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The Origins of Specific Phobias: Influential Theories and Current Perspectives

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Fears are quick and adaptive responses that permit powerful reply to imminent threats. Less adaptive, phobias are extreme manifestations of fear to objects or situations in the absence of a proportional danger. Although the utility of fear is accepted, the nature of phobias is controversial. Initial theories favored a fear conditioning-based explanation, with vicarious and information learning pathways subsequently included as additional routes to the development of specific phobias. More recently, an important group of investigations strengthened the case for a nonassociative account of fear acquisition proposing that evolutionarily relevant fears can occur without any need of critical learning experiences. In parallel, there is some evidence for a dedicated fear module in the detection of threats, involving the amygdala, which is relatively independent from conscious cognitive control. Nonetheless, cognitive models stress learning and developmental factors and their role in the etiology and maintenance of phobic behavior. This article critically reviews each of these views and theories stressing their recent developments, weaknesses, and controversies with an aim to provide the groundwork for the construction of a more integrated position. Finally, the authors suggest encouraging trends in recent research.

Keywords: specific phobias, etiology, fear, emotion, development

Initial theories regarding the acquisition of specific phobias favored a fear conditioning-based explanation (Watson & Rayner, 1920). Rachman (1978) added vicarious and information learning as additional pathways leading to the development of specific phobias. The behavioral/learning models of phobias, understood in terms of traumatic experiences and aversive classical conditioning, persisted from the early 1920s until around the 1970s. Nonetheless, behavioral and cognitive models continued to improve, and new perspectives emerged. Recently, Poulton and Menzies (2002a, 2002b) suggested that evolutionarily relevant fears could manifest without any need of critical learning experiences. In parallel, Öhman and Mineka (2001) proposed a model in which the amygdala and the hippocampus have emotional and cognitive functions, respectively, allowing an encapsulated and automatic fear response to certain threats.

Among these perspectives, the two distinct and opposite frameworks are the associative and the nonassociative accounts that propose either learning or innate nature as wholly responsible. The associative perspective holds that fear and phobias occur mainly as a consequence of learning experiences, whereas the nonassociative perspective assumes that some fears and phobias reflect an innate spontaneous reaction to relevant evolutionary cues. Whereas a preparedness viewpoint allows that a learning episode is required to initialize an innately hardwired response, in its more extreme version, the nonassociative point of view assumes that certain fears, such as fear of heights and water, represent evolutionarily relevant threats that may evoke fear without the need of critical learning experiences. This perspective has recently gained support from data accrued in longitudinal studies of common fears (e.g., Poulton & Menzies, 2002a).

This article provides an overview of these perspectives on the etiology and development of fears and phobias as well as the contribution of recent findings that, once merged, can contribute to the foundation of an integrated model of fear and phobias.

1. The Classical, Vicarious, and Informative Pathways for Fear Acquisition

The classical conditioning model of specific phobias had its origins in animal research laboratories and gained strength in the second half of the 20th century. Watson and Rayner (1920) observed that they could teach (i.e., condition) an animal or a child to respond with fear to a harmless situation by repeatedly linking a harmless conditioned stimulus (CS) with a frightening unconditioned stimulus (US). Given that participants with specific phobias had unrealistic fears of harmless situations or stimuli, it was suggested that this behavior could be the result of a similar process and, therefore, that phobias were instances of conditioned fear (see Fyer, 1998). Later, Mowrer suggested a theory of fear in which the development of avoidance is crucial to the persistence of conditioned anxiety by preserving the exposure to the CS and preventing the extinction of fear responses (Mowrer, 1960).

Wolpe (1958) used Mowrer's theory in phobia treatments, creating the well-known systematic desensitization treatment and providing a rationale for behavioral therapies. With some variations (see Field, 2006), the behavioral/learning models of phobias, understood in terms of traumatic experiences and aversive classical conditioning, persisted from the early 1920s until around the 1970s. The theory was exhaustively tested with the conclusion that it was easy to generate fear reactions in animals by exposing them

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to a set of neutral and aversive stimuli, usually electric shocks. Modulation in the strength of the fear was explained in terms of the number of repetitions of the association (s-r) and by the intensity of fear or pain felt when in the presence of the stimulus. Moreover, fears were said to generalize to stimuli that had similar properties (Rachman, 1991). In the late 1970s, more complexity was added to this model with the addition of the vicarious and informative acquisition pathways.

In keeping with Bandura's (1977) findings and social learning theory, Rachman (1978) suggested vicarious conditioning as an important factor in phobia acquisition and added that information and instructions from parents and other family members influenced the acquisition of fear. After numerous studies, nontraumatic experiences were increasingly viewed as an important part of models, especially when describing the etiology of phobias in childhood. Nowadays, the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*) acknowledges traumatic events, unexpected panic attacks, modeling, and information transmission as processes that may facilitate the development of phobias (American Psychiatric Association, 1994).

Perhaps one of the most important and cited contributions to the role of vicarious conditioning in phobia acquisition comes from Mineka and Cook's experiments (e.g., Cook & Mineka, 1989; Cook & Mineka, 1990; Mineka & Cook, 1986). In 1986, Mineka and Cook assessed 22 rhesus monkeys that observed, on video, other monkeys of their species reacting with intense fear to relevant stimuli (snakes or artificial crocodiles) and irrelevant stimuli (flowers or artificial rabbits). The primates that observed the videos had no previous experience with any of the stimuli. The research confirmed that, whereas observer monkeys did not initially show fear to any of the stimuli, after 12 sessions they had acquired fear of evolutionarily relevant stimuli but not of nonevolutionarily relevant stimuli. Moreover, Mineka and Cook noted that when they exposed monkeys to other monkeys that interacted with snakes without showing fear, this group did not acquire fear after a subsequent exposure to phobic models. This effect was even stronger than the latent inhibition effect (simple exposure to snakes by themselves). A later experiment indicated that participants acquired a fear of fear-relevant stimuli (toy snakes and toy crocodile), but not of fear-irrelevant stimuli (flowers and toy rabbits; Cook & Mineka, 1989). This vicarious conditioning occurred simply through watching videotapes of models behaving fearfully (Cook & Mineka, 1990). This research provided strong evidence that fear can be learned vicariously, although it also addressed the notion of preparedness (Seligman, 1971), which claimed that certain stimuli are evolutionally predisposed to evoke fear responses. This concept is addressed later.

Reinforcing the importance of vicarious learning, a study with 40 children (25 boys, 15 girls) between 9 and 12 years of age (Muris, Steerneman, Merckelbach, & Meesters, 1996), found a significant positive relationship between the mother's and the child's fears and a relationship with the frequency with which the parent usually expressed their fear in front of their children. Those who showed fear more frequently had more fearful children; mothers who did not express their fears had less fearful children, and those who showed moderate fear had children with moderate levels of fearfulness.

Ollendick and King (1991) explored Rachman's model with the help of 1,092 Australian and American children between 9 and 14 years of age. Similarly to other studies, the results indicated an important role for information and modeling. Most of the children attributed the beginning of their fears to informative and vicarious factors (56% and 39%, respectively) more than to direct conditioning events (37%). The majority of high fear levels in these children resulted from a combination of learning sources, suggesting that fear is more likely to develop as a result of synergistic effects of various sources or pathways. In a study by Merckelbach, Arntz, and de Jong (1991), 42 participants identified as having spider phobia, completed the Phobic Origins Questionnaire by Öst and Hugdahl (1981), which offers three response alternatives, and found results that favored modeling (71%) as the main learning experience for this type of fear. Direct conditioning experiences were recalled by 57% of participants, and 45% mentioned the information pathway. Once again, the majority of fears (57.1%) were attributed to more than one cause, and a combination of modeling and conditioning (47.6%) was the most commonly mentioned dual cause. However, when Öst (1991) assessed participants ($n = 137$) classified with blood or injection phobia, 52% of the participants stated conditioning experiences as the cause of their phobia, 24% recalled vicarious experiences, 7% recalled negative instructions/information, and 17% did not recall specific circumstances of fear initiation. These studies suggest that different types of fears may be produced or magnified by different pathways.

Several studies also support the influence of information in fear acquisition. In a study by Field, Argyris, and Knowles (2001), 40 children between 7 and 9 years of age were exposed to novel stimuli (dolls/monsters). Before the exposure, positive or negative information was given, either through telling a story or by a video that presented a woman interacting positively or negatively with the monsters. Later, in questioning the children about the monsters, it was demonstrated that the type of information given influenced the children's beliefs regarding the new stimulus. The verbal information was more effective than the video presentation. Recently, Lawson, Banerjee, and Field (2007) also explored the role of verbal information in fear-related beliefs about social situations and found support for the effect of verbal information, particularly when the information given was negative.

Fear acquisition through the visual observation of another's fear, the vicarious dimension proposed by Rachman (1978), is also well documented. Olsson and Phelps (2004) found in a functional magnetic resonance imaging (fMRI) study that observational fear learning (a partner undergoing fear conditioning) resulted in activation of the bilateral amygdala both when the presentation was supraliminal and when it was subliminal. According to Phelps (2006), these results evidence a neural substrate for the acquisition and expression of fears acquired through the vicarious experience of social observation. These results favor Bandura's (1965, 1971, 1977) pioneer work in vicarious learning, substantiating the idea that no direct consequences need to be delivered to the observer for acquisition to occur and that the principles of reinforcement and punishment operate when consequences are delivered to another individual. Additionally, Olsson and Phelps (2004) showed that fear learning after observation does not need to be accompanied by explicit awareness.

In summary, this research supports that conditioning, vicarious experience, and information can provide avenues for fear acquisition. The three possible routes postulated by Rachman are likely interactive, and in most cases, more than one is implicated in the

etiology of a fear. As is subsequently presented, this model attracts similar criticisms as the earlier conditioning model that reflect on the powerlessness of these early learning approaches to account completely for the varied factors related to the origins of human fears.

