become familiar with during supervised treatment of these individuals by student therapists.

² The reader may note that the subtitle of our article ("It's Not What You Thought It Was") is similar to the subtitle used in Rescorla's (1988) *American Psychologist* article.



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municated to clinical scientists who advance the theories of etiology and maintenance. Therefore, we attempt to advance such communication by describing how certain insights from this area of research overcome the shortcomings of earlier, overly simplistic behavioral approaches. We show how early learning histories serve as vulnerability (or invulnerability) factors that can dramatically affect the emotional consequences of traumatic and stressful life events often implicated in the origins of anxiety disorders. Such early learning histories, when considered together with temperamental vulnerabilities, can serve as diatheses that make certain individuals more susceptible to adverse and stressful experiences that sometimes lead to the development of anxiety disorders. Consideration of these diatheses, as well as of various contextual variables during and following traumatic learning events, can lead to a rich and nuanced understanding of the etiology and course of anxiety disorders (Mineka & Zinbarg, 1996).

Overall, then, we believe that a contemporary learning theory approach can account for such complexity at least as well as the other two prominent contemporary psychological approaches to anxiety disorders—that is, the cognitive (e.g., Beck & Emery, 1985) and the psychodynamic (e.g., Michels, Frances, & Shear, 1985) approaches. Moreover, we believe that these new contemporary learning approaches have several advantages over the other two approaches: The learning approaches are better grounded in the theories and methods of experimental psychology, they provide more comprehensive formulations of the etiology of anxiety disorders, and they provide a more explicit analysis of factors promoting or inhibiting the development of different anxiety disorders. In this article we summarize some of the available research illustrating these points for each of the six primary anxiety disorders.^{3,4}

Specific Phobia

Individuals with specific phobias show intense and irrational fears of certain objects or situations that they usually go to great lengths to avoid. Watson and Rayner (1920) originally argued that phobias are simply intense classically conditioned fears that develop when a neutral stimulus is paired with a traumatic event, such as occurred when their Little Albert acquired an intense fear of rats after hearing a frightening gong paired with the presence of a rat several times. Indeed, a large number of studies using retrospective recall, although fraught with interpretive pitfalls (Mineka & Öhman, 2002a; Mineka & Sutton, in press), have confirmed that many people with phobias can recall a traumatic conditioning event when their phobia began (e.g., Öst & Hugdahl, 1981; see Muris & Mercklebach, 2001, for a review). This straightforward view of phobia acquisition was later criticized for several reasons we will discuss. However, each of the cited problems with such views generally reflects some ignorance of a wide variety of vulnerability and invulnerability factors that are predicted by contemporary learning theory to strongly influence who develops phobias among all those who have had the seemingly requisite conditioning experiences. For example, consider Marian and Emily, the two teenage girls described at the outset who were both attacked by dogs, yet only Marian developed dog phobia. Marian was a bit shy and timid and had had minimal experience with dogs before having been chased and cornered by three dogs. Emily, by contrast, was not shy and had had a lot of experience with dogs prior to having been badly bitten. It thus makes more intuitive sense that only Marian developed dog phobia, but why exactly?

Vicarious Conditioning of Fears and Phobias

One criticism of early conditioning approaches centered on the observation that many people with phobias do not appear to have had any relevant history of classical conditioning (Rachman, 1978, 1990). How can one account for the origins of fears and phobias in these individuals? Clinicians have long speculated that simply observing others experiencing a trauma or behaving fearfully could be sufficient for some phobias to develop. The results of some retrospective studies are consistent with this idea (e.g., Muris & Mercklebach, 2001; Öst & Hugdahl, 1981). One such case involved a boy who had witnessed his grand-

³ Because of space constraints, our reviews are less thorough for generalized anxiety disorder and obsessive-compulsive disorder.

⁴ A significant amount of the research that we review is based on animal studies. We recognize the long-standing controversies about the relevance of animal research to understanding human emotional disorders. We and many others have reviewed these issues extensively elsewhere (e.g., Abramson & Seligman, 1977; McKinney, 1974; Mineka, 1985, 2002; Mineka & Zinbarg, 1991; Pavlov, 1927). We and many (but not all) others have concluded that there are far more advantages than disadvantages in relying on animal research to advance knowledge about etiology, treatment, and prevention, as long as careful attention is paid to documenting compelling similarities as well as differences between the animal and the human observations and findings.



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father vomit while dying; shortly thereafter the boy developed a strong and persistent vomiting phobia. Indeed, in middle age he even contemplated suicide one time when he was nauseous and feared vomiting.

The strongest experimental evidence for the role of vicarious conditioning in phobia acquisition stems from a primate model showing that strong and persistent phobic-like fears can indeed be learned rapidly through observation alone. In Mineka and Cook's series of experiments on observational conditioning of snake fear (e.g., Cook, Mineka, Wolkenstein, & Laitsch, 1985), laboratory-reared young adult rhesus monkeys who initially were not afraid of snakes served as observers who watched unrelated wild-reared model monkeys reacting very fearfully in the presence of live and toy snakes. These lab-reared observer monkeys showed rapid acquisition of an intense phobic-like fear of snakes that did not diminish over a three-month follow-up period. This vicarious conditioning also occurred simply through watching videotapes of models behaving fearfully (Cook & Mineka, 1990), suggesting that humans are also susceptible to acquiring fears vicariously simply through watching movies and TV (as expected from numerous anecdotal observations). Thus, traditional conditioning models erred in not attending to the important role that vicarious conditioning can play in the origins of phobic fears.

Sources of Individual Differences in the Acquisition of Fears and Phobias

Another problem with traditional views is how to explain why many individuals who do undergo traumatic experiences do not develop phobias (e.g., Mineka & Sutton, in press; Mineka & Zinbarg, 1996; Rachman, 1990). For example, in studies using retrospective recall, many non-

phobic individuals report having had traumatic experiences in the presence of some potentially phobic object without having acquired a fear or phobia (e.g., Poulton & Menzies, 2002). From our diathesis-stress perspective, such findings are actually expected. First, there seems to be a modest genetically based vulnerability for phobias (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1992; Kendler et al., 1995). This genetic vulnerability may well be mediated through genetic contributions to fear conditioning (Hettema, Annas, Neale, Kendler, & Fredrikson, 2003), which may in turn be mediated through personality variables such as high trait anxiety that also seem to serve as vulnerability factors, affecting the speed and strength of conditioning (e.g., Levey & Martin, 1981). Moreover, children categorized as behaviorally inhibited (excessively timid, shy, etc.) at 21 months of age have been found to be at higher risk for the development of multiple specific phobias (an average of three to four per child) by seven to eight years of age than were uninhibited children (32% vs. 5%, respectively) (e.g., Biederman et al., 1990). In our example at the outset, Marian was at higher risk than Emily for developing phobias partially because of her timidity.

Differences in life experiences among individuals can also strongly affect the outcome of conditioning experiences. Such experiential factors may serve as vulnerability (or invulnerability) factors for the development of phobias. The relevant differences in life experiences may occur before, during, or following a fear-conditioning experience, and they can act singly or in combination to affect how much fear is experienced, acquired, or maintained over time (e.g., Forsyth & Eifert, 1996a, p. 90; Mineka, 1985; Mineka & Zinbarg, 1996). This point was not appreciated by proponents of earlier conditioning models because they extrapolated directly from the results of experiments on naive animals studied in isolated conditioning chambers to how conditioning experiences might affect humans in their everyday lives. But humans are neither "naive" blank slates nor isolated from the world during conditioning events.

