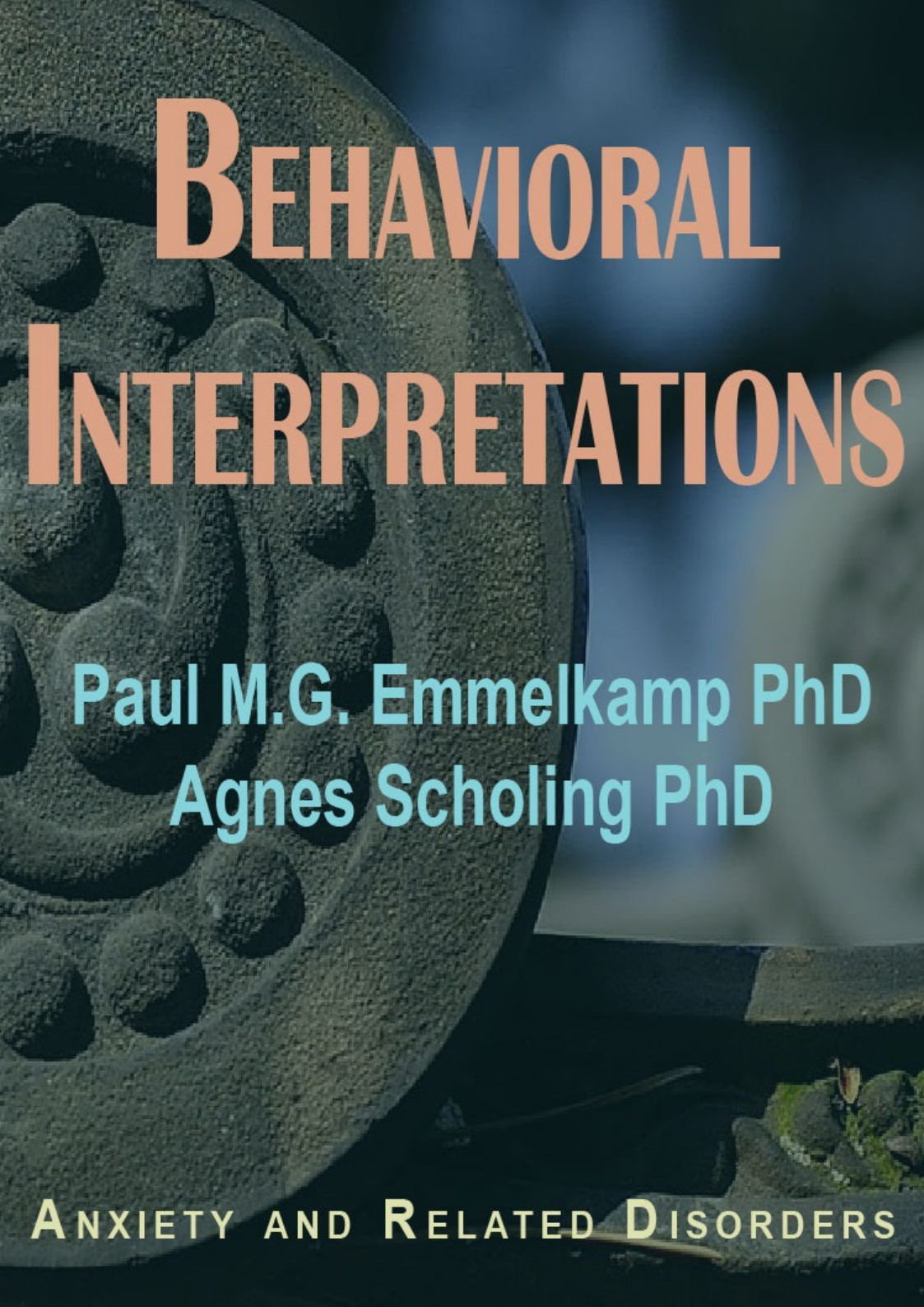
BEHAVIORAL
INTERPRETATIONS
Paul M.G. Emmelkamp PhD
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ANXIETY AND RELATED DISORDERS

Behavioral Interpretations

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e-Book 2015 International Psychotherapy Institute

From *Anxiety and Related Disorders* edited by Benjamin Wolman & George Stricker

Orig. Publisher: John Wiley & Sons

Created in the United States of America

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Behavioral Interpretations

PAUL M.G. EMMELKAMP, PhD, and AGNES SCHOLING. PhD

In this chapter an overview is given of behavioral theories with respect to the etiology and maintenance of anxiety disorders. After a discussion of the current status of learning theories in explaining the acquisition and maintenance of fears and phobias, separate sections are devoted to the development of panic disorder, social phobia, generalized anxiety disorder, post-traumatic stress disorder, and obsessive-compulsive disorder.

LEARNING THEORIES

The two-stage theory (Mowrer, 1960) of fear acquisition has been highly influential, and despite some serious criticisms (e.g., Mineka, 1979), it still plays a prominent role in current thinking of the development of phobias. Mowrer explicitly distinguished between a classical conditioning process, responsible for the conditioning of fear, and an operant conditioning or instrumental learning process, responsible for the conditioning of the avoidance response. The model was developed on the basis of animal experiments. In training procedures, animals receive repeated pairings of a warning signal (for example, a tone (CS)) and an aversive stimulus (for example, a shock (UCS)). After some time, the tone will acquire aversive properties and the animal will experience anxiety (CR) on tone presentation

when no shock is applied. This phase of the experimental procedure represents the first stage of learning, in which anxiety is attached to previously neutral cues through classical conditioning. In the second stage, the animal learns to terminate the tone by making escape responses, reducing thereby the anxiety. The termination or avoidance of aversive stimuli leads to negative reinforcement (anxiety reduction), thus strengthening the avoidance behavior. This second stage of learning involves operant conditioning. In summary, it is assumed that fear is acquired through a process of classical conditioning and motivates avoidance behavior.