1.1 Critique of the Conditioning and Vicarious/Informative Models

The conditioning models show considerable face validity, despite the difficulty in finding all the components needed for the occurrence of a specific phobia in an individual (see Rachman, 1991, for a review). However, studies by English (1929), Bregman (1934), and Valentine (1930) failed to reproduce Watson and Rayner's (1920) findings (but see Delprato, 1980, for a critical review). In fact, there are difficulties in extrapolating fears and phobias that are conditioned in a laboratory to those in the real world. As a largely animal model, the conditioning theory was criticized as to its generalization to humans and to situations outside the laboratory (Rachman, 2002). Clinicians often have difficulty in discovering aversive conditioning events, which is not surprising given that this requires the identification of the US and its unconditioned response and discovering when and where there was a matching between the US and the CS (Herbert, 1994).

Rachman's (1978) model attracts similar criticisms. First, a significant number of participants with phobias do not remember traumatic or conditioning events that might have contributed to fear acquisition (e.g., Graham & Gaffan, 1997; Kleinknecht, 1994; Menzies & Clarke, 1993a, 1995; Ollendick & King, 1991). Second, a small number of stimuli (e.g., snakes, spiders, heights, thunderstorms, dark, blood, strangers, social scrutiny, separation and distancing from home; Marks & Nesse, 1994) comprise almost all phobias without any consistency in distribution (e.g., Mineka & Cook, 1986; Seligman, 1971). Third, not all individuals who have aversive encounters with stimuli develop a phobia (e.g., Gafford, Silva, & Langley, 1996; Poulton, Davies, Menzies, Langley, & Silva, 1998; Poulton & Menzies, 2002a, 2002b). Finally, phobias have proven hard to train in laboratory settings, and the effects of training tend to be weak and transitory (e.g., Mineka & Öhman, 2002b; Öhman, 1996; Rachman, 1991).

Although the addition of the informative and vicariant learning pathways improved the robustness of the classical conditioning model, certain researchers (e.g., Mineka & Zinbarg, 2006; Rachman, 1991) stressed the need to also recognize the concept of preparedness to answer some of the questions that classical conditioning was unable to answer. This perspective proposed that some individuals may be more or less biologically prepared to acquire some disorders. Thus early learning histories, when considered together with temperamental vulnerabilities, could serve as diatheses that make certain individuals more or less susceptible to the adverse experiences that lead to the development—or lack of development—of anxiety disorders (Mineka & Zinbarg, 2006).

In parallel with the advances in the behavioral models was the increasing popularity of cognitive models, which peaked with the idea that fears may be acquired from a cognitive perspective, in absence of any direct conditioning. The emergence of a neoconditioning perspective precipitated this new viewpoint, which had as its main feature the recognition of the occurrence of noncontiguous conditioning. Researchers noticed that they could develop conditioned responses even when the CS and the unconditioned event were separated in time (see Mackintosh, 1983). This finding removed most of the objections to the conditioning theory of fear acquisition (Rachman, 1991). However, before going further into the cognitive perspectives of fear, we explore the notion of preparedness more fully.

2. The Preparedness Framework

From the biological perspective of preparedness, particular stimulus configurations are evolutionally predisposed to evoke fear responses in certain animals. Seligman (1971) initially proposed this account as a reformulation of the conditioning model. Seligman found that human fears are not randomly distributed (see Herbert, 1994; Rachman & Bichard, 1988), challenging the traditional conditioning theory that assumed the equipontential acquisition of fears—that any stimulus can acquire the capacity to evoke a fear response. Seligman (1971) proposed that the premise of equipotentiality was flawed and that associations such as taste and nausea (see Garcia & Koelling, 1966), which have an obvious survival benefit, may represent associations biologically prepared to be learned. Thus CS–US associations operated along a continuum that Seligman termed *preparedness*.

It did not take long before the notion of prepared associations was applied to the learning of phobias. According to Seligman, ontogenetic and phylogenetic selection creates tendencies to respond with fear to certain threatening stimuli, favoring the survival of some characteristics of given species through the course of evolution. As such, the most common fears are more stable, more easily acquired, or both. This theory can account for the phobia distribution, as the more commonly feared stimuli are usually evolutionarily old threats (Marks & Nesse, 1994). Nowadays, the *DSM–IV* highlights this fact, noticing that the feared objects or situations tend to involve situations that might have presented a threat in some point of human evolution (American Psychiatric Association, 1994).

This appreciation is not recent. In 1877, Charles Darwin noticed that some fears might appear by natural selection. After observing his 2-year-old son being afraid of large animals in the zoo, Darwin questioned whether fears in children, that seem quite independent of experience, are effects of the hereditariness of real dangers during prehistoric times (Darwin, 1877). More recently Rachman (2002) included the preparedness model in his view of fear acquisition. In his perspective, the child gradually acquires the competencies needed to deal with the existent predispositions and actual fears by habituation and experience. The remaining fears are those that are most resistant to extinction or habituation and those acquired through conventional learning processes, the uncommon or rare fears. The environment can work, therefore, toward eliminating biologically relevant fears. Additionally, the same vicariant and informational processes that are at work in building fears can extinguish them. Rachman's three-route theory is both compatible and complementary with that of Seligman. People with more opportunity for contact with (harmless) feared stimuli should have less fear than those who never or rarely deal with these stimuli. In fact, urban children tend more to be afraid of snakes than rural children, and during World War II, rural populations were more afraid of air strikes than urban ones, who were repeatedly bombarded (Rachman, 1991, 2002).

2.1 Critique of the Preparedness Model

The main challenge to Seligman's perspective is in the identification and discrimination of plausibly prepared fears. Certain fears, such as the fear of spiders, are frequently referred to as "biologically prepared." However, only approximately 200 species of spiders worldwide can cause severe human envenoming, out of the more than 30000 species of spiders (Diaz, 2004). The contrary is true of mushrooms, which are usually viewed as not biologically relevant to fear. Nevertheless, some mushrooms, such as the *Amanita phalloides*, are one of the toxic agents most responsible for fatal cases in Poland (Kotwica & Czerczak, 2007), and unintentional poisoning with plants is common in small children (Eddleston & Persson, 2003). Approximately 100 species of poisonous mushrooms have been identified in the United States alone (Lincoff & Mitchell, 1977), which makes it reasonable to suspect that mushrooms have posed a greater threat to the survival of the human species than have spiders and snakes combined (Delprato, 1980).

Additionally, it seems important to identify the specific elements of stimuli that provoke a fear response:

> For example, what is it that water phobic individuals react to? Clearly it is not water per se as they likely seek water to drink, to bathe in, and to nourish their plants. Is it deep water? If so, how deep is deep enough? Or is it water in which they might drown, or in which they might not be able to negotiate their way to safety? (Kleinknecht, 2002, p. 162)

These etiological factors have implications in exposure techniques, which can be modified to address the specific response tendency (e.g., Coelho, Santos, Silva, Tichoon, Hine, & Wallis, 2008). Muris, Merckelbach, de Jong, and Ollendick (2002) also drew attention to the dangers of formulating plausible evolutionary scenarios to justify the occurrence of prepared fears. The fear of insects, for example, could be considered an evolutionarily relevant fear, but historians remind us that we only discovered the role of insects in spreading diseases around 1900. That marked the beginning of a remarkable change in people's attitude toward insects, especially flies (Muris et al., 2002). Additionally, Seligman's model requires a conditioning episode for a prepared fear to be developed, which makes it also subject to the criticisms leveled at conditioning theory.

An alternative view of preparedness was soon proposed (e.g., Poulton & Menzies, 2002a), suggesting that many fears emerge from the very first time that the living organism meets the stimulus. This is the nonassociative theory of fear acquisition.

3. The Nonassociative Theory

The nonassociative model derives from the observation that each species seems to have certain fears that are part of their development. However, the theory hypothesizes that these might occur in individuals even without direct or indirect experience (e.g., information) with the phobic stimulus. Thus, the majority of members of a species will show fear to a set of relevant stimuli from the first encounter. This immediate fear response favored survival, compared with acquiring the fear from experience (Poulton & Menzies, 2002a; Stein & Bouwer, 1997). Recent work has demonstrated that numerous participants diagnosed with phobia consider that the origins of their fear go back to their first contact with the stimulus.

Menzies and Clarke (1993a) reported the first studies supporting the nonassociative model while investigating the origins of water phobia. The authors noticed that only 1 of 50 parents identified a classical conditioning episode to explain the emergence of their child's phobia, whereas 13 parents identified possible vicarious learning and none attributed the emergence to instruction/information. Menzies and Clarke proposed the nonassociative theory as an explanation for fear of water. Adding some support to this hypothesis, Graham and Gaffan (1997) assessed groups of children with and without fear of water and concluded that the groups did not differ significantly in the incidence of aversive experiences related to water. According to their mothers, the majority of children with water fear (7 of 9) had displayed fear since their first contact.

Additionally, Poulton, Menzies, Craske, Langley, and Silva (1999) noticed that participants with fear of water did not differ from those without this fear in the age at which they learned to swim, in the amount of water exposure (number of times they went swimming in the previous year), or in the age at which they had their first water-related accident, excluding latent inhibition as the possible mechanism that prevented fear learning. Such studies suggest that lesion, injury, or pain situations do not necessarily provide a sufficient incentive for fearless individuals to avoid dangerous situations or to learn to fear. Later, other studies explicitly studied links between aversive experiences and other fears claimed as evolutionarily relevant. Poulton et al. (1998) reported that participants with less fear of heights seemed to be those who had sustained more injuries due to falling (see also Menzies & Parker, 2001; Poulton & Menzies 2002b).

The nonassociative model therefore proposes that conditioning events are not required for the onset of fear responses to stimuli that have evolutionary relevance. Additionally, the model acknowledges that a number of common fears, such as dental fear, seem unlikely to have a basis in evolution. The nonassociative model integrates the associative model to explain fears with no apparent evolutionary relevance, stating that conditioning experience is required to acquire fear to evolutionarily irrelevant stimuli. Accordingly, Poulton, Thomson, Davies, Kruger, Brown, and Silva (1997) studied the relationship between conditioning experiences and fear through the severity of teeth cavities. The results convey a relationship between cavities at the age of 15 and phobia development to dentists, but not other fears, at the age of 18. Poulton et al. (1997) suggested that poor dental health that requires a more demanding treatment enhances the likelihood of aversive conditioning and seems to have a causal relationship with fear of dental treatment situations at 18 years of age. These results are seen by the authors to be consistent with conditioning theory and also with the nonassociative model as it relates to nonevolutionary fears. This model predicts that fears with no evolutionary basis, such as fear of dentists and fear of driving automobiles, must be connected with conditioning events.