Impact of prior experiences. A phenomenon known as latent inhibition demonstrates that simple prior exposure to a conditioned stimulus (CS) before the conditioned and unconditioned stimulus (US) are ever paired together reduces the amount of subsequent conditioning to the CS when paired with the US (e.g., Lubow, 1998). Consistent with this, several studies have shown that children who have had more previous nontraumatic encounters with a dentist are less likely to develop dental anxiety if subsequently traumatized at the dentist's office than are those with fewer prior encounters when they are traumatized (e.g., Kent, 1997). Moreover, Mineka and Cook (1986) also showed that most monkeys who had initially simply watched a nonfearful model monkey behaving nonfearfully with snakes were completely immunized against acquiring a fear of snakes when subsequently exposed to fearful monkeys behaving fearfully with snakes. By analogy, if a child has extensive preexposure to a nonfearful parent behaving nonfearfully with the phobic object or situation (e.g., heights) of the other parent, this may immunize the child against the later effects of seeing the

phobic parent behaving fearfully with heights. Using Marian and Emily as examples, we note that Emily's own extensive prior exposure to dogs probably immunized her from developing dog phobia.

A person's history of control over important aspects of his or her environment is another important experiential variable strongly affecting reactions to frightening situations. For example, Mineka, Gunnar, and Champoux (1986) found that infant monkeys reared with a sense of mastery and control over their environments for 7–10 months later adapted more readily to frightening events and novel anxiety-provoking situations than did monkeys reared in identical environments except without the experiences with control. This experimental evidence supports claims long made by developmentalists using correlational data that infants and children raised in environments in which they gain a sense of control over their environment are less frightened by (and better able to cope with) novel and frightening events. Such research suggests that children reared with a stronger sense of mastery over their environments should be more invulnerable to developing phobias following traumatic experiences (see also Chorpita & Barlow, 1998).

Impact of contextual variables during conditioning. Several different features of conditioning events themselves also have a strong impact on how much fear is acquired. For example, having control over a traumatic event (such as being able to escape it) has a major impact on how much fear is conditioned to CSs paired with that trauma. Far less fear is conditioned when the aversive event is escapable than when it is inescapable (e.g., Mineka, Cook, & Miller, 1984). Thus, consider our example of Marian, who was not able to end the dog attack herself. Instead, the dog's owner pulled the dog off her. Our perspective suggests that if she had instead been able to escape on her own at the same point during the attack that the dog owner pulled the dog off her, she would have been less likely to have developed dog phobia, even though the dog attack would have been of equal duration and intensity.

Impact of postevent variables. Different kinds of experiences that people can have following conditioning also affect the strength of the conditioned fear that is maintained over time. For example, animal and human research suggests that a person who is exposed to a more intense traumatic experience (not paired with the CS) after conditioning of a mild fear is likely to show an increase in fear of the CS (e.g., Rescorla, 1974; White & Davey, 1989). This so-called inflation effect suggests that a person who acquired a mild fear of automobiles following a minor crash might develop full-blown driving phobia if he or she were later physically assaulted even though no automobile was present during the assault (Mineka, 1985; Mineka & Zinbarg, 1996). Davey (1997) has also described a US reevaluation process that can occur when a person receives verbally or socially transmitted information about the US being more dangerous than when she or he originally experienced it paired with the US. This can result in an inflated level of fear to the CS. Finally, Davey and

Matchett (1994) also found that simple mental rehearsal of CS-US relationships can lead to enhanced strength of the conditioned fear response.

In sum, a number of factors occurring before, during, and following potentially traumatic conditioning experiences (direct or vicarious) affect the strength of fear that is experienced, conditioned, and maintained over time. These examples of many individual differences in vulnerability (and invulnerability), as well as various contextual factors during and after noxious events, go a long way toward addressing the limitations of earlier conditioning models.

Selective Associations in the Conditioning of Fears and Phobias

Early conditioning models predicted that fears and phobias would occur to any random group of objects associated with trauma. However, clinical observations show that people are much more likely to have phobias of snakes, water, heights, and enclosed spaces than of bicycles, guns, or cars, even though today the latter objects (not present in our early evolutionary history) may be at least as likely to be associated with trauma. Seligman (1971) and Öhman (e.g., Öhman, Dimberg, & Öst, 1985) argued that primates may be evolutionarily prepared to rapidly associate certain kinds of objects (such as snakes, spiders, water, heights) with aversive events. This is because there may have been a selective advantage in the course of evolution for primates who rapidly acquired fears of certain objects or situations that posed threats to humans' early ancestors (see also Cook & Mineka, 1990). Consistent with this, several studies have shown that the content of most phobias is rated by independent raters as "prepared" in the sense that it was probably dangerous to pretechnological humans (e.g., see Öhman & Mineka, 2001).

Thus, prepared fears are not seen as inborn or innate but rather as very easily acquired and/or especially resistant to extinction. Öhman and his colleagues conducted an important series of human conditioning experiments supporting this theory. Using slides of snakes and spiders as fear-relevant CSs and mild shock as the US, they have consistently found superior conditioning compared with what is found when more fear-irrelevant CSs, such as slides of flowers, mushrooms, or electric outlets, are used (e.g., Öhman et al., 1985; Öhman & Mineka, 2001). More recent studies have also shown that with fear-relevant CSs (but not with fear-irrelevant CSs), conditioning can even occur when subliminal CSs are used (i.e., CSs that cannot be consciously identified; e.g., Öhman & Soares, 1998). Other research shows that fear learning with fear-relevant stimuli is more impenetrable to conscious cognitive control than is fear learning with fear-irrelevant stimuli (see Mineka & Öhman, 2002b). Such results may help explain the irrationality of phobias. If the fears of individuals with phobias arise automatically from cognitive structures not under the control of conscious intentions, the fears will remain unaffected by rational analysis (Öhman & Soares, 1993).

In addition, Cook and Mineka's experiments (e.g., 1989, 1990) using videotaped model monkeys showed that naive monkeys can easily acquire fears of such fear-rele-

vant stimuli as a toy snake or a toy crocodile but not of such fear-irrelevant stimuli as flowers or a toy rabbit. It is important to note that these lab-reared monkeys had no prior exposure to any of the fear-relevant or fear-irrelevant stimuli before participating in these experiments. Thus, the monkey experiments more strongly support the role of evolutionary factors in the biased associations seen with preparedness effects because there is no possibility that the monkeys had preexisting negative associations to snakes, as may be the case in human experiments on this topic (see Mineka & Öhman, 2002b).

In both monkeys and humans, therefore, evolutionary fear-relevant stimuli more readily enter into selective associations with aversive events, and these same stimuli seem more likely than others to become the objects of human phobias. Moreover, the special characteristics of fear learning seen with fear-relevant (but not fear-irrelevant) stimuli (e.g., its automaticity and its resistance to higher cognitive control) suggest that the acquisition of phobias involves a primitive basic emotional level of learning that humans share with many other mammalian species (Öhman & Mineka, 2001).

In summary, the origins and maintenance of fears and phobias are considerably more complex than was assumed by traditional conditioning models. Nevertheless, these complexities are expected from the standpoint of contemporary research on conditioning, which reveals a large variety of vulnerability, invulnerability, and contextual variables that have a strong impact on the outcome of direct and observational conditioning experiences. Moreover, evolutionary pressures seem to have affected the kinds of objects and situations that humans are most prone to learn to fear.

