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Fears, Phobias, and Preparedness: Toward an Evolved Module of Fear and Fear Learning

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An evolved module for fear elicitation and fear learning with 4 characteristics is proposed. (a) The fear module is preferentially activated in aversive contexts by stimuli that are fear relevant in an evolutionary perspective. (b) Its activation to such stimuli is automatic. (c) It is relatively impenetrable to cognitive control. (d) It originates in a dedicated neural circuitry, centered on the amygdala. Evidence supporting these propositions is reviewed from conditioning studies, both in humans and in monkeys; illusory correlation studies; studies using unreportable stimuli; and studies from animal neuroscience. The fear module is assumed to mediate an emotional level of fear learning that is relatively independent and dissociable from cognitive learning of stimulus relationships.

Mammalian evolution has required the successful development of defense systems to cope with dangers that threatened to disrupt the transport of genes between generations. In the early mammalian environment of evolutionary adaptiveness (Tooby & Cosmides, 1990), disaster could strike fast and without warning, primarily through hunting predators but also through aggressive conspecifics and from physical events such as falling objects, floods, thunder and lightning, and sudden lack of oxygen. Escape and avoidance were common strategies designed by evolution to deal with such exigencies. At a minimum, they required a perceptual system to identify threats and a reflexively wired motor system to move the organism away from the danger. With more sophisticated nervous systems, the effectiveness could be expanded both at the sensory and the motor ends, and the relation between stimulus and response could be rendered less stereotyped by inserting a central motive state between the two. In this way, the motive state could be activated from innate danger stimuli and serve to promote escape through the flexible tailoring of responses to environmental contingencies (e.g., Archer, 1979). For example, depending on circumstances, the animal would freeze, escape, or attack (e.g., Blanchard & Blanchard, 1988). It is this central motive state that we commonly identify as *fear* (e.g., Mineka, 1979; Öhman, 1993a). An essential characteristic of fear, therefore, is that it motivates avoidance and escape (Epstein, 1972).

Potentially disastrous events sometimes do not strike without notice, however, but may be heralded by subtle cues. For example, to the attentive observer, a predator may announce its presence by faint sounds or odors. By using the contingency between such cues and the potentially deadly consequence, the central motive state of fear could be conditioned to the cue (e.g., Rescorla & Solomon, 1967), which would promote further flexibility in the relationships between stimulus and response. Furthermore, conditioned fear cues could recruit defensive responses in anticipation of the predator's strike, which provides a decisive edge in the arms race between predator and prey (see Hollis, 1982). From this perspective, it is likely that survival-relevant relationships between cues and consequences could be used by natural selection to promote their preferential and selective association in the brains of animals (e.g., Bolles, 1970; Seligman, 1970). The emergence of more advanced nervous systems assured that the outcome that evolution selected for, avoidance of potentially deadly events or situations, could be achieved through more sophisticated and selective mechanisms, such as inborn defense responses, Pavlovian conditioning, instrumental learning, and eventually cognition and conscious deliberation (e.g., Razran, 1971).

Viewed from the evolutionary perspective, fear is central to mammalian evolution. As a product of natural selection, it is shaped and constrained by evolutionary contingencies. It is a central thesis of this article that this evolutionary history is obvious in the fear and phobias exhibited and readily learned by humans. We are more likely to fear events and situations that provided threats to the survival of our ancestors, such as potentially deadly predators, heights, and wide open spaces, than to fear the most frequently encountered potentially deadly objects in our contemporary environment, such as weapons or motorcycles (e.g., Marks, 1969; Seligman, 1971).

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Purpose and Overview

The purpose of this article is to develop a concept of an evolutionarily evolved fear module (Tooby & Cosmides, 1992) that helps to organize and explain important aspects of human fear and fear learning. The article is organized into eight parts. The first one develops the concept of the fear module. Briefly, a fear module is proposed to be a relatively independent behavioral, mental, and neural system that is specifically tailored to help solve adaptive problems prompted by potentially life-threatening situations in the ecology of our distant forefathers. The second and third parts discuss a central feature of the fear module: its relative selectivity regarding the input to which it responds and preferably enters into associations. In the fourth part, we evaluate two alternatives, one evolutionary but nonassociative and one quasi-associative but nonevolutionary, to evolutionarily shaped selective associations as a basis for fear learning. In the fifth and sixth parts, we analyze the role of cognition in fear learning, particularly with regard to the automaticity with which particular stimuli activate the fear module and with regard to the encapsulation of the module from cognitive input. In the seventh part, we review research that delineates the neural basis for the fear module. In the eighth part, finally, we develop a levels-of-learning concept to reconcile data from human conditioning with the database from animal research on fear and its neural mechanisms.

An Evolved Fear Module

The Status of Evolutionary Explanations

From the evolutionary perspective, behavior is more likely to be organized into relatively independent modules than in terms of general-purpose mechanisms (Tooby & Cosmides, 1992). Just as our body is composed of a number of independent organs serving survival and procreation, behavioral and mental systems can be thought of as composed of organs or independent modules (e.g., Fodor, 1983). As a result of natural selection, such modules were tailored to solve specific adaptive problems that were recurrently encountered in the environment of evolutionary adaptiveness (Tooby & Cosmides, 1990).

In our view, evolutionarily based explanations of psychological phenomena have no special status that set them apart from other types of explanations. Although evolutionary hypotheses are not amenable to direct empirical tests in psychological research, basic postulates that are testable only indirectly, in terms of their consequences, are commonplace in science. To take a classic example, Newton's concept of attraction perhaps did not appear to make sense to his contemporaries, but it did make sense of the data on the paths of planets when used in his axiomatic system. In psychology, however, the widespread commitment to "the standard social science model" (Tooby & Cosmides, 1992, pp. 24–34), with its emphasis on learned behavior, collectively known as *culture,* has planted skepticism among many regarding the role of phylogenetic influences on human behavior (see, e.g., Delprato, 1980, with regard to fear). Partly, the skepticism is nourished by popular misuse of evolutionary arguments, in which virtually any psychological phenomenon can be declared post hoc to represent evolutionarily shaped adaptations. But if hedged by some reasonable caveats, we think that evolutionarily based theorizing provides a fruitful avenue for analyzing psychological phenomena.

A putative evolutionary explanation must, just as any scientific explanation, be open to empirical tests; to be more precise, it must be integrated into a conceptual network with testable consequences. Here, the distinction between *theoretical* and *metatheoretical* statements may be helpful (Johnston & Turvey, 1980). The purpose of a scientific theory is to explain a set of empirical phenomena, whereas a "metatheory is concerned with justifying the asking of certain kinds of questions in a particular area of inquiry" (Johnston & Turvey, 1980, p. 149). For example, the questions about fear that we pose in this article are different from those that would be posed by someone inspired by, for example, a social constructivist or psychodynamic metatheory of psychology. The theory of evolution by natural selection is overwhelmingly supported by data from many different fields of science that provide its legitimate testing grounds. Its use in psychology, however, is not motivated by the ambition to subject it to further empirical tests but by its usefulness in posing problems that can be addressed in research, given the methodological constraints of the particular subfield. Merely interpreting something as an evolutionarily shaped adaptation is a dead end unless it stimulates testable ideas that connect the putative adaptation to other concepts in abstract structures known as *scientific theories.*

As a metatheory for psychology, the theory of evolution starts from the premise that humans (as members of an animal species) are part of the biological universe, which implies that not only their anatomy and physiology but also their behavioral capacity have been shaped by evolutionary contingencies. Thus, it is assumed that behavior serves biologically useful functions and that evolutionary processes are helpful in explaining the characteristics of human behavior and human mental life. These statements are broad generalizations that are of little use in accounting for specific phenomena. To analyze specific units of behavior, more circumscribed theoretical statements are needed. First, it must be ascertained that the specific behavior under scrutiny is a meaningful unit in a functional–evolutionary perspective. For example, our argument starts with the premise that fear is anchored in defense systems that are central to evolution. Thus, fear is likely to reflect evolutionary influences. Second, the characteristics of the behavioral units must be specified. For example, what are their response components and activation characteristics? In the case of fear, we argue that it is composed of behavioral, psychophysiological, and verbal–cognitive components, that the activation of the system is automatic, and that it is relatively immune to cognitive influences. Third, as in any scientific enterprise, hypotheses must be generated as to the matrix of causal influences that controls the behavior. With regard to specifying both the characteristics and the causal matrix, the evolutionary metatheory is helpful in suggesting questions that should be addressed and in delineating sets of potential causal factors to consider. For example, we argue that the postulated fear module is particularly sensitive to stimuli that are fear relevant in an evolutionary perspective because they were related to threats that had to be coped with for organisms to survive and leave genes to coming generations. Fourth, in empirically evaluating an evolutionarily based theory, the strategy conforms to standard scientific canon: as stringent empirical tests as possible and the successive ruling out of competing interpretations. The product of this endeavor will be a theory that not only specifies the proximal mechanisms controlling (e.g., fear and fear behavior) but

also provides some insight into the ultimate causal factors that shaped and characterized the underlying adaptations.

The theoretical structure that we end up with, the fear module, comprises four characteristics: *selectivity* with regard to input, *automaticity, encapsulation,* and a *specialized neural circuitry* (see Fodor, 1983). Each of these characteristics is assumed to be shaped by evolutionary contingencies. The selectivity results to a large extent from the evolutionary history of deadly threats that have plagued mammals. The automaticity is a consequence of the survival premium of rapid defense recruitment. Encapsulation reflects the need to rely on time-proven strategies rather than recently evolved cognitions to deal with rapidly emerging and potentially deadly threats. The underlying neural circuitry, of course, has been crystallized in evolution to give the module the characteristics that it has. Thus, even though most of the research has addressed the first characteristic of the module, its theoretical validity as well as the fruitfulness of its origin in evolutionary theory should be judged from the complete set of statements that it generates.

Judging the status of the claim that the fear module represents an evolutionary adaptation is formally similar to the diagnosing of medical conditions. The diagnostic signs of an evolved module that we discuss (relative selectivity to input, automaticity, encapsulation, and dedicated neural circuitry) are not independent, and not one in itself is sufficient to allow the conclusion that the system it characterizes is an evolutionary adaptation. The odds for this inference improve if the system shows all these features, and they can be further improved by considering the functionality of the system in a likely ecology of evolutionary adaptiveness (Tooby & Cosmides, 1990) and by comparative analyses across related species and families. The characteristics, therefore, should be regarded as diagnostic signs; each in itself is insufficient for diagnosing adaptations. Nevertheless, a particular configuration of signs may eventually provide a compelling case for such a diagnosis.

Characteristics of the Fear Module

Selectivity. Basically, the fear module is a device for activating defensive behavior (e.g., immobility or fight–flight) and associated psychophysiological responses and emotional feelings to threatening stimuli. A common requirement of evolutionarily shaped behavioral systems is their relative selectivity with regard to the input to which they respond. Rather than being open to any stimulus, the fear module is assumed to be particularly sensitive to stimuli that have been correlated with threatening encounters in the evolutionary past. By limiting the set of effective stimuli, ready-made neural mechanisms could be devised for identifying critical events after only minimal neural processing, which would serve to facilitate rapid initiation of defense maneuvers. The module may be selectively sensitized to respond to evolutionarily primed stimuli by aversive stimuli or aversive states. Less evolutionarily primed events, on the other hand, would require more extensive neural processing to activate the module and would be less sensitized by aversive states. The range of stimuli that can activate the fear module can be vastly expanded through Pavlovian conditioning, which may give stimuli that happen to predict and coincide with the activation of the module power to activate it by themselves. But rather than positing a general associative apparatus that is independent of the specific to-be-associated events, an evolutionary approach expects constraints on associative learning depending on the specific contexts in which the events have typically been encountered during evolution (Domjan, 1983; Garcia, McGowan, & Green, 1972; Revusky, 1977; Seligman, 1970; Seligman & Hager, 1972). When it comes to fear learning, the context involves situations of recurrent survival threats, and the presupposition would be that events defining such contexts are particularly easy to associate in the interest of promoting effective avoidance of danger. Thus, even though evolutionary fear stimuli would have an advantage, associations between arbitrary cues and fear are by no means precluded but would be more difficult to learn or would be less resistant to extinction.

Automaticity. Evolution may long ago have designed mechanisms to identify stimuli related to recurrent survival threats after a minimum of neural computations and to immediately give them priority (e.g., in terms of efficient attention capture). Because of their origin in animals with primitive brains, behavioral modules that have a deep evolutionary origin typically are not under voluntary control but are directly elicited by stimuli. Thus, the behavior is likely to be elicited whether we want it or not and whether the stimulus has been represented in consciousness. Evolutionarily fear-relevant stimuli, therefore, show characteristics of preconscious automaticity (i.e., they may trigger responses in the absence of any conscious awareness of the stimulus event; Bargh, 1989, p. 11). Automaticity in itself may suggest an evolutionary origin, but it is also clear that automaticity of mental function can be achieved through extensive training (e.g., Shiffrin & Schneider, 1977), even though such automaticity may reflect *postconscious* rather than *preconscious* automaticity in Bargh's (1989) terminology. However, rather than pitting evolutionarily derived automaticity against learned automaticity, it is important to realize that evolution frequently uses extensive experience as a means of shaping neural architecture (e.g., Elman et al., 1996).

Encapsulation. A third characteristic of a behavioral module is encapsulation (i.e., that it is relatively impenetrable to other modules with which it lacks direct connections; Fodor, 1983). Thus, once activated, a module tends to run its course with few possibilities for other processes to interfere with or stop it. In particular, evolutionarily shaped modules will be resistant to conscious cognitive influences because their origin typically precedes recent evolutionary events such as the emergence of conscious thought and language. However, even though the module is relatively impenetrable to conscious influences, influences may be possible in the other direction. When such influence is manifested at the level of conscious cognition, it is likely to be distorting, resulting, for example, in exaggerated expectancies of bad outcomes when the fear module is activated to promote persistent coping attempts. Encapsulation may appear closely related to automaticity. However, automaticity is primarily related to the initiating of activity whereas *encapsulation* refers more to the maintaining of activity over time. For example, behaviors can be automatically elicited yet immediately compensated for by voluntary acts. Electromyographically assessed facial responses, for example, appear to be automatically elicited by certain stimuli, even if the stimulus is not consciously perceived (Dimberg, Elmehed, & Thunberg, 2000). However, this automatic response may be immediately masked by a different facial gesture reflecting culturally determined "display rules" (Ekman, 1972).

Specific neural circuitry. At the neural level, an evolved module is likely to be controlled by a specific neural circuit that has been shaped by evolution because it mediates the functional relationship between ecological events and behavior. In the case of modules that are of ancient evolutionary origin, such brain circuits are likely to be located in subcortical or even brainstem areas. For fear and fear learning, a neural circuit that appears to be shared by mammals has been delineated through important discoveries by several investigators during the past decade (see reviews by Davis, 1992; Davis & Lee, 1998; Fanselow, 1994; Kapp, Whalen, Supple, & Pascoe, 1992; Lang, Davis, & Öhman, 2001; LeDoux, 1996; Rosen & Schulkin, 1998). It is organized around the amygdala, a limbic structure in the medial anterior temporal lobe that mediates input from cortical and thalamic sites to hypothalamic and brain stem nuclei that control various aspects of overt fear behavior. Its sharing among mammals and its subcortical location suggest that it has an ancient evolutionary origin and that it served animals with primitive brains long before more recent biological families with more well developed cortices emerged. In particular, this circuit was firmly established at the base of the brain, which eventually, during relatively recent hominid evolution, became the site of cortical neural networks serving language and advanced cognition. Thus, its ancient origin and location in the brain makes it automatic and relatively impenetrable to cognition (LeDoux, 1996). Indeed, "although the experience of fear can be conscious, the brain mechanisms generating fear and the appraisal of stimuli as fearful are unconscious and automatic, similar to the workings of any other body organ" (Rosen & Schulkin, 1998, p. 326).

Effective Stimuli for Activating and Learning to Activate the Fear Module

Categories of Phobic's Fears

Even though fear may be elicited from many stimuli (e.g., Russell, 1979), intense fears and phobias tend to cluster around objects and situations that are fear relevant in a phylogenetic rather than an ontogenetic perspective (Marks, 1969; Seligman, 1971). Two epidemiological studies (Agras, Sylvester, & Oliveau, 1969; Costello, 1982) established that some stimuli (e.g., thunder, snakes) do indeed become the objects of fears and phobias more often than others. A number of subsequent studies examined this issue in a different way by having raters assess on 5-point scales the evolutionary *preparedness* of the content and behavior of phobic patients' fears. Ratings of 5 were given to objects or situations that were probably dangerous to pretechnological man under most circumstances, and ratings of 1 were given to objects or situations that were unlikely to have ever been dangerous to pretechnological man. Three studies conducted in this manner reported that the content of most clinical phobias was rated in the 4 or 5 range (i.e., de Silva, Rachman, & Seligman, 1977, who had 69 cases in London; de Silva, 1988, who had 88 cases in Sri Lanka; and Zafiropoulou & McPherson, 1986, who had 49 cases in Scotland). Somewhat different conclusions were reached by Merckelbach, van den Hout, Jansen, and van der Molen (1988), who used a different methodology. They asked students and biology researchers to rate the survival relevance of a variety of items that had been proposed as evolutionarily relevant in the literature on fears and phobias. However, given that survival relevance was not restricted to survival promoted by fear-activated defense, the results are not relevant in the present context. For example, poisonous mushrooms no doubt have threatened survival, but the relevant defense system here is taste aversion, not fear.