Although Seligman's preparedness theory is sometimes grouped with nonassociative theories, there are important differences. The preparedness model assumes a latent, evolutionarily derived potential for certain stimuli to be associated with fear. Nevertheless this model does not predict that a fear response would be evident on first contact with a prepared stimulus. This differentiates pre-

paredness theories in that associative learning (by conditioning, vicarious learning, or information) is still required at some point of the species' and organism's learning history, but with a relevant match between conditioned stimulus and US, for fear acquisition. The nonassociative point of view omits the need for this ontogenetic learning (Menzies & Clarke, 1993b, 1995; Poulton & Menzies, 2002a) at least for some fears, like heights or water.

3.1 Critique of the Nonassociative Theory

Poulton and Menzies's studies ignore many crucial dynamics of children's fear development that have a strong impact on the results of any learning experience (see Mineka & Öhman, 2002a). Several studies support the role of control in predicting a vulnerability to fear and anxiety (Chorpita & Barlow, 1998; Craske, 2003; Mineka, Cook, & Miller, 1984; Mineka, Gunnar, & Champoux, 1986; Rapee, 1997), and many other additional factors can influence fear acquisition (e.g., the level of development as a child, trace anxiety, previous experience with uncontrollable events, the beliefs about the stimulus, the previous quantity of non-noxious experience, and fear and pain experienced during the event).

Besides the complexities of fear acquisition that are ignored by the nonassociative account, there are also noticeable methodological concerns with the questionnaire that assessed the origin of phobias used by Menzies and Clarke (1993b). Allowing a response choice such as "I was always this way" reinforces the hypothesis of nonassociative etiology. Failure to remember any experience related to the emergence of the disturbance inadvertently supports the nonassociative perspective, as this item might easily be interpreted as, "I don't know/remember how I became phobic," pointing to problems inherent to self-report and memory more than to a nonassociative etiology. Thus, this style of questioning inflates estimations of nonassociative etiology (Muris et al., 2002).

Retrospective recall carries problems recognized and acknowledged in the literature on traumatic events. Several of those problems can be related to the nonassociative model. The first question relates to the use of verbal reports. Since the Nisbet and Wilson (1977) studies, it is clear that verbal reports are not very useful to reveal causal processes, especially when learning and conditioning can happen without awareness (Öhman & Soares, 1994). The human memory is highly prone to changes or revisions based on experience (Loftus, 2004). Prospective studies, such as the one in Dunedin, New Zealand (see Gafford et al., 1996), are also a sequence of retrospective memories, and several studies suggest that memory reliability of true events decays in the space of weeks or months (Brainerd & Reyna, 2004), especially in children (Brainerd & Reyna, 1996), in whom false memories can be created and persist (Brainerd & Mojardin, 1998). The second issue is that phobias to stimuli such as water and heights might be the result of the accumulation of subtle, nontraumatic experiences (e.g., bathing and small falls at young ages) that are not very memorable (Graham & Gaffan, 1997) but that can model and influence long-term behaviors (Forsyth & Chorpita, 1997). These could give rise to a gradual fear (Emmelkamp, 1982; Forsyth & Chorpita, 1997), which is especially hard to attribute to direct conditioning. Poulton et al. (1999) also pointed to the existence of insidious conditioning associations that could result from a series of experiences rather than from a specific traumatic event. In fact, the notion that a series of pairings of a CS with a mild US may give rise to "silent learning" (de Jong, Muris, & Merckelbach, 1996, p. 228) is not recent. A subtle influence of the US has been noted in similar research with the inflation effect (Rescorla, 1974) and the effect of US reevaluation (Davey, 1997). A third and related question concerns the fact that direct conditioning experiences seem easier to memorize than indirect conditioning experiences (Field et al., 2001; Taylor, Deane, & Podd, 1999; Withers & Deane, 1995). A final and important aspect underlined by Muris et al. (2002) is related to the genetic implications of the nonassociative point of view. This theory should imply a substantial genetic contribution in the etiology of phobias; however, this has not yet been supported. For example, Kendler, Neale, Kessler, Heath, and Eaves (1992) concluded on the basis of the study of twins that, with regard to anxiety disorders, specific phobias present the lowest hereditability rates and the highest specific environmental influences. Thus, they concluded that, in simple phobias, pathogenic environmental experiences are generally highly specific (e.g., being locked in a dark room, being bitten by a snake, being close to falling out of a window; Kendler et al., 1992; see also Craske & Waters, 2005, for a review).

4. The Fear Module Theory

To account for the uneven distribution of fears, but discounting the notion of purely nonassociative fear acquisition, Öhman and Mineka (2001; see also Mineka & Öhman, 2002b) suggested the concept of an evolutionally created fear module, a system specifically adjusted to solve adaptive problems provoked by life-threatening situations in our distant ancestors' ecology. On the basis of Razran's (1971) study, the authors argued that higher levels of learning (e.g., cognitive and contingency learning) emerged in later phylogenic development, generating dissociable emotional and cognitive learning pathways (Öhman & Mineka, 2001). The first (emotional) reflects the operation of the amygdale, and the second reflects the operation of the hippocampus circuits. The hippocampus has associative cognitive functions centered in collecting information about possible relationships between cues and consequences (LeDoux, 1996). The main role of the cortical tissue expansion was to provide humans with new ways of keeping their distance from potentially life-threatening situations. Whereas the automatic system is involuntary and hard to access through introspection, controlled processing is governed by intentions and is consciously available. Cognition might also create expectations that allow an avoidance of potential threats in the absence of explicit cues that signal danger (Mineka & Öhman, 2002b). Nonetheless, this model conceptualizes cognitive factors as nondeterminant of fear response. Cognitions are an effect of these responses, because of the need to give meaning to an experience and a justification for the fear (e.g., Foa, Steketee, & Rothbaum, 1989). According to the fear module theory, more detailed information about the stimulus goes through the temporal cortex to confirm, or disconfirm, the activation that is on its way through the subcortical route. The conscious mind can therefore be used to retrospectively check the unconscious information and provide some meaning to it.

The fear module includes four characteristics, each one formed by natural selection, namely: relative selectivity to stimuli, automaticity, encapsulation, and a specific neuronal circuit. Selectiveness concerns the extent to which a certain stimulus is effective in

activating the module and varies both with evolutionary relevance and with the previous aversive experiences with the situation. Effectiveness is operationalized as faster acquisition and more resistance to extinction of associations between the stimulus and fear (Mineka & Öhman, 2002b). Although potentially dangerous objects such as guns (see Fox, Griggs, & Mouchlianitis, 2007) can be efficiently detected and strongly associated with fear, prepared stimuli presumably need only to be linked with moderately aversive events to elicit fear (Öhman & Mineka, 2001).

Automatism is the fast, reflexive activation of defense responses, independent of the neocortex (Mineka & Öhman, 2002b), in a fast process (LeDoux, 1996; Smith, Cacioppo, Larsen, & Chartrand, 2003) that prefers to risk false positives than false negatives (Nesse, 2005). The hypothesis that stimuli related to evolutionarily relevant fears can be processed automatically and preconciously (Öhman & Mineka, 2001) has been tested with the backward masking paradigm. This procedure prevents the recognition of a target stimulus by presenting it immediately (<30 ms) before another stimulus that acts as a mask (e.g., Öhman, 1996; Öhman & Soares, 1994, 1998). The measures of galvanic skin responses to the backward-masked presentations of pictures of snakes and spiders were found to be larger and more resistant to extinction than responses conditioned to neutral stimuli (Öhman & Soares, 1993).

Encapsulation defines the relative independence and resistance of the fear response, once initiated, from conscious cognitive control (Mineka & Öhman, 2002b). The amygdala receives inputs about the emotional significance of a stimulus and then, through its projections, modulates further attentional and perceptual processes (Anderson & Phelps, 2001). Nevertheless, attention and awareness seem to have little impact on the amygdala response to feared stimuli (Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003). Therefore, after its activation, the module is said to process the stimulus automatically, with relatively little interference from other processes. Although the response is resistant to conscious cognitive influences, the module might influence cognitions resulting in, for example, exaggerated expectations of aversive consequences (Öhman & Mineka, 2001).

The last domain of the model is the proposal that, at a neuronal level, the fear module is controlled by a specific circuit. Öhman and Mineka (2001) largely based this aspect of their work around amygdala-related findings. Data about this neuroanatomical region converge with reports that patients with bilateral amygdala lesions generally demonstrate selective deficits for recognizing facial expressions of fear and anger despite normal recognition of other emotional expressions (e.g., Broks et al., 1998; Calder, Young, Rowland, Perrett, Hodges, & Etcoff, 1996). It is also well known that amygdala damage produces deficits in fear conditioning in humans (Bechara, Tranel, Damasio, Adolphs, Rockland, & Damasio, 1995). Furthermore, fear conditioning leads to increases in amygdala functional activity, as measured by fMRI (Buchel, Morris, Dolan, & Friston, 1998), and these effects also occur with subliminal stimulus presentations (Morris, Öhman, & Dolan, 1998). Additionally this region has a higher efferent than afferent density of connections to the cortex (LeDoux, 1996). On this basis, it has been argued that fear exerts a higher influence over cognition than it is capable of handling. Projections from the amygdala to the brainstem are involved in the expression of fear responses, and projections from the amygdala to the cortex are believed to contribute to the experience of fear and other cognitive aspects of emotional processing (LeDoux, 2003; see also Barrett, Mesquita, Oschner, & Gross, 2007).