Social Phobia

Individuals with social phobias show excessive fears of situations in which they might be evaluated or judged by others, and they either avoid such situations or endure them with marked distress. Several theorists have suggested that social phobia can arise as a result of direct traumatic conditioning, and several retrospective studies (with all their inherent limitations) have produced evidence consistent with this idea. For example, McCabe, Antony, Summerfeldt, Liss, and Swinson (2003) reported that 92% of their adult sample with social phobia reported a history of severe teasing in childhood, compared with only 50% and 35% in their panic disorder and obsessive-compulsive disorder groups, respectively. Another study found that 56% of those with specific social phobias recalled direct traumatic conditioning experiences as having played a role in the origins of their social phobia (Stemberger, Turner, Beidel, & Calhoun, 1995). One relevant case involved a young man who came home one day to find his fiancée in bed with his best friend (a humiliating and traumatic experience for him). Over the next few months he developed severe social phobia.

Social Learning and Social Phobia

Another potentially important pathway to the development of social phobia is through social learning. Vicarious conditioning—that is, simply observing another being ridiculed or humiliated or behaving in a very anxious way in some social situation—is one potent form of social learning that may be sufficient to make the observer develop social phobia of similar situations. For example, a vulnerable student might begin to develop social phobia after watching a high school friend give a speech in front of class and then be sharply criticized by the teacher and laughed at by the class. Öst and Hughdahl (1981) found that 13% of individuals with social phobia recalled vicarious learning experiences as having played a role in the origins of their social phobia. Additionally, there is evidence of modeling of social anxiety in families of those with social phobia. Retrospective studies show that adults with social phobia report that their parents were more likely than the parents of those without social phobia to have been avoidant of social interactions (e.g., Bruch & Heimberg, 1994). It is noteworthy that Rapee and Melville (1997) extended these findings by including reports from the mothers of individuals with social phobia and found that the reports from both the offspring and the mothers indicated more social avoidance among the families of patients with social phobia than among those of the nonclinical controls.

Direct social reinforcement and verbal instruction are also likely to play a role. For example, Dadds, Barrett, Rapee, and Ryan (1996) found that parents of anxious children were more likely than other parents to reciprocate their children's proposals of avoidant solutions. It is important to note that discussion of the potentially threatening situations with the parents appears to strengthen the anxious children's avoidant tendencies (Barrett, Rapee, Dadds, & Ryan, 1996).

Finally, culturally transmitted display rules and norms are another form of social learning that is likely to exert powerful influences on the expression of social anxiety, as is illustrated by the Japanese disorder *taijin kyofusho*, which is related to the Western diagnosis of social phobia. People with this disorder fear that they will do something to embarrass or offend others. For example, they may fear they will offend others by blushing, emitting an offensive odor, or staring inappropriately into someone else's eyes (Barlow, 2002; Kirmayer, 1991). By contrast, Westerners with a diagnosis of social phobia are afraid of other people because they believe they will be the object of scrutiny by others and will act in ways that will be personally humiliating or embarrassing. Kirmayer (1991) and Tanaka-Matsumi, Seiden, and Lam (1996) have argued that the pattern of symptom expression in *taijin kyofusho* and social phobia has clearly been shaped by cultural factors. In Japan, a collectivistic society that reinforces interdependence and social affiliation, there is a great deal of emphasis on implicit communication—being able to guess another's thoughts and feelings and being sensitive to them. People who make too much eye contact are likely to be considered aggressive and insensitive; children are taught to look at the

throats of people with whom they are conversing rather than into their eyes.

Preparedness and Social Phobia

Another fruitful perspective comes from Öhman et al.'s (1985) extension of preparedness theory to social anxiety. Öhman et al. proposed that social anxiety is a by-product of the evolution of dominance hierarchies and therefore predicted that social stimuli signaling dominance and intraspecific threat should be fear-relevant or prepared CSs for social anxiety. Because angry facial expressions are prominent signals during dominance conflicts among primates, Öhman et al. tested whether angry facial expressions would function as fear-relevant CSs for social fears in humans. Öhman and Dimberg (1978) indeed found superior conditioning when slides of angry faces were used as CSs, compared with slides of happy faces or neutral faces (all paired with mild shocks). Subsequent studies have reported other intriguing findings, parallel to those found for specific phobias. For example, fear CRs can even be conditioned to subliminal presentations of angry faces—that is, to angry faces that cannot be consciously perceived (e.g., Esteves, Parra, Dimberg, & Öhman, 1994). Thus, a person can claim to “know” rationally that a social situation is safe and yet still experience anxiety that is automatically activated in response to subtle cues that are not consciously processed.

Behavioral Inhibition as a Temperamental Diathesis for Social Phobia

Obviously not everyone who has experienced a socially traumatic episode or observed a socially anxious model will develop social phobia. For example, Stemberger et al. (1995) reported that 20% of their control sample without social phobia nevertheless recalled socially traumatic experiences. As for specific phobias, one source of individual differences involves the temperamental diathesis known as *behavioral inhibition*, which may influence the outcome of exposure to socially traumatic experiences and/or socially anxious models (e.g., Gray, 1982; Kagan, 1994). Indeed, early behavioral inhibition not only predicts the onset of multiple specific phobias in childhood (when they are most likely to emerge) but also social phobias in adolescence (when they are most likely to develop; e.g., Hayward, Killen, Kraemer, & Taylor, 1998; Kagan, 1997).

Uncontrollability and Social Phobia

Perceptions of uncontrollability are another likely source of individual differences in vulnerability to the effects of socially traumatic episodes or exposure to socially anxious models. For example, animal research has shown that uncontrollable (but not controllable) electric shock increases submissiveness (e.g., Williams & Lierle, 1986). Second, animal studies (e.g., Uhrich, 1938) of repeated social defeat (another uncontrollable stressor) show that it also leads to increased submissiveness to any other conspecific behaving in an aggressive manner. Moreover, repeated social defeat in animals also produces many of the classic learned helplessness effects usually associated with uncontrollable shock, including escape deficits (Hebert, Evenson, Lumley,

& Meyerhoff, 1998) and exaggerated fear CRs (Williams & Scott, 1989). We concur with Williams and colleagues, who concluded from such findings that the deleterious effects of social defeat are probably mediated by perceptions of uncontrollability. Moreover, such research suggests that perceptions of uncontrollability are likely to play a role in the origins of social anxiety. Indeed, cross-sectional evidence documents a moderate to strong association between perceptions of uncontrollability and social phobia in people (e.g., Leung & Heimberg, 1996).

In summary, a model of social phobias based on contemporary learning principles can account for many of the complexities involved in their etiology because it acknowledges both temperamental diatheses such as behavioral inhibition, and experiential diatheses such as learning histories, including those that lead to perceptions of uncontrollability. Our model of social phobia also takes into account the preparedness of certain cues to become CSs for social anxiety, as well as social learning experiences as alternative pathways to the acquisition of social phobia.