To organize the large range of stimuli and situations that may serve as objects of fears and phobias in humans, Öhman, Dimberg, and Öst (1985; see also Öhman, 1986) used an evolutionarily based categorization of behavior proposed by Mayr (1974). First, he distinguished between behavior directed toward the living and the nonliving world, called *communicative* and *noncommunicative* behavior, respectively, because only the former would elicit active responses from the environment. Second, within the communicative category, Mayr distinguished between behavior directed toward members of one's own and other species (*intraspecific* and *interspecific* fears, respectively). In this way, Öhman et al. (1985) distinguished between fears of physical objects or events (e.g., heights, thunder; noncommunicative fears), fears of other humans (social fears; communicative intraspecific fears), and fears of animals (communicative interspecific fears), noting that these classes of fear corresponded to three important classes of human phobia: *nature* phobias, *social* phobia, and *animal* phobias (cf. *Diagnostic and Statistical Manual of Mental Disorders,* fourth edition; *DSM–IV*; American Psychiatric Association [APA], 1994).

The Evolutionary Origin of Fear Systems

Predation has provided an important force in moving the evolutionary frontier, from the enormous proliferation of species in the Cambrian era to the emergence first of mammals and eventually of primates several hundreds of millions of years later (Allman, 1999). Where there is predation, there is by evolutionary necessity its complement, predatory defense. These two processes were tied together in an evolutionary arms race in which the emergence of more effective hunting strategies of predators forced potential prey animals to evolve more effective predatory defense and vice versa (Dawkins & Krebs, 1979). Thus, the need for effective predatory defense systems was the likely origin of the fear module, which has shaped its characteristics and explains the prominent role of animals in human fears (e.g., Arrindell, Pickersgill, Merckelbach, Ardon, & Cornet, 1991).

Early and reliable recognition of the predator is a prerequisite for effective defense. For example, Öhman, Flykt, and Esteves (in press) reported a preattentively controlled bias for picking out threatening animals (snakes and spiders) from complex visual displays and that this bias was specifically exaggerated in participants who were fearful of one of the animal categories. Öhman et al. (1985) suggested that the predatory defense system has its evolutionary origin in a prototypical fear of reptiles in early mammals who were targets for predation by the then dominant dinosaurs. Thus, because of this system, contemporary snakes and lizards remain powerful actual fear stimuli (e.g., Agras et al., 1969; Costello, 1982), and for the same reason, dragons have served as mythical embodiments of fear-arousing creatures throughout the history of mankind (Öhman, 1986; Sagan, 1977).

Öhman et al. (1985) proposed that social fears and social phobia originated from a second evolved behavioral system related to conspecific attack and self-defense (see Blanchard & Blanchard, 1988). This system controls the interaction among individuals in a

group by defining which group members boss or yield to which others. The resulting dominance hierarchy provides a vehicle for bringing order into the group and for minimizing further aggressive encounters. Because the focus here is on the relevance of this system for fear, Öhman et al. (1985) talked about the defense or submissiveness part of it, relating social fear and anxiety to exaggerated social submissiveness.

Facial expressions of threat and submissiveness provide an important channel of communication in dominance contests among primates, which makes facial threat a powerful fear stimulus (Dimberg & Öhman, 1996; Öhman & Dimberg, 1984). Like animal stimuli (Öhman, Flykt, & Esteves, in press), facial stimuli suggesting threat are powerful attention catchers in humans. Thus, using carefully matched schematic faces, Öhman, Lundqvist, and Esteves (2001) reported that normal nonanxious participants were quicker to detect a discrepant threatening face among neutral and friendly distractors than a friendly face against neutral or threatening distractors.

Even though Öhman et al. (1985) invoked the differential evolutionary background of these two defense systems to understand differences between animal and social phobias, both interspecific defense and intraspecific defense appear to rely on the same neural system (Blanchard & Blanchard, 1988). Thus, the same basic fear module may serve in the different behavioral contexts provided by the predatory defense and the social submissiveness systems (as well as for the much less studied nature fears). For example, antipredator strategies tend to rely on active defenses that tax metabolic resources, thus prompting cardiac accelerations (see Öhman & Wiens, in press), whereas social submissiveness may rest on behavioral immobility and prolonged risk assessment (Blanchard & Blanchard, 1988), putting less demand on the cardiovascular system. The relative balance between these two types of defenses (active, such as fight–flight, vs. passive, such as immobility) is also related to the imminence of the threat (Fanselow, 1994; Fanselow & Lester, 1988).

Perceptual Mechanisms for Activating the Fear Module

Because of the time constraints of predatory–prey encounters, the more rapid the defense recruitment, the more likely the potential prey is to survive the encounter. Thus, the fear module's judgment of the fear relevance of stimuli is likely to rely on a quick and dirty process that rather risks false positives than false negatives (LeDoux, 1996). Because the fear module originates in primitive brains with limbic cortex rather than neocortex at the top of stimulus-processing hierarchies, recognition of fear-relevant stimuli in limbic structures may be possible in primates. Thus, there are populations of cells in the primate amygdala that respond selectively to faces (Desimone, 1991; Rolls, 1992), and representations of emotional faces in the amygdala may be accessed directly from midbrain and thalamic nuclei (Morris, Öhman, & Dolan, 1999). Furthermore, there is at least one report of amygdaloid cells in monkeys that respond specifically to spiders (Ono & Nishijo, 1992).

Öhman (1993a) argued that there must be an initial stage of perceptual processing involving feature extraction to allow preliminary identification of stimuli that (innately or because of learning) convey information about threatening circumstances. Thus, identifying simple stimulus features that could access and activate the fear module is an important research priority. They may be simple, such as rapid stimulus onsets (Yantis & Johnson, 1990) or directed movements (e.g., Bernstein, Taylor, Austen, Nathanson, & Scarpelli, 1971). However, they may also include more complex characteristics, such as sinusoidal shapes related to snakes or hairy bodies with many protruding legs such as in spiders. For example, Aronoff, Barclay, and Stevenson (1988) reported that participants rated curved lines converging on a joint point, suggesting the legs and body of a spider, as more negative and active than when the same curved lines were regularly arranged in parallel with no point of convergence. Similarly, Lundqvist, Esteves, and Öhman (1999) reported that frowned eyebrows composed of diagonal V-shaped lines provided decisive information for the negative evaluation of schematic faces. Following up on these findings, Öhman et al. (2001) reported that such faces were rapidly and accurately detected among distractor faces in visual search tasks. Further work to identify features or configurations of features that can activate the fear module is important because it would promote understanding of the psychological information-processing mechanisms that control the fear module.

Sensitization and Learning of Fear Responses

The predatory defense and social submissiveness systems discussed by Öhman et al. (1985; Öhman, 1986) suggest that there are stimuli (e.g., snakes and threatening faces) that owe part of their potential to activate the fear module to evolutionary contingencies. However, they are not necessarily innate fear stimuli in the sense that they automatically and invariably activate the module in all individuals. Rather, other conditions such as the presence of other aversive stimuli or a preexisting state of fear or anxiety in the organism may enhance the likelihood of an evolutionarily primed stimulus to elicit fear. Accordingly, the fear of such stimuli could be selectively sensitized to be displayed only in aversive contexts (see Gray, 1982, 1987; Lovibond, Siddle, & Bond, 1993).

As traditionally conceived, sensitization would result in a relatively time-limited enhanced responsiveness to evolutionarily relevant fear stimuli when the fear state is already activated (e.g., Groves & Thompson, 1970). More permanent changes in the tendency of a stimulus to elicit fear would require learning. Thus, in contrast to sensitized fear, learned fear denotes a relatively permanent change in response readiness to stimuli that have been previously encountered in aversive contexts (e.g., Kimble, 1961). Learning is an evolutionarily derived adaptation to cope with environmental changes that occur within the life span of individuals and allows individual organisms to tailor their behavior to the specific environmental niche they occupy (e.g., Öhman & Dimberg, 1984; Plotkin & Odling-Smee, 1981). Learning is costly in terms of the relatively advanced neural circuitry that it requires and in terms of the time needed to get the adaptive response in place (Öhman et al., 1985). For example, if effortful trial-and-error learning was the only learning mechanism available, most animals would be dead before they knew which predators and circumstances to avoid (Bolles, 1970).

The evolutionary cost of learning, however, must be balanced against its potential benefits in solving specific adaptive problems (Johnston, 1982). In general, survival-critical responses to aspects of the environment that remain stable across aeons of time can be efficiently controlled from the gene pool. The panic elicited by

choking may be a human case in point (Klein, 1993). But survival threats also vary across time and space, and there is an enormous number of stimuli and stimulus dimensions that have been connected with fear in different species (e.g., Marks, 1987; Russell, 1979). In such circumstances, as long as it occurs rapidly, learning which specific objects and situations to fear may provide a better solution to adaptive problems than inborn fears. It is better in the sense that it allows the organism to deal with relatively rapid environmental changes, such as new predation pressures, and with the selection of which specific stimuli out of many similar ones to fear and avoid. For example, predators may specialize on a particular prey, and so avoidance of those predators by individuals from other species may only provide unnecessary restrictions in those species' foraging behavior.

The Preparedness Theory of Phobias

Organisms are likely to be conservative in their dealing with potentially fatal situations (Hendersen, 1985; Mineka, 1992). Given the lurking deadly consequences of failures to elicit fear responses, the evolutionary perspective makes it likely that organisms quickly (i.e., with minimal training) would learn to fear potentially deadly stimuli. These premises were incorporated into a theory of fear acquisition by Seligman (1970, 1971). This theory combined the insight that intense fear may result from Pavlovian conditioning (e.g., Watson & Rayner, 1920), the evolutionary requirement of survival contingencies, and the empirical fact that phobias primarily occur to stimuli that are survival relevant in an evolutionary perspective (e.g., Marks, 1969). Seligman (1970, 1971) assumed that evolutionary pressures have predisposed primates to condition fear more readily to stimuli related to recurrent survival threats (phylogenetically fear-relevant stimuli) than to stimuli that never have threatened survival (fear-irrelevant stimuli) or to fear-relevant stimuli that emerged only recently in our evolutionary history (e.g., ontogenetically fear-relevant stimuli such as guns and electric outlets). Seligman (1970) further proposed that prepared associations not only should be easy to acquire (often in as little as one trial) but also should obey different laws of learning than do nonprepared associations. For example, he proposed that prepared associations, relative to nonprepared associations, should be more resistant to extinction—another index of robust conditioning important for associations with survival relevance. Seligman and Hager (1972) added that different cognitive mechanisms and physiological substrates also varied with the preparedness dimension, with prepared associations being less "cognitive" (e.g., less influenced by rational input; p. 5) and being mediated by brain areas of more ancient evolutionary origin.

Selective Associations

The concept of selective associations is the primary feature of Seligman's original preparedness theory of phobias that has remained viable within the area of animal learning (e.g., Domjan & Galef, 1983; Rozin & Kalat, 1971; Schwartz, 1974). The empirical validity of this concept has been supported by many studies in many different species with many different types of learning (i.e., not simply fear conditioning; see Domjan, 1983; Logue, 1979; LoLordo, 1979; LoLordo & Droungas, 1989; Revusky, 1977, for reviews). Other aspects of Seligman's (1970) original theory, such as the assumption that the laws of learning varied across the preparedness continuum, were from early on criticized by learning researchers (e.g., Rozin & Kalat, 1971; Schwartz, 1974) and have not received any serious attention in that field since the 1970s.

A selective association is said to occur when organisms show superior conditioning with certain combinations of conditioned and unconditioned stimuli (CSs and UCSs, respectively) for reasons other than their simple salience. The basic assertion is that there are differences between CSs in the learning they generate when paired with a UCS. If there are two CSs (CS_1, CS_2) and one UCS (UCS_1), this can be formally represented as $(CS_1 - UCS_1) > (CS_2 - UCS_1)$. However, there are two further requirements to conclusively demonstrate that the association is selective. First, it must also be shown that the comparison stimulus (CS_2) can enter into association with some other qualitatively different UCS, say UCS_2, rather than simply being an inadequate stimulus for any conditioning, that is, $(CS_2 - UCS_2) > 0$ (LoLordo, 1979; LoLordo & Droungas, 1989). Second, the organism should not show superior conditioning to CS_1, relative to CS_2, when it is paired with another, qualitatively different, UCS, that is, $(CS_1 - UCS_2) \leq (CS_2 - UCS_2)$. The existence of selective associations clearly challenges what has been termed the *equipotentiality premise* (Seligman, 1970) of Pavlov (1927) and Thorndike (1898). On the basis of this earlier premise, learning theorists presumed that all CS–UCS combinations (or response–reinforcer combinations) are learned about with comparable ease (given comparable salience and intensity of the CSs and UCSs).

The index of selective associations can either be faster acquisition of the conditioned response (CR), the acquisition of a larger response, or enhanced resistance to extinction of that response. Although this point is typically disregarded in the current literature on preparedness and phobias, there is no a priori reason to regard any of these (or other) potential indices of associative conditioning as inherently more valid than any of the other indices (e.g., Kimble, 1961, p. 113; Rescorla, 1980, p. 12).

Selectivity of Input: Basic Findings on Selective Associations From Three Experimental Paradigms

A central thesis of this article is that the fear module is differentially sensitive to different kinds of stimuli. Furthermore, although learning is an important determinant of these differences, evolutionary contingencies moderate the ease with which particular stimuli may gain control of the module. Thus, the likelihood for a given stimulus to be effective in activating the module is a joint function of evolutionary preparedness and previous aversive experiences in the situation. This proposition is supported by behavioral genetic data showing that animal fears and phobias are jointly determined by a genetic factor for phobias and unique individual experiences (Kendler, Neale, Kessler, Heath, & Eaves, 1992).

This section of the article is directed at the role of evolutionarily constrained selective associations in connecting the fear module to new stimuli. Three distinct experimental paradigms have provided converging lines of evidence supporting a role for selective associations in primate fear learning. First, there are a large number of experiments with human participants that have used classical conditioning paradigms, a variety of different psychophysiological indices of conditioning, and occasionally evaluative measures as well (see Öhman, 1993b, for a review). Second, there are a smaller

number of experiments with rhesus monkeys as participants that have used a vicarious or observational fear-conditioning paradigm and several different measures of fear (e.g., M. Cook, Mineka, Wolkenstein, & Laitsch, 1985; Mineka, Davidson, Cook, & Keir, 1984). Third, there are a number of experiments with human participants that have used an illusory correlation paradigm first developed by Tomarken, Mineka, and Cook (1989). The index of selective associations in this paradigm is the demonstration of a covariation bias, that is, a tendency of research participants to overestimate the association between (randomly related) fear-relevant stimuli and aversive outcomes. Before we go into these substantive issues, a brief comment on the measurement of fear is needed.

The Measurement of Fear

The fear module generates a complex output of relatively independent overt fear manifestations that may be grouped into three response systems: *verbal–cognitive, behavioral,* and *physiological* (Lang, 1971). Most experiments on selective associations, selective sensitization, and latent inhibition have used measures from only one or two of these response systems. However, across the three converging lines of experimentation supporting selective associations in fear learning, evidence for its selectivity has been provided using measures from all three fear-response systems. Specifically, some experiments have used subjective or cognitive measures of fear (e.g., subjective ratings of acquired aversiveness [Öhman, Eriksson, & Olofsson, 1975], expectancy ratings of the probability of an aversive UCS occurring [Dawson, Schell, & Banis, 1986], or inflated retrospective estimates of event correlations [Tomarken et al., 1989]); others have used behavioral–expressive measures of fear (e.g., behavioral avoidance or distress [Mineka, Davidson, et al., 1984]); and yet others have used psychophysiological measures of fear (e.g., skin conductance, finger-pulse volume, heart rate [e.g., Fredrikson & Öhman, 1979] and cortical slow wave [Regan & Howard, 1995]). The majority of the human-conditioning studies have used skin conductance responses (SCRs) as an index of conditioning (see Öhman, 1993b, for review). However, like most psychophysiological indices, the SCR does not reflect only fear. Rather, it reflects processes such as attention (Öhman, 1979b), interest, and general emotional arousal, which are related to fear but to other emotional processes as well (Lang, Bradley, & Cuthbert, 1997). Thus, even though SCRs are necessarily evoked by fear stimuli, they are not specific to fear. Fear is perhaps most closely related to a conglomerate of defensive responding that includes heart rate acceleration, increased activity in the corrugator muscle of the face, and potentiation of the startle reflex as the most important components (Lang et al., 1997; Öhman, Hamm, & Hugdahl, 2000).