The assumption of the two-stage theory that avoidance is mediated by fear is supported neither by everyday experiences nor by experimental results. There is ample evidence that avoidance behavior can be acquired and maintained in the absence of fear as a mediating factor (Gray, 1975). A more serious difficulty for the theory is the observation that often extinction of avoidance responses does not occur (Mineka, 1979). If the first stage of the theory is correct, it would be expected that rapid extinction of fear, and hence of avoidance behavior, should occur when the UCS is no longer present. However, there is abundant evidence that avoidance behavior, when established, is highly resistant to extinction, despite the fact that, according to the extinction paradigm, conditioned fear itself must start to extinguish as soon as the shocks are no longer given. Modifications of the two-stage theory have been proposed involving the safety-signal theory (Mowrer, 1960) and

Herrnstein's (1969) theory of expectancies. In current theorizing, instrumental conditioning is held responsible for maintaining the avoidance behavior for some of the time only.

The safety signal theory assumes that it is not anxiety reduction *per se* but safety signals that positively reinforce avoidance behavior. This version differs from Mowrer's original position in that it includes, in addition to the negative reinforcement (anxiety reduction), the presence of secondary rewarding stimuli (i.e., safety signals) as a potential source of reinforcement for avoidance behavior (Gray, 1975). According to this model, safety signals acquire positive reinforcement properties, so that avoidance behavior is motivated not only by an escape from fear, but also by an approach to safety, and resistance to extinction is explained by the reinforcement of the avoidance behavior through the simultaneous presence of safety signals.

Research by Herrnstein and his colleagues led to another important modification of the two-stage theory (Herrnstein, 1969). In Mowrer's original view, avoidance behavior is maintained by the reinforcing escape from the anxiety-provoking conditioned stimulus. Herrnstein (1969) argued that CS termination is an unnecessary feature of avoidance procedures. In a particularly well-designed study, Herrnstein and Hineline (1966) were able to demonstrate that the classically conditioned fear responses are not a requirement for the instrumental behavior. In their study, rats were offered a

choice between two frequencies of being shocked at unpredictable intervals. Lever pressing resulted in the lower shock frequency, but after some time control reverted to the schedule with the higher shock frequency until the animal's next lever press. Thus, animals could learn to choose being shocked at a lower frequency, rather than escape or avoid shock. Most animals learned to respond. Herrnstein (1969) argued that the reinforcement for avoidance behavior is a reduction in time of aversive stimulation. Further experiments suggested that the conditioned stimuli may function as discriminative stimuli for avoidance responses.

As shown in studies by Herrnstein (1969), avoidance behavior can be ruled by preferences rather than by anxiety *per se.* This could explain the persistence of avoidance behavior in the absence of fear, as is often seen in clinical cases.

Theoretical Innovations

Paradigmatic behaviorists (e.g., Eifert & Evans, 1990; Hekmat, 1987) hold that phobias are not acquired exclusively through a first-order conditioning process. In their view, phobic reactions are not only acquired through aversive classical conditioning, but may also be acquired by higher-order semantic conditioning processes. Thus, language conditioning is given an important role in the development of human fear reactions. In this view, a

snake phobia does not necessarily develop through classical conditioning or vicarious learning, but "The word 'snake' by virtue of being semantically accrued to negatively valued words such as 'ugly, poison, disgusting, slimy, etc.' may have indirectly acquired negative reactions through higher-order semantic conditioning" (Hekmat, 1987, p. 201).

Other authors have attempted to amplify the conditioning hypotheses with recent insights of cognitive theories (Baeyens, Eeelen, Crombez, & Van den Bergh, 1992; Davey, 1992; Martin & Levey, 1985). Levey and Martin (1983, 1985) distinguished between two ways of learning, the first being a relatively immediate registration of stimuli associated with (positive or negative) events, the second being a more cognitive process, in which experiences of the past are summarized and repeated. They stated that these summaries form the basis of the hypotheses and schemata that are assumed in cognitive theories. In the same way, Baeyens et al. (1992) described signal learning versus evaluative learning, the second being conceived as a kind of referential learning in which the CS activates a (cognitive) UCS-representation. Davey (1992) proposed a model of human conditioning, in which outcome expectancy, expectancy evaluation, cognitive representations of the UCS, and revaluation processes play a major role. According to Davey, this model differs from more traditional conditioning theories in the following aspects: (1) the association between CS and UCS can be influenced by factors other than the experienced contingency, and (2) a performance

component is added that suggests that the strength of the CS is determined by nonassociative factors, which influence the evaluation of the UCS. Models assuming cognitive representations may be promising in that they have better explanations for phenomena that could not be explained by the more traditional models, like the absence of clear traumatic experiences at the onset of a fear reaction, the failure to develop a phobia after clear traumatic experiences, and incubation effects.

More recently, Barlow (1988) has formulated a model in which both biological vulnerability and learning factors play a role. In this model, it is assumed that in biologically vulnerable individuals severe stress will lead to alarm reactions (fear or panic attack) that prepare the individual for immediate action (fight or flight). The stressor is experienced as uncontrollable ("I may not be able to deal effectively with it") and unpredictable ("It might happen again"), resulting in a preparatory coping set that is manifested as chronic arousal and anxious apprehension. Apart from the biological vulnerability, which is possibly genetically transmitted, the model also assumes a psychological vulnerability, namely a specific attribution style. This style is characterized by thoughts of uncontrollability and unpredictability and is considered to be a consequence of specific developmental experiences. Thus individuals who were raised by parents who were oversensitive to illnesses are likely to interpret physical sensations as threatening, meaning that something is wrong with their body.

Alternatively, individuals raised by parents who were overly concerned with scrutiny of others may focus their anxious apprehension on social evaluation. The focus of anxious apprehension in individuals with post-traumatic stress disorder is on cognitive and physiological cues associated with the original trauma.

Barlow (1988) differentiated three types of alarm: (1) true alarm, (2) false alarm, and (3) learned alarm. Reactions during a real life-threatening event are seen as true alarm, whereas false alarms occur in the absence of real life-threatening events. The association of false alarms with internal or external cues results in the phenomenon of learned alarm. Conditioned responses to either interoceptive or external cues are considered learned alarms. Such learned alarms may be only partial responses, such as cognitive representations without the physiological component of anxiety.

Thus, according to Barlow, anxious apprehension is the result of a complex interaction of biological, psychological, and environmental events. In individuals with a biological and psychological vulnerability, an anxious circle develops: " . . . once this cycle begins, it becomes self-perpetuating: a sense of unpredictability and uncontrollability increases emotionality, which in turn increases the probability of learned alarms . . ." (p. 276).