Panic Disorder With and Without Agoraphobia

People with panic disorder (PD) experience recurrent unexpected panic attacks that occur without their being aware of any cues or triggers, and they must also experience worry, anxiety, or behavioral change related to having another attack. Many, but not all, people with PD also go on to develop some degree of agoraphobic avoidance of situations in which they perceive that escape might be either difficult or embarrassing if they were to have a panic attack. An early conditioning approach to panic disorder with agoraphobia (PDA) was proposed by Goldstein and Chambless (1978), who argued, as had others, that *exteroceptive conditioning* (where CSs impinge on the external sensory receptors like eyes and ears) is central to the development of agoraphobia. But Goldstein and Chambless also described a process they termed “fear of fear” developing from *interoceptive conditioning*. In interoceptive conditioning, the CSs are the body's own internal sensations (e.g., Razran, 1961). Thus, Goldstein and Chambless proposed that when low-level somatic sensations of anxiety precede and are paired with higher levels of anxiety/panic, the low-level somatic sensations of anxiety come to elicit high levels of anxiety and panic.⁵

Such early conditioning approaches to the etiology of panic disorder were criticized for several reasons not discussed here (e.g., Clark, 1988; McNally, 1994). However, Bouton, Mineka, and Barlow (2001) reviewed these criticisms and presented a contemporary learning theory perspective that overcomes these limitations. Bouton et al.'s perspective also builds on growing evidence from two

⁵ The distinction between panic disorder and agoraphobia was not clearly stated until the *DSM-III* in 1980 (American Psychiatric Association, 1980)—two years after the article by Goldstein and Chambless (1978) appeared. Moreover, knowledge of the psychometric and neurobiological differences between panic and anxiety was not developed until even later.

sources that anxiety and panic are at least partially unique emotional experiences. First, psychometric analyses of panic- and anxiety-related symptomatology reveal two separable factors: Panic is accompanied by strong autonomic arousal, extreme fear, and fight-or-flight action tendencies, and anxiety is accompanied by apprehension, worry, and tension (Barlow, 1988, 2002; Craske, 1999). Second, neurobiological research supports the existence of these two partially distinct emotional states of panic and anxiety (e.g., Fanselow, 1994). In what follows, it is important to keep this distinction between anxiety and panic in mind because both are central to PD and PDA.

Exteroceptive and Interoceptive Conditioning in Panic Disorder

Bouton et al. (2001) argued that the conditioning that may occur in vulnerable individuals (see the *Vulnerability Factors for Panic Disorder* section) during initial panic attacks sets the stage for the development of PD and PDA. The initial attacks become associated with initially neutral interoceptive and exteroceptive CSs through conditioning. Specifically, because of their status as terrifying emotional events, replete with strong interoceptive stimuli, panic attacks are capable of supporting conditioning, according to contemporary research (e.g., Bouton et al., 2001; Forsyth & Eifffert, 1996b). Prototypic CSs during initial (as well as later) panic attacks include heart palpitations and dizziness (interoceptive CSs), as well as escalators and malls (exteroceptive CSs). Bouton et al. proposed that the primary effect of this conditioning is that anxiety becomes conditioned to these CSs, but another effect is that panic attacks themselves are also likely to be conditioned to certain internal cues. For example, one young man with PD, who was particularly sensitive to signs of his heart racing, surprisingly experienced a panic attack one night when he got excited while watching an announcement that his favorite presidential candidate had won a hotly contested state. Thus, the panic attack occurred at a time when he was happy and excited; however, this was also a time when his heart was racing, which probably served as an interoceptive CS.

On the basis of the premise that the conditioned response (CR) must resemble the unconditioned response (UR), some earlier critics objected that panic (the UR) cannot be involved in conditioning of anxiety (the CR) (e.g., McNally, 1994), but this criticism is based on outdated views of conditioning (Bouton et al., 2001). Today it is known that although some CRs are very similar to certain components of the UR, other CRs are actually opposite in nature to the UR, although in all cases they serve to “prepare” for an upcoming US and UR. For example, aversive conditioning in rats usually involves a CR like freezing (anxiety) rather than the activity burst that constitutes the UR to a shock US (analogue of a panic attack) (e.g., Fanselow, 1994).

Bouton et al. (2001) also noted that different CSs paired with the very same US often result in qualitatively different CRs to the different CSs. For example, in fear conditioning, Cook, Hodes, and Lang (1986) showed that when fear-relevant CSs (e.g., snake slides) are paired with

mild electric shock (US), one component of the CR is heart rate acceleration, in contrast to heart rate deceleration seen when fear-irrelevant CSs (e.g., flower slides) are paired with the shock US (see Öhman & Mineka, 2001). Thus, some internal and external cues present before and during panic attacks may become conditioned to elicit anxiety (different from the panic UR), and others may become conditioned to elicit panic itself. Interoceptive cues in close proximity to panic (as opposed to more distal external cues) may serve as “prepared” or fear-relevant CSs and may be especially likely to elicit panic, just as other fear-relevant CSs seem especially likely to condition CRs that strongly resemble URs (see Öhman & Mineka, 2001).

Interoceptive conditioning is robust and stable (Razran, 1961). In one example, Razran described a dog that received exposure to distention of the intestine (interoceptive CS) paired with a mixture of 10% carbon dioxide delivered directly to the trachea (interoceptive US). Fairly quickly, the CS of intestinal distention came to elicit hypercapnic respiratory changes (very labored breathing) as the CR. This example is particularly relevant to PD and PDA because various internal sensations like intestinal contractions (interoceptive CSs) might be paired with sensations of hyperventilation (interoceptive USs—often occurring during panic); later the contractions might come to elicit hyperventilation or even panic by themselves (Bouton et al., 2001).

Interoceptive conditioning has also been observed when small doses of a drug like morphine serve as CSs for larger doses of morphine USs (e.g., Kim, Siegel, & Patenall, 1999). Moreover, small increases in blood pressure can become associated with larger increases in blood pressure (e.g., Dworkin, 1993). So weak versions of some event can become conditioned by pairing it with a strong version of the same event—much as when early internal signs of panic become conditioned when paired with full-blown panic attacks (Bouton et al., 2001).

The Development of Agoraphobia

As noted, many people who develop panic attacks and panic disorder go on to develop some degree of agoraphobic avoidance of situations in which they fear or expect they may have a panic attack in the future (e.g., Barlow, 2002; Craske, 1999). Many of these situations are the commonly observed ones such as shopping malls, driving, standing in line, sitting in a theater, and so forth. Anxiety and avoidance of such situations have long been thought to develop as a result of exteroceptive conditioning of anxiety to these situations when panic attacks have occurred there in the past, followed by learned avoidance of these situations to minimize anxiety. Moreover, as the disorder develops, these agoraphobic fears and avoidance often generalize to other similar situations, as would be expected on the basis of the principles of classical conditioning. In addition, for many individuals, numerous everyday activities also come to be avoided, such as aerobic exercise, sauna baths, scary movies, caffeine intake, and even sexual activity because the interoceptive cues created by these

activities resemble those experienced at the beginning of a panic attack (and often trigger panic).

Who is most vulnerable to developing agoraphobia? Two major risk factors are gender and employment: Women are far more likely to than men to develop agoraphobia, and people who must leave the house to work are less likely than those who do not work or who work from home to develop agoraphobia (Barlow, 2002; Craske, 1999). The learning theory approach can explain both of these factors because in each case the person is allowed to avoid his or her feared situations rather than be exposed to them, which would extinguish his or her anxiety. For example, in American culture it is more acceptable for women than men to stay home and adopt a traditionally feminine role (Chambless & Mason, 1986). We might also expect that panic attacks perceived as particularly unpredictable and uncontrollable might well lead to the development of greater agoraphobic avoidance because these factors are known to be associated with greater conditioning of anxiety (e.g., Bouton et al., 2001; Mineka & Zinbarg, 1996; see also Craske, Glover, & DeCola, 1995).

Further Complexities of the Role of Conditioning in Panic Disorder and Agoraphobia

Conditioned stimuli not only come to elicit conditioned responses but also to facilitate or inhibit responses controlled by other events (Bouton et al., 2001). For example, for individuals with PD or PDA, inhibitory CSs for safety (such as the presence of a trusted companion) can attenuate panic symptoms ordinarily elicited by carbon dioxide inhalation (a US which is widely used as a panic provocation agent; Carter, Hollon, Carson, & Shelton, 1995). By contrast, CSs for anxiety (e.g., a warning about facing a stressful day at work) can strengthen ongoing avoidance behavior (such as agoraphobic avoidance like calling in sick) or engaging in “safety behaviors” (such as carrying talismans).