4.1 Critique of the Fear Module Theory

Contrary to the prevailing view, Pessoa, McKenna, Gutierrez, and Ungerleider (2002) showed that the amygdala only responded to emotional faces when sufficient attentional resources were available to process the faces. Pessoa (2005) also noticed effects of task context and attention, as well as large intersubject differences in sensitivity to the detection of masked fearful faces. Phillips et al. (2004) did not observe any response in the amygdala during unaware conditions. These results suggest automatism as a far too inflexible characteristic to provide humans with an adaptive fear response, as automatic behaviors are resistant to change even when paired with negative consequences. As stated by Mesulam (1998), behavioral flexibility is vital:

> With the exception of some autonomic, brainstem and spinal reflexes, the behavior of primates displays much greater latitude in translating sensation into action, so that identical sensory events can potentially trigger one of many different reactions, depending on the peculiarities of the prevailing context. A stimulus that deserves to be approached in one setting may need to be avoided in another . . . . (Mesulam, 1998, p. 1014)

The role of the retino-collicular-pulvinar-amygdala pathway proposed by Morris, Öhman, and Dolan (1999) as a neural substrate for the automatic processing of visual threats has been questioned, because superior colliculus neurons are unable to discriminate high spatial resolutions and thus would be unable to compute more complex visual stimuli (Rodman, Gross, & Albright, 1989). In fact, the amygdala responds more strongly to low spatial frequency information (Vuilleumier, Armony, Driver, & Dolan, 2003). Also, pictures of guns and other emotional stimuli that were low-pass filtered still caused interference on an auditory discrimination task (De Cesarei & Codispoti, 2008). Therefore, we could make the distinction that, whereas low-pass information may be enough to discriminate these images to allocate attention, it may not be enough for an emotional response.

Accordingly, Purkis and Lipp (2007) showed that, although snakes and spiders are preferentially attended, negative evaluations are not automatically elicited during this processing. In their study, which used an implicit emotional index, control participants evaluated both dangerous and nondangerous snakes and spiders as more negative than other animals, whereas expert participants evaluated only dangerous but not nondangerous snakes and spiders as more negative than other animals. The authors claimed that their findings are "inconsistent with the idea of an automatic and negative response to fear-relevant stimuli" (Purkis & Lipp, 2007, p. 322).

Pessoa, Kastner, and Ungerleider (2002) proposed that, for visual stimuli, initially neutral and emotional stimuli are processed equally by the occipitotemporal cortex. Only later, after feedback from the amygdala (and other structures) would the responses incorporate the valence of the stimulus.

To this discussion, it is also important to notice that earlier history of exposure with a particular stimulus can delay or impede the occurrence of a subsequent episode of conditioning (latent

inhibition; Lubow, 1973). Studies with facial expressions (Bond & Siddle, 1996), fear of dogs (Doogan & Thomas, 1992), and dental fear (Berge Ten, Veerkamp, & Hoogstraten, 2002; Kent, 1997) noticed that the acquisition of fear was less likely after a history of nontraumatic experiences, supporting the latent inhibition hypothesis. The implication of latent inhibition to the fear module is that, given that humans more frequently see joyful than angry expressions, as well as more flowers than snakes, it may be harder to associate a happy face or a flower with fear. Thus, the effects observed in Öhman's studies (e.g., Öhman & Soares, 1993, 1994) might have resulted from the inequality in participants' previous experiences with the stimuli. As is discussed next, the meaning that people give to what happens to them can have a very important role in the genesis and development of specific phobias.

5. Cognitive Models

Thorpe and Salkovskis (1995) considered that Seligman's (1971) work relegated to second place the role of cognitions in phobias. Conditioning can be conceptualized as a cognitive process, during which the participant learns that a determined event or stimulus precedes an aversive outcome. As far back as 1949, Tolman advocated that the basic acquisition in a learning experiment is an expectation, nowadays considered crucial in the development of human fear responses (e.g., Davey & Dixon, 1996). From a cognitive perspective, fear is related not only to a biological preparation or stimulus response association but also to the attributions regarding the safety and danger of the stimulus; the perception of control over the situation and the attribution made about the bodily alarm signal that the stimulus elicits (Arntz, Rauner, & Van Den Hout, 1995). Mineka and Kihlstrom (1978) considered that the characteristic common to experimental neurosis literature, on the basis of experiments with animals since the 20th century, is the unpredictability and uncontrollability of important events in people's lives. Mineka and Cook (1986) observed that baby monkeys raised with a sense of mastery and control over their environment habituated to scary events faster than monkeys raised in identical environments but with no experience of control. Moreover, children with a sense of mastery and control over their environment were less fearful when faced with new events and more capable of dealing with scary situations. Chorpita and Barlow (1998) suggested that recent life experiences with uncontrollable events can be a first path toward the development of fear and anxiety, as this type of experience can increase the probability of processing events as being out of one's control (i.e., it creates a psychological vulnerability). Children with an increased sense of control have higher access to information that foresees the possibility of avoiding negative consequences. However, children with lower control over events during development mainly stored information supporting beliefs that nothing could be done to prevent negative results (see Craske, 2003).

Maltzman and Boyd (1984) noticed that the results of Öhman's early experiments, which compared pictures of snakes and spiders with those of flowers and mushrooms (Öhman, Eriksson, Fredrikson, Hughdahl, & Olofsson, 1974; Öhman, Eriksson, & Olofsson, 1975; Öhman, Fredrikson, Hugdahl, & Rimmo, 1976), could reflect ontogenetic factors rather than support a preparedness account. Subsequently, Davey (e.g., Davey, 1989, 1992, 1995, 2002) systematically studied possible ontogenetic factors and the evaluative processes related to fear. Davey argued that human participants hold exaggerated expectations that aversive events will occur after the presentation of relevant stimuli, when compared with the presentation of irrelevant stimuli, from the beginning of the conditioning experience. This cognitive aspect of the processing of threat seems to be stable (Kindt & Brosschot, 1998).

Cognitive models could explain Öhman and colleagues' results on the basis of expectations arising from experience, instructions, and vicariant learning. When there is a strong expectation of covariation between two classes of events, people frequently overestimate their contingency. Therefore, in an experiment in which the aversive consequence distribution (among fear-relevant, -neutral or -irrelevant stimuli) is the same, participants tend to report that the aversive stimulus (e.g., electric shock) occurred more frequently paired with the fear-relevant stimulus. Several studies have noted that phylogenetic fear-relevant stimuli seem to engender stronger biases than ontogenetic fear-relevant stimuli, although there is an ongoing debate. It is proposed that this bias allows humans to attend to and learn about not only phylogenetic stimuli but also any stimulus that might—according to personal experience, instructions, or social learning—be labeled as dangerous. A bias in reporting the correlation of experimental stimuli is termed *covariation bias*, whereas a bias in expected outcomes before the experiment is referred to as *expectancy bias*.

Tomarken and colleagues (Tomarken, Mineka, & Cook, 1989) used an illusory correlation paradigm, presenting various uncorrelated stimuli across trials. Flower, mushroom, snake, and spider stimuli were randomly paired with aversive (shock) and nonaversive (nothing/tone) outcomes. Participants were previously selected for high or low fear of the snake and spider stimuli. High-fear participants overestimated the number of trials on which the fear-relevant stimulus had been paired with shock but were relatively accurate in estimating the other stimulus probabilities. Low-fear participants showed a similar but less significant pattern. To examine whether the covariation bias effect held for nonevolutionary stimuli, Kennedy, Rapee, and Mazurski (1997) used pictures of electrical outlets, snakes, and spiders and found that, in high-fear participants, covariation bias was specific to phylogenetic fear-relevant stimuli and shock.

Davey and Dixon (1996) conducted a series of studies to assess participants' foresight before and after presenting images of phylogenetically (e.g., spider, tiger, blood, fire, feces, angry faces, dirt, thunder, snake, and darkness) and ontogenetically (splintered glass, scissors, dentists, guns, gas fire, kettle, ladder, chainsaw, electric plug, and car) relevant stimuli. The procedures were developed to assess the expectation of aversive consequences, as well as a postexperimental estimate of actual aversive consequences. The aversive stimulus was a noise (115 db) and a vibration (through a metallic vibrator in contact with the arm). Each image could be followed by an aversive stimulus, a luminous flash, or an absence of stimulus. Although the frequency of consequence presentation was evenly distributed among the different images, the authors found a bias in both a priori and a posteriori judgments for ontogenetically and phylogenetically relevant stimuli and aversive outcomes. The authors also observed a significantly higher expectation of aversive consequence in participants who had perceptions of danger and fear of the presented stimuli (see also Davey & Craigie, 1997).

Davey and Dixon (1996) considered that the most parsimonious interpretation of these results came from the perspective of the cog-

nitive processes involved in the judgment of fear-relevant stimuli and their potential results. Although the authors acknowledged that evolutional associative predispositions might coexist with expectation biases, they suggested that these are sufficiently flexible to accommodate phylogenetically and ontogenetically relevant stimuli, allowing the potential dangers of new stimuli to be learned quickly. Similarly, Mineka (1992) proposed that phobias are an enduring example of a fear or danger schema, but that a short-term activation of a fear or danger schema in nonphobic participants might result in a similar covariation bias as is observed with phobic participants. This was illustrated in de Jong and Peters's (2007) and in Pury and Mineka (1997)'s experiments with blood-injection-injury fears, in which a covariation bias of harm or disgust was found in all participants and not only in blood-injection-injury-fearful participants.

According to Davey (1989, 1992, 1995), expectancy bias allows rapid learning to occur for any stimulus (ontogenetic or phylogenetic) that the participant has reason to believe is dangerous. A loud sound is an example of a possible prepared stimulus. It can even be a primary aversive type of stimuli (Rolls, 2007) nonetheless: "[D]efensive reactions should not be completely mobilized until the hearing cortex analyses the localization, frequency and intensity of the sound, in order to specifically determine the nature and extension of this potentially threatening sound signal" (LeDoux & Phelps, 2000, p. 160). The authors consider that, after freezing or expressing a physiologic response to a dangerous stimulus, cognitive processes will have control over behavior, taking into account the expectations regarding what is more likely to happen next, considering past experiences in similar circumstances. However, people diagnosed with phobia tend to overestimate the predictive relationship between a particular stimulus and its probable outcome.

Davey (1995) proposed a conditioning-based theory that includes expectations as an alternative to the prepared fear model. These expectations reflect previously acquired information regarding the link between the stimulus and its consequences. As such, participants may enter an experiment with preexisting expectations that may influence their responses to experimental stimuli. The confirmation or disconfirmation of expectancies leads to fear acquisition or extinction, with learning biased in favor of acquisition when stimuli are regarded as potentially threatening (see also Hosoba, Iwanaga, & Seiwa, 2001).