Human Experiments With Classical Conditioning Paradigms

In the 1970s, Öhman and coworkers (see an early review by Öhman, 1979a) published a series of papers that supported some of the basic assertions of the evolutionary perspective on fear. These studies used classical conditioning paradigms in which pictures with fear-relevant content (e.g., snakes or spiders) were compared with pictures with fear-irrelevant content (e.g., houses, flowers, mushrooms) as CSs for an aversive electric shock UCS in normal nonfearful human participants. The dependent variables were autonomic responses such as skin conductance or finger-pulse-volume responses (FPVRs). Preliminary work indicated that SCRs sometimes were larger to fear-relevant than to fear-irrelevant stimuli even before conditioning and that this difference was sharply potentiated in aversive contexts occasioned by a shock workup procedure and threat of shock—seemingly due to a process of selective sensitization (Öhman, Eriksson, Fredrikson, Hugdahl, & Olofsson, 1974). Thus, to demonstrate enhanced conditioning to fear-relevant as compared with fear-irrelevant stimuli, it was necessary to control for sensitization (i.e., nonassociative) effects of noncontingent shock on responses to the pictorial stimuli.

The between-subject control procedure. Öhman et al. (1975) used a between-subject control procedure in which participants receiving paired presentations of the pictorial CSs and the shock UCSs were compared with participants receiving the CSs and the UCSs unpaired (a control for sensitization) or with other participants given only the CSs. The shock UCS, the intensity of which was determined individually for each participant to an intensity level perceived as uncomfortable but not painful, followed at the offset of the 8-s CS. Regardless of whether they were given one or five shock UCSs, the participants exposed to the CS–UCS contingency showed larger SCRs to CS onset during 10 extinction trials than participants given unpaired CSs and UCSs or only the CS, but only if the CS was fear relevant. Thus, 1 conditioning trial was sufficient to induce resistance to extinction of responses conditioned to a fear-relevant CS, whereas 5 conditioning trials were insufficient to induce resistance to extinction of responses to fear-irrelevant CSs. Furthermore, participants conditioned to the fear-relevant CS were the only ones to increase their fear rating of the CS from before to after conditioning, supporting the idea that verbal–subjective components of fear were also selectively conditioned in addition to the autonomic components (cf. Lang, 1971).

The within-subject control procedure. Öhman, Fredrikson, Hugdahl, and Rimmö (1976) then developed a within-subject control procedure that has been used most often in subsequent research. They used a differential conditioning paradigm in which one stimulus (e.g., a picture of a snake or a mushroom) was followed by a shock UCS, whereas another equally fear-relevant (or fear-irrelevant) stimulus (e.g., a spider or a flower) was presented without any shocks. Because both the stimuli were presented in the aversive context provided by the shock UCS, it was assumed that nonspecific sensitization effects would affect both stimuli to the same degree, given that they were equalized for fear relevance. Öhman et al. (1976) reasoned that if the difference in response to the shock-associated stimulus (the CS+) and the no-shock stimulus (the CS−) for fear-relevant stimuli exceeded the CS+/CS− difference observed for fear-irrelevant stimuli, then the superior conditioning to fear-relevant stimuli predicted by Seligman's (1971) preparedness theory would be confirmed (see Öhman, 1983, for a general discussion of control procedures in human autonomic conditioning). This was the result reported for extinction but not for acquisition in two independent experiments by Öhman et al. (1976). Experiments revealed similar, very rapid rates of acquisition for both groups, quite possibly obscuring potential group differences because of a ceiling effect. These findings were subsequently confirmed in an extensive series of experiments from Öhman's laboratory (e.g., Fredrikson & Öhman,

1979; Hugdahl & Öhman, 1977, 1980; Hugdahl, Fredrikson, & Öhman, 1977; Öhman, Fredrikson, & Hugdahl, 1978; see reviews by Öhman, 1979a, 1993b; see the two upper panels of Figure 1 as an example).

Different conditioned responses to fear-relevant and fear-irrelevant stimuli. Most of these studies used SCRs as the dependent variable, but similar results were reported for other indices of conditioning. For example, although Öhman et al. (1975) failed to observe larger resistance to extinction to fear-relevant stimuli when FPVRs were measured in a between-subject single-cue paradigm, Fredrikson and Öhman (1979) reported reliably better resistance to extinction of FPVRs to fear-relevant stimuli for this measure in a within-subject differential conditioning paradigm.

Furthermore, E. W. Cook, Hodes, and Lang (1986) measured heart rate responses in groups of participants conditioned to fear-relevant (snakes, spiders) or fear-irrelevant (flowers, mushrooms, houses, household objects) stimuli using the standard differential conditioning paradigm with an electric shock UCS (but for some groups also a vibro-tactile/noise UCS). With data collapsed over several experiments, they reported very robust conditioning of heart rate accelerations to fear-relevant stimuli during acquisition trials, whereas the heart rate CR to fear-irrelevant stimuli during acquisition was the typically observed deceleration (see, e.g., Öhman, 1983; Öhman, Hamm, & Hugdahl, 2000). Similar heart rate results were reported from Öhman et al. (1985, pp. 149–154). Thus, the heart rate response conditioned to fear-relevant stimuli was similar to the response shown by animal-fearful participants when exposed to pictorial feared material, which contrasted with the decelerations typically seen to other stimuli (e.g., Globisch, Hamm, Esteves, & Öhman, 1999; Hamm, Cuthbert, Globisch, & Vaitl, 1997).

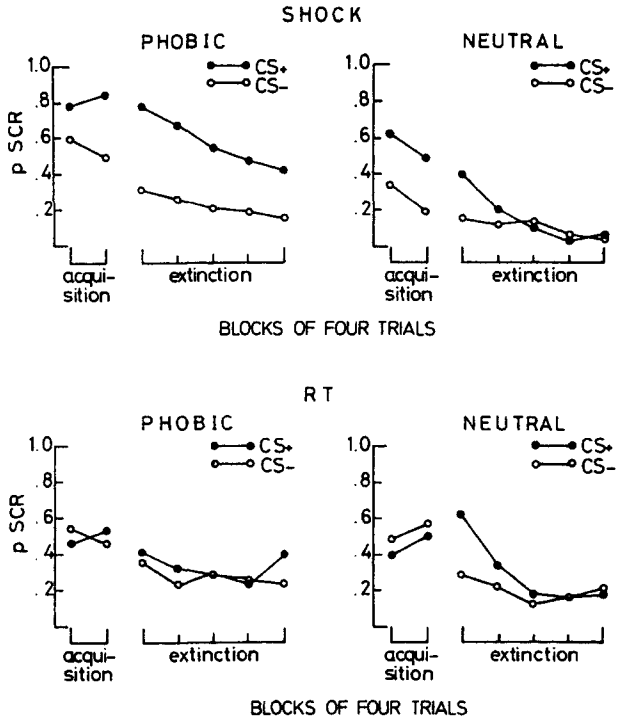

Figure 1. Probability of a skin conductance response (p SCR) for groups of participants conditioned to potentially phobic (e.g., snakes or spiders) or neutral (e.g., flowers or mushrooms) conditioned stimuli (CSs). One of the CSs was followed by an electric shock (upper panels) or the imperative stimulus for a reaction time (RT) task (lower panel) and the other by nothing (the CS+ and the CS−, respectively). Trials are blocked by four. Note that the resistance to extinction (differential responding to the CS+ and the CS−) was considerably larger with phobic than with neutral stimuli in the shock condition and larger with neutral than with phobic stimuli in the RT condition. From "Orienting and Defensive Responding in the Electrodermal System: Palmar–Dorsal Differences and Recovery Rate During Conditioning to Potentially Phobic Stimuli," by A. Öhman, M. Fredrikson, and K. Hugdahl, 1978, *Psychophysiology, 15,* p. 96. Copyright 1978 by Cambridge University Press. Reprinted with the permission of Cambridge University Press.

The pattern of heart rate responses to fear-relevant stimuli suggests that a genuine defensive response was conditioned (Graham & Clifton, 1966; Öhman, Hamm, & Hugdahl, 2000) to these stimuli, which contrasts to the heart rate orienting response apparently conditioned to fear-irrelevant stimuli (see Öhman, 1983; Öhman, Hamm, & Hugdahl, 2000). Even though currently somewhat controversial (E. W. Cook & Turpin, 1997; Öhman, Hamm, & Hugdahl, 2000), the cardiac defense response as traditionally conceived (Graham & Clifton, 1966; Sokolov, 1963) denotes an adaptive response whose function is to down regulate the intensity of aversive stimuli to protect their central processing and to facilitate motor output to cope with them. Thus, the data show that qualitatively different responses are conditioned to biologically fear-relevant and fear-irrelevant stimuli, the former eliciting a defensive and the latter an enhanced orienting response after conditioning. Such findings of qualitatively different responses being conditioned to different CSs are not an anomaly. Rather, findings that the nature of the CS determines the qualitative nature of the CR have been observed widely in the animal-learning literature on classical conditioning (see Bouton, Mineka, & Barlow, 2001, for illustrative examples).

Conditioning to social stimuli. Öhman and Dimberg (1978) extended this finding to a new set of fear-relevant stimuli related to social fears, namely, threatening angry faces. They also used a differential conditioning paradigm, in which one group of participants was required to differentiate between pictures of two different persons showing an angry facial expression by having one of the angry faces followed by shock during the acquisition phase of the experiment. Another group differentiated between two different happy faces, and a third group differentiated between two different neutral faces. The results conformed to those previously demonstrated for animal stimuli by showing reliable acquisition effects in all three groups but reliable resistance to extinction only in participants conditioned to the angry face. In addition, a later experiment demonstrated that conditioning to angry (but not happy or neutral) faces was also evident in enhanced fear ratings, conditioned heart rate increases, and enhanced activity of the corrugator muscle controlling the frowning eyebrow, suggesting the conditioning of a true defensive response only with the fear-relevant angry faces (Dimberg, 1987). (See Dimberg & Öhman, 1996, for an extensive review of conditioning to facial stimuli.)

Controversy concerning replicability. In spite of the consistency of the findings reported by Öhman's group, they quickly acquired a reputation of being fragile and hard to replicate (e.g., Marks, 1981, p. 200; Marks, 1987, pp. 236–237; Menzies & Clarke, 1995b, p. 32), and failures to replicate the effect have been

widely cited (e.g., McNally, 1981; McNally & Foa, 1986; Merckelbach, van der Molen, & van den Hout, 1987). However, as shown in a review by Öhman (1993b), most of these failures to replicate are unconvincing. For example, Merckelbach et al. (1987) used a differential conditioning procedure that differed in details (e.g., number of trials during acquisition and extinction) but was overall similar to the one used by Öhman et al. (1976). The basic failure of their results, however, was that they obtained very weak conditioning effects to both fear-relevant and fear-irrelevant stimuli. For the most relevant SCR measure (the response to CS onset), there was no overall significant conditioning effect, and the reliable interaction between CS and trials was attributable to unsystematic differences in trends over trials in extinction for responses to the CS+ and CS−. Because human differential conditioning with a shock UCS is a highly robust phenomenon (e.g., Dawson & Schell, 1985; Öhman, 1983; Prokasy & Kumpfer, 1973), this indicates that something was seriously wrong with their procedure. Given the failure to obtain a conditioning effect, there is, of course, no basis for expecting differential resistance to extinction of responses to fear-relevant stimuli such as reported by Öhman's group (see e.g., Öhman, 1979a). Similar problems are evident in another study using a differential conditioning procedure (McNally & Foa, 1986; see Öhman, 1993b, for a critique), whereas other alleged replication studies that have failed to produce a preparedness effect (e.g., Deitz, 1982; Emerson & Lucas, 1981; Maltzman & Boyd, 1984; McNally, 1981; McNally & Reis, 1982, 1984) have used dissimilar procedures (sometimes not even including an aversive UCS!; e.g., Maltzman & Boyd, 1984) or primarily have addressed different issues (McNally & Reiss, 1982, 1984).

Moreover, by now there are a large number of experiments from many different laboratories reporting more resistance to extinction of conditioned SCRs to fear-relevant animal stimuli (e.g., E. W. Cook et al., 1986; Davey, 1992, Experiments 3 and 4; Dawson et al., 1986; Schell, Dawson, & Marinkovic, 1991; see Öhman, 1993b, for review) and to fear-relevant social stimuli (e.g., Dimberg, 1986, 1987; Dimberg & Öhman, 1983; Johnsen & Hugdahl, 1991, 1993; Mazurski, Bond, Siddle, & Lovibond, 1996; Öhman & Dimberg, 1978; Pitman & Orr, 1986; see review by Dimberg & Öhman, 1996) than to fear-irrelevant stimuli. It is unfortunate, however, that a wider range of fear-relevant stimuli have not been used yet in these kinds of experiments.

Interaction between phylogenetic and ontogenetic factors in the origins of selective associations. The relative consistency of the results showing superior conditioning to fear-relevant as compared with fear-irrelevant stimuli is encouraging, but it is by no means conclusive regarding the issue of the phylogenetic basis of these differences. This is because the fear-relevant stimuli used may owe their fear relevance to cultural, ontogenetically mediated information rather than to evolutionary, genetically mediated effects. Even in the absence of a significant fear of snakes, spiders, or angry faces, participants may have generally negative associations to such stimuli that could be potentiated when they are encountered as stimuli in a fear-conditioning experiment. In efforts to rule out this competing ontogenetic interpretation, several investigators have performed the more stringent test of comparing stimuli having only cultural connotations of fear and danger (e.g., weapons, broken electrical equipment) with stimuli that also have such connotations (e.g., snakes and spiders) but for which, in addition, phylogenetic factors are likely to operate. If superior conditioning occurs only with the phylogenetic fear-relevant stimuli, and not with ontogenetic or cultural fear-relevant stimuli, this constitutes presumptive evidence for the evolutionary hypothesis as to the origin of the selective associations.

Using classical conditioning paradigms, comparisons of aversive conditioning to phylogenetic and ontogenetic fear-relevant stimuli have been reported in three experiments. First, Hugdahl and Kärker (1981) used electrical outlets as ontogenetically based fear-relevant stimuli. They compared differential conditioning with the electrical outlets with conditioning with phylogenetically based fear-relevant stimuli (snakes and spiders) and with conditioning with fear-irrelevant stimuli (geometric shapes). All three groups showed reliable conditioning effects in the acquisition phase. However, in the second phase, the results supported the evolutionary hypothesis by showing enhanced resistance to extinction for snakes and spiders, relative to both electrical outlets and geometric figures.

Second, E. W. Cook et al. (1986) compared conditioning to snakes, guns (handguns and rifles), and household objects; the UCS was a loud noise rather than shock. They reported superior conditioning, as evidenced by enhanced resistance to extinction to the snakes relative to the guns, in spite of the fact that guns and loud noise should belong together better than snakes and loud noise (see Hugdahl & Johnsen, 1989, below). However, the snake versus household object comparison was not significant (a failure to replicate the traditional preparedness effect probably attributable to the use of a loud noise UCS, because subsequent experiments demonstrated the traditional preparedness effect with snakes when shock was used; see below).

Third, Hugdahl and Johnsen (1989) presented the results of a complex experiment in which SCR conditioning to slides of snakes and guns was compared. The stimuli were either directed straight at or to the side of the participant. The participants were required to discriminate between the direction of the stimuli by having one direction followed either by a shock or a loud noise. Hugdahl and Johnsen hypothesized that conditioning would be best in the groups where the CS and UCS most closely belonged together and that this would occur for both phylogenetic (snake) and ontogenetic (gun) stimuli. Specifically, they predicted (and found) the best conditioning in groups where the CS+ was the snake directed at the participant with the shock UCS and where the CS+ was the gun pointing at the participant with the loud noise UCS (which probably belonged better with a gun as a CS+ than with shock as a UCS). This experiment was interpreted by the authors and by others (e.g., Davey, 1995) as supporting the claim that conditioning to ontogenetic fear-relevant stimuli may be as strong as to phylogenetic fear-relevant stimuli given that they are paired with an adequate UCS that belongs well with the CS+.

However, there are good reasons to question whether the apparent conditioning to the pointed gun in the Hugdahl and Johnsen (1989) study in fact reflected associative conditioning rather than selective sensitization. First, pointed guns are extremely potent stimuli. Emotional ratings from the standardization sample of the International Affective Picture Systems (Lang, Bradley, & Cuthbert, 1996) showed that the pointed guns used in this experiment did not differ from directed snakes in arousal but were rated as inducing more negative valence and less dominance than pointed snakes (i.e., one feels smaller and in a more negative mood in front

of a pointed gun than in front of pointed snakes). For most people in industrialized societies, pointed guns have been frequently and strongly associated with danger (including death) through literature, newspapers, television, and movies. Furthermore, a pointed gun is a deadly threat that can act at a distance, whereas even a snake threatening to bite often can be coped with by withdrawal from the immediate danger zone. In other words, pointed guns are more imminent fear stimuli than directed snakes (cf. Fanselow, 1994; Flykt, 1999). Therefore, based on their ontogenetic history, it does not seem surprising that pointed guns might have come to operate like phylogenetic fear-relevant stimuli in terms of gaining access to the fear module. Second, in support of the potency of pointed guns, the results from the Hugdahl and Johnsen study showed elevated responding to these stimuli even before conditioning, during the habituation phase. Third, during conditioning and the outset of extinction, the participants who had the noise UCS following the gun directed to the side (CS+) nevertheless showed larger responses to the pointed gun, even though it formally served as a CS−! Thus, the initial elevated responding to the pointed gun seems to have been selectively sensitized by the occurrence of loud noise in a very specific manner because the gun pointed to the side was an ineffective CS+. With snakes, however, the direction factor was less potent, according to the interaction between direction and type of CS. Given the independent evidence of selective sensitization to the pointed gun, therefore, the statistically equivalent conditioning to the pointed gun paired with loud noise and to snakes paired with shock may actually have reflected the additive effects of selective sensitization and a weaker associative effect for the pointed gun–noise group (cf. Lovibond et al., 1993, for a discussion of how such effects theoretically may combine).