Conditioned anxiety also serves to increase startle responses (e.g., Lang, 1995), and Bouton et al. (2001) have argued that is quite likely that CSs for anxiety similarly lower the threshold for, or exaggerate, panic reactions (see also Zinbarg, Barlow, Brown, & Hertz, 1992). Indeed, baseline levels of anxiety strongly predict who will experience panic in response to various panic provocation agents as USs (e.g., Bouton et al., 2001). Moreover, several prospective monitoring studies have shown that anxiety is very often a precursor to (and may potentiate) panic attacks. For example, Başoğlu, Marks, and Sengün (1992) had 39 patients with PDA carefully monitor their panic and anxiety over three 24-hour periods. Over two thirds of their patients reported that elevated anxiety preceded all their panic attacks, and 87% reported that this happened at least some of the time. Finally, because the same CR (anxiety) elicited from two different sources can summate (e.g., baseline anxiety and anxiety elicited by a shopping mall), one would also expect that baseline anxiety could potentiate agoraphobic anxiety and avoidance.

Vulnerability Factors for Panic Disorder

Far more people experience panic attacks than go on to develop panic disorder. Among those individuals who experience panic attacks, what determines who will develop PD or PDA? Bouton et al. (2001) considered three types of vulnerability factors that seem likely to predispose some people who experience panic attacks to develop PD or PDA. First, they discussed a moderate nonspecific genetic vulnerability for PD and PDA (that overlaps especially with the genetic vulnerability for phobias) that may well be mediated by temperamental or personality vulnerability factors like neuroticism or trait anxiety (e.g., Kendler et al., 1995). As discussed earlier, these personality variables serve to potentiate conditioning of anxiety responses as well as aversive expectations (e.g., Zinbarg & Mohlman, 1998). Thus, genetic and temperamental variables seem to serve as nonspecific vulnerability factors that increase the likelihood of developing panic (e.g., Hayward, Killen, Kraemer, & Taylor, 2000) as well as various other anxiety disorders.

A second set of nonspecific vulnerability factors discussed by Bouton et al. (2001) are prior learning experiences that lead to perceptions of lack of control and helplessness and that may serve as psychological vulnerability factors influencing the development of panic, agoraphobia, and other emotional disorders. Early experience with uncontrollable stressful life events such as death and divorce can enhance vulnerability to PD and depression (see Bouton et al., 2001). Conversely, as noted earlier, early experiences with control and mastery in infancy and childhood are important for developing the ability to cope with stress and anxiety-provoking situations (invulnerability factors) (e.g., Chorpita & Barlow, 1998; Mineka et al., 1986).

Finally, Bouton et al. (2001) also reviewed some more specific learning experiences that may play a more unique role in creating risk for developing PD and PDA per se. For example, Ehlers (1993) found that people who experienced panic attacks as adults (relative to controls) were likely to have stronger learning histories of having been encouraged to engage in sick role behavior when experiencing panic symptoms (but not cold symptoms). Adults who had experienced some panic attacks were also more likely than controls to report others having had chronic illnesses in their households while growing up, suggesting that observing a lot of physical suffering may contribute to the evaluation of somatic symptoms as dangerous.

In summary, Bouton et al.'s (2001) perspective on PD and PDA argues that initial panic attacks set the stage for conditioning of anxiety to external and internal cues associated with panic, thus explaining the origins of anticipatory and agoraphobic anxiety. Moreover, the process of interoceptive conditioning also allows certain bodily sensations to become conditioned to elicit panic itself such that, for example, heart palpitations occurring early in an attack become predictors of the rest of the attack and acquire the capacity to provoke panic. Finally, people with certain genetic/temperamental and/or experiential vulnerabilities will show stronger conditioning of anxiety and

panic, explaining why only a subset of those who have panic attacks develop PD or PDA.

Posttraumatic Stress Disorder

The symptoms of posttraumatic stress disorder (PTSD) include reexperiencing the trauma, passively avoiding reminders of the trauma, numbing of affect, and heightened general arousal. Although a traumatic event is necessary for the diagnosis of PTSD, there are many puzzles in the development of PTSD that require explanation. Consider the cases of Ahmet and Hasan mentioned at the outset—both were torture victims in Turkey (M. Başoğlu, personal communication, 1997). Ahmet had been a political activist who was imprisoned by the government for his political activities for 48 months, during which he was tortured approximately 300 times. By contrast, Hasan was an ordinary citizen who was not politically active. One day he had called the police to report what he thought was a burglar lurking in his neighborhood. Unable to find the alleged burglar, the police arrested Hasan instead, charging him with having intentionally made a crank call. Despite the fact that Hasan was imprisoned and tortured for only a few weeks and experienced far less torture than Ahmet, Hasan developed PTSD, whereas Ahmet did not. Why? We believe this seemingly puzzling fact and many other complexities associated with PTSD are entirely consistent with contemporary learning theory research on the effects of uncontrollable and unpredictable stress in animals (sometimes known as learned helplessness effects).

Before exploring the implications of this perspective, we examine some of the compelling similarities between the symptoms of PTSD and the effects of uncontrollable and unpredictable stress, as well as the stressors involved in producing each. Regarding symptoms, animals exposed to uncontrollable and/or unpredictable stress, like individuals with PTSD, show heightened generalized anxiety and arousal. They also show enhanced passive avoidance behavior (e.g., Rush, Mineka, & Suomi, 1982) resembling the avoidance symptoms of PTSD (Foa, Zinbarg, & Rothbaum, 1992). In addition, the opioid-mediated analgesia (relative insensitivity to pain) produced by cues associated with uncontrollable stress in animals may resemble certain numbing symptoms seen in PTSD. For example, Vietnam veterans with PTSD who watched a video of a combat scene (a reminder cue for their uncontrollable stressful experiences) showed analgesia (Pitman, Van der Kolk, Orr, & Greenberg, 1990). Finally, as discussed by Foa et al., the reexperiencing symptoms of PTSD include fear and distress at exposure to reminders of trauma (as well as nightmares and flashbacks), and these emotional reactions can be seen as conditioned emotional responses elicited by reminder cues (CSs previously paired with trauma).

In addition, the intense physical stressors typically used in animal studies of the effects of uncontrollable and unpredictable stress (e.g., electric shocks, defeats in physical fighting) are similar to several forms of human trauma often associated with PTSD—torture (e.g., electric shocks, beatings), child abuse, and assault. Considered together, the symptom similarities and resemblance of physical stressors

used to produce them lead us to believe that the uncontrollable, unpredictable stress animal model is highly relevant for understanding PTSD. Specifically, to aid in the understanding of the onset, severity, and course of PTSD symptoms, Başoğlu and Mineka (1992) argued for the relevance of animal research on variables occurring before, during, or following exposure to unpredictable, uncontrollable stress, the topic to which we now turn.

Trauma Phase

The most obvious prediction from the animal model is that traumas that are perceived to be uncontrollable and unpredictable are more likely to result in PTSD. Results from retrospective studies consistent with this prediction have been reported (e.g., Bolstad & Zinbarg, 1997; Regehr, Cadell, & Jansen, 1999). In addition, organisms differ in how they respond to the experience of uncontrollable stress, and such differences can strongly affect the outcome. For example, animal research on social defeat shows that the analgesia induced by extensive attacks from a conspecific (analgesia is a characteristic facet of learned helplessness) is more highly correlated with the extent to which the animal assumed the characteristic posture of defeat than with the number of bites received from the conspecific (e.g., Rodgers & Hendrie, 1983). Conversely, Weiss, Glazer, and Pohorecky (1976) found that allowing animals to express aggression during uncontrollable stress attenuated the effects of the stress.