Classical Conditioning of Fear

What evidence is available that fears and phobias are acquired through a process of classical conditioning? The classical conditioning paradigm states that neutral stimuli, when associated with fear or pain, elicit fear reactions, and that the strength of the fear is determined by (1) the number of repetitions of the association between the stimuli and the emotional reactions, and (2) the intensity of the emotion experienced. Central to the model are one or more traumatic experiences in which the association between stimulus and fear reaction is learned.

A number of studies obtained information about the acquisition of simple phobias and specific fears; and results suggest that classical conditioning may be involved in a number of cases. Research done by Rimm, Janda, Lancaster, Nahl, and Ditmar (1977) showed that 16 out of 45 phobic volunteers reported direct experiences of a more or less traumatic nature. In a study by Fazio (1972), on the genesis of insect phobias, similar results were found. In contrast, Murray and Foote (1979), studying the origins of snake phobia, found very few frightening experiences with snakes in their phobic group. These studies are, however, of questionable relevance, since the subjects consisted of normal subjects with fears, rather than patients with simple phobics.

Lautch (1971) found that patients with dental phobia *(n* = 34) reported having had a traumatic dental experience on at least one occasion in

childhood. However, all patients were diagnosed as generally neurotic, whereas 10 control subjects with comparable traumatic experiences showed little sign of dental fear. Goldstein and Chambless (1978) compared 32 agoraphobics with 36 patients with simple phobias and found that only 4 agoraphobics reported conditioning events at the onset of the phobia, in contrast with 17 simple phobics, suggesting that conditioning events are etiologically more important in simple phobias than in agoraphobia. McNally and Steketee (1985) reported data on the etiology of severe animal phobias. All patients *(n* = 22) stated that the phobia had begun in early childhood and had remained stable or worsened with age. The majority of the patients (77%) could not remember the onset of the phobia. Two of them had parents who witnessed the origin of the phobia, which left 68% of the cases unclassifiable. Out of the 7 patients who remembered the onset, 6 patients attributed it to experiences interpretable as conditioning events (which were frightening encounters, no patient reported that the animal had inflicted pain). Only 1 patient reported indirect learning experiences, like watching frightening movies. In a study by DiNardo, Guzy, Jenkins, Bak, Tomasi, et al. (1988) on fear of dogs, conditioning events were reported by 56% of the fearful subjects and 66% of nonfearful subjects. All fearful subjects believed that fear and physical harm were likely consequences of confrontation with a dog, while few nonfearful subjects had such expectations. The study suggested that painful experiences seem to be common in the history of dog

fears, in contrast with the results of McNally and Steketee (1985) and results found with snake fears (Murray & Foote, 1979). However, no evidence was found for etiological significance, because those experiences were at least equally common among nonfearful subjects. Exaggerated expectation of harm, a cognitive factor, seemed to play a role in the maintenance of the fear.

Ost and Hugdahl (1981, 1983, 1985) studied how patients with clinical phobias remembered the onset of their fears. Patients rated the way they acquired their fear on a phobic origin questionnaire, based on the three pathways to fear theory that was proposed by Rachman (1978). The first pathway, *conditioning,* includes exposure to traumatic experiences. The second, *vicarious learning,* refers to direct or indirect observations of people displaying fear. The third pathway is by *transmission of fear-inducing information.* The results showed that conditioning experiences were most frequently mentioned in all subgroups of simple phobias studied: claustrophobics (67.7%), dental phobics (65.6%), animal phobics (50.0%), and blood phobics (50.0%). Hekmat (1987) investigated the factors leading to development and maintenance of human fear reactions in animal phobics *(n* = 56) and nonphobic undergraduate controls *(n* = 18). Evidence was found for fear acquisition through conditioning, vicarious processes, and information/instruction pathways. Ollendick and King (1991) obtained data from 556 female and 536 male Australian and American children and adolescents. The majority attributed onset of their fears to vicarious and

instructional factors, although these indirect sources of fear were often combined with direct conditioning experiences. The findings suggested that the three pathways to fear are interactive rather than independent.

In sum, although the results suggest that conditioning processes may play a role in the onset of some simple phobias, it is more and more recognized that other factors, like indirect conditioning processes, seem to be important as well.

Several studies have investigated the role of classical conditioning in the etiology of *agoraphobia.* As noted, Goldstein and Chambless (1978) found that the onset of the agoraphobia was marked by a conditioning event in only 4 out of 32 agoraphobics. In the Buglass, Clarke, Henderson, Kreitman, and Presley (1977) study, results showed that in only 7 out of 30 agoraphobics discrete events at the time of the onset of the agoraphobia could be identified. Also, only two of these events were "specific," meaning that the event occurred in the setting in which the patient was subsequently phobic. Similar results were found by Solyom, Beck, Solyom, and Huger (1974) and Bowen and Kohout (1979). In contrast, research by Ost and his colleagues (Ost & Hugdahl, 1983; Ost, 1985, 1987) suggested that conditioning was much more frequently involved in the development of agoraphobia (ranging from 81-89%).

To summarize the studies reviewed so far, there is conflicting evidence that classical conditioning is an important factor to account for development of agoraphobia. There is more evidence provided that classical conditioning of fear is involved in the development of specific phobias. The latter conclusion, however, needs to be qualified by the finding that even with specific phobias in a substantial number of cases no traumatic experiences could be identified in relation with onset of the phobia. A problem in these studies is the definition of a traumatic conditioning event. A minimum requirement of the classical conditioning paradigm is that not only should a traumatic experience be identified, but also that the subject should have experienced pain or anxiety in the situation that subsequently led to the phobia. Unfortunately, most studies we reported on did not provide data with respect to this point. Thus, the occurrence of traumatic incidents in the history of a phobic patient, even when in some way related to the development of the phobia, is by itself insufficient evidence that classical conditioning can be held responsible for the acquisition of the phobia.

Vicarious Learning

In an attempt to explain the development of fears that are not associated with traumatic learning experiences, one might argue that in these cases fears are acquired through vicarious learning (Rachman, 1978). According to this paradigm, observing others experiencing anxiety in specific

situations might lead to fear of those situations for the observer.