Finally, even if true selective associations could be demonstrated with pointed guns, we would not consider this very damaging to our account, because ontogenetic and phylogenetic accounts of fear relevance are not inherently incompatible or mutually exclusive. For example, if they are either paired with a very intense trauma (or if paired many times with aversive stimuli), culturally specific objects such as weapons or motor vehicles may come to be strongly associated with fear. This is part of what happens in posttraumatic stress disorder (see Barlow, 1988; Craske, 1999, for reviews) and may also occur in some unprepared fears or phobias (see Rachman & Seligman, 1976, for discussion). However, from the assumption that only degraded input is needed to form selective associations (Seligman & Hager, 1972), it follows that evolutionarily prepared fear stimuli may need to be coupled only with mildly aversive events to come to elicit unreasonably strong fear, such as in phobias (an issue that appears never to have been investigated). Moreover, it follows from the assumption that culture is partly shaped by evolution (Öhman & Dimberg, 1984; Tooby & Cosmides, 1992) that phylogenetic and ontogenetic factors should be mutually supporting in predisposing humans to learn to fear and avoid potentially deadly situations. Thus, although most fear-relevant stimuli may have preferential access to the fear module because of their evolutionary history, some with very strong shared cultural connotations of fear may have access to the module because of a strong ontogenetic history.

To summarize, of the three reported conditioning experiments comparing phylogenetic and ontogenetic CSs, two have found better conditioning to snakes or spiders than to electrical outlets (Hugdahl & Kärker, 1981) and guns (E. W. Cook et al., 1986). The results from the third experiment (Hugdahl & Johnsen, 1989) are more difficult to interpret. There is no doubt that pointed guns are very strong fear stimuli, but to us it appears that their effectiveness when paired with an aversive UCS is better interpreted in terms of selective sensitization than in terms of selective associations.

Selective associations? The enhanced resistance to extinction of autonomic responses conditioned to phylogenetically fear-relevant as compared with ontogenetically fear-relevant and fear-irrelevant CSs is what would be expected for a phylogenetically based selective association. As noted earlier, however, it must also be ruled out that the enhanced conditioning to fear-relevant stimuli does not simply reflect a characteristic of the stimulus, such as salience, but concerns the specific CS–UCS contingency under study. This is most conveniently done by showing that the putative evolutionarily primed fear CS conditions well only in aversive contexts. In addition, it must be shown that the control stimuli (i.e., the fear-irrelevant stimuli) are effective CSs when paired with a different UCS (e.g., LoLordo & Droungas, 1989). Thus, what is needed are experiments comparing conditioning to fear-relevant and fear-irrelevant stimuli with different UCSs, expecting the fear-relevant CS to surpass the fear-irrelevant one only with an aversive UCS. Furthermore, to rule out that the fear-irrelevant CS simply is generally ineffective, significant conditioning to this stimulus with the nonaversive UCS is required.

It is difficult to come up with nonaversive stimuli that can serve as UCSs for human autonomic conditioning, primarily because it is difficult to experimentally control emotionally positive stimuli with an immediate and strong impact. One alternative, albeit one that involves informative rather than emotional stimuli, is to use the imperative stimulus for a reaction time task as the UCS with the CS serving as a warning signal. With this arrangement, differential SCR and heart rate responding to the warning signal (the CS+) and a similar control stimulus not signaling the imperative stimulus (the CS−) can readily be demonstrated (Baer & Fuhrer, 1968; Öhman, Nordby, & d'Elia, 1986). Öhman et al. (1978) adapted this procedure for use with fear-relevant and fear-irrelevant stimuli in an SCR conditioning paradigm. Two groups of participants were conditioned to fear-relevant (snakes, spiders) and fear-irrelevant (flowers, mushrooms) CSs, respectively, with a shock UCS. Two other groups had fear-relevant or fear-irrelevant stimuli as signals for a reaction time imperative stimulus in a differential conditioning procedure. The shock UCS supported differential conditioning irrespectively of fear relevance during acquisition, whereas only the fear-relevant group showed substantial and reliable resistance to extinction, replicating previous findings (see Figure 1). With the reaction time task, there was little evidence of differential responding during acquisition, but during the early extinction trials, there was reliably larger differential responding to the fear-irrelevant stimuli than to the fear-relevant stimuli. Thus, for the extinction data, the fear-relevant stimuli were more effective than the fear-irrelevant ones when the UCS was the aversive shock. With the nonaversive procedure, however, it was the other way around, with better resistance to extinction of fear-irrelevant than of fear-relevant stimuli. In other words, the other requirements for a demonstration of selective associations were met.

LoLordo and Droungas (1989) criticized this conclusion on the basis that the effectiveness of the imperative stimulus UCS ap-

peared doubtful, given that it did not support differential responding during acquisition. Two points can be raised to counter this criticism. First, only eight acquisition trials were used, which may have been insufficient with this UCS. It is unquestionable that a longer acquisition series can result in reliable differential response to a neutral warning stimulus in a reaction time task (Öhman et al., 1986). Second, the reliable differential response during extinction to the fear-irrelevant stimulus that had signaled the imperative stimulus must be attributed to associations formed during acquisition, and as previously indicated, resistance to extinction is as valid an index of conditioning as response amplitude during acquisition (e.g., Kimble, 1961). Thus, on the balance, we are inclined to accept the data reported by Öhman et al. (1978) as evidence supporting the conclusion that conditioning to pictures of snakes and spiders with an aversive shock UCS represents a selective association.

Further support for the selectivity of the snake/spider–shock association comes from results reported by E. W. Cook et al. (1986). They failed to obtain superior resistance to extinction of the SCR to fear-relevant stimuli when the UCS was a loud aversive noise. However, such evidence was obtained when a vibratory stimulus was added to the noise, particularly when the noise was exchanged with an electrical shock UCS. Thus, enhanced differential SCR conditioning to fear-relevant stimuli was observed only with UCSs that were aversive and included a tactile component. E. W. Cook et al. (1986) interpreted this result as suggesting that the selective association for snakes and spiders concerned an insult to the skin with accompanying activation of skin pain receptors. Indeed, there is a direct input from this nociceptive system to the periaqueductal grey area of the midbrain controlling the fight–flight response (Fanselow, 1994). Thus, the selective association may not be between animal stimuli and aversiveness in general but between animal stimuli and aversive stimuli evolutionarily related to injuries produced by small animals.[1]

Hamm, Vaitl, and Lang (1989) pointed out that belongingness between the CS and the UCS had been presumed rather than rigorously tested in previous experiments on selective associations in human conditioning. To rectify this deficit, they used psychophysical scaling methods to ascertain that an angry human face combined with a loud human scream was perceived as belonging better together than any of several other pictorial CSs and more or less aversive UCSs. In a subsequent experiment, they showed reliable FPVR differential conditioning and resistance to extinction for angry faces presented against a background of a light stimuli and where one of the faces provided discriminative information about the scream UCS (i.e., high belongingness). However, for participants given landscape pictures rather than angry faces (and a scream UCS—low belongingness), little differential conditioning was observed. In a third experiment, the roles of the light and pictorial stimuli were reversed. Thus, when the angry face was presented repeatedly with background light stimuli that provided the discriminatory information about scream occurrence, research participants showed less differential responding to the light–face combinations signaling or not signaling the scream than to the similarly informative light–landscape combination. Thus, the highly belonging face–scream relation effectively overshadowed the background light stimulus that provided the discriminatory information about the aversive scream. Hamm et al. (1989) took this as a demonstration of the operation of selective associations in human conditioning. However, they avoided taking a stand on the issue of whether the selective associations they demonstrated reflected ontogenetic (i.e., prior learning) or phylogenetic (i.e., evolutionary predispositions) influences on conditioning, an issue to which we return later.

Taken together, the results from human-conditioning experiments favor the conclusion that participants conditioned to biologically fear-relevant stimuli show larger resistance to extinction than participants conditioned to fear-irrelevant stimuli, as well as participants conditioned to culturally fear-relevant stimuli. Furthermore, there is evidence that this represents the operation of selective associations in that this superior conditioning to fear-relevant stimuli is observed only when the UCS is aversive and especially when it has a tactile component for snakes and spiders. Note, however, that there is a need for more experiments both on the stringent requirements for demonstrating selective associations and the phylogenetic basis of such selective associations.

Monkey Experiments With a Vicarious Conditioning Paradigm

The most important shortcoming of the human studies reviewed so far is that we cannot be certain that similar findings would occur using measures of more significant and intense fears such as occur in real human fears and phobias. Because of self-evident ethical constraints, the level of aversiveness that human research participants can be exposed to is rather limited, and hence it could be questioned whether the electrical shocks typically used are of sufficient intensity to generate a genuine fear response. As a consequence, the eventual CR may be only remotely related to the intense level of fear observed in real phobics. Furthermore, the fact that the psychophysiological index used in most of these studies, the SCR, appears to be more related to attention and to general emotional arousal than specifically to fear (e.g., Öhman, Hamm, & Hugdahl, 2000) casts further doubt on the conclusiveness of a number of these studies. With the goal in mind of examining more intense and unequivocal fear responses, Mineka, Davidson, et al. (1984) and M. Cook et al. (1985) developed an observational, or vicarious conditioning, paradigm to study the origins of snake fear in rhesus monkeys. The choice of a vicarious conditioning procedure was motivated by ecological validity considerations, as well as a previous demonstration of superior vicarious conditioning to fear-relevant stimuli in humans by Hygge and Öhman (1978). They had demonstrated superior conditioning of SCRs to fear-relevant stimuli (snakes, spiders, rats), relative to fear-irrelevant stimuli, preceding another stimulus (UCS equivalent) to which a confederate (an alleged fellow participant) expressed intense (but faked) fear.

The vicarious conditioning paradigm. An additional factor motivating the Mineka, Davidson, et al. (1984) and M. Cook et al. (1985) paradigm was to understand why wild-reared, but not

[1] Note, however, that investigators have sometimes found evidence for superior conditioning with snakes or spiders, relative to fear-irrelevant stimuli, using loud noise as a UCS (e.g., E. W. Cook et al., 1986, Experiment 1, for the phylogenetic vs. ontogenetic fear-relevant stimulus comparison; Hugdahl & Johnsen, 1989; Regan & Howard, 1995). However, it is also quite clear that the results are more reliably obtained with a shock UCS.

lab-reared, rhesus monkeys showed an intense phobiclike fear of snakes (Joslin, Fletcher, & Emlen, 1964; Mineka & Keir, 1983; Mineka, Keir, & Price, 1980). The hypothesis was that monkeys reared their entire lives in the Wisconsin Primate Laboratory (Madison, WI) lacked some requisite learning experience that monkeys reared in India during their first few years of life had gained before capture. Moreover, Mineka and colleagues (1984) and M. Cook and colleagues (1985) reasoned that the requisite learning experience was likely to have been vicarious, because few wild-reared monkeys would have survived direct snake attacks, given the prevalence of poisonous snakes (including deadly cobras) in India.

The results supported this hypothesis, as well as the goal of conditioning more intense fear responses. Lab-reared monkeys who watched wild-reared monkeys behaving fearfully with snakes (real and toy) and nonfearfully with neutral objects (wood blocks) rapidly acquired a fear of snakes that was nearly as intense as that of their wild-reared models (M. Cook et al., 1985; Mineka, Davidson, et al., 1984). Learning was asymptotic after 8 min of exposure to a wild-reared model monkey behaving fearfully with snakes (Mineka & Cook, 1993) and occurred independently of whether the observer and model were related or even acquainted. Moreover, it was evident using three different fear measures and in two different contexts. In addition, the fear showed no signs of diminution after a 3-month follow-up period during which the monkeys were not exposed to snakes. Finally, the mechanisms involved in vicarious conditioning of fear appeared to be highly similar, if not identical, to those involved in traditional classical conditioning (Mineka & Cook, 1993).

The question of primary interest here is whether this rapid, strong, and persistent learning of snake fear in lab-reared rhesus monkeys was an example of a selective association. That is, would lab-reared observer monkeys acquire a fear of other fear-irrelevant objects as readily as they acquired a fear of real and toy snakes?

The selectivity to fear-relevant stimuli. Using an overshadowing design, M. Cook and Mineka (1987) exposed observer monkeys to models behaving fearfully in reaction to simultaneously presented snake and flower stimuli, with no obvious way to know which of the two stimuli the model was reacting to. As would be expected if conditioning of fear to snake stimuli represents a selective association, observer monkeys tested alone exhibited a fear of snake stimuli presented alone in the follow-up tests, whereas they did not exhibit any fear of flower stimuli presented alone.

Although consistent with the selective association concept, overshadowing data by themselves do not constitute conclusive tests of selective associations because they could simply reflect differential salience of the two stimuli; it is well known that a more salient stimulus overshadows a less salient one (cf. Mackintosh, 1983). To conclusively attribute the observed fear acquisition to selective associations, it was first necessary to demonstrate that observer monkeys watching model monkeys behaving fearfully with flower stimuli alone (or some other fear-irrelevant stimulus) would not show as good conditioning as observers watching model monkeys behaving fearfully with toy snake stimuli (which were used to avoid potentially confounding movement cues with a live snake). This result by itself, however, would not rule out differential salience as a possible explanation of the result. Thus, it was also necessary to demonstrate with an appetitive paradigm that monkeys could learn about flowers at least as readily as they learned about toy snake stimuli, thereby ruling out a simple differential salience explanation of the results.

Unconfounded comparison of fear conditioning to snakes and flowers requires equation of the fear exhibited by the model to these two stimuli because the fear exhibited by the model determines the amount of fear learned in these experiments (M. Cook et al., 1985; Mineka & Cook, 1993; Mineka, Davidson, et al., 1984). To control the level of fear exhibited by the model, videotapes of models reacting fearfully (and nonfearfully) were used. First it was ascertained that observer monkeys who watched videotapes of models reacting fearfully to snakes and nonfearfully to neutral woodblocks did indeed acquire a fear of snakes (M. Cook & Mineka, 1990, Experiment 1). Then video-editing techniques were used to make two different types of videotapes from the original tape. On one, the image of the real snake associated with the model's fear performance was edited out, and an image of brightly colored artificial flowers was edited in. On this same videotape, the image of the neutral woodblocks associated with the model's nonfear performance was edited out, and the image of a toy snake was edited in. This was called the reinforced flower (FL+) and nonreinforced snake (SN−), or FL+SN−, videotape. On the second videotape, the image of the real snake associated with the model's fear performance was edited out, and the image of a toy snake was edited in. In addition, the image of the neutral woodblocks associated with the model's nonfear performance was edited out, and the image of the brightly colored artificial flowers was edited in. This was called the SN+FL− videotape. One group of observer monkeys watched the FL+SN− videotape, and another group watched the SN+FL− videotape for 12 sessions of observational conditioning. When tested individually to determine whether they had acquired a fear of toy snakes or flowers, the monkeys in the SN+FL− group evidenced a significant fear of toy snakes but not of flowers (see Figure 2). By contrast, monkeys in the FL+SN− group did not acquire a significant fear of either the flower stimuli or the toy snake stimuli (M. Cook & Mineka, 1990, Experiment 2).

The paradigm used in this initial videotape experiment required the learning of complex stimulus contingencies in the FL+SN− group. Not only was the CS+ a fear-irrelevant stimulus but also there were three kinds of conditioning trials on these videotapes (FL+ trials, SN− trials, and nonfear trials with neutral stimuli). Thus, in a second experiment, discrimination was simply required between FL+ (or SN+) trials and nonfear trials with the neutral woodblock stimuli (i.e., there were no SN− or FL− trials). Again monkeys in the SN+ group acquired a fear of toy snakes, but monkeys in the FL+ group did not acquire a fear of the flower stimuli (M. Cook & Mineka, 1989, Experiment 1). These results eliminated the possibility that the fear-acquisition failure of the FL+SN− monkeys in the first experiment was due simply to the complexity of the conditioning paradigm.