5.1 Critique of the Cognitive Models

The main criticism of cognitive theories comes from the viewpoint that fears are evoked before cognitions and, thus, that cognitions, although important in the interpretation of fear responses, cannot affect their initial production. According to Öhman, cognitions about fear occur to make sense of, or be congruent with, the fear that participants already have. Arntz et al. (1995) suggested that, in fact, participants might infer danger on the basis of their anxiety response. As previously noted, expectancy bias regarding the possibility of an imminent danger can be conceptualized from a cognitive point of view. However, the fear model considers that the physical response to a potentially threatening stimulus is elicited before, and is more or less independent from, the later cognitive meaning attributed by the participant.

Davey (1995) argued that the speed with which certain stimuli (e.g., snakes, spiders) are associated with aversive results is related to the existence of biases in information processing of threatening stimuli rather than with associative phylogenetic predispositions. This cognitive bias consists of a high expectation that aversive results will occur with fear-relevant stimuli. Regarding studies such as Öhman and Soares's (1993, 1994), which demonstrate preattentive processing of fear-relevant stimuli, Davey and Craigie (1997) argued that there is no reason to suppose that information related to expectations cannot be quickly assessed and determined by preattentive processes. It is nonetheless difficult for cognitive theories to conceptualize cognitive processes as influencing such early processing. Several paradigms provide evidence for the preferential attention given to threatening material. Moreover, participants with specific phobias are faster in processing stimuli relevant to their fear, such as snakes and spiders (Öhman, Flykt, & Esteves, 2001) or threatening faces (Mogg, Millar, & Bradley, 2000).

Summary and Conclusions

Marks (2002) wisely stated that observations concerning distinct models of fear acquisition are better understood when they are considered as part of a continuum. In the purely innate extreme of this continuum are the defensive reactions that are so potent or prepared that they appear with no type of traumatic experience, such as blinking with the fast approach of an object toward the face. In the opposite extreme of the continuum are the situations that should theoretically become the target of aversion but that have to be associated with a large quantity of aversive experiences before becoming feared. Kendler et al. (1992) also suggested that phobia subtypes can be placed throughout an etiological continuum. A considerable number of studies support that irrational fears and phobias represent different points in the same continuum (e.g., Kendler, Karkowski, & Prescott, 1999; Kendler, Myers, Prescott, & Neale, 2001; Kendler et al., 1992; Kendler, Walters, Neale, Kessler, Heath, & Eaves, 1995). The question, therefore, is not whether a given fear is associative but how much learning is needed to evoke that particular fear (Marks, 2002). The smaller the need for aversion in a stimulus–response link to provoke the appearance of fear, the more predisposed the fear can be considered. However, taking a dichotomist approach in classifying fear has some obvious pitfalls because, for example, fear of suffocating on food might qualify as having evolutionary significance, yet choking phobia frequently originates in a conditioning episode (McNally, 1994).

Accumulated evidence suggests that, at some point of processing, functional specialization is lost and emotion and cognition conjointly contribute to the control of thought and behavior (Gray, Braver, & Raichle, 2002). This means that behavior cannot be plainly separated into cognitive or emotional categories. Individual brain areas do not work in isolation but instead are part of connected networks (Pessoa, 2008). Cognitively changing the meaning of emotionally evocative stimuli greatly affects amygdala responses (Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Ochsner & Gross, 2005). There is evidence that major prefrontal territories are involved in emotion and that the left prefrontal cortex may be an area in which cognition and emotion are integrated (Pessoa, 2008).

In most of the models addressed in this review, there is a tendency to assume the primacy of either cognitive or emotional factors in triggering fear responses. In this way, the majority of models for the

etiology of fears and phobias highlight a specific domain as the protagonist for fear acquisition. With the realization of the inefficacy of separating emotions from cognitions, current perspectives merge several elements from different theories of fear etiology. Rachman (2002) clearly used elements of the preparedness model to clarify why fears are not evenly distributed, which strongly links his theory with Seligman's (see Rachman, 1991). Mineka and Zinbarg (2006) used learning theories and preparedness to describe a contemporary learning perspective on the etiology and maintenance of anxiety disorders. Davey's (1995) model acknowledges that evolutional associative predispositions might coexist with expectation biases. Nevertheless, Davey suggests that these are sufficiently flexible to accommodate phylogenetically and ontogenetically relevant stimuli, allowing the potential dangers of new stimuli to be learned quickly. Purkis and Lipp (2007) demonstrated that not all preferentially attended and potentially threatening stimuli evoke a fear response and that previous experience plays a vital role. Both day-to-day learning and natural selection are implied in the genesis of fear, and it is probable that, whereas phylogenetic evolution is responsible for general predispositions, ontogenetic learning is responsible for the more specific aspects of fears.

6. Future Directions

The next step for phobia researchers is to independently categorize the biological relevance of a stimulus, incorporating evolutionary concepts into the understanding of mental disorders (Siegert & Ward, 2002). When identifying the evolutionary pressures that lead to prepared associations, it is important to avoid dividing behavior into merely innate and learned. The challenge is to identify both vulnerability to phylogenetic predispositions and ontogenetic–experiential processes and to ignore neither the subtle aspects of participants' developmental histories (Delprato, 1980) nor the pressures related to each stimulus in its relation to humans (Mineka & Zinbarg, 2006). Reflecting on the evidence covered in this review, it seems unlikely that specific animal threats are selected and encoded by evolutionary processes. As stressed by Davey (1995), there is a great variety of situations that might endanger a human organism. Given that potential predators can change more quickly than the genetic configuration of fear of a specific predator could be encoded, it is more likely that the encoding of more general configurations occur, such as quick approaches, sudden shadows, or being stared at or followed (Davey, 1995). As such, the specific features of stimuli encoded during ontogenetic learning require further investigation. In this quest, it is essential to keep in mind that preparedness is not a stimulus but a relationship between an organism and the environment. The fear of driving, for example, could be considered a nonevolutional fear because cars are recent in human evolution. However, the same fear could be mediated by the fear of sudden movements toward the individual (e.g., a car getting closer). This latter might be a specific configuration selected through evolutional pressures (Davey, 1995; see also Brosch & Sharma, 2005).

As an example of a general stimulus configuration related to fear, reaction to certain types of movement was initially studied from the perspective of the hawk–goose effect. This effect was initially thought to be an inborn ability in chicks to tell the difference between a goose or nonpredatory bird flying overhead from a hawk or other predatory bird (Tinbergen, 1951). Later, Schneirla (1965) hypothesized that the hawk–goose effect was instead due to the result of differences in the rate of change of retinal stimulation produced by the type and speed of movement and stimulus size. These features were more reliably related to fear than were the perceptual characteristics of the visual stimulus (hawk or goose). This hypothesis does not require an innate, qualitative perceptual schema (short-neckedness) to account for fear reactions of birds to selected stimuli (see Delprato, 1980). Additionally, it was reported that birds reacted equally to a variety of shapes, including hawk, goose, and geometric figures (McNiven, 1960; Melzack, Penick, & Beckett, 1959; Schleidt, 1961). Sudden swooping and fast movements were more important than configuration in eliciting fear in ducklings (see also Mueller & Parker, 1980).

More recently, Riskind, Williams, Gessner, Chrosniak, and Cortina (2000) found a unique feature of the mental scenarios generated by anxious individuals, in which movement plays an essential role. The looming vulnerability, or looming cognitive style (LCS), is conceptualized as a distinctive cognitive phenomenology of threat or danger characterized by a tendency to construct, generate, maintain, and attend mental scenarios that elicit anxiety and sensitize the individual to signs of movement and threat, which biases cognitive processing and impedes habituation to threat stimuli (Riskind, 1997; Riskind & Williams, 2005; Riskind, Williams, & Joiner, 2006). It is interesting to note that the fear-relevant stimuli repeatedly chosen for experiments are snakes and spiders, which were originally compared with flowers and mushrooms (e.g., Öhman et al., 1974; Öhman et al., 1975; Öhman et al., 1976; Öhman et al., 2001; Öhman & Mineka, 2003; Öhman & Soares, 1993, 1994). However, among backgrounds of flowers and mushrooms, pictures of all animals are found faster (Lipp, Darakshan, Waters, & Logies, 2004). Threatening and nonthreatening animals (e.g., horses, cats, and rabbits) are, in general, easily found (Tipples, Young, Quinlan, Broks, & Ellis, 2002). Although these experiments cannot entirely rule out the possibility of low-level artifacts affecting the results, flowers and mushrooms differ from animals in their moving behavior. In fact, animals' movement behavior is obviously more unpredictable than that of flowers and mushrooms. More recent experiments designed to evaluate the potential for preparedness in different stimuli have started to control for this feature. However, the LCS may well have contributed to previous findings, and this interesting possibility needs further study.

Other examples stress the importance of studying specific stimuli and situations that might convey fear-related information. Recently, motion sickness susceptibility and postural stability were identified as relevant features for several anxiety disorders. These findings can help differentiate specific phobias from other anxiety disorders, such as panic and agoraphobia (see Coelho, Waters, Hine, & Wallis, 2009; Jacob, Redfern, & Furman, 1995; Redfern, Yardley, & Bronstein, 2001). Similarly, some aspects of driving phobia and height phobia may also result from a common underlying vulnerability to motion sickness (DiNardo et al., 1997). These findings might help us to classify and differentiate fears of heights, planes, and cars and agoraphobia, as they share several features. The question raised by McNally (2002) regarding the classification of airplane phobia is pertinent here: Is airplane phobia a contemporary fear, like driving phobia, or is it a subset of height phobia?

There is already a trend toward regained interest in latent inhibition (Lubow, 1973). Bond and Siddle (1996) measured the

differential conditioning of skin conductance responses when facial expressions of joy, anger, and surprise were paired with electric shocks. As discussed before, fear-based theories would predict more robust conditioning for angry facial expressions. However, subsequent extinction of skin conductance responding was slower for expressions of surprise than for expressions of anger or happiness. This result was contrary to expectations from the prepared fear hypothesis, and the authors concluded that the latent inhibition hypothesis was implicated here.