Indirect evidence in favor of a vicarious learning interpretation for the acquisition of phobias came from studies demonstrating that children often share the fears of their parents. Particularly, mothers may be an important etiological factor in children's fear (Emmelkamp, 1982). Several studies indicated that mother and children are frequently fearful of the same situation, which can be considered to support a vicarious learning interpretation of the etiology of children's fears. On the other hand, it should be noted that a relationship between fears of mother and child can also be the result of processes other than vicarious learning, for example, informational processes, genetic influences, or similar traumatic experiences.

That fears can be acquired through modeling was demonstrated by Mineka and her colleagues. For example, laboratory reared monkeys who initially were not fearful of snakes, developed a snake phobia as a result of observing wild monkeys displaying fear in the presence of a snake (Cook & Mineka, 1991). Other indirect evidence in favor of the vicarious transmission of fear came from retrospective patient reports. Several investigations of war neurosis showed that in certain cases fear of war experiences was caused by observing accidents of other soldiers (e.g., Kipper, 1977). Kipper, in analyzing the circumstances surrounding the development of fears in soldiers in the Yom Kippur War, identified three sets of conditions under which these fears

were acquired. The first set of conditions involved a sudden realization of danger. In the second group, fears developed "more or less accidentally." A third group of conditions "involved fears acquired vicariously while observing the unfortunate fate of fellow soldiers" (p. 218).

The studies by Fazio (1972) and Rimm et al. (1977) provided further evidence that vicarious learning might be responsible for the acquisition of fears in only a few cases: 13% of the subjects with an insect phobia (Fazio, 1972) and 7% of the subjects with other specific phobias (Rimm et al., 1977) reported vicarious learning experiences. Further, the Murray and Foote (1979) study presented "only marginal evidence of vicarious experiences in the acquisition of fear of snakes" (p. 491) and, in the studies by Ost and his colleagues discussed above, vicarious learning was less prevalent than conditioning. Finally, results of a study by Merkelbach, de Ruiter, Van den Hout, and Hoekstra (1989) suggested that in most phobic patients both conditioning factors and vicarious learning are involved.

In sum, studies on etiology of phobias have demonstrated that patients attribute onset of the fears to traumatic experiences, vicarious learning, and fearful information or instruction about the feared stimuli. However, studies that compared phobic patients with controls without such fears did not show clear differences in the frequency of one of these factors, pointing to the fact that conditioning experiences, even those of a painful nature, do not

automatically lead to phobic reactions. Development of fear seems to be determined by other factors, for example neuroticism (Eysenck & Rachman, 1965; Lautch, 1971) or expectations (DiNardo et al., 1988), interpretations (Rachman, 1991a,b) and, more generally, spoken, cognitive representations of the feared stimulus (Davey, 1992). A major drawback of all studies is their retrospective character. Only prospective studies will be able to give more reliable information on acquisition of phobic reactions.

Preparedness

Some hold that individuals differ in genetically based predispositions to acquire fears to specific situations. From an evolutionary perspective, phobias represent examples of evolutionally primed predispositions to acquire fears for situations involving danger. What evidence is available that there are predispositions to acquire phobias for specific situations over other situations? According to the classical conditioning theory, any stimulus that is paired with an unconditioned stimulus that invokes pain or anxiety should result in a conditioned emotional reaction after a number of pairings. However, this assumption is no longer adequately defensible. Consider, for example, experiments of the kind in which one has attempted to condition fear in infants. Several such experiments have been reported and they show that the nature of the conditioned stimulus is of paramount importance for conditioning of fear to occur. English (1929) was unable to condition fear to a

wooden toy duck, but he succeeded in producing conditioned fear to a stuffed black cat. Bregman (1934) also failed to condition fear in infants. In this study, the conditioned stimuli consisted of shapes and colored clothes. Taken together, the variable results of the studies by Bregman (1934), English (1929), and Watson and Rayner (1920) seem to indicate that fear might be much more easily conditioned to animals and furry objects than to wooden objects, shapes, and clothes. This finding suggests that there might be an innate base for some fear development. Marks (1969) suggested the concept of "prepotency" of certain stimuli to explain the development of some human phobias. Along similar lines, Seligman (1971) viewed phobias as instances of highly "prepared" learning. According to Seligman, the majority of clinical phobias concerns objects of natural importance to the survival of the species. In his view, evolution has preprogrammed the human species to easily acquire phobias to potentially dangerous situations. Such prepared learning is selective, highly resistant to extinction, probably noncognitive, and can be acquired in one trial.

In recent years, Ohman and his colleagues (Ohman, 1987) have tested the preparedness theory experimentally. The studies by Ohman and his associates demonstrated that the stimulus content variable plays a major role with respect to resistance to extinction and far less so with respect to the acquisition phase, thus partially supporting the preparedness theory. As phrased by Ohman (1987):

> Potentially phobic stimuli such as pictures of snakes or angry faces have special effects, compared to those of neutral stimuli, when they are presented in a Pavlovian contingency with electric shock US. The results, therefore, are in accord with the basic premise of the preparedness theory that these types of stimuli have a biologically determined readiness to become easily imbued with fear. (p. 148)

On the theoretical side, Ohman (1987) argued that social fears are associated with a social submissiveness system, whereas animal fears are associated with a predatory defense system. As yet, this differentiation is purely speculative and not substantiated by experimental evidence. Despite some evidence in favor of preparedness provided by Ohman and his colleagues, the results of these studies need to be qualified in several ways (Emmelkamp, 1982; McNally, 1987). Although Ohman provides some evidence in support of the role of preparedness in laboratory fear extinction, it is a very bold claim to generalize these findings to human phobias. All subjects in their experiments were normal nonphobic college students. Further, only psychophysiological data were used as the dependent variable. Phobic anxiety is usually conceptualized as three different systems—subjective, physiological, and behavioral—which do not always covary.

Another line of research was followed by Mineka (1979). She investigated whether fear of snakes in monkeys had a prepared basis. Laboratory-reared animals with no fear of snakes were exposed to a wild-reared animal displaying fear of snakes. As a result of vicarious learning, the

laboratory-reared animals developed a phobia for snakes. Related studies on monkeys showed that the fear was conditioned only to snakes and not to flowers, even when the model monkey had displayed fear to both snake and flower. Similar results were found with the acquisition of fear of crocodiles in contrast with the nonacquisition of fear of rabbits.