A second concern centered around whether there were inherent characteristics of the inanimate flower stimuli that rendered them unlikely to be associated with aversive outcomes. In a third experiment, therefore, parallel videotapes to those in the FL+SN−/SN+FL− experiment were constructed in which the fear-relevant stimulus was a toy crocodile (C) and the fear-irrelevant stimulus was a toy rabbit (R). The results indicated that monkeys in the C+R− group did indeed acquire a fear of the toy crocodile but

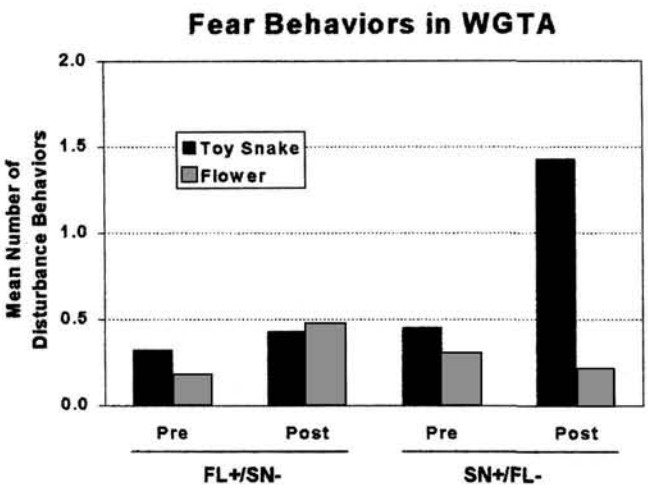

Figure 2. Mean number of fear–disturbance behaviors exhibited by monkeys in the Wisconsin General Test Apparatus (WGTA) in the presence of three stimulus objects (toy snakes, artificial brightly colored flowers, and neutral wood object) for one group exposed to a videotaped model acting fearfully with flowers but not with snakes (FL+/SN−) and another group exposed to a fearful model in connection with snakes but not with flowers (SN+/FL−). Data are presented for pretest and posttest (before and after observational conditioning with the edited videotapes). All monkeys were lab reared. Adapted from "Selective Associations in the Observational Conditioning of Fear in Rhesus Monkeys," by M. Cook and S. Mineka, 1990, *Journal of Experimental Psychology: Animal Behavior Processes, 16,* p. 378, Figure 3. Copyright 1990 by the American Psychological Association.

that monkeys in the R+C− group did not acquire a fear of the toy rabbit (M. Cook & Mineka, 1989, Experiment 2). Together these three experiments provide strong evidence that monkeys are predisposed to acquire a fear of certain fear-relevant stimuli such as toy snakes and crocodiles, when exposed to a model showing manifest signs of fear of these objects.

Selective associations? As already discussed, by themselves such results are not sufficient to demonstrate that superior conditioning to fear-relevant stimuli reflects the formation of a selective association (e.g., LoLordo, 1979). In addition, it is necessary to demonstrate that monkeys are capable of learning about the fear-irrelevant stimuli used in these experiments (flowers or toy rabbit). Thus M. Cook and Mineka (1990, Experiment 3; see also M. Cook & Mineka, 1991) designed an appetitive-discrimination-learning experiment in which the previously used flower and toy snake stimuli were used as discriminative stimuli cuing the availability of food reward. The instrumental learning paradigm used for this purpose had to be quite complex to show that the monkeys used information provided by exactly the same flower and snake stimuli as used in the fear-conditioning experiments to solve the discrimination (see M. Cook & Mineka, 1990, 1991, for further details). Nevertheless, the experiment was successful in demonstrating that monkeys learned about the flower stimuli as readily as they learned about the snake stimuli when they both served as cues for the availability of food reward. Thus, according to these results, the conditions proposed by LoLordo as necessary for demonstration of a selective association appeared to be met for the vicarious conditioning of snake fear in rhesus monkeys.

Phylogenetic versus ontogenetic origin of the effect. The rhesus monkeys used in the vicarious-fear-conditioning experiments on selective associations by M. Cook and Mineka (1989, 1990, 1991) were completely naive with regard to the stimuli used in the experiments. They had lived their entire lives inside the Wisconsin Primate Laboratory and had never been exposed to toy (or real) snakes, toy (or real) crocodiles, toy (or real) rabbits, or brightly colored artificial (or real) flowers. That is, their participation in the experiments involved their first exposure to any of these stimuli (live or on videotape). Thus, it seems extremely likely that the differences observed in the conditionability of fear-relevant and fear-irrelevant stimuli (superior conditioning to a toy snake vs. artificial flowers and to a toy crocodile vs. a toy rabbit) derived from phylogenetic as opposed to ontogenetic factors. Given that the pattern of results from the monkey experiments was generally consistent with that of the results from the human classical conditioning studies, the most parsimonious explanation for these effects seems to be that they do indeed derive from phylogenetic factors.

Countering criticisms of the monkey experiments. Even though the monkey experiments generally have been accepted as the strongest available evidence that phobias represent phylogenetically based selective associations in fear conditioning, Davey (1995) claimed that they were less than conclusive. His primary criticism was that the UCS used in these studies (a model monkey's fear behavior on videotape originally exhibited to a snake stimulus) might have relevance only for snakes and that this might be why the observer monkeys did not show any significant acquisition of a fear of flowers. From the premise that some species of primates have discriminably different fear reactions for different predators (e.g., Cheney & Seyfarth, 1990, for vervet monkeys living in the wild), Davey (1995) argued that the observer rhesus monkeys might have found the signaling information about snakes to be "irrelevant to flowers and toy rabbits" (p. 292). He further argued that although the observers were laboratory reared, they could nevertheless have learned this signaling system through interactions with adults during development. Even though the monkeys lacked prior experience with snakes, Davey (1995) suggested that they could have learned the signaling system through abstract features such as, for example, sinusoidal shapes. Finally, he noted that in one study using squirrel monkeys, experience with certain stimuli like live insects seemed to differentially sensitize a fear of snakes (Masataka, 1993). (See discussion of selective sensitization in next section.)

Several arguments can be raised to counter these criticisms (see Mineka & Cook, 1995, for a more complete account). First, there is no evidence that rhesus monkeys (even wild-reared ones) have a representational signaling system for predators such as that observed, for example, in vervet monkeys in the wild (Cheney & Seyfarth, 1990; C. T. Snowdon, personal communication, 1994). Second, even if there was such a signaling system, there is no evidence that the monkeys reared in the laboratory had learned any fear of sinusoidal shapes such as electrical cords (or real or toy snakes) before observational conditioning (M. Cook et al., 1985; Mineka, Davidson, et al., 1984; Mineka et al., 1980). Third, it also seems that even if there were such a signaling system (so that the fear repertoire of rhesus monkeys differed across situations and that this difference determined ease of conditioning to new stim-

uli), this would seem to strengthen rather than weaken the evolutionary argument (Öhman, 1995).

Finally, the argument concerning selective sensitization by stimuli such as insects, as seen in squirrel monkeys (Masataka, 1993), remains unconvincing. The rhesus monkeys in M. Cook and Mineka's (1989, 1990) experiments clearly did not show selective sensitization (see the next section, Alternatives to Selective Associations: Selective Sensitization and Latent Inhibition). There is also evidence that a fear of snakes in squirrel monkeys (New World monkeys) is probably somewhat more hardwired than is a fear of snakes in Old World monkeys such as rhesus monkeys (cf. Levine, Atha, & Wiener, 1993). Thus, Masataka's findings are irrelevant to what occurs in the acquisition of snake fear in rhesus monkeys. Moreover, this experiment also has been seriously criticized on methodological grounds by Taylor (1997). In summary, Davey's (1995) critique of the M. Cook and Mineka studies does not seem compelling (see also three separate critical commentaries to Davey's, 1995, article by Klein [1995], Öhman [1995], and Tomarken [1995]).

Human Experiments Using a Covariation Bias Paradigm

Tomarken et al. (1989) developed a new paradigm for studying fear-relevant selective associations in humans that did not depend on Pavlovian conditioning (direct or vicarious). The basic idea was that the enhanced resistance to extinction to fear-relevant stimuli seen in classical conditioning studies may reflect a more general covariation bias for fear-relevant stimuli and aversive outcomes. If so, selective associations would also be found in tasks where participants were asked to judge explicitly the association (or covariation) between fear-relevant stimuli and aversive outcomes. This proposition was based on evidence from animals and humans that the perception of a contingency between a CS and a UCS often seemed necessary for conditioning to occur (e.g., Dawson & Schell, 1985; Mackintosh, 1983; although some counterexamples are discussed later). In support of this notion, it has been shown that conditioned responses are often sensitive to the same factors that affect humans' covariation judgments (Alloy & Tabachnik, 1984; Dickinson & Schanks, 1985).

To test the covariation bias hypothesis, Tomarken et al. (1989) developed an illusory correlation paradigm that borrowed many of the central features of Öhman's classical conditioning paradigm (e.g., Öhman et al., 1976). Participants were exposed to three categories of slides (snakes or spiders, flowers, and mushrooms), with each slide followed immediately by one of three types of outcomes (shocks, tones, or nothing). Across 72 slide–outcome trials, the three categories of slides were randomly related to which of the three types of outcomes followed the slides. At the end of the sequence, participants were asked to make judgments concerning the degree of covariation between the slide categories and outcomes. In addition, because individual differences in fear and anxiety tend to be associated with selective processing of threat-related cues (e.g., Mathews & MacLeod, 1994; Williams, Watts, MacLeod, & Mathews, 1988, 1997), Tomarken et al. (1989) also studied two groups of participants: those preselected for fear of the fear-relevant stimulus (snake or spider) and those preselected for little fear of these stimuli. They hypothesized that any covariation bias for fear-relevant stimuli and aversive outcomes might be enhanced in participants already highly fearful of the fear-relevant stimulus.

The results indicated that high-fear participants markedly overestimated the covariation between fear-relevant slides and aversive outcomes relative to their veridical relationship ($p = .33$; Tomarken et al., 1989). In contrast, they were fairly reliable in their estimates of the other categories of slides and outcomes (including the fear-relevant stimuli and nonaversive outcomes and the fear-irrelevant stimuli and aversive outcomes). Moreover, participants were quite accurate in their estimation of the simple base rates of each category of slide and each outcome type. Thus, the overestimation of the snake (or spider)–shock relationship could not be attributed to any general tendency to overestimate the occurrence of snakes (or spiders) or shocks; rather it was simply their covariation that was overestimated, suggesting a true selective association. Low-fear participants showed a tendency toward the same bias, but it was not significant for all of the relevant comparisons, and the bias was significantly lower than that seen in high-fear participants.

In a second experiment, Tomarken et al. (1989) replaced the tone outcome with a highly salient stimulus (a chime used in public settings as an alert signal for messages, plus flashing colored lights) to exclude the possibility that it was the salience rather than the aversiveness of the shock that produced the covariation bias. Although the chime–light was rated as salient a stimulus as the shock, high-fear participants again overestimated the snake–shock contingency but not the snake/chime–light outcome, thus confirming that the covariation bias was specific for the contingency between fear-relevant and aversive stimuli (see Figure 3).

Since these initial experiments, numerous studies have extended the generality of the covariation bias phenomenon in a variety of

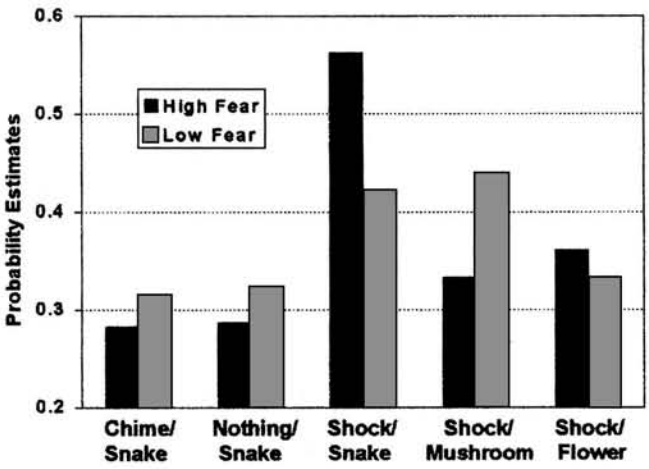

Figure 3. Mean estimates of the conditional probability of outcomes (shock, chime–flashing light, or nothing) given the different slide categories (phobic [snake or spider], flowers, or mushrooms) for high- and low-snake- (or spider-) fear participants. Only the pertinent estimates to demonstrate covariation bias are shown. Shock and the chime–flashing light outcomes were equated on salience. Adapted from "Fear-Relevant Selective Associations and Covariation Bias," by A. J. Tomarken, S. Mineka, and M. Cook, 1989, *Journal of Abnormal Psychology, 98,* p. 387, Figure 2. Copyright 1989 by the American Psychological Association.

ways. For example, De Jong, Merckelbach, Arntz, and Nijman (1992) demonstrated this effect in untreated spider-phobic participants and reported some (albeit not entirely consistent) evidence that the effect disappeared after successful treatment of the spider phobia. This result would be expected given the high–low fear differences typically seen in previous studies. Moreover, De Jong, Merckelbach, and Arntz (1995) reported a significant correlation between any residual covariation bias after treatment and return of fear 2 years later; that is, the larger the covariation bias at the end of treatment, the greater the return of fear at 2-year follow-up. The covariation bias effect with snakes or spiders in high-snake or spider-fear participants was also replicated by Tomarken et al. (1989, Experiment 3), Tomarken, Sutton, and Mineka (1995, Experiment 1), Sutton and Mineka (cited in Mineka, 1992), Amin and Lovibond (1997) and Kennedy, Rapee, and Mazurski (1997).

In addition, several studies have extended the generality of the covariation bias phenomenon by studying other categories of fear-relevant stimuli. For example, Pury and Mineka (1997) examined a new category of fear-relevant stimuli not yet used in this line of research, specifically, stimuli relevant to blood–injury–injection phobia. In three different experiments, they used surgery slides, mutilation slides, and minor injury slides as fear-relevant stimuli, compared with conceptually related fear-irrelevant stimuli (e.g., fixing the inside of an automobile or computer for the surgery slides). They consistently found that participants overestimated the covariation between blood–injury stimuli and an aversive outcome (shock). By contrast, their estimates of aversive outcomes with fear-irrelevant stimuli and shock were quite accurate. However, unlike in previous studies with small-animal phobics, the covariation bias was not affected by participants' level of blood–injury (or mutilation) fear. In addition, Sutton, Luten, Pury, and Mineka (1990) found evidence of covariation bias for socially fear-relevant stimuli such as slides of angry or disgusted facial expressions and aversive outcomes in four separate experiments; the fear-irrelevant stimuli in these experiments were happy and neutral faces. As in the blood–injury studies, however, this effect was not affected by participants' level of social anxiety or trait anxiety (see also De Jong et al., 1998, for related results).

Thus, many studies show that selective associations can be demonstrated in humans using an illusory correlation paradigm rather than a classical conditioning paradigm and judgments of covariation rather than autonomic CRs as the dependent variable. With snakes and spiders as stimuli, the primary difference between the two phenomena seems to be that the illusory correlations to aversive outcomes are often limited to high-fear participants whereas classical conditioning to such stimuli is not dependent on prior fear (McNally & Foa, 1988). Nevertheless, more similar results across the two paradigms have been obtained with other categories of fear-relevant stimuli such as blood–injury and socially fear-relevant stimuli, where the covariation bias effect is not moderated by participants' prior level of fear. The source of these differences, however, is not well understood and remains to be specified (but see Mineka, 1992; Tomarken et al., 1995, for relevant discussions).

Phylogenetic Versus Ontogenetic Basis of the Illusory Correlation Effect

Tomarken et al. (1995) extended Hugdahl and Kärker's (1981) conditioning findings that suggested a phylogenetic origin of the enhanced resistance to extinction to fear-relevant stimuli by comparing covariation bias in high- and low-snake-fear participants using snakes or damaged electrical outlets as the fear-relevant stimuli (a between-groups manipulation). Slides of damaged electrical outlets were used rather than nondamaged electrical outlets (cf. Hugdahl & Kärker, 1981) to maximize the perceived danger potential of these ontogenetic fear-relevant stimuli. High-snake-fear participants showed the expected covariation bias for snakes and shock. However, they did not show a bias for damaged electrical outlets and shocks, even though in a separate experiment the damaged electrical outlets and shock were rated as belonging together better in a semantic sense (cf. Hamm et al., 1989) than did snakes and shocks. Moreover, ratings of affective responses to the snakes and the damaged electrical outlets did not differ on arousal, negative affect, and physical symptoms, suggesting comparable salience and prior fear of the stimuli.

Sutton and Mineka (cited in Mineka, 1992) also showed that under ordinary circumstances, participants did not show significant covariation bias for knives and shock, by using a similar paradigm. The knife was presented in the hand of a male dressed in a black leather jacket, to enhance perceived danger potential. However, a reliable covariation bias was found in participants who had recently been accidentally and naturalistically primed by the presence on campus of an at-large serial rapist who threatened his victims with a knife or by a highly publicized incident involving a fatal stabbing of a fellow student. In both cases, it was hypothesized that the naturalistic priming incident temporarily activated a danger schema regarding knives in the hands of a male who possibly could be an assailant.