Such findings led Başoğlu and Mineka (1992) to hypothesize that the amount of trauma inflicted during torture (or assault) may be less predictive of the long-term emotional effects of the torture than is the victim's psychological state of resistance and fighting back versus giving up and conceding defeat. Consistent with this, studies of political prisoners and assault survivors have shown that survivors with PTSD were more likely than those without PTSD to retrospectively report having experienced mental defeat during their traumatization (e.g., Ehlers, Maercker, & Boos, 2000). It is important to note that Dunmore, Clark, and Ehlers (2001) also found that perceived mental defeat measured within four months of an assault predicted PTSD symptom severity at follow-up assessments six and nine months after the assault.

Pretrauma Phase

The effects of prior experiences with control or lack of control on reactions to subsequent uncontrollable trauma are complex. Sometimes prior uncontrollable stress *sensitizes* an organism to the harmful effects of subsequent exposure to such trauma (e.g., Moye, Hyson, Grau, & Maier, 1983). This means that as repeated exposures to trauma occur, the adverse effects get larger rather than smaller (as in *habituation*). Thus, a history of prior trauma (especially repeated trauma, which is more likely to be perceived as uncontrollable) should be associated with increased risk of developing PTSD to a recent trauma. For example, one retrospective study showed that a history of repeated childhood abuse had an even greater sensitizing effect than a single incident of child abuse (Bolstad &

Zinbarg, 1997). Moreover, the results of a recent meta-analysis support this prediction, although prior trauma was more strongly related to PTSD when the prior trauma involved interpersonal violence such as child abuse rather than combat exposure or an accident (Ozer, Best, Lipsey, & Weiss, 2003). We should also note that there are a few studies supporting a moderate genetic contribution to liability for PTSD (e.g., True et al., 1993), which might perhaps be mediated by sensitivity to uncontrollable and unpredictable stress (an interesting question for future research).

By contrast, a prior history of control over stressful events can immunize against the harmful effects of subsequent uncontrollable stress (e.g., Williams & Maier, 1977). Hence, we also predict that a prior history of control over stress can immunize against the development of PTSD to a recent trauma. Consistent with this prediction (and the case studies at the outset), there is suggestive evidence from a study of torture survivors that psychological readiness prior to being tortured (such as the psychological readiness Ahmet had because of his background as a political activist) is associated with a decreased likelihood of subsequently developing PTSD despite very high levels of torture (Başoğlu, Mineka, et al., 1997). Psychological readiness was measured by a questionnaire asking about the participants' knowledge of torture methods, their expectations of being imprisoned and tortured, and their training in stoicism techniques, among other things. By contrast, torture victims like Hasan, who were ordinary citizens arrested "out of the blue," were much less prepared for their torture experiences. Therefore, despite the fact that they experienced far less torture, they were more likely to develop PTSD and other forms of psychopathology than were the torture victims who had been political activists. Given that the psychological readiness measure used was multifaceted, the active ingredients responsible for this protective effect are unclear. However, the protective effects of readiness were probably mediated at least in part by rendering the subsequent torture more predictable. In addition, the stoicism training may have led the torture to be perceived as partially controllable and therefore less likely to produce mental defeat; indeed the nonactivists reported less perceived control but more distress than the activists.

Posttrauma Phase

One potential discrepancy between the effects of uncontrollable, unpredictable stress and the symptoms of human PTSD is that at least some of the effects of uncontrollable, unpredictable stress in animals are short-lived (usually dissipating over several days), in contrast to the sometimes chronic nature of PTSD, which often lasts many years. However, this discrepancy may be more apparent than real. First, only a small number of individuals who develop initial signs of PTSD go on to develop chronic PTSD (e.g., in one study, 0% of male assault victims and approximately 30% of female assault victims developed PTSD; Riggs, Rothbaum, & Foa, 1995). Second, if rats are simply exposed at two- to three-day intervals to the context in which they had previously experienced uncontrollable stress (without actually experiencing the

stress), the usual time course for at least some of the effects of the uncontrollable stress is prolonged "indefinitely" (Maier, 2001, p. 763). Such contexts associated with stress become CSs for anxiety, and we consider such exposure to anxiety CSs as eliciting conditioned emotional responses that are analogous to the anxiety-provoking aspects of the reexperiencing symptoms in PTSD.

On the basis of earlier reports of Maier's (2001) findings with rats, Mineka and Zinbarg (1996) predicted that the severity of reexperiencing symptoms after a trauma should influence the course of the symptoms of PTSD; greater reexperiencing would be associated with a more persistent course. At least six prospective studies of trauma survivors now support this prediction (e.g., Ehlers, Mayou, & Bryant, 1998). Moreover, in several studies, early reexperiencing symptoms significantly predicted later PTSD, but early avoidance (e.g., McFarlane, 1992) or numbing symptoms (e.g., Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992) did not, suggesting some degree of specificity between early reexperiencing symptoms and later PTSD.

What other kinds of posttrauma events can affect the course and intensity of PTSD symptoms? The inflation and US reevaluation effects, described earlier, can also affect the course of PTSD. Thus, mild PTSD symptoms could become full-blown PTSD when there is reason to reevaluate the danger posed by the original trauma (e.g., finding out weeks after an assault that one's assailant was in fact a convicted murderer). In addition, *reinstatement of fear* is the term for the phenomenon in which after a CS has extinguished, the CS can regain its ability to elicit a CR by simply exposing the animal to the US (not paired with the CS) (cf. Rescorla & Heth, 1975). Thus, a posttherapy trauma not linked with any of the cues associated with the original trauma could lead to a reinstatement of the original trauma symptoms.

In summary, there are compelling parallels between the literature on uncontrollable, unpredictable stress in animals and human PTSD. These include striking similarities in the symptoms of each as well as significant resemblance between the kinds of stressors. These parallels suggest that perceptions of uncontrollability and unpredictability play a role in the development and course of PTSD. Moreover, striking parallels have been observed between the trauma and pre- and posttrauma variables affecting the outcome of exposure to unpredictable, uncontrollable stress in animals and several forms of human trauma associated with PTSD.

Generalized Anxiety Disorder

People with generalized anxiety disorder (GAD) are primarily characterized by chronic excessive worry about a number of events or activities for at least six months, and the worry must be experienced as difficult to control (see Footnote 3). Several theorists have suggested that uncontrollable and unpredictable aversive events may play an important role in the development of GAD (e.g., Barlow, 2002; Mineka, 1985; Mineka & Zinbarg, 1996). As reviewed earlier, such events are much more stressful and create more fear and anxiety than do controllable and predictable aversive events. Although the unpredictable

and uncontrollable events involved in GAD are generally not as severe and traumatic as those involved in the origins of PTSD, there is some evidence (albeit limited) that people with GAD may be more likely to have a history of childhood trauma than are people with several other anxiety disorders (Borkovec, Alcaine, & Behar, 2004). Moreover, people with GAD have far less tolerance for uncertainty than do nonanxious controls (Dugas, Buhr, & Ladouceur, 2004), which suggests that they are especially disturbed by not being able to predict the future (Roemer, Orsillo, & Barlow, 2002). People with GAD may also have a relative lack of safety signals in their lives (indicators telling them when bad things are very unlikely to happen); this may help explain why they feel constantly tense and vigilant for possible future threats (Mineka & Zinbarg, 1996; Rapee, 2001).

Conversely, there is evidence relevant to individual differences suggesting that people with extensive experience controlling important aspects of their lives may be immunized against developing GAD (Barlow, 2002; Chorpita & Barlow, 1998; Mineka & Zinbarg, 1996). For example, as noted earlier, infant monkeys reared in controllable as opposed to uncontrollable environments were much better able to cope with frightening and novel situations later in their first year of life (Mineka et al., 1986). Moreover, because neuroticism and trait anxiety are known risk factors for anxiety disorders, it is possible that people high on neuroticism or trait anxiety may be especially susceptible to the effects of uncontrollable and unpredictable aversive events.