Although the results of the studies by Ohman and associates and Mineka are intriguing and provided support for the preparedness theory, it is still unclear what the implications are for the development of clinical phobias in humans. To make this theory clinically relevant, it needs to be demonstrated that phobias of phobic patients are of a prepared nature. In retrospective analyses, it was found that most of the phobias of phobic patients could be classified as prepared both in Western (De Silva, Rachman, & Seligman, 1977; Zalfiropoulo & McPherson, 1986) and non-Western cultures (De Silva, 1988). However, results of a study by Merkelbach, Van den Hout, Hoekstra, and Van Oppen (1988) did not corroborate these findings. In their study among Dutch severe phobics, no evidence was found that most of the phobias were of a prepared nature.

PANIC DISORDER

Until the 1980s, panic had not been of central interest to behavioral researchers. In behavioral research, the emphasis was more on the avoidance

behavior (agoraphobia) than on the panic associated with it. Klein (1981) argued that panic was mediated by a discrete biological mechanism and that the emphasis in treatment should be on remedying the dysfunctional biological structure, preferably by tricyclic-antidepressants, rather than on dealing with the avoidance behavior.

Behavioral clinicians have been aware for decades that in a number of agoraphobics the development of the agoraphobia was preceded by an episode or a series of episodes of panic attacks, but—forced by the emphasis on panic in the biological psychiatry camp—only recently panic has been studied more directly. This research, more extensively discussed elsewhere (e.g., Ehlers & Margraf, 1989; McNally, 1990), emphasizes psychological factors in the development and course of panic disorder.

Interoceptive Conditioning

Both Van den Hout (1988) and Wolpe and Rowan (1988) suggested that panic may be acquired through interoceptive conditioning. According to Van den Hout (1988), anxiety is the aversive event and the associated bodily sensations act as conditioned stimuli. As a result of repeated pairings between panic and bodily sensations, the latter evoke anxiety as a conditioned response. Similarly, Wolpe and Rowan (1988) hold that "the initial panic is an unconditioned response to a bizarre stimulus complex

produced by excessive hyperventilation, and panic disorder is the result of contiguous stimuli, especially endogenous stimuli, being conditioned to the elicited anxiety" (p. 441). The interoceptive conditioning model, however, is not without problems. First, as noted by Van den Hout (1988) the notion that panic patients fear bodily sensations is not unique for interoceptive conditioning but also plays a crucial role in the cognitive explanatory account of panic, a model in which catastrophic misinterpretations of bodily sensations account for the acquisition and maintenance of panic. Further, McNally (1990) has criticized the vagueness of the definition of the CS and UCS in the interoceptive conditioning model. It is indeed difficult to define exactly which interoceptive cue might be regarded as UCS or CS, making it difficult to verify the theory.

Separation Anxiety

It has been suggested that childhood separation from parents and associated anxiety are precursors of panic disorders in adults. The separation anxiety hypothesis has some face validity, given the apparent similarities between the two conditions. A number of studies suggested that panic patients or agoraphobics were in childhood more frequently separated from their parents than patients with other anxiety disorders, like generalized anxiety and social phobia (Persson & Nordlund, 1985; Raskin, Peek, Dickman, & Pinsker, 1982). There is no evidence, however, that actual separation in

childhood occurred more often in panic patients or agoraphobics than in simple phobics (Thyer, Himle, & Fischer, 1988) or in patients with other psychiatric disorders (Van der Molen, Van den Hout, Van Dieren, & Griez, 1989). Further, Raskin et al. (1982) did *not* find differences in separation *anxiety* in childhood. Similarly, Thyer, Nesse, Cameron, and Curtis (1985) and Thyer, Nesse, Curtis, and Cameron (1986) found no difference in childhood separation anxiety between panic patients and simple phobics. Finally, Van der Molen et al. (1989) found no more childhood separation anxiety in panic patients than in psychiatric controls and normal controls. In sum, although the results are inconclusive, there is little support yet for the notion that childhood separation and separation anxiety are precursors of panic or agoraphobia in adults.

Life Events

Life events have been hypothesized to be related to the onset of panic. Results of studies in this area are inconclusive: some (e.g., Faravelli, 1985; Hibbert, 1984; Last, Barlow, & O'Brien, 1984; Ost & Hugdahl, 1983; Ottaviani & Beck, 1987; Sheehan, Sheehan, & Minichiellon, 1981) found an excess of life events before the onset of the panic disorders or agoraphobia, but others did not (e.g., Roy-Byrne, Geraci, & Uhole, 1986). Foa, Steketee, and Young (1984), reviewing the literature, found that the most frequent stressors preceding agoraphobia were loss of a significant other and physical threat. Kleiner and

Marshall (1987), however, found marital conflict and family conflict as the two most frequent precipitants. Research in this area has a number of problems. Both the onset of the panic disorder and the occurrence of the life events can often not be dated exactly. Further, most studies did not involve a control group, thus it is unclear whether this incidence of life events exceeds that in a normal population. Moreover, even though a number of stressors are involved, they cannot completely account for the development of panic disorders, because (1) such stressors have also been found to be associated with other psychiatric and psychosomatic disorders, and (2) many individuals who experience these stressors do not develop any disorder at all.

How does stress relate to the development of panic disorders in behavioral terms? The most parsimonious explanation is in terms of interceptive conditioning. Anxiety, as a result of such stressors, may result in a panic attack, which may be experienced as coming "out of the blue." As a consequence, persons with such unexpected attacks may develop fear of somatic sensations associated with a panic attack (e.g., heart palpitations, shortness of breath) and become quite sensitive to somatic manifestations, which can result in a vicious circle. They tend to interpret somatic sensations as evidence of a serious medical problem ("catastrophizing"), which will increase the anxiety level and may accumulate into other panic attacks.