Several additional experiments also demonstrated covariation bias with phylogenetic but not technological or ontogenetic fear-relevant stimuli. These experiments were also concerned with the extent to which covariation bias would derive from a priori expectancies (at the outset of the experiment) that shocks might follow fear-relevant stimuli (either phylogenetic or ontogenetic). Each of these experiments found elevated expectancies for shock after both ontogenetic and phylogenetic fear stimuli at the outset of an experiment (termed *a priori* covariation bias), suggesting that at least cognitive expectancies of the fear relevance of the ontogenetic and phylogenetic stimuli were comparable. However, by the end of the illusory correlation experiment, when estimates of covariation between the various stimulus categories and outcomes were made (termed *a posteriori* covariation bias), the bias was exhibited only for the phylogenetic fear-relevant stimuli and shock (Amin & Lovibond, 1997; Kennedy et al., 1997).

Kennedy et al. (1997) also manipulated whether the participants had prior fear of damaged or exposed electrical outlets as well as of spiders. Their purpose with this manipulation was to determine whether covariation bias for ontogenetic fear-relevant stimuli might occur if the participants were highly fearful of the specific stimulus (as in the Tomarken et al., 1989, 1995, studies with snake fear). Nonetheless, even participants highly fearful of electrical outlets did not exhibit a posteriori covariation bias for the electrical outlets and shock.

De Jong et al. (1995) conducted a similar experiment in which snakes and weapons were both included as fear-relevant stimuli (along with one fear-irrelevant stimulus) within the same paradigm. Spider-phobic participants (but not treated phobics) showed elevated expectancies for both weapons and spiders at the outset of the experiment, but by the end of the illusory correlation sequence, they exhibited covariation bias only for the spider–shock covariation. In a sense, these findings are parallel to the conditioning findings reported by Hugdahl and Kärker (1981) and E. W. Cook et al. (1986), who found comparable acquisition with phylogenetic and ontogenetic fear-relevant stimuli but enhanced resistance to extinction only with the phylogenetic stimuli. Analogously, in the covariation bias studies, there were comparable a priori biases exhibited at the outset of the experiment. However, after the random presentations of fear-relevant stimuli, fear-irrelevant stimuli, and aversive and nonaversive outcomes during the covariation bias paradigm, the expected extinction of the bias occurred only for the ontogenetic stimuli, whereas the bias remained to phylogenetic stimuli, which resulted in differential a posteriori biases after the experiment.

In summary, a series of studies comparing ontogenetic and phylogenetic fear-relevant stimuli has consistently demonstrated that a posteriori covariation bias is present only for phylogenetic fear-relevant stimuli. The only exception to this has been when knives were used as stimuli and naturalistic priming incidents accidentally happened to occur closely in time and near the campus where the experiment took place (perhaps thus temporarily sensitizing the participants to a potential male assailant in a way similar to how pointed guns may be chronically sensitized). Thus, human studies using both classical conditioning and covariation bias paradigms converge on the conclusion that selective associations between fear-relevant stimuli and aversive outcomes are restricted to stimulus classes for which an evolutionary background of their fear-evocative power appears likely.

Selective Associations: Concluding Discussion

On balance, across the different sets of results we have considered, we think that it is justified to conclude that selective associations between evolutionarily fear-relevant stimuli and aversive UCSs have been demonstrated for primate fear learning. The most conclusive set of results no doubt is provided by the monkey vicarious conditioning data (M. Cook & Mineka, 1991; Mineka, 1987, 1992). Here it is clearly demonstrated that (a) conditioning of genuine fear responses is stronger to fear-relevant (e.g., snake or toy crocodile) than to fear-irrelevant stimuli (flower or toy rabbit) that are presented in the context of a fear responses from a conspecific (M. Cook & Mineka, 1989, 1990); (b) both these types of stimuli can support learning in a nonaversive context—that is, the association is selective (M. Cook & Mineka, 1990); (c) the vicarious conditioning process conforms to ordinary Pavlovian conditioning with, for example, the snake serving as the CS and the fear response of the conspecific serving as the UCS (Mineka & Cook, 1993); and (d) the basis for these selective associations is extremely likely to be phylogenetic rather than ontogenetic because the monkeys had no prior experiences with any of the stimuli used as CSs in these experiments.

The strength of this conclusion notwithstanding, its primary domain of generalizability is restricted to the rhesus monkey. The question then becomes whether there are sufficient data to warrant a conclusion that similar principles are valid for human fear learning. We believe that this is the case. There are relatively consistent data to support that more robust fear conditioning, as well as conditioning of qualitatively different (defensive) responses, is obtained when fear-relevant stimuli such as snakes, spiders, or angry faces are paired with aversive events than when fear-irrelevant stimuli (e.g., flowers, mushrooms, or happy faces) are presented in a similar contingency.

Regarding the potential preponderance of phylogenetic rather than ontogenetic contributions to selective associations, the comparisons between conditioning to phylogenetically and ontogenetically fear-relevant CSs in humans are suggestive but not conclusive in their own right. However, the converging data from the monkey vicarious conditioning and the illusory correlation paradigms again warrant the conclusion that phylogenetic contributions are the most important ones. It is striking that human participants report a priori biases to the effect that they judge shock as more likely to follow both phylogenetically and ontogenetically fear-relevant stimuli before being exposed to any contingency but that they, after actually experiencing a random relation between an aversive shock and these stimuli, overestimate only the contingency between phylogenetically fear-relevant stimuli and the shock (Amin & Lovibond, 1997; Kennedy et al., 1997). Thus, there is something special with phylogenetically fear-relevant stimuli such as snakes and spiders, which is not there for ontogenetically fear-relevant stimuli such as guns or broken electrical equipment (and at least the damaged electrical equipment seems of comparable salience to snakes; cf. Tomarken et al., 1995). In combination with the monkey vicarious conditioning data and the few human-conditioning experiments, we take these sets of findings as support for the hypothesis that there is selectivity in the relation between evolutionarily fear-relevant stimuli and aversive outcomes when it comes to controlling the fear module. That is, there is strong convergent evidence from these three lines of research not only for the operation of selective associations in primate fear conditioning but also that phylogenetic fear-relevant stimuli have preferential access to the fear module in humans.

Even though these findings are relatively consistent, one must consider whether differences in stimulus salience could account for the differences in strength of conditioning. There are three sets of arguments against differential salience as critical for producing these differences in conditionability. First, this explanation does not easily explain why qualitatively different CRs are conditioned to fear-relevant and fear-irrelevant stimuli (E. W. Cook et al., 1986; Dimberg, 1987). Second, the double dissociation findings reported by Öhman et al. (1978), showing superior resistance to extinction with snakes or spiders with a shock UCS and with flower or mushrooms when the CSs signaled a reaction time task, are very important. Similar results were reported by E. W. Cook et al. (1986) and by Hamm et al. (1989), and the weight of these findings conforms to the selective association results reported for the rhesus monkey by M. Cook and Mineka (1989, 1990).

The final point regarding the differential salience argument concerns the nature of the argument itself. In a sense, saying that fear-relevant stimuli are more salient and that this accounts for all the results on selective associations begs the question of why they may be more salient. Traditionally, salience is a concept defined by the physical characteristics of a stimulus (e.g., brightness,

loudness, color intensity; e.g., Kamin, 1965; Mackintosh, 1974) that represents a parameter influencing the rate of learning in some current models of conditioning (e.g., Rescorla & Wagner, 1972). Defined in this way, salience is nonassociative; that is, it is a characteristic of the CS rather than of the CS–UCS contingency, as is required for a selective association.

In the research on selective associations that we have reviewed, however, salience cannot be defined in this way, because for the stimuli used, the pictorial content is much more important than any simple physical dimension. Rather than being definable through simple stimulus characteristics, the salience that is relevant in these experiments concerns the effects of the stimuli, for example, in catching attention. Indeed, Öhman, Flykt, and Esteves (in press) showed across three experiments that the latency for finding pictures of snakes and spiders among flower or mushroom distractors was shorter than when the search concerned flowers and mushrooms among snakes or spiders. Similarly, across five experiments, Öhman et al. (2001), using perceptually well controlled schematic faces, showed shorter detection latencies for angry faces than for happy faces among both neutral and emotional distractors. These latter results cannot be attributed to simple stimulus characteristics, because the schematic angry and happy faces had physically identical individual features organized in different patterns (see Lundqvist et al., 1999). Therefore, the detection advantage for snakes–spiders and angry faces must reflect something more abstract, such as threat potential. Öhman and coworkers (2001) argued that these findings reflect an evolutionarily derived bias for automatically attending to potentially threatening stimuli.

This conclusion rested on two important findings. First, in the studies with schematic faces (Öhman et al., 2001), potentially confounding factors to the threat interpretation, such as novelty and negative valence, were ruled out in an experiment showing more rapid detection of angry than of a relatively novel scheming expression or another negatively valenced but nonthreatening sad expression. Second, in the experiment using snakes and spiders (Öhman, Flykt, & Esteves, in press) the bias for rapid detection of snakes and spiders was present in all participants, but it was specifically enhanced in participants selected to be fearful of snakes but not of spiders and vice versa. Particularly, the finding that the threat bias was enhanced in fearful participants is important. Presumably, the feared stimuli induced an aversive emotional state of fear or anxiety in the fearful participants, and this state then specifically sensitized them to their feared animal. Thus, the salience was not simply a characteristic of the stimulus, but was enhanced by the emotional context in which the stimulus occurred. As a result, the attention of the participants was automatically and effectively focused on the fear-relevant stimulus, which might facilitate learning about its consequences. Rather than being a confounding factor to be ruled out by appropriate experimental design, therefore, salience could be a mechanism used by evolution to assure rapid learning of fear signals in an aversive context.

Alternatives to Selective Associations: Selective Sensitization and Latent Inhibition

In the previous section, we have reviewed a large set of results that we take to support the conclusion that there are selective associations operating between evolutionarily fear-relevant stimuli and aversive events, thus supporting that fear of such stimuli results from evolutionarily prepared associative learning. However, investigators have argued that associative mechanisms need not be invoked to account for such fear. Two such alternative mechanisms are evaluated in the light of existing empirical data in this section. The first one is *selective sensitization,* which preserves the evolutionary hypothesis but does away with associative learning, and the second one is *latent inhibition,* which uses a quasi-associative mechanism to explain differential efficiency of stimuli in fear conditioning without recourse to evolutionary arguments.

The Selective Sensitization Hypothesis

Gray (1982, 1987) and others have argued that the enhanced responding to biologically fear-relevant stimuli that results from their pairing with aversive events more parsimoniously can be attributed to a nonassociative process, namely, selective sensitization rather than to selective associations. According to this argument, fear-relevant stimuli are genetically encoded directly to elicit fear, but a state of anxiety or arousal is required for actual manifestations of fear to emerge. Note that our concept of selectivity of input to the fear module does not exclude selective sensitization as a factor that may promote enhanced responding to evolutionarily fear-relevant stimuli. However, we do maintain that selective associability is the primary means by which new stimuli acquire their lasting ability to activate the fear module. From a theoretical standpoint, therefore, it is important to know whether selective sensitization or selective associability is the more prominent route to the acquisition of clinical fears and phobias (see, e.g., Menzies & Clarke, 1995b, for a nonassociative account of acquisition of fears and phobias).

There is no question that selective sensitization occurs. Indeed, one of the first publications on preparedness by Öhman and his colleagues' research on this topic did demonstrate selective sensitization with fear-relevant stimuli (Öhman et al., 1974). They showed that fear-relevant stimuli sometimes elicit larger SCRs than do fear-irrelevant stimuli and that this difference is much augmented by the threat of electrical shock (see also Davey, 1992, for related results). Similarly, as already noted, Hugdahl and Johnsen (1989) reported what we believe to be powerful selective sensitization of SCRs to pictures of directed guns in the context of aversive noises that was not manifested for undirected guns (see also Flykt, 1999).

However, early experiments on conditioning also demonstrated that sensitization was insufficient to account for what occurs when a fear-relevant stimulus is presented before an aversive event. In perhaps the most convincing demonstration discussed earlier, Öhman et al. (1975; within a larger design) gave a conditioning group of participants a single-shock UCS after a fear-relevant CS (picture of a snake) and a sensitization group the single shock unpaired with the CS. A third group had only the CS without any shocks. Although initially the unpaired group responded more than the no-shock group during the following extinction trials, the conditioning group maintained a level of responding above the unpaired group throughout these trials. Three comparison groups given fear-irrelevant stimuli (houses) did not differ among themselves as a function of a single UCS administration. Thus, this rapid and sustained conditioning effect was observed only when the CS was fear relevant. A simple selective sensitization inter-

pretation of the preparedness effect cannot account for the more sustained responding to a fear-relevant CS in the conditioning group than in the unpaired group.

As discussed earlier, because of such findings, later experiments on this topic typically have controlled for selective sensitization effects by using a CS+ and a CS− stimuli from the same category (both fear relevant or both fear irrelevant). Therefore, any tendency for selective sensitization (more sensitization to fear-relevant than to fear-irrelevant stimuli) will result in larger responses but poorer differentiation between the CS+ and CS− in a group conditioned with fear-relevant stimuli than in a group conditioned with fear-irrelevant stimuli. Such a result indeed was reported for a group of participants selected for high levels of electrodermal arousal by Hugdahl et al. (1977). These highly aroused participants showed elevated responding to both the fear-relevant CS+ and the fear-relevant CS−, with little differential responding to the two. Participants with lower levels of arousal, on the other hand, were able to suppress their responding to the fear-relevant CS−, which gave room for excellent differential responding to the fear-relevant CS+. For participants conditioned to fear-irrelevant stimuli, on the other hand, differential conditioning was better for those with high than those with low electrodermal arousal (see also Öhman & Bohlin, 1973). These findings suggest that selective sensitization may cause problems under conditions of very high arousal but has little effect in unselected groups of participants.

Lovibond et al. (1993) challenged the adequacy of differential conditioning designs to assure that fear relevance can be attributed to associative processes, by developing a more sophisticated version of the selective sensitization argument. They noted that "this design depends on responses to the fear-relevant CS− being augmented (by selective sensitization) to the same extent as responses to the fear-relevant CS+" (Lovibond et al., 1993, p. 450). During the course of differential conditioning, they argued that a CS− develops fear-inhibitory properties, which they assumed to be comparable for fear-relevant and fear-irrelevant stimuli. As a result, this inhibition may protect the fear-relevant CS− from an augmenting selective sensitization process (see also Davey, 1992). Lovibond et al. (1993) concluded that "there is the same degree of conditioning to the fear-relevant and fear-irrelevant CS+ stimuli, but the fear-relevant CS+ benefits from selective sensitization that is not expressed in the fear-relevant CS−" (p. 451). Thus, they did not deny the role of associative processes in producing differential fear conditioning but argued that the enhanced response observed to a fear-relevant CS+ reflects a combination of conditioning and selective sensitization, whereas that to a fear-irrelevant CS+ reflects conditioning only. In other words, the normal fear conditioned to a CS+ is sufficient to sensitize enhanced responding if the CS+ is fear relevant. This is an ingenious argument, but it is hard to distinguish empirically from the alternative possibility, namely, that enhanced conditioning accounts for the enhanced responses to fear-relevant CSs.

Even though the pattern of responding to fear-relevant and fear-irrelevant CSs+ and CSs− sometimes conforms to these expectations from the selective sensitization hypothesis, this is not the typical finding (although for one consistent example, see the response probability data reported by Öhman et al., 1978, illustrated in Figure 1). Specifically, this hypothesis would predict a higher level of responding to a fear-relevant CS+ than to a fear-irrelevant CS+, but comparable responding to fear-relevant and fear-irrelevant CSs−. A typical finding bearing on this hypothesis comes from a combined analysis incorporating data from 292 participants by E. W. Cook et al. (1986). With tactile UCSs (shock or noise plus vibratory stimulus), there were substantially elevated extinction response probabilities to the fear-relevant CS− (.36) compared with the fear-irrelevant CS− (.25; even though no statistical test was reported). The corresponding CS+ probabilities were .43 and .27, respectively, which resulted in statistically reliable differential responding to fear-relevant ($t = 2.94$, $p < .005$) but not to fear-irrelevant ($t < 1$) stimuli. Note that the significant difference between the fear-relevant CS+ and CS− (.07) actually is smaller than the nontested difference between the fear-relevant and fear-irrelevant CS− (.11). It is hard to see that these data show the protective inhibition to the fear-relevant CS− required by the selective sensitization hypothesis. Rather, responses to the CS− often appear to be augmented by sensitization exactly as expected from the logic behind the differential conditioning control procedure (i.e., greater responding to a fear-relevant CS− than to a fear-irrelevant CS−).

Lovibond et al. (1993) proceeded to test their selective sensitization interpretation in two experiments that are interesting and provocative. However, each used very complex designs that differ from the standard conditioning paradigms in many ways. Overall, in our view, the results of both experiments were partly inconsistent across dependent measures and had several interpretive difficulties, all of which did not make them amenable to straightforward conclusions.[2] Moreover, the effects that could be due to selective sensitization seem far too fragile and short-lived to account for the high degree of resistance to extinction seen with fear-relevant stimuli in most traditional prepared conditioning studies (including one that did not even include CS− in the design—the focus of Lovibond et al.'s [1993] critique; cf. Öhman et al., 1975; e.g., McNally, 1987).