The expression that is seen less frequently in day-to-day situations (surprise) is the one that shows higher resistance to extinction (Bond & Siddle, 1996). Doogan and Thomas (1992) observed a significant number of participants with fear of dogs who reported having less experience of contact with dogs before the beginning of the fear, as compared with fearless participants. The participants who had previous experiences with dogs have more knowledge of how dogs behave and may view their behavior as predictable and controllable (Doogan & Thomas, 1992). Similarly, Kent (1997) and Berge Ten et al. (2002) noticed that the acquisition of dental fear was less likely after a history of noninvasive dental visits. Given that the amygdala is sensitive to ambiguous stimuli, the same neuronal circuits as the ones claimed to defend the fear module corroborate these findings. In fact, amygdala activity is observed mainly when the contingencies between a stimulus and a negative outcome are altered or unpredictable (LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998). The previous quantity of nonnoxious experience with an event is able to explain the lack of equipotentiality in the fear distribution and why some aversive encounters do not produce phobias (see Mineka & Öehlberg, 2008). A nonnormal fear distribution can exist if some people fail to develop phobia after an aversive encounter. Because fear-relevant stimuli are typically more infrequent and salient than are non-fear-relevant stimuli, they are more prone to be feared, compared with usual stimuli perceived as safe on the basis of extensive experience and familiarity.

New lines of research into the potential physiological causes for particular fears may well provide a far greater insight into the mechanics of fear and phobia. An approach that seeks to determine such causes may also avoid the major problem with grouping fears together under one banner, which is that deeper causes for specific fears may be overlooked. Thus, further investigation into the mechanics of specific phobias, as well as more rigorous assessment of the specific features of stimuli that activate phobias, will provide better insight. The aim of future research then is to work toward a more accurate delineation of the specific conditions currently grouped together as specific phobias.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Anderson, A. K., Christoff, K., Panitz, D., De Rosa, E., & Gabrieli, J. D. E. (2003). Neural correlates of the automatic processing of threat facial signs. *Journal of Neuroscience, 23,* 5627–5633.

Anderson, A. K., & Phelps, E. A. (2001). Lesions of the human amygdala impair enhanced perception of emotionally salient events. *Nature, 411,* 305–309.

Arntz, A., Rauner, M., & Van Den Hout, M. (1995). "If I feel anxious, there must be danger": Ex-consequentia reasoning in inferring danger in anxiety disorders. *Behaviour Research and Therapy, 33,* 917–925.

Bandura, A. (1965). Influence of model's reinforcement contingencies on the acquisition of imitative responses. *Journal of Personality and Social Psychology, 1,* 589–595.

Bandura, A. (1971). Vicarious and self-reinforcement processes. In R. Glaser (Ed.), *The nature of reinforcement* (pp. 228–278). New York: Academic Press.

Bandura, A. (1977). *Social learning theory.* Englewood Cliffs, NJ: Prentice Hall.

Barrett, L. F., Mesquita, B., Oschner, K. N., & Gross, J. J. (2007). The experience of emotion. *Annual Review of Psychology, 58,* 7.1–7.31.

Bechara, A., Tranel, D., Damasio, H., Adolphs, R., Rockland, C., & Damasio, A. R. (1995). Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science, 269,* 1115–1118.

Berge Ten, M., Veerkamp, J. S. J., & Hoogstraten, J. (2002). The etiology of childhood dental fear: The role of dental and conditioning experiences. *Anxiety Disorders, 16,* 321–329.

Bond, N., & Siddle, D. A. (1996). The preparedness account of social phobia: Some data and alternative explanations. In R. M. Rapee (Ed.), *Current controversies in the anxiety disorders,* (pp. 291–316). New York: Guilford Press.

Brainerd, C. J., & Mojardin, A. H. (1998). Children's false memories for sentences: Long-term persistence and mere testing effects. *Child Development, 69,* 1361–1377.

Brainerd, C. J., & Reyna, V. F. (1996). Mere memory testing creates false memories in children. *Developmental Psychology, 32,* 467–476.

Brainerd, C. J., & Reyna, V. F. (2004). Fuzzy-trace theory and memory development. *Developmental Review, 24,* 396–439.

Bregman, E. O. (1934). An attempt to modify the emotional attitudes of infants by the conditioned response technique. *Journal of Genetic Psychology, 45,* 169–198.

Broks, P., Young, A. W., Maratos, E. J., Coffey, P. J., Calder, A. J., Isaac, C., Mayes, A. R., et al. (1998). Face processing impairments after encephalitis: Amygdala damage and recognition of fear. *Neuropsychologia, 36,* 59–70.

Brosch, T., & Sharma, D. (2005). The role of fear-relevant stimuli in visual search: A comparison of phylogenetic and ontogenetic stimuli. *Emotion, 5,* 360–364.

Buchel, C., Morris, J., Dolan, R. J., & Friston, K. J. (1998). Brain systems mediating aversive conditioning: An event-related fMRI study. *Neuron, 20,* 947–957.

Calder, A. J., Young, A. W., Rowland, D., Perrett, D. I., Hodges, J. R., & Etcoff, N. L. (1996). Facial emotion recognition after bilateral amygdala damage: Differentially severe impairment of fear. *Cognitive Neuropsychology, 13,* 699–745.

Chorpita, B. F., & Barlow, D. H. (1998). The development of anxiety: The role of control in the early environment. *Psychological Bulletin, 124,* 3–21.

Coelho, C. M., Santos, J. A., Silva, C. F., Tichoon, J., Hine, T. J., & Wallis, G. (2008). The role of self-motion in acrophobia treatment. *Cyberpsychology & Behavior, 11,* 723–725.

Coelho, C. M., Waters, A. M., Hine, T., & Wallis, G. (2009). The use of virtual reality in acrophobia research and treatment. *Journal of Anxiety Disorders, 23,* 563–574.

Cook, M., & Mineka, S. (1989). Observational conditioning of fear-relevant versus fear-irrelevant stimuli in rhesus monkeys. *Journal of Abnormal Psychology, 98,* 448–459.

Cook, M., & Mineka, S. (1990). Selective associations in the observational conditioning of fear in monkeys. *Journal of Experimental Psychology: Animal Behavior Processes, 16,* 372–389.

Craske, M. G. (2003). *Origins of phobias and anxiety disorders: Why more women than men?* Oxford, England: Elsevier.

Craske, M. G., & Waters, A. W. (2005). Panic disorder, phobias, and generalized anxiety disorder. *Annual Review of Clinical Psychology, 1,* 197–225.

Darwin, C. (1877). A biographical sketch of an infant, *Mind, 2,* 285–294.

Davey, G. C. L. (1989). UCS revaluation and conditioning models of acquired fears. *Behaviour Research and Therapy, 27,* 521–528.

Davey, G. C. L. (1992). Classical conditioning and the acquisition of human fears and phobias: A review and synthesis of the literature. *Advances of Behaviour Research and Therapy, 14,* 29–66.

Davey, G. C. L. (1995). Preparedness and phobias: Specific evolved associations or a generalized expectancy bias? *Behavioral and Brain Sciences, 18,* 289–325.

Davey, G. C. L. (1997). A conditioning model of phobias. In G. C. L. Davey (Ed.), *Phobias*: *A handbook of theory, research and treatment.* Chichester, England: Wiley.

Davey, G. C. L. (2002). "Nonspecific" rather than "nonassociative" pathways to phobias: A commentary on Poulton and Menzies. *Behaviour Research and Therapy, 40,* 151–158.

Davey, G. C. L., & Craigie, P. (1997). Manipulation of dangerousness judgements to fear-relevant stimuli: Effects on *a priori* UCS expectancy and *a posteriori* covariation assessment. *Behaviour Research and Therapy, 35,* 607–617.

Davey, G. C. L., & Dixon, A. L. (1996). The expectancy bias model of selective associations: The relationship of judgements of CS dangerousness, CS-UCS similarity and prior fear to *a priori* and *a posteriori* covariation assessments. *Behaviour Research and Therapy, 34,* 235–252.

De Cesarei, A., & Codispoti, M. (2008). Fuzzy picture processing: Effects of size reduction and blurring on emotional processing. *Emotion, 8,* 352–363.

de Jong, P. J., Muris, P., & Merckelbach, H. (1996). UCS inflation and human aversive autonomic conditioning. *Biological Psychology, 43,* 227–239.

de Jong, P. J., & Peters, M. L. (2007). Blood-injection-injury fears: Harm- vs. disgust-relevant selective outcome associations. *Journal of Behavior Therapy and Experimental Psychiatry, 38,* 263–274.

Delprato, D. J. (1980). Hereditary determinants of fears and phobias: A critical review. *Behavior Therapy, 11,* 79–103.

Diaz, J. H. (2004). The global epidemiology, syndromic classification, management, and prevention of spider bites. *American Journal of Tropical Medicine and Hygiene, 71,* 239–250.

DiNardo, P. A., Gilbert, S. J., Amatuccio, D., Fox, A., Garland, J., Bucalos, F. J., Pate, J., et al. (1997). *Motion sickness susceptibility and height fears.* Paper presented at the Association for Advancement of Behavior Therapy, Miami Beach, FL.

Doogan, S., & Thomas, G. V. (1992). Origins of fear of dogs in adults and children: The role of conditioning processes and prior familiarity with dogs. *Behaviour Research and Therapy, 30,* 387–394.

Eddleston, N., & Persson, H. (2003). Acute plant poisoning and antitoxin antibodies. *Journal of Toxicology and Clinical Toxicology, 41,* 309–315.

Emmelkamp, P. M. G. (1982). *Phobic and obsessive-compulsive disorders: Theory, research, and practice.* New York: Plenum Press.

English, H. B. (1929). Three cases of the "conditioned fear response." *Journal of Abnormal and Social Psychology, 24,* 221–225.

Field, A. P. (2006). Is conditioning a useful framework for understanding the development and treatment of phobias? *Clinical Psychology Review, 26,* 857–875.

Field, A. P., Argyris, N. G., & Knowles, K. A. (2001). Who's afraid of the big bad wolf? A prospective paradigm to test Rachman's indirect pathways in children. *Behaviour Research and Therapy, 39,* 1259–1276.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy, 20,* 155–176.

Forsyth, J. P., & Chorpita, B. F. (1997). Unearthing the nonassociative origins of fears and phobias: A rejoinder. *Journal of Behavior Therapy & Experimental Psychiatry, 28,* 297–305.