Panic and Agoraphobic Avoidance

Theoretically, one would expect that patients who experience panic attacks in a particular situation will try to escape that situation and will avoid that situation subsequently. Indeed, for most patients, panic precedes the development of agoraphobic avoidance behavior (Lelliott, Marks, McNamer, & Tobema, 1989; Rapee & Murrell, 1988; Schneieret al., 1991; Thyer & Himle, 1985). According to the two-stage theory, a linear relationship between severity of the panic attack and severity of the avoidance behavior is expected. Clum and Knowless (1991) reviewed the research in this area and found support neither for the hypothesis that severity of attacks was related to severity of avoidance, nor that more frequent attacks and attacks that persist over a long period of time predicted severity of avoidance behavior. There is, however, some evidence that cognitions are related to avoidance. Anticipation of panic in specific situations leads to avoidance of that situation. As phrased by Rachman (1991b), "They do not engage in avoidance behavior simply because of past panics; rather . . . they avoid because of their current prediction that they are likely to panic in a particular place during a particular period of time" (p. 188). Further, strong expectations of negative (social) consequences were found to be related to avoidance of situations in which these consequences may occur. In this respect, results of a study of Rapee and Murrell (1988) are also of interest. They found that panic disorder patients with extensive avoidance were less assertive, less extravert, and more socially anxious than patients with minimal avoidance. More recently, Robinson and

Brichwood (1991) found that panic patients with marked avoidance have significantly stronger social-evaluative concerns. There is some evidence that gender is related to avoidance. The prevalence of female panic patients with avoidance behavior is four times as high as the prevalence of panic disorder with avoidance among males, whereas panic disorder without avoidance behavior is more equally distributed across the sexes. The reasons why more females develop avoidance behavior after a (series of) panic attack(s) are unclear. Chambless (1989) found that severity of avoidance behavior was associated with femininity scores on measures of gender role.

SOCIAL PHOBIA

Few studies have been conducted on the etiology and maintenance of social phobia—an irrational and excessive fear of social evaluation. It has been suggested that this state of affairs is caused by a number of factors, for example, lack of recognition of the disorder and symptom overlap with other anxiety disorders (Bruch, 1989) and problems in distinguishing social phobia from "normal" social anxiety on the one hand (Scholing & Emmelkamp, 1990) and from related disorders like avoidant personality disorder or shyness (Turner, Beidel, Dancu, & Keys, 1986) on the other. Initially, fear of social situations was conceptualized as a conditioned response, acquired by traumatic experiences in social situations (Wolpe, 1958). Another influential paradigm, more exclusive for social phobia, is the skills deficit model, in

which a central role is ascribed to inadequate social skills, provoking aversive reactions from other people, which in turn lead to anxiety (Trower, Bryant, & Argyle, 1978). In fact, the models are not mutually exclusive. They both emphasize traumatic or aversive experiences in social contacts, the skills deficit model placing more emphasis on the origin of those experiences. Results from studies that were conducted to test the models are equivocal. Some support for the conditioned response hypothesis was found by Ost and Hugdahl (1981), who found that 58% of social phobics recalled a traumatic social experience before the onset of the complaint. As was noted earlier, results from these studies should be interpreted cautiously, because memories often yield a distorted picture of the past, and because results must be compared with results about childhood memories of normals. In addition, results of such studies are heavily dependent on definitions of what qualifies as a conditioning event. Apart from this, the classical conditioning explanation has other shortcomings. Although this paradigm can explain the development of a phobia after a traumatic experience, the model is inadequate in explaining the gradual development of social phobias that is often reported by social phobic patients (Amies, Gelder, & Shaw, 1983). Evidence for the skills deficit model was partly inferred from the fact that social skills training led to a decrease of fear in social situations (Marzillier, Lambert, & Kellett, 1976). However, the effectiveness of social skills training is on its own not sufficient to conclude that inadequate skills play a role in the etiology of the

complaints. Results of studies investigating whether social phobics are less socially competent than normals are inconclusive (Arkowitz, 1977; Beidel, Turner, & Dancu, 1985; Dow, Biglan, & Glaser, 1985), and it has been suggested that social skills deficits are of less importance in the etiology of social anxiety than once thought (Edelmann, 1985; Newton, Kindness, & McFudyen, 1983). As a consequence of the shortcomings of the conditioned anxiety and skills-deficits models in explaining the origin of social phobia, attention shifted to more cognitively oriented models (Hartman, 1983; Leary, 1988; Lucock & Salkovskis, 1988), which will be discussed in Chapter 5 of this volume. However, the recent developments and refinements of the conditioning hypothesis, placing more emphasis on indirect ways of conditioning and cognitive representations of conditioned stimuli, may be especially useful for social phobics. Although onset age of social phobia was found to be early adolescence (Marks & Gelder, 1966; Ost, 1987), it has been suggested that factors predisposing individuals to such fears originate from early socialization processes. The fact that parental attitudes may play a prominent role in these processes has led to family studies and research on child-rearing practices.

Windheuser (1977) found a remarkable similarity between phobias of children and those of their mothers, especially for social phobics. Bruch, Heimberg, Berger, and Collins (1989) also reported that parents of social phobics avoided certain social situations. These results could be interpreted

in terms of vicarious learning.

Buss (1980) stated that social anxiety is the result of negative childhood and adolescent experiences in situations in which evaluation by other people plays a major role. He assumed specific parental rearing styles to be responsible for hypersensitivity for social evaluation. As a matter of fact, studies on parental rearing styles have yielded differences in perceived parental behaviors between social phobics and agoraphobics. Parker (1979) found that social phobics (retrospectively) described both of their parents as showing little affection and being overprotective, whereas agoraphobics only reported that their mother had been low on emotional support but not overprotective. This finding led Parker to conclude that "... parental overprotection, by restricting the usual developmental processes of independence, autonomy and social competence, might further promote any diathesis to a social phobia . . .” (p. 559). Arrindell, Emmelkamp, Monsma, and Brilman (1983) compared memories of parental rearing styles of social phobics and agoraphobics, and found the same results as Parker did. Bruch et al. (1989) again found differences between social phobics and agoraphobics in that the social phobics reported that their parents had isolated them from social events and often worried about other people’s opinions of the family.