Finally, the selective sensitization account has difficulty accounting for certain aspects of the results of M. Cook and Mineka's (1989, 1990) second experiments. In those experiments, monkeys in the group that watched a videotape of model monkeys responding fearfully to flowers (or a toy rabbit) and nonfearfully to toy snakes (or a toy crocodile) had ample opportunity for selective sensitization to the toy snake (or toy crocodile) stimuli to occur. In the 1990 experiment, flower and snake trials were intermixed, so monkeys often observed a snake stimulus shortly after watching the model's fear behavior with the flower stimulus. Similarly in the 1989 experiment, toy rabbit and toy crocodile trials were intermixed, so monkeys often observed a toy crocodile stimulus shortly after watching the model's fear behavior with the toy rabbit

[2] There are additional interpretive difficulties for both experiments that are not be detailed here but can be provided by Susan Mineka. As one example, for the second experiment, the traditional prolonged extinction effect that essentially defines a prepared conditioning effect with this paradigm was not replicated. Specifically, the lack of differences between the two groups in the test phase must be interpreted in the context that for the preparedness group, consistent differential responding to the combination of a fear-relevant CS+ and a novel fear-irrelevant CS and the combination of a fear-irrelevant CS+ and a novel fear-irrelevant CS was maintained only on 3 of the first 4 trials of extinction, as opposed to the effect that typically lasts for 10 to 20 extinction trials.

stimulus. Yet, results indicated that monkeys in these groups did not show any fear of the toy snakes, or the toy crocodile, respectively, in the final posttests, as would be expected from a selective sensitization viewpoint.

In summary, although it is clear that selective sensitization can occur (cf. Öhman et al., 1974, 1975), it also seems equally clear that such a nonassociative process cannot account for all of the prepared conditioning effects that have been reported in the literature. Thus, some stimuli can come to activate the fear module quite readily either through a process of selective sensitization or selective associations, but only in the latter case are the effects long-lasting and likely to be relevant to acquisition and maintenance of real clinical fears and phobias.

Preconditioning Exposure to Fear-Relevant Stimuli: Latent Inhibition

There is one further alternative nonevolutionary account of basic findings regarding selective associations that also needs to be addressed: Differential latent inhibition (retarded conditioning) to fear-irrelevant versus fear-relevant stimuli (Bond & Siddle, 1996). This account starts with the observation that many of the stimuli used to represent biologically fear-relevant stimuli are relatively rare in the typical ecology of contemporary humans. In particular, these stimuli (e.g., angry faces, snakes) may be less frequently encountered than those used to represent fear-irrelevant stimuli (e.g., happy faces, flowers). This provides a potential confounding factor in many experiments comparing conditioning and extinction to fear-relevant and fear-irrelevant stimuli. As pointed out by Davey (1997), "if the individual has had many trauma-free experiences with a stimulus, it will then be much harder to subsequently associate that stimulus with a trauma" (p. 305). For example, several studies have shown that children who have had more previous nontraumatic encounters with a dentist are less likely to develop dental anxiety if subsequently traumatized than are those with fewer previous nontraumatic encounters before they are traumatized (e.g., Kent, 1997). This is the familiar phenomenon of latent inhibition, which denotes retarded or reduced conditioning to CSs that have been preexposed in the absence of the UCS (Lubow, 1973; Mackintosh, 1983).

Bond and Siddle (1996) have argued that latent inhibition provides an alternative account to evolutionarily constrained selective associations in explaining the effects of fear-relevant stimuli in general, and facial stimuli in particular, in human conditioning. To boost their argument, they measured the frequency with which different facial expressions were encountered in the ecology of their undergraduate experimental participants. Across six different measures, happy faces were the most frequently encountered, followed by angry, surprise, sadness, disgust, and fear. Arguing that more frequent preexposure to happy faces provided for more latent inhibition to them than to angry faces, they went on to test the latent inhibition hypothesis by comparing conditioning to happy, angry, and surprised faces. In keeping with the latent inhibition prediction, they reported larger resistance to extinction of both SCRs and expectancy ratings for participants conditioned to surprised than for those conditioned to angry and happy faces. (There was also at least a small trend for angry faces to show better resistance to extinction than happy faces although the appropriate comparison is not reported.)

The data reported by Bond and Siddle (1996) are interesting, but it is hard to see that the hypothesis they support provides a viable alternative to the selective association account of selectivity of input to the fear module. The primary problem with the latent inhibition hypothesis is that it is dubious whether the experimental results reported by Bond and Siddle can be attributed to latent inhibition at all. In laboratory studies of latent inhibition, great care is taken to present the CS alone in the preexposure treatment defining latent inhibition. In real life, however, exposure to different facial expressions is not independent of outcomes, which more likely are pleasurable after happy and aversive after angry facial expressions. Thus, in this context, preexposure would reduce to prior conditioning: Better conditioning (and more resistance to extinction) to fear-relevant stimuli can be attributed to prior aversive conditioning (e.g., Delprato, 1980). However, in M. Cook and Mineka's (1989, 1990) experiments on monkeys, this factor was controlled because the monkeys had no prior exposure to the CSs before the outset of the experiment (when they were given minimal preexposure to both flowers and snake stimuli to establish that there were no baseline differences in responding). Yet there was more rapid and persistent fear learning to snakes than to flowers and to toy crocodiles than to toy rabbits. Thus, we do not find the latent inhibition account a compelling alternative to a selective association account based on evolutionary contingencies.

Alternatives to Selective Associations: Concluding Discussion

In our view, neither selective sensitization (an evolutionary but nonassociative account) nor differential latent inhibition (a quasi-associative but nonevolutionary account) provides viable alternatives to selective associations (an evolutionary associative account) as explanations for selectivity of input to the fear module or for the nonrandom distribution of fears and phobias that are observed clinically. Both selective sensitization and latent inhibition are very well documented processes in the learning literature, and both may affect learning mediated by the fear module, but neither is sufficient to explain the range of phenomena covered by a selective association account.

A primary problem for selective sensitization as the sole factor accounting for the type of results we have discussed is that at least as traditionally conceived, sensitization is a relatively time-limited process (Groves & Thompson, 1970). Both in the laboratory and in everyday life, however, fears and phobias tend to be persistent and long-lasting. Moreover, none of the experiments on selective sensitization to date provide compelling evidence that it is the primary process operating in these fear-conditioning experiments. Of course, someone might argue that sensitization could be chronic, as in chronic generalized anxiety disorder, and that as a consequence of the chronically elevated level of anxiety, evolutionarily primed fear stimuli would tend persistently to evoke strong fear responses because of selective sensitization. Thus, in this case, the primary problem would be the elevated level of anxiety, and the excessive fear responses to a particular stimulus, such as in phobia, would be only secondary to the elevated generalized level of anxiety. This notion seems contradicted by two lines of evidence. First, specific phobics do not generally have elevated levels of generalized anxiety. For example, results from the *DSM–IV* field trials showed that only 6% of 115 individuals with a primary

diagnosis of specific phobia had a comorbid diagnosis of generalized anxiety disorder (Brown, Campbell, & Grisham, 2000). Moreover, another recent study reported that 20 specific phobics scored within the range of nonclinical samples (and lower than all other anxiety-disordered groups) on a scale measuring negative affect and general distress (Brown, Chorpita, Korotitsch, & Barlow, 1997). The second line of evidence not supporting the idea that phobias are secondary to generalized anxiety comes from the consistent data on the efficacy of exposure therapy for phobic disorders (see the reviews in Davey, 1997). If the primary problem was the underlying anxiety, then extinguishing or habituating the fear evoked by a specific stimulus would have little long-lasting effect but would simply result in substituting the treated stimulus by other sensitized stimuli that would continue to elicit fear and anxiety. On the other hand, if the response to the specific stimulus was the result of a selective association in fear conditioning, the primary problem would be the fear elicited by the stimulus, and the cure of the fear would be its extinction through exposure, a notion that is much more in accordance with the data (e.g., Barlow, 1988; Craske, 1999).

The Automaticity of Fear Activation

We now turn to our second hypothesized feature of the proposed fear module: its tendency to be automatically activated by the kinds of fear-relevant stimuli discussed in the previous section. As will be seen, there is now a large research literature supporting this hypothesis.

The Evolved Dissociation Between Fear Evocation and Cognition

According to the *DSM–IV* (APA, 1994), one of the defining features of phobias is that the victim recognizes the fear as excessive and unreasonable. Thus, at the heart of phobia, there is a dissociation between fear and cognitive understanding that is consistent with the automaticity and encapsulation of fear characterizing the evolved module. Such dissociation was also postulated by preparedness theory, by the presumed noncognitive nature of prepared selective associations (Seligman & Hager, 1972).

If phobias result from a defense system of ancient origin in the class of mammals, this system must have evolved to serve organisms with much more primitive brains than those of contemporary phobics. This happened hundreds of millions of years before the emergence of language and thought in the recently evolving hominids. From this perspective, it is not surprising that strong fears and phobias may not be amenable to cognitive control.

The fear module was postulated to be automatically activated by specific fear stimuli. Indeed, instantaneous activation is very important for defensive responses from an adaptive standpoint. Particularly when interacting with an active agent, be it a predator or a jealous rival in rage, a few milliseconds' difference in defense activation may mean the difference between continued procreation and the disruption of potential gene transports to new generations. Whatever the reason for the evolution of cognition, it did not evolve to support fast responding. Thus, in fearful circumstances in which rapid defense recruitment is called for, it would have been counterproductive to design the system for requiring a complete cognitive analysis of the situation before defense was activated. Rather the defense response should be automatically activated after only minimal analysis of the stimulus. Clearly, in this type of situation, false negatives (i.e., failure to elicit the defense response in a dangerous situation) would be more evolutionarily costly than false positives (i.e., elicitation of a response to a stimulus that turned out to be harmless; e.g., LeDoux, 1990; Lorenz, 1966). The former situation could be deadly, whereas the latter would involve only wasted resources on unnecessary defense responses.

Automatic Elicitation and Conditioning of Fear

Activation of the fear module by nonreportable stimuli: The backward-masking paradigm. The evolutionary scenario suggests that the fear module should be possible to activate automatically, more or less independently of conscious analysis of the stimulus. Öhman and colleagues have developed this argument to imply that it should be possible to activate the fear module from subliminal or nonconsciously presented stimuli, or, to use a more descriptive language, from nonreportable stimuli (e.g., Öhman, 1986, 1993a; Öhman, Dimberg, & Esteves, 1989). This fits with an emerging consensus that there are many types of psychological processes that do not require conscious recognition of the stimuli that control them (see Bornstein & Pitman, 1992; Öhman, 1999, for overviews).

Backward masking is a preferred method of presenting nonreportable stimuli to research participants (Holender, 1986). In this procedure, a target stimulus is very briefly presented and then immediately followed by a second, masking stimulus. With suitable stimulus–onset asynchronies (SOAs) between the two stimuli and with suitable choice of the masking stimulus, the second stimulus may effectively block the first one from conscious recognition. Esteves and Öhman (1993) and Öhman and Soares (1993) adapted a backward-masking procedure for use with biologically fear-relevant and fear-irrelevant stimuli. A fear-relevant stimulus (picture of a snake or a spider, Öhman & Soares, 1993, or an angry face, Esteves & Öhman, 1993) was presented as a target stimulus and was followed by a masking stimulus, which was either a randomly composed picture based on fragments of the snake or spider pictures (Öhman & Soares, 1993) or a neutral face (Esteves & Öhman, 1993). There were also similar trials with fear-irrelevant (flower, mushroom, happy face) stimuli and appropriate masking stimuli. These nonfearful participants were presented with a long series of target–mask pairs with varying SOAs, and they were required to guess the content of the target stimulus as well as to rate their confidence in the response. The results showed that participants both performed at chance level and felt that they were only guessing when the SOA was 30 ms or less. When the SOA was extended beyond this range, performance improved, and when the SOA was above about 100 ms, performance was at a high level, and participants were very confident in their responses.

Activation of the fear module by nonreportable stimuli: Responses of fearful participants. Öhman and Soares (1994) used the backward-masking procedure to test the hypothesis that SCRs can be automatically elicited, in the absence of a full conscious analysis of the stimulus. They selected participants highly fearful of snakes (but not of spiders) or spiders (but not of snakes), as well as a control group of nonfearful participants. A pilot experiment ascertained that both fearful and nonfearful participants were un-

able to report correctly the target stimulus when it was exposed for 30 ms and immediately followed by a masking stimulus. In the main experiment, they first exposed the three groups of participants to masked pictures of snakes, spiders, flowers, and mushrooms and then to nonmasked presentations of these stimuli while SCRs were measured. In spite of the effective masking interval (30-ms SOA), the snake-fearful participants responded more to snakes, and the spider-fearful ones more to spiders, than to any of the other stimuli, whereas participants in the control group did not show differential responding to the different types of stimuli (see Figure 4). Moreover, there were few significant differences in SCR for any of the groups between the masked and the nonmasked stimulus series. Thus, this experiment supported the thesis that stimuli that are denied access to conscious recognition nevertheless can activate at least one physiological component of the fear response. The activation appears to be automatic rather than voluntarily controlled and to reflect nonconscious rather than conscious psychological processes.

Activation of the fear module by nonreportable stimuli: Conditioned responses. From the preparedness perspective, one would expect that many of the participants examined by Öhman and Soares (1994) had been conditioned to fear snakes or spiders, either directly or vicariously, at some point earlier in their life. Thus, similar responses to nonreportable stimuli would be possible to elicit in participants who had been exposed to the standard preparedness conditioning procedure. To test this hypothesis, Öhman and Soares (1993) conditioned research participants either to fear-relevant (snakes, spiders) or fear-irrelevant (flowers, mushrooms) stimuli, using a differential conditioning procedure in which one of the stimuli was followed by an electrical shock UCS using a 0.5-s CS–UCS interval. After a series of acquisition trials, the participants were exposed in extinction to nonreportable CSs+ and the CSs− (each 30 ms long and masked by randomly cut and reassembled pictures). In two independent experiments, the participants conditioned to fear-relevant stimuli continued to show reliable differential responses to the masked stimuli during extinction, whereas masking removed differential responding to fear-irrelevant stimuli. These results were replicated by Soares and Öhman (1993a, 1993b).

Esteves, Dimberg, and Öhman (1994) showed that another class of fear-relevant stimuli, angry faces, also survived backward masking after having served as CS+ in a differential conditioning paradigm (cf. Öhman & Dimberg, 1978). Across three independent experiments, they found that masking the CSs with neutral faces after nonmasked conditioning was ineffective in removing differential responding when the CS+ was an angry face (but not when it was a happy face). The survival of differential SCRs to masked angry CS+ was replicated in three further experiments by Parra, Esteves, Flykt, and Öhman (1997).

In a constructive replication, Wong, Shevrin, and Williams (1994) reported that responses conditioned to negatively evaluated schematic facial stimuli survived backward masking. Thus, participants conditioned to a negative face continued to show differential SCRs in spite of masking. Wong et al. (1994) also measured slow cortical potentials during their experiment. They reported a slow negative shift to the masked negative stimuli (but not the positively evaluated stimuli) that peaked at the point in time of UCS delivery, as if their brains, unbeknownst to the participants, were expecting the shock to appear after this nonrecognized stimulus (cf. Lang, Öhman, & Simons, 1978).

However, the apparent selectivity of the effect to biologically fear-relevant stimuli may be due partly to the general fear irrelevance of the control stimuli used. A more stringent test would involve culturally fear-relevant stimuli such as guns. Such studies were recently reported in a dissertation by Flykt (1999), who conditioned different groups of participants to snakes and guns with electrical shock and noise, respectively, as the UCS, using direction of the CSs as the discriminandum, following the procedure of Hugdahl and Johnsen (1989). Thus, in his first experiment, the CS+ was always directed at the observers, whereas the CS−

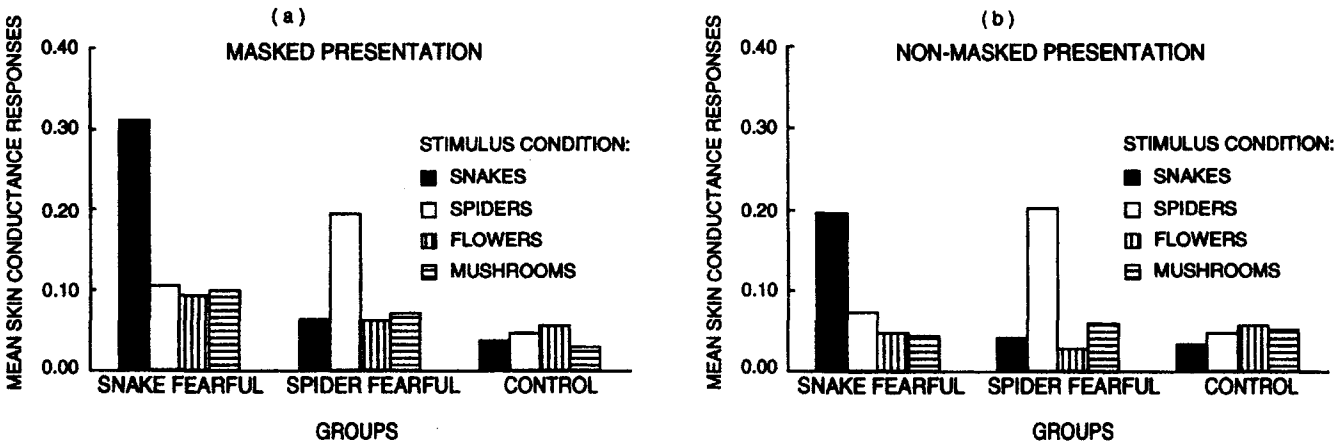

Figure 4. Skin conductance responses to (a) effectively masked and (b) nonmasked presentations of pictures of snakes, spiders, flowers, and mushrooms in groups of participants selected to be highly fearful of snakes (but not spiders) or spiders (but not snakes) and nonfearful control participants. The nonmasked condition always followed the masked condition. From " 'Unconscious Anxiety': Phobic Responses to Masked Stimuli," by A. Öhman and J. J. F. Soares, 1994, Experiment 2, *Journal of Abnormal Psychology, 103,* p. 236. Copyright 1994 by the American Psychological Association.

was always directed perpendicular to the observer. After a number of acquisition trials, the CSs were presented masked in extinction. The results showed reliable resistance to extinction to masked stimuli of conditioned SCRs for both the directed snakes and the directed guns (but somewhat stronger in the former than the latter condition). Furthermore, in a second experiment, when the directed-away stimuli were used as CSs+ there was no evidence of resistance to extinction for either stimulus category.