In recent years worry has increasingly come to be seen as the central feature of GAD. Borkovec (1994) and colleagues (e.g., Borkovec et al., 2004) have incorporated ideas from learning theory to help explain why worry is such a persistent process. They have investigated both the perceived benefits of worry as well as what actual functions worry serves. Most of the perceived benefits of worry center around people's beliefs that worry helps avoid catastrophe (either superstitiously or in reality) and deeper emotional topics that they do not want to think about. Investigations of how worry actually functions help reveal why the worry process can become so self-sustaining. First, when people with GAD worry, their emotional and physiological responses to aversive imagery are actually suppressed. Borkovec et al. argued that this suppression of emotional and physiological responses serves to reinforce the process of worry (i.e., increases its probability). Because worry suppresses physiological responding, this also prevents the person from fully experiencing or processing the topic that is being worried about; such processing is necessary if extinction of anxiety is to occur. Thus, the threatening meaning of the topic will be maintained. Finally, Borkovec et al. noted that worry may also be reinforced superstitiously because the vast majority of things people worry about never happen. The theory thus proposes that worry serves as a cognitive avoidance response. Because avoidance responses in animals are notoriously resistant to extinction (e.g., see Mineka, 1979, and Mineka, Yovel, & Pineles, 2002, for reviews), Borkovec argued by

analogy that this helps to explain why worry is so self-sustaining.

But why do people with GAD come to perceive worry as uncontrollable—a defining feature in the *DSM-IV* (APA, 1994)? Worry is not an enjoyable activity and can actually lead to a greater sense of danger and anxiety because of all the catastrophic outcomes the worrier envisions. Moreover, it can actually lead to more negative intrusive thoughts (e.g., Wells & Papageorgiou, 1995), and for these and other reasons people sometimes try to suppress or control their worrying (e.g., Craske, Rapee, Jackel, & Barlow, 1989). But research has also shown that attempting to control thoughts and worry may paradoxically lead to increased experience of intrusive thoughts and enhanced perception of being unable to control them (e.g., Abramowitz, Tolin, & Street, 2001; Wells, 1999). As we have noted, perceptions of uncontrollability are known to be associated with increased anxiety, and further intrusive thoughts can serve as trigger topics for more worry. Thus a vicious circle of anxiety, worry, and intrusive thoughts may develop, leading to this sense of being unable to control worry in GAD (Mineka, 2004; Mineka et al., 2002; Zinbarg, Craske, & Barlow, 1994).

In summary, people who have a history of uncontrollable and unpredictable life stress may be especially prone to developing GAD. Worry about possible bad outcomes or dangerous events, the central characteristic of GAD, seems to serve as a cognitive avoidance response that is reinforced because it suppresses emotional and physiological responding. Because attempts to suppress or control worry may lead to more negative intrusive thoughts, perceptions of uncontrollability over worry may develop, which is in turn associated with greater anxiety, leading to a vicious cycle.

Obsessive-Compulsive Disorder

The central features of obsessive-compulsive disorder (OCD) are unwanted and intrusive thoughts, impulses, or images that cause marked anxiety or distress; these are usually accompanied by compulsive behaviors or mental rituals that are performed to neutralize or prevent the distressing thoughts or images. Even “normal” people experience occasional cognitive intrusions that do not differ in content from those seen in OCD (e.g., Rachman & de Silva, 1978). What distinguishes people with OCD is that clinical intrusions/obsessions are (a) associated with greater distress, (b) more frequent, and (c) more strongly resisted. Thus, one critical question regarding the development of OCD is why intrusions are more distressing, frequent, and strongly resisted in certain people.

Verbal Conditioning and Social Learning

Although research thus far has failed to find good evidence that a traumatic conditioning history is involved in the origins of many cases of OCD (Mineka & Zinbarg, 1996; Rachman & Hodgson, 1980), verbal transmission of dangerous thoughts (as discussed for specific phobias) may occur (e.g., “my mother told me that dirt and contamination may be on doorknobs or toilet seats”). In addition, direct

verbal conditioning may also occur in which a neutral idea is paired with some scary idea that a person may have (e.g., "I saw that filthy person use the toilet and wash her hands before me so I should be very careful in all bathrooms"). In addition, there is some evidence to suggest that social learning factors may play a role in the greater levels of distress and resistance associated with clinical intrusions. One idea is that parents (or teachers) who encourage a broad sense of responsibility and rigid rules for duty and conduct may create a vulnerability for the development of OCD in the child (e.g., Salkovskis, Shafran, Rachman, & Freeston, 1999). Similar factors may lead to beliefs referred to as *thought-action fusion* in which people are taught the idea that thoughts, desires, and impulses are morally equivalent to actions (e.g., "it is just as immoral to think about harming someone as it is to actually cause harm") (Shafran, Thordarson, & Rachman, 1996). A second type of thought-action fusion involves the idea that thoughts of a specific catastrophe increase the probability that it will actually occur (e.g., "thinking about my mother getting cancer increases the risk that she actually will"). Evidence suggests that each of these factors may lead to greater distress and attempts to resist intrusions (see Salkovskis et al., 1999, for several relevant cases).

Social learning is also involved in culturally transmitted beliefs and norms from the teachings of various religions that may exert influences on the expression of some obsessions and compulsions. One hypothesis is that highly religious individuals may be at slightly higher risk for developing OCD. Although only correlational data are available, Steketee, Quay, and White (1991) found that religiosity was significantly correlated with measures of OCD symptom severity (see also Rassin & Koster, 2003). Moreover, Rassin and Koster found that religiosity is strongly ($r = .71$) correlated among Protestants with beliefs about thought-action fusion (e.g., that thoughts and desires are morally equivalent to actions) ($r = .42$ among Catholics).

The Role of Avoidance in the Maintenance of OCD

Most patients with OCD engage in either behavioral or mental rituals intended to neutralize or prevent their obsessions, and it is therefore not surprising that rituals have long been construed as avoidance responses (e.g., Rachman & Hodgson, 1980). As noted in the section on GAD, learned avoidance responses are remarkably resistant to extinction (for reviews, see Mineka, 1979, 1985) and thus provide a useful analogue to compulsive rituals in humans with OCD. Moreover, the best way to extinguish avoidance responses is by preventing them from occurring during prolonged exposure to the conditioned aversive stimulus; this procedure is highly analogous to the exposure and response-prevention procedures long considered to be the most effective treatment for OCD (e.g., Rachman & Hodgson, 1980; Steketee & Barlow, 2002). Mineka and Zinbarg (1996) also summarized other findings from the avoidance learning literature that are relevant for understanding the

fluctuations in frequency of occurrence of compulsive rituals as a person goes through varying levels of stress.

Preparedness and OCD

Finally, the preparedness concept discussed earlier in the context of specific and social phobias is also relevant to understanding OCD in an evolutionary context. As demonstrated years ago by De Silva, Rachman, and Seligman (1977), the contents of most obsessions and the forms of most compulsive rituals are nonrandom (thoughts about dirt, contamination, and danger are the most common obsessions; cleaning and checking for danger are the most common rituals). Indeed, humans' obsessions with dirt, contamination, and danger have deep evolutionary roots (Mineka, 1985; Mineka & Zinbarg, 1996). Moreover, the displacement activities that many animal species engage in under situations of conflict or high arousal (such as grooming, nesting, or tidying) bear a significant resemblance to the compulsive rituals seen in OCD. Whether these fears are based on nontraumatic conditioning (verbal or vicarious verbal conditioning) or on social learning, the idea is that people with OCD do not typically obsess about random scary thoughts and do not show arbitrary ritualistic behaviors; rather, their thoughts and behaviors have deep evolutionary roots.