Fox, E., Griggs, L., & Mouchlianitis, E. (2007). The detection of fear-relevant stimuli: Are guns noticed as quickly as snakes? *Emotion, 7,* 691–696.

Fyer, A. B. (1998). Current approaches to etiology and pathophysiology of specific phobia. *Biological Psychiatry, 44,* 1295–1304.

Gafford, J. E., Silva, P. A., & Langley, J. D. (1996). Injuries. In P. A. Silva & W. R. Stanton (Eds.), *From child to adult: The Dunedin Multidisciplinary Health and Development Study* (pp. 98–112). Auckland, New Zealand: Oxford University Press.

Garcia, J., & Koelling, R. A. (1966). Relation of cue to consequence in avoidance learning. *Psychonomic Science, 4,* 123.

Graham, J., & Gaffan, E. A. (1997). Fear of water in children and adults: Etiology and familial effects. *Behaviour Research and Therapy, 35,* 91–108.

Gray, J. R., Braver, T. S., & Raichle, M. E. (2002). Integration of emotion and cognition in the lateral prefrontal cortex. *Proceedings of the National Academy of Sciences, USA, 99,* 4115–4120.

Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry, 53,* 494–501.

Herbert, M. (1994). Etiological considerations. In T. H. Ollendick, N. J. King, & W. Yule (Eds.), *International handbook of phobic and anxiety disorders in children and adolescents* (pp. 3–20). New York: Plenum Press.

Hosoba, T., Iwanaga, M., & Seiwa, H. (2001). The effect of UCS inflation and deflation procedures on "fear" conditioning. *Behaviour Research and Therapy, 39,* 465–475.

Jacob, R. G., Redfern, M. S., & Furman, J. M. (1995). Optic flow-induced sway in anxiety disorders associated with space and motion discomfort. *Journal of Anxiety Disorders, 9,* 411–425.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999). Fears and phobias: Reliability and heritability. *Psychological Medicine, 29,* 539–553.

Kendler, K. S., Myers, J., Prescott, C. A., & Neale, M. C. (2001). The genetic epidemiology of irrational fears and phobias in men. *Archives of General Psychiatry, 58,* 257–265.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). The genetic epidemiology of phobias in women: The interrelationship of agoraphobia, social phobia, situational phobia and simple phobia. *Archives of General Psychiatry, 49,* 273–281.

Kendler, K. S., Walters, E. E., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1995). The structure of the genetic and environmental risk factors for six major psychiatric disorders in women. *Archives of General Psychiatry, 52,* 374–383.

Kennedy, S. J., Rapee, R. M., & Mazurski, E. J. (1997). Covariation bias for phylogenetic versus ontogenetic fear-relevant stimuli. *Behaviour Research and Therapy, 35,* 415–422.

Kent, G. (1997). Dental phobias. In G. C. Davey (Ed.), *Phobias: A handbook of theory, research and treatment* (pp. 107–127). Chichester, England: Wiley.

Kindt, M., & Brosschot, J. F. (1998). Stability of cognitive bias for threat cues in phobia. *Journal of Psychopathology and Behavioral Assessment, 20,* 351–367.

Kleinknecht, R. A. (1994). Acquisition of blood, injury and injection fear and phobia. *Behaviour Research and Therapy, 32,* 817–823.

Kleinknecht, R. A. (2002). Comments on non-associative fear acquisition: A review of the evidence from retrospective and longitudinal research. *Behaviour Research and Therapy, 40,* 159–163.

Kotwica, M., & Czerczak, S. (2007). Acute poisonings registered since 1970: Trends and characteristics. Analysis of the files collected in the

National Poison Information Centre, Lodz, Poland. *International Journal of Occupational Medicine and Environmental Health, 20,* 38–43.

LaBar, K. S., Gatenby, C., Gore, J. C., LeDoux, J. E., & Phelps, E. A. (1998). Human amygdale activation during conditioned fear acquisition and extinction: A mixed trial fMRI study. *Neuron, 20,* 937–945.

Lawson, J., Banerjee, R., & Field, A. P. (2007). The effects of verbal information on children's fear beliefs about social situations. *Behaviour Research and Therapy, 45,* 21–37.

LeDoux, J. E. (1996). *The emotional brain.* New York: Simon & Schuster.

LeDoux, J. E. (2003). The emotional brain, fear, and the amygdala. *Cellular and Molecular Neurobiology, 23,* 727–738.

LeDoux, J. E., & Phelps, E. A. (2000). Emotional networks in the brain. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of emotions* (pp. 157–172). New York: Guilford Press.

Lincoff, G., & Mitchel, D. H. (1977). *Toxic and hallucinogenic mushroom poisoning.* New York: Van Nostrand, Reinhold.

Lipp, O. V., Darakshan, N., Waters, A. M., & Logies, S. (2004). Snakes and cats in the flower bed: Fast detection is not specific to pictures of fear-relevant animals. *Emotion, 4,* 233–250.

Loftus, E. F. (2004). Memories of things unseen. *Current Directions in Psychological Science, 13,* 145–147.

Lubow, R. E. (1973). Latent inhibition. *Psychological Bulletin, 79,* 398–407.

Mackintosh, N. J. (1983). *Conditioning and associative learning.* New York: Oxford University Press.

Maltzman, I., & Boyd, G. (1984). Stimulus significance and bilateral SCRs to potentially phobic pictures. *Journal of Abnormal Psychology, 93,* 41–46.

Marks, I. M. (2002). Innate and learned fears are at opposite ends of a continuum of associability. *Behaviour Research and Therapy, 40,* 165–167.

Marks, I. M., & Nesse, R. M. (1994). Fear and fitness: An evolutionary analysis of anxiety disorders. *Ethnology and Sociobiology, 15,* 247–261.

McNally, R. J. (1994). Choking phobia: A review of the literature. *Comprehensive Psychiatry, 35,* 83–89.

McNally, R. J. (2002). On nonassociative fear emergence. *Behaviour Research and Therapy, 40,* 169–172.

McNiven, M. A. (1960). "Social-releaser mechanism" in birds—A controlled replication of Tinbergen's study. *Psychological Record, 10,* 259–265.

Melzack, R., Penick, E., & Beckett, A. (1959). The problem of "innate fear" of the hawk shape: An experimental study of mallard ducks. *Journal of Comparative and Physiological Psychology, 52,* 694–698.

Menzies, R. G., & Clarke, J. C. (1993a). The etiology of childhood water phobia. *Behaviour Research and Therapy, 31,* 499–501.

Menzies, R. G., & Clarke, J. C. (1993b). The etiology of fear of heights and its relationship to severity and individual response patterns. *Behaviour Research and Therapy, 31,* 355–365.

Menzies, R. G., & Clarke, J. C. (1995). The etiology of phobias: A non-associative account. *Clinical Psychology Review, 15,* 23–48.

Menzies, R. G., & Parker, L. (2001). The origins of height fear: An evaluation of neoconditioning explanations. *Behaviour Research and Therapy, 39,* 185–199.

Merckelbach, H., Arntz, A., & de Jong, P. (1991). Conditioning experiences in spider phobics. *Behaviour Research and Therapy, 29,* 333–335.

Mesulam, M.-M. (1998). From sensation to cognition, *Brain, 121,* 1013–1052.

Mineka, S. (1992). Evolutionary memories, emotional processing, and the emotional disorders. In D. Medin (Ed.), *The psychology of learning and motivation* (Vol. 28, pp. 161–206). San Diego, CA: Academic Press.

Mineka, S., & Cook, M. (1986). Immunization against the observational conditioning of snake fear in rhesus monkeys. *Journal of Abnormal Psychology, 95,* 307–318.

Mineka, S., Cook, M., & Miller, S. (1984). Fear conditioned with escapable and inescapable shock: The effects of a feedback stimulus. *Journal of Experimental Psychology: Animal Behavior Processes, 10,* 307–323.

Mineka, S., Gunnar, M., & Champoux, M. (1986). Control and early socioemotional development: Infant rhesus monkeys reared in controllable versus uncontrollable environments. *Child Development, 57,* 1241–1256.

Mineka, S., & Kihlstrom, J. F. (1978). Unpredictable and uncontrollable events: A new perspective on experimental neurosis. *Journal of Abnormal Psychology, 87,* 256–271.

Mineka, S., & Öehlberg, K. (2008). The relevance of recent developments in classical conditioning to understanding the etiology and maintenance of anxiety disorders. *Acta Psychologica, 127,* 567–580.

Mineka, S., & Öhman, A. (2002a). Born to fear: Non-associative vs. associative factors in the etiology of phobias. *Behaviour Research and Therapy, 40,* 173–184.

Mineka, S., & Öhman, A. (2002b). Phobias and preparedness: The selective, automatic, and encapsulated nature of fear. *Biological Psychiatry, 52,* 927–937.

Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders. It's not what you thought it was. *American Psychologist, 61,* 10–26.

Mogg, K., Millar, N., & Bradley, B. P. (2000). Biases in eye movements to threatening facial expressions in generalized anxiety disorder and depressive disorder. *Journal of Abnormal Psychology, 109,* 695–704.

Morris, J. S., Öhman, A., & Dolan, R. J. (1998). Conscious and unconscious emotional learning in the human amygdala. *Nature, 393,* 467–470.

Morris, J. S., Öhman, A., & Dolan, R. J. (1999). A subcortical pathway to the right amygdale mediating "unseen" fear. *Proceedings of the National Academy of Sciences, USA, 96,* 1680–1685.

Mowrer, O. H. (1960). *Learning theory and the symbolic processes.* London: Wiley.

Mueller, H. C., & Parker, P. G. (1980). Naïve ducklings show different cardiac response to hawk than to goose models. *Behaviour, 74,* 101–112.

Muris, P., Merckelbach, H., de Jong, P. J., & Ollendick, T. H. (2002). The etiology of specific fears and phobias in children: A critique of the non-associative account. *Behaviour Research and Therapy, 40,* 185–195.

Muris, P., Steerneman, P., Merckelbach, H., & Meesters, C. (1996). The role of parental fearfulness and modeling in children's fear. *Behaviour Research and Therapy, 34,* 265–268.