GENERALIZED ANXIETY

There is no specific behavioral theory with respect to the development of generalized anxiety disorder (GAD). GAD is often conceptualized as a life-long characteristic, so if learning factors are involved, these have to be located in childhood. Not surprisingly, the few studies into the etiology of GAD are of a retrospective nature (Rapee, 1991) and have no direct bearing on an interpretation of the development of GAD in terms of learning theories. Since GAD does not have a specific focus, but is characterized by free-floating anxiety, it has many similarities with trait anxiety (Eysenck & Mathews, 1987), which is defined as an individual's disposition to perceive a wide range of stimulus situations as dangerous or threatening (Spielberger, 1972). Recent experimental work has considered the information processing of GAD patients. Mathews and his colleagues consistently have shown in a number of laboratory experiments that selective attentional attraction to threat cues is characteristic for generalized anxiety patients (Mathews, 1989). It remains to be shown whether selective emotional processing is cause, consequence, or just one of the cognitive symptoms of GAD. A detailed discussion of these cognitive studies is outside the scope of this chapter.

Another important theoretical development from a behavioral perspective are studies into worrying (Borkovec & Inz, 1990). Worrying is one of the characteristic components of GAD. According to DSM-III-R (APA, 1987), GAD is characterized by unrealistic or excessive anxiety and worry (apprehensive expectation) about two or more life circumstances, for

example, worry about possible misfortune to one's child (who is in no danger) and worry about finances (for no good reason), for a period of six months or longer, during which the person has been upset more days than not by these concerns. Worries are characterized by having a continuous stream of thoughts and images with respect to future negative events and the (un)ability to cope with them. The focus of the anxious apprehension is usually rather diffuse and often on "daily hassles." Such persons are oversensitive to relatively minor events. Borkovec and his colleagues found, however, that worriers were unable to solve the problem or come to a definite solution. Worriers are further differentiated from nonworriers in terms of distraction: worriers are unable to stop their ruminations and to engage in distracting thoughts, images, or activities. It is tempting to assume that such worrying increases arousal which in turn increases the worrying so that the individual is no longer able to shut off worry activity. According to Borkovec and Inz (1990), this vicious circle is self-perpetuating. Actually, the worrier may have the illusory feeling that by worrying he or she is doing something to deal with the negative future events (Barlow, 1988). It has further been suggested that worry "may represent an avoidance of affect in general or emotional experience in particular" (Borkovec & Inz, 1990, p. 158). By engaging in worrying, actual exposure to fearful situations is prevented, resulting in maintenance of the anxiety. Thus, worrying is conceptualized as an inadequate coping device (cognitive avoidance), which impedes emotional

processing of fear stimuli and actually maintains GAD.

Barlow (1988) has argued that GAD is the end result of a process in which multiple etiological factors are involved. According to Barlow (1988), GAD patients have a biological vulnerability and experience external stressors (life events and daily hassles) as uncontrollable and unpredictable, eventually culminating in a spiral of worrying. Although this model has some appeal, it is far from proven yet.

POST-TRAUMATIC STRESS DISORDER (PTSD)

According to Foa, Steketee, and Olasov Rothbaum (1989), it is tempting to consider PTSD as a prototype for etiology and symptomatology of phobia. " . . . there is a recognizable traumatic stimulus, following which an individual shows fear reactions when confronted with situations associated with or similar to the original trauma" (p. 156). As they themselves note, however, there are many important differences between phobias and PTSD. Anxiety is sufficient for the diagnosis of phobia, whereas in PTSD other emotions as hostility and numbness of feelings are also important. Other characteristic symptoms of PTSD as flashbacks, nightmares, sleep disturbance, startle responses, and feeling of detachment from others are not characteristic of phobias.

Most behavioral researchers use Mowrer's two-stage theory to explain

post-traumatic stress disorder (e.g., Kilpatrick, Veronen, and Best (1985) on PTSD in rape victims and Keane, Zimmerling, and Caddell (1985) on PTSD in Vietnam veterans). Two other learning processes are also involved; higher order conditioning and stimulus generalization. In higher order conditioning stimuli that were originally conditioned to the traumatic event are paired with other unconditioned stimuli, which eventually may result in a new conditioned response. Through this process of higher order conditioning, many stimuli, including thoughts and images, may evoke anxiety. Stimulus generalization refers to the tendency of an organism to transfer its acquired response to new stimulus situations as a function of stimulus similarity. Taken together, these two learning processes may explain the gradual worsening of symptoms over time. As a result, often many more cues than actually present at the time of the traumatic event are capable of eliciting traumatic memories and emotions. Further, Keane et al. (1985) stressed the importance of lack of social support in the development of PTSD. They hypothesize that patients who have an adequate social support system are less likely to develop a (severe) PTSD than patients who have less adequate or no social support at all.

The conditioning explanation of PTSD has some appeal, but is not without problems. Why does exposure to traumatic memories as occurs in re-experiencing the original trauma not lead to habituation and extinction of anxiety? It has been suggested by Keane et al. (1985) that such exposure is

incomplete because not the whole stimulus complex is included. Indeed, most PTSD sufferers tend to avoid thinking of important aspects of the traumatic situations because of the aversiveness of the situation and the anxiety it evokes. Thus, it does not come as a surprise that such occasional exposure is ineffective. In addition, patients are inclined to shut off memories and re-experiences when these occur, resulting in a too short exposure time to be effective (Emmelkamp, 1982). Further, the hostility, numbness of affect, startle response, and the occurrence of nightmares are not easily explained in conditioning terms. Part of these problems may be solved by taking into account research on experimental neurosis. Mineka and Kihlstrom (1978) interpreted results of studies into experimental neurosis in terms of uncontrollability and unpredictability of the stimuli. Many of the symptoms of laboratory animals who are exposed to a loss of predictability or controllability, symptoms such as agitation, sudden outbursts of aggressive behavior, passivity, and lethargy, resemble characteristics of PTSD (Foa et al., 1989). Foa et al. (1989) suggest that other characteristic phenomena in PTSD as re-experiencing of the trauma via intrusive thoughts, images, flashbacks, and nightmares are better accounted for by information processing theories. Discussion of these theories, however, is outside the scope of this chapter (see Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988; Foa et al., 1989; and Litz & Keane, 1989).

OBSESSIVE-COMPULSIVE DISORDER

In obsessive-compulsives, it is useful to differentiate between active and passive avoidance. With passive avoidance, the individual avoids stimuli, situations, and so on, that might provoke anxiety and discomfort. Active avoidance usually refers to the motor component of obsessive-compulsive behavior, for example, checking and cleaning. Active avoidance can be explained by the escape learning paradigm, whereas passive avoidance fits the avoidance paradigm. Washing, cleaning, and checking can be regarded as escape responses, in the sense that performance of the washing ritual terminates anxiety.