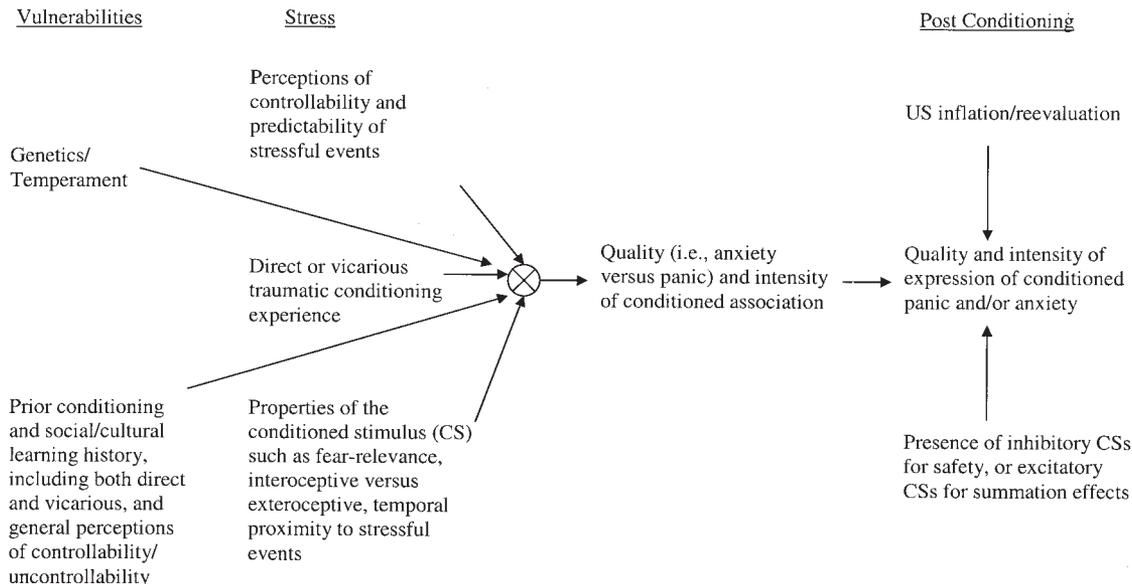
In summary, we have argued that verbal learning and social learning factors are involved in the acquisition of beliefs that lead to heightened levels of distress and resistance to intrusions; these beliefs tend to center on a few evolutionarily relevant themes. Moreover, once compulsive rituals have developed as methods to neutralize or prevent obsessive thoughts, they show the same high resistance to extinction seen in learned avoidance responses in animals.

Conclusions

The influence of early, rather simplistic learning approaches to understanding the etiology and course of anxiety disorders had begun to wane around the mid-1970s following some forceful criticisms of such models for phobias. However, contemporary learning theory and research provide a foundation for the development of models of the anxiety disorders that can capture the richness and complexity associated with the development and course of these disorders (see Figure 1). Not only do contemporary learning models of the etiology of anxiety disorders have a great deal of explanatory power but they are also testable. We have outlined several falsifiable predictions derived from these models here and elsewhere (e.g., Başoğlu & Mineka, 1992; Bouton et al., 2001; Foa et al., 1992; Mineka, 1985; Mineka & Zinbarg, 1996). Some of these predictions have already been tested and supported. Only time and additional research will tell if the remaining predictions are also supported or refuted.

Whereas some of the predictions derived from learning theory models of the etiology of anxiety disorders overlap with predictions made by models emanating from other theoretical perspectives, we believe that the learning

Figure 1
Overview of Major Elements Incorporated in Contemporary Learning Models of the Etiology of the Anxiety Disorders



Note. US = unconditioned stimulus.

theory models often have the advantage of being more comprehensive. Consider as an example two psychological models of PDA. Both the leading cognitive perspective in which catastrophic cognitions about the consequences of feared bodily sensations are thought to play a causal role in panic attacks (e.g., Clark, 1988, 1996) and the learning theory perspective on etiology outlined here predict the occurrence of panic attacks in many of the circumstances in which they tend to occur (see Bouton et al., 2001). However, the cognitive perspective does not distinguish clearly between panic and anxiety as partially distinct emotional states as does the learning theory approach. Therefore the cognitive model of etiology is silent about the ways in which anxiety can potentiate panic as outlined previously and does not do as thorough a job outlining the exact processes by which occasional panic attacks only sometimes develop into panic disorder. Moreover, the cognitive model is silent about the variety of different vulnerability factors that the learning theory approach explicitly addresses affecting which individuals with panic attacks are most likely to develop PD or PDA.

Finally, although our primary focus here is on etiology, it is also necessary to note that contemporary learning models of the anxiety disorders have important implications for treatment and prevention. We start with the old premise that what can be learned can also be unlearned and perhaps also prevented. Thus it is not surprising that treatment approaches based on learning principles have been shown to be effective for each of the anxiety disorders

(e.g., Barlow, 2002; Barlow, Allen, & Coate, 2004; Craske, 1999; Craske & Mystkowski, in press). More specifically, behavioral/learning research on habituation, extinction, counterconditioning, and, most recently, safety signals sowed the seeds for the development of exposure therapy—a central component of many empirically supported treatment packages for the anxiety disorders as they are practiced today. In addition, more recent studies on the important role that contextual shifts have on enhancing the return of fear following extinction in animals (e.g., Bouton & Swartztruber, 1991) have provided new insights into the similar role that contextual shifts play in the return of fear in individuals who have undergone exposure treatment for specific phobias and probably other anxiety disorders as well, although there is not yet much research from this perspective (e.g., Craske & Mystkowski, in press; Mystkowski & Mineka, in press).

Regarding prevention, by incorporating the role of genetic and temperamental variables, contemporary learning models can help identify which individuals are at highest risk for the development of anxiety disorders. However, contemporary learning models can also provide a theoretical and empirical basis for targeting individuals at risk on the basis of their prior learning histories as well. Thus, perhaps as a result of both genetic transmission and vicarious learning of specific fears, as well as avoidant coping styles and patterns of attentiveness to somatic cues, children of parents with anxiety disorders seem to be at elevated risk for the development of anxiety disorders.

Contemporary learning models predict that rearing aimed at facilitating the development of a strong sense of mastery, as well as extensive exposure to nonanxious models, from an early age should provide powerful immunization in such children.

Detailing how to implement the immunization principles suggested by contemporary learning models of anxiety disorders is beyond the scope of the current article (see Feldner, Zvolensky, & Schmidt, 2004, for a review of the status of prevention research for anxiety disorders). However, one example would be how to address the not-uncommon tendency of parents in treatment for anxiety disorders today to voice concerns that their children will develop similar problems. In discussing such concerns, the therapist might offer several suggestions that these parents could implement to lower their children's risk, in addition to providing the usual reassurance that many such children do not go on to develop anxiety disorders. For example, the therapist might discuss various parenting behaviors and strategies that should facilitate the development of mastery and a nonavoidant coping style. In addition, the therapists might suggest to parents with phobias that they ensure that their child has extensive direct exposure to nonfearful models interacting with the phobic object.

It is certainly the case that much work remains to be done on the etiology, course, treatment, and prevention of anxiety disorders. We have focused here on the implications for etiology and course of anxiety disorders because of space constraints and have only briefly noted implications for treatment and prevention. It should be clear, however, that we believe contemporary learning theory has much to offer in guiding the efforts to solve the remaining mysteries associated with anxiety disorders.

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