Nesse, R. M. (2005). Natural selection and the regulation of defenses: A signal detection analysis of the smoke detector principle. *Evolution and Human Behavior, 26,* 88–105.

Nisbett, R., & Wilson, T. (1977). Telling more than we can know: Verbal reports on mental processes. *Psychological Review, 84,* 231–259.

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9,* 242–249.

Öhman, A. (1996). Preferential preattentive processing of threat in anxiety: Preparedness and attentional biases. In R. M. Rapee (Ed.), *Current controversies in the anxiety disorders* (pp. 253–290). London: Guilford Press.

Öhman, A., Eriksson, A., Fredrikson, M., Hughdahl, K., & Olofsson, C. (1974). Habituation of the electrodermal orienting reaction to potentially phobic and supposedly neutral stimuli in normal human subjects. *Biological Psychology, 2,* 85–93.

Öhman, A., Eriksson, A., & Olofsson, C. (1975). One-trial learning and superior resistance to extinction of autonomic responses conditioned to potentially phobic stimuli. *Journal of Comparative and Physiological Psychology, 88,* 619–627.

Öhman, A., Flykt, A., & Esteves, F. (2001). Emotion drives attention: Detecting the snake in the grass. *Journal of Experimental Psychology: General, 130,* 466–478.

Öhman, A., Fredrikson, M., Hugdahl, K., & Rimmo, P. A. (1976). The premise of equipotentiality in human classical conditioning. Conditioned electrodermal responses to potentially phobic stimuli. *Journal of Experimental Psychology, General, 105,* 313–336.

Öhman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review, 108,* 483–522.

Öhman, A., & Mineka, S. (2003). The malicious serpent: Snakes as a prototypical stimulus for an evolved module for fear. *Current Directions in Psychological Science, 12,* 5–9.

Öhman, A., & Soares, J. J. F. (1993). On the automatic nature of phobic fear: Conditioned electrodermal responses to masked fear-relevant stimuli. *Journal of Abnormal Psychology, 102,* 121–132.

Öhman, A., & Soares, J. J. F. (1994). "Unconscious anxiety": Phobic responses to masked stimuli. *Journal of Abnormal Psychology, 103,* 231–240.

Öhman, A., & Soares, J. J. F. (1998). Emotional conditioning to masked stimuli: Expectancies for aversive outcomes following nonrecognized fear-relevant stimuli. *Journal of Experimental Psychology: General, 127,* 69–82.

Ollendick, T. H., & King, N. J. (1991). Origins of childhood fears: An evaluation of Rachman's theory of fear acquisition. *Behaviour Research and Therapy, 29,* 117–123.

Olsson, A., & Phelps, E. A. (2004). Learned fear of "unseen" faces after Pavlovian, observational, and instructed fear. *Psychological Science, 15,* 822–828.

Öst, L.-G. (1991). Acquisition of blood and injection phobia and anxiety response patterns in clinical patients. *Behaviour Research and Therapy, 29,* 323–332.

Öst, L.-G., & Hugdahl, K. (1981). Acquisition of phobias and anxiety response patterns in clinical patients. *Behaviour Research and Therapy, 19,* 439–447.

Pessoa, L. (2005). To what extent are emotional visual stimuli processed without attention and awareness? *Current Opinion in Neurobiology, 15,* 188–196.

Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews Neuroscience, 9,* 148–158.

Pessoa, L., Kastner, S., & Ungerleider, L. G. (2002). Attentional control of the processing of neutral and emotional stimuli. *Cognitive Brain Research, 15,* 31–45.

Pessoa, L., McKenna, M., Gutierrez, E., & Ungerleider, L. G. (2002). Neural processing of emotional faces requires attention. *Proceedings of the National Academy of Sciences, USA, 99,* 11458–11463.

Phelps, E. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology, 57,* 27–53.

Phillips, M. L., Williams, L. M., Heining, M., Herba, C. M., Russell, T., Andrew, C., Bullmore, E. T., et al. (2004). Differential neural responses to overt and covert presentations of facial expressions of fear and disgust. *Neuroimage, 21,* 1484–1496.

Poulton, R., Davies, S., Menzies, R. G., Langley, J. D., & Silva, P. A. (1998). Evidence for a non-associative model of the acquisition of a fear of heights. *Behaviour Research and Therapy, 36,* 537–544.

Poulton, R., & Menzies, R. G. (2002a). Fears born and bred: Toward a more inclusive theory of fear acquisition. *Behaviour Research and Therapy, 40,* 197–208.

Poulton, R., & Menzies, R. G. (2002b). Non-associative fear acquisition: A review of the evidence from retrospective and longitudinal research. *Behaviour Research and Therapy, 40,* 127–149.

Poulton, R., Menzies, R. G., Craske, M. G., Langley, J. D., & Silva, P. A. (1999). Water trauma and swimming experiences up to age 9 and fear of water at age 18: A longitudinal study. *Behaviour Research and Therapy, 37,* 39–48.

Poulton, R., Thomson, W. M., Davies, S., Kruger, E., Brown, R. H., & Silva, P. A. (1997). Good teeth, bad teeth and fear of the dentist. *Behaviour Research and Therapy, 35,* 327–334.

Purkis, H. M., & Lipp, O. V. (2007). Automatic attention does not equal automatic fear: Preferential attention without implicit valence. *Emotion, 7,* 314–323.

Pury, C. L. S., & Mineka, S. (1997). Covariation bias for blood-injury stimuli and aversive outcomes. *Behaviour Research and Therapy, 35,* 35–47.

Rachman, S. J. (1978). *Fear and courage.* San Francisco: Freeman.

Rachman, S. J. (1991). Neo-conditioning and the classical theory of fear acquisition. *Clinical Psychology Review, 11,* 155–173.

Rachman, S. J. (2002). Fears born and bred: Non-associative fear acquisition? *Behaviour Research and Therapy, 40,* 121–126.

Rachman, S. J., & Bichard, S. (1988). The overprediction of fear. *Clinical Psychology Review, 8,* 303–312.

Rapee, R. M. (1997). Perceived threat and perceived control as predictors of the degree of fear in physical and social situations. *Journal of Anxiety Disorders, 11,* 455–461.

Razran, G. (1971). *Mind in evolution: An east-west synthesis of learned behavior and cognition.* Boston: Houghton Mifflin.

Redfern, M. S., Yardley, L., & Bronstein, A. M. (2001). Visual influences on balance. *Journal of Anxiety Disorders, 15,* 81–94.

Rescorla, R. A. (1974). Effect of inflation following conditioning. *Journal of Comparative and Psychological Psychology, 86,* 101–106.

Riskind, J. H. (1997). Looming vulnerability to threat: A cognitive paradigm for anxiety. *Behaviour Research and Therapy, 35,* 685–702.

Riskind, J. H., & Williams, N. L. (2005). The looming cognitive style and generalized anxiety disorder: Distinctive danger schemas and cognitive phenomenology. *Cognitive Therapy and Research, 29,* 7–27.

Riskind, J. H., Williams, N. L., Gessner, T., Chrosniak, L. D., & Cortina, J. (2000). The looming maladaptive style: Anxiety, danger, and schematic processing. *Journal of Personality and Social Psychology, 79,* 837–852.

Riskind, J. H., Williams, N. L., & Joiner, T. E. (2006). The looming cognitive style: A cognitive vulnerability for anxiety disorders. *Journal of Social and Clinical Psychology, 25,* 779–801.

Rodman, H. R., Gross, C. G., & Albright, T. D. (1989). Afferent basis of visual response properties in area MT of the macaque: I. Effects of striate cortex removal, *Journal of Neuroscience, 9,* 2033–2050.

Rolls, E. T. (2007). *Emotion explained.* Oxford, England: University Press.

Schleidt, W. M. (1961). Reaktionen von Truthühnern auf fliegende Raubvögel und Versuche zur Analyse ihres AAM's [Reactions of turkeys to flying raptors and experiments to analyze their innate releasing mechanism]. *Zeitschrift fur Tierpsychologie, 18,* 534–560.

Schneirla, T. C. (1965). Aspects of stimulation and organization in approach/withdrawal processes underlying vertebrate behavioral development. In D. S. Lehrman, R. A. Hinde, & E. Shaw (Eds.), *Advances in the study of behavior* (pp. 1–71) New York: Academic Press.

Seligman, M. E. P. (1971). Phobias and preparedness. *Behaviour Therapy, 2,* 307–320.

Siegert, R. J., & Ward, T. (2002). Clinical psychology and evolutionary psychology: Toward a dialogue. *Review of General Psychology, 6,* 235–259.

Smith, N. K., Cacioppo, J. T., Larsen, J. T., & Chartrand, T. H. (2003). May I have your attention please: Electrocortical responses to positive and negative stimuli. *Neuropsychologia, 41,* 171–183.

Stein, D. J., & Bouwer, C. (1997). A neuro-evolutionary approach to the anxiety disorders. *Journal of Anxiety Disorders, 11,* 409–429.

Taylor, J. E., Deane, F. P., & Podd, J. V. (1999). Stability of driving fear acquisition pathways over one year. *Behaviour Research and Therapy, 37,* 927–939.

Tinbergen, N. (1951). *The study of instinct.* New York: Oxford University Press.

Tipples, J., Young, A. W., Quinlan, P., Broks, P., & Ellis, A. W. (2002). Searching for threat. *Quarterly Journal of Experimental Psychology: Human Experimental Psychology, 55*A, 1007–1026.

Tomarken, A. J., Mineka, S., & Cook, M. (1989). Fear-relevant selective associations and covariation bias. *Journal of Abnormal Psychology, 98,* 381–394.

Valentine, C. W. (1930). The innate bases of fear. *Journal of Genetic Psychology, 37,* 394–420.

Vuilleumier, P., Armony, J. L., Driver, J., & Dolan, R. J. (2003). Distinct spatial frequency sensitivities for processing faces and emotional expressions. *Nature Neuroscience, 6,* 624–631.

Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology, 3,* 1–14.

Withers, R. D., & Deane, F. P. (1995). Origins of common fears: Effects on severity, anxiety responses and memories of onset. *Behaviour Research and Therapy, 33,* 903–915.

Wolpe, J. (1958). *Psychotherapy and reciprocal inhibition.* Stanford, CA: Stanford University Press.

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