The criticism of the process learning theory of fear acquisition applies equally well in the case of explaining obsessive-compulsive behavior.

As to the classical conditioning component of the two-stage theory, there is little evidence that this type of learning plays a crucial role in the development of obsessive-compulsive behavior. According to a classical conditioning interpretation, a traumatic event should mark the beginning of the obsessive-compulsive disorder. An analysis of the history of our obsessive-compulsive cases revealed that in a significant number of cases the onset of the obsessive-compulsive behavior was gradual. Generally speaking, patients related the onset of their problems to life stress in general rather than to one or more traumatic events (Emmelkamp, 1982). Further, many

patients do not mention traumatic experiences associated with the onset of the symptoms. When such traumatic events were reported, they often took place much earlier than the onset of the obsessive-compulsive problems, thus making an explanation in terms of classical conditioning less credible. Finally, clinical observations clearly demonstrate the occurrence of several obsessions together as well as the regular change of obsessions in some patients, unrelated to new traumatic learning experiences. Based on patients' accounts of the course of the problem, it can be assumed that in a stressful period ritualistic activities have powerful anxiety-reducing effects.

Although there is little evidence that classical conditioning plays an important role in the development of obsessive-compulsive behavior, there is some evidence that the rituals may serve to reduce anxiety. Rachman and Hodgson (1980) studied the provocation of compulsive acts and the effects of performance of the rituals under controlled laboratory conditions to test the anxiety-reduction theory of obsessive-compulsive behavior. The design of these studies was usually as follows: Obsessive-compulsive behavior was provoked and measurements of subjective anxiety were taken before and after provocation, and after performance of the (checking or cleaning) ritual. In addition, patients' reactions were tested when the performance of the ritual was interrupted and when it was delayed.

The results of these studies can be summarized as follows. With patients

whose primary problem was obsessive-compulsive washing arising out of fears of contamination or dirt, contamination led to an increase of subjective anxiety/discomfort, while the completion of a washing ritual had the opposite effect. Spontaneous decrease in discomfort occurred when the performance of the hand-washing ritual was postponed for half an hour. The interruption of the ritual produced neither an increase nor a decrease in subjective anxiety/discomfort (Rachman & Hodgson, 1980). The results of studies on checkers were along the same line but more variable. Taken together, the findings of these studies support the anxiety-reduction theory, as far as the maintenance of obsessive-compulsive behavior is concerned. With only a few exceptions among checkers, provocation of rituals led to an increase in subjective anxiety/discomfort and performance of rituals reduced discomfort.

Rachman (1976) postulated that differences in ritualistic behavior arise from differences in rearing practices. According to this theory, checking rituals are most likely to arise from families where the parents set high standards and are over-critical. Checking compulsions can therefore be identified with active avoidance behavior in order to avoid errors, motivated by fear of criticism or guilt. On the other hand, cleaning rituals will emerge in families where the parents are over-controlling and overprotective. Cleaning rituals can be considered as passive avoidance behavior, in order to avoid danger or anxiety-provoking situations in which the coping abilities of the patient might not be sufficient. Four studies have been reported to test this

theory. Turner, Steketee, and Foa (1979) investigated whether checkers are more sensitive to criticism than washers. No differences in fear of criticism were found between washers and checkers. Using the same checklist, Thyer, Curtis, and Fechner (1984) reported no differences between obsessive-compulsives, agoraphobics, and social phobics; some slight differences appeared between these patient groups and simple phobics. Steketee, Grayson, and Foa (1985) found that checkers more often perceived their mothers as meticulous and demanding than washers did.

In a study by Hoekstra, Visser, and Emmelkamp (1989), Rachman's theory was tested on a large sample of obsessive-compulsives, using a validated questionnaire to assess rearing practices (Perris, Jacobsson, Lindstrom, Van Knorring, & Perris, 1980). The results partially supported Rachman's theory: Washers reported a more overprotective father than checkers. They also rated their mothers as more rejecting than checkers, which is not in line with the theory. Taking together the four studies discussed, there is insufficient evidence yet to support a rearing practice specificity in the etiology of compulsive rituals.

Emmelkamp and Rabbie (1982) and Hoekstra et al. (1989) postulated that those occupations for which persons hold themselves responsible are crucial for the type of compulsions they might develop. If activities are in the area of hygiene and tidiness (e.g., housekeeping, nursing) those persons are

expected to develop cleaning rituals. On the other hand, people who have to be punctual and accurate at work (e.g., administrator) are expected to develop checking rituals. According to this theory, the profession will have a great influence on the type of compulsion that develops. In most Western societies, women are responsible for housekeeping and according to this sex-role pattern one may expect more women to exhibit cleaning rituals than men. Both in the Emmelkamp & Rabbie (1982) study and in the Hoekstra et al. study (1989), clear support was found for this hypothesis. In the Hoekstra et al. (1989) study, for most of the patients the type of compulsion could be predicted by their profession.

CONCLUDING REMARKS

The emphasis in this chapter has been on behavioral factors involved in the etiology and maintenance of anxiety disorders. As discussed, learning theories are inadequate as a uniform theory for the development of anxiety disorders. Even in the case of phobias and post-traumatic stress disorder, centered around clear stimuli that trigger the anxiety, simple learning theories are inadequate in explaining the acquisition and maintenance. In other anxiety disorders like obsessive-compulsive disorder, generalized anxiety, and social phobia, there is even less evidence that conditioning plays a crucial role. Recent developments have stressed cognitive representations and cognitive schemata as important determinants of anxiety disorders. For

example, Beck, Emery, and Greenberg (1985) hold that cognitive schemata and automatic thoughts that are typical of anxiety play a central role in the etiology and maintenance of anxiety disorders. Other research has shown that biological factors may be involved in the etiology of anxiety disorders. Although discussion of these developments was outside the scope of this chapter, we wholeheartedly agree with an interactional perspective, in which biological, cognitive, and behavioral factors all play an important role. There are likely to be several factors operating at different levels and influencing each other in the etiology and maintenance of anxiety disorders. A comprehensive account of the acquisition and maintenance of fears and anxiety disorders is much more complex than once thought and needs to integrate biological, behavioral, and cognitive factors.

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