Overall, these results strongly suggest that responses conditioned to biologically fear-relevant stimuli are automatic in the sense that they can be elicited independent of conscious cognition. Thus, they provide relevant evidence for the automaticity diagnostic criterion of the fear module. However, the results reported by Flykt (1999) cast some doubt on whether this automaticity exclusively pertains to biologically fear-relevant stimuli. They suggest that similar effects may be obtained with extremely potent fear-relevant stimuli of cultural origin when they are presented as part of a CS–UCS contingency very high in belongingness (pointed gun and noise) and when they have very strong ontogenetically based associations with danger and death, as pointed guns do in most societies.

Conditioning of responses to nonreportable fear-relevant stimuli. An obvious question raised by these findings is whether fear-related responses can also be conditioned to nonreportable stimuli. Given the heretofore nearly unanimous view in the human-conditioning literature that participants must be able to report the CS–UCS relation to show autonomic evidence of conditioning (see reviews by Dawson & Schell, 1985; Öhman, 1983), this may appear a remote possibility.

To examine this question, Esteves, Parra, Dimberg, and Öhman (1994) conditioned research participants to masked (by a neutral face) pictures of angry or happy faces using SCRs as the dependent variable. Their first experiment compared differential responding to nonmasked presentations of angry and happy faces during extinction, as a function of different treatments during acquisition. One experimental group was conditioned with an effectively masked (30-ms SOA) angry face as the CS+ (i.e., followed by shock after 1 s) and a similarly masked happy face as the CS− (i.e., without any shocks). A second group was given the same conditioning procedure but with ineffectively masked (330-ms SOAs) CSs. This group was included to make sure that conditioning to a clearly perceived CS was possible even with a disrupting stimulus presented in the CS–UCS interval. A third group was simply given the masking stimuli with no preceding CSs. Thus, similar to what was perceivable by the first conditioning group, this group of participants was exposed to a series of masking (neutral) faces, some of them consistently followed by shock. During the nonmasked extinction trials, both conditioning groups responded more to the angry than to the happy faces (i.e., to the CS+ than to the CS−), whereas the third group showed no differential response to these stimuli. Thus, in obvious contradiction to the thesis that human conditioning requires that participants can verbally report the CS–UCS contingency (Dawson & Schell, 1985; Öhman, 1983), these data demonstrated conditioning under conditions precluding awareness of the CS+ and thus of the CS–UCS contingency.

In a second experiment, Esteves, Parra, et al. (1994) included a set of groups that were exposed to effectively and ineffectively masked faces as CSs but where the happy rather than the angry face served as the CS+ (and the angry face served as CS−). These groups were compared with a set of groups that replicated the first experiment (i.e., an angry face as the CS+ and a happy face as the CS−). As in the previous experiment, only participants who had been exposed to masked angry CSs+ during acquisition showed larger responses to the angry faces than to the happy faces during extinction. There were no differences in conditioning between groups given effectively and ineffectively masked angry CSs. Participants exposed to (effectively or ineffectively) masked happy faces as the CS+ showed no evidence of conditioning. Thus, this experiment replicated the effectiveness of masked angry faces to serve as CSs+ and showed that a masked happy face did not result in conditioning when used as the CS+.

Öhman and Soares (1998) also examined conditioning to masked stimuli using pictures of snakes and spiders as fear-relevant CSs and pictures of flowers and mushrooms as fear-irrelevant CSs. In one experiment, they presented masked acquisition trials in which one group of participants received masked presentations of snakes and spiders and another group received masked presentation of flowers and mushrooms. The masking interval was 30 ms, and the shock UCS followed one of the target–mask pairs by a CS–UCS interval of 0.5 s. A given mask could occur both on CS+ and CS− trials, so there was no way for the participant to know whether the shock UCS was imminent. Conditioning was assessed on masked test trials with no shock during acquisition as well as on nonmasked extinction trials. There was unequivocal evidence of conditioning on both these types of trials, but only for the participants exposed to fear-relevant (and not fear-irrelevant) CSs (see Figure 5).

In a second experiment, Öhman and Soares (1998) used the same procedure as that used with the fear-relevant group in the previous experiment but extended the CS–UCS interval to allow measurement of anticipatory SCRs on every trial of acquisition rather than on test trials only, as in the previous experiment. To ascertain that the participants remained unable to report the CSs throughout the experiment, one group of participants was asked to guess after each trial whether a snake or a spider had been presented. A second group of participants was asked to manipulate a small lever to indicate their expectancy of shock on a continuous scale from −100 (*absolutely sure of no shock*) to 100 (*absolutely sure of shock*), with 0 indicating that shock and no shock were equally likely. A third group was exposed to the experimental contingency without any extra task. All three groups showed reliable conditioning of SCRs both during masked acquisition and nonmasked extinction trials. For participants asked to guess the nature of the CS during the masked acquisition, the proportion of correct responses was .505. However, participants required to explicitly state their shock expectancies actually showed small but significant differential expectancies of shock to the CS+ and the CS− both during acquisition and extinction. Thus, even though, as judged from the performance of the previous group, the nature of the CSs was clearly nonreportable, these participants appeared to have access to some aspect of the CS that they could use to guide their expectancy ratings.

Katkin, Wiens, and Öhman (in press) replicated the procedure used by Öhman and Soares (1998), and in close agreement with the previous results, they reported both reliable SCR conditioning to masked snakes and spiders and small but reliable differential expectancy ratings to the masked CSs+ and CSs−. Using the

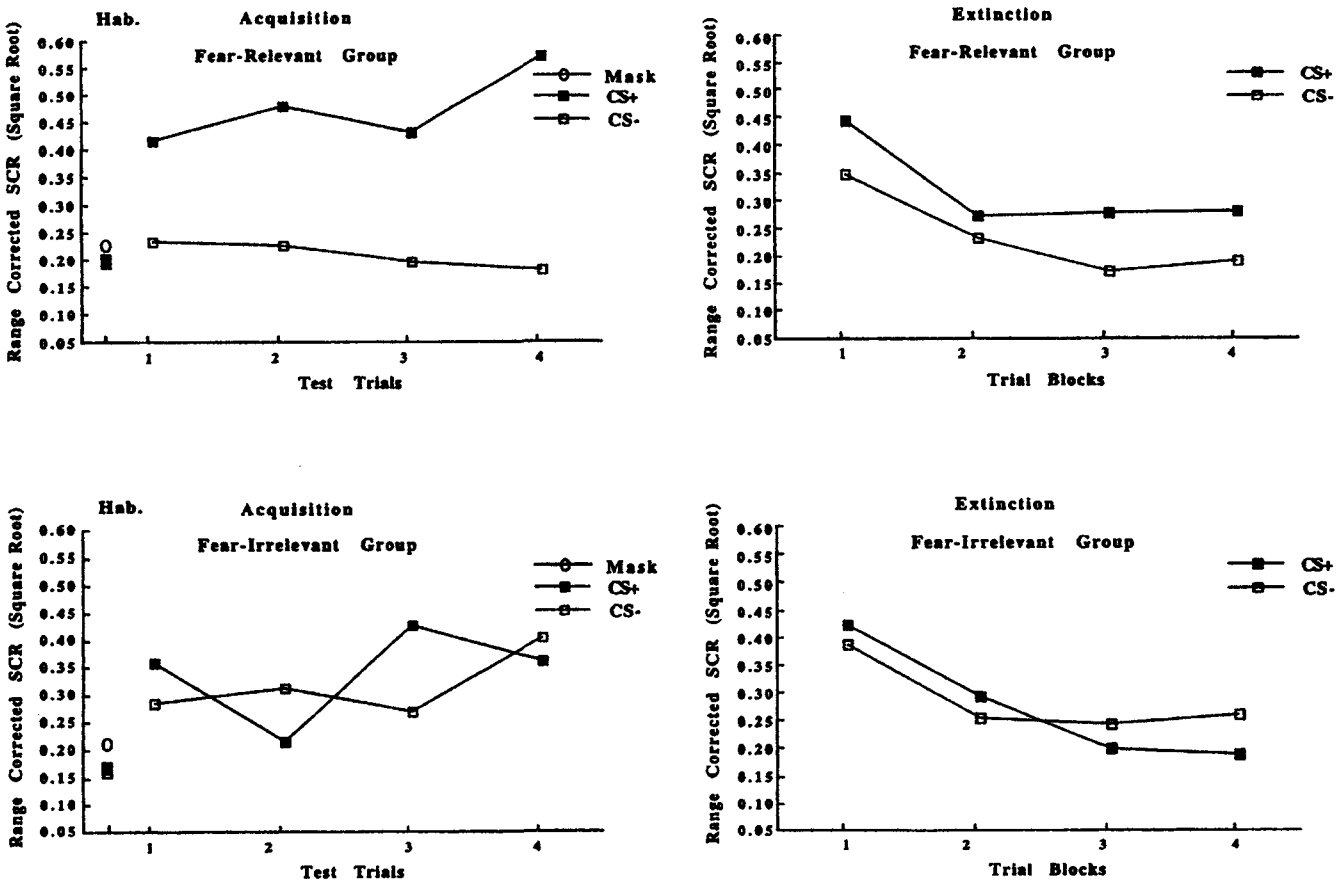

Figure 5. Skin conductance responses (SCRs) to masked acquisition test trials (left panels) and nonmasked extinction trial blocks (right panels) in participants aversively conditioned to snakes or spiders (fear-relevant group; upper panels) and flowers or mushrooms (fear-irrelevant group; lower panel). One of the pictorial conditioned stimuli (CSs) was followed by an electrical shock unconditioned stimulus during acquisition (the CS+), and the other was followed by nothing (CS−). Note the reliable conditioned differential response both during (masked) acquisition and (nonmasked) extinction in the fear-relevant group. Hab. = habituation. From "Emotional Conditioning to Masked Stimuli: Expectancies for Aversive Outcomes Following Nonrecognized Fear-Relevant Stimuli," by A. Öhman and J. J. F. Soares, 1998, *Journal of Experimental Psychology: General, 127,* p. 73. Copyright 1998 by the American Psychological Association.

procedure developed by Katkin (1985; Eichler & Katkin, 1994) to assess autonomic perception, Katkin et al. (1999) regrouped their participants into those that were good or poor heart beat discriminators (assessed in a separate experiment) and found that only the good heart beat discriminators were able to differentiate the masked CS+ and CS− in their expectancy ratings. Thus, because differential expectancy ratings were evident only in participants showing skills in autonomic perception, these results imply that feedback from conditioned autonomic responses has a decisive role in providing information about the emotional response that can be used to guide verbal ratings. From this interpretation, it appears that the participants were unconsciously conditioned to masked fear-relevant stimuli and that this CR gave perceivable feedback that could be consciously accessed to guide ratings of shock imminence, at least for participants skilled at autonomic perception.

Automatic Fear Responding: Concluding Discussion

The results indicate that human participants can learn to associate fear (or at least some level of aversiveness) with a stimulus that they do not consciously perceive. This effect is primarily observed with biologically fear-relevant stimuli such as snakes, spiders, and angry faces, and for such stimuli, conditioning to effectively masked CSs does not appear to be poorer than conditioning to ineffectively masked CSs. With clearly fear-irrelevant stimuli such as flowers, mushrooms, or happy faces, no conditioning is observed when they are masked and presented with an aversive UCS. Thus, in the past decade, many experiments have clearly illustrated that the fear module meets the second diagnostic criterion for an evolved behavioral module. Specifically, it is mostly not under direct voluntary control but rather is activated extremely rapidly and automatically, independent of intentions, and often without conscious awareness. We now turn to evidence

supporting the third and related hypothesized characteristic of the fear module: its encapsulation from higher cognitive influences.

Cognition and Conditioning: The Encapsulation of the Fear Module

LeDoux (1996) has emphasized that the central structure in the neural fear circuitry, the amygdala, has more dense efferent than afferent connections with the cortex. Thus, it sends more information to the cortex than it receives. This neuroanatomical fact has been used by LeDoux (1996) to argue that fear affects cognition more than it is controlled by it. Once activated, fear runs its course, with limited possibilities for cognitive interventions. Indeed, animal data suggest that there is a monoamine-mediated shutdown of the prefrontal cortex in stressful circumstances, as the control of behavior is relinquished to subcortical defense circuits (Arnsten, 1998). A similar deactivation of prefrontal areas related to working memory during strong fear has been reported from human positron-emission tomography (PET) studies of specific phobics (Rauch et al., 1995). When in danger, we are designed for relying on the evolutionary wisdom distilled in the fear module rather than for trusting our own thinking (LeDoux, 1996). Thus, there is a neuroanatomical underpinning for the encapsulation of the fear module.

The next section of the article is devoted to the role of cognition in human fear. The encapsulation, particularly to higher cognitive influences, that was postulated as an important characteristic of the fear module suggests that fear responding, at least that evoked by fear-relevant stimuli, should be quite immune to influences from cognitive factors such as expectancy. Thus, regardless of whether participants expect bad or good things to happen, the fear response should run its course, once it has been activated. Even though in agreement with preparedness theory, this postulate is contrary to contemporary research giving expectancy a key role in understanding human fear responses (e.g., Davey, 1995, 1997). Therefore, it is important to evaluate these two opposing perspectives on the relationship between cognition and fear responding.

The Role of Expectancy in Human Fear Responding

Hugdahl and Öhman (1977) first attempted to capture the irrationality of phobias in an experimental paradigm in which participants were instructed that no more shock UCSs would be presented during extinction. They reported that for participants conditioned to neutral fear-irrelevant stimuli (circles and triangles), SCRs were immediately extinguished after such instructions but that the same instructions had no effect on participants conditioned to fear-relevant stimuli. Soares and Öhman (1993b) replicated this findings by combining masking–no masking of the CSs in extinction with instruction–no instruction that no more shock UCSs would be presented during extinction. The effects of the masking and instruction manipulations were essentially additive. Thus, in agreement with the hypothesis that the processing of fear-relevant stimuli is less dependent on conscious cognition, both manipulations removed differential responding to fear-irrelevant stimuli but left reliable differential responding to fear-relevant stimuli. In fact, even participants given masked presentations and explicit instructions that no more UCSs would be presented showed reliable differential responding to the fear-relevant CS+ and CS−. Hugdahl and Öhman (1977) and Soares and Öhman (1993b) excluded participants who admitted that they had doubted the validity of the instruction, but they made no effort to actually measure expectancies of the UCS after fear-irrelevant and fear-relevant CSs—a topic to which we now turn.

Different types of expectancies about aversive UCSs. From ratings of UCS expectancy before and during conditioning, it appears that human participants enter conditioning experiments with an exaggerated a priori expectancy that aversive events will follow fear-relevant stimuli even when instructed that no aversive stimulus will be presented (Davey, 1992). This tendency is enhanced in participants selected to be fearful of the specific fear-relevant stimulus they are exposed to (Diamond, Matchett, & Davey, 1995). Moreover, when the experimental instructions include an explicit threat that an aversive UCS may follow a specific cue, expectancy ratings for the aversive outcome are much more inflated if the cue is fear relevant than if it is fear irrelevant (Davey, 1992, Experiment 2). This effect, however, is independent of whether the fear-relevant stimulus derives its fear-evoking power from phylogenetic (snakes, spiders) or ontogenetic (guns) sources (Lovibond, Hanna, Siddle, & Bond, 1994). Finally, the a priori differential expectancy of aversive outcomes between fear-relevant and fear-irrelevant stimuli was maintained in trial-by-trial (or on-line) expectancy ratings in spite of repeated exposures to equally probable aversive outcomes after fear-relevant and fear-irrelevant stimuli in a modified covariation bias paradigm (Amin & Lovibond, 1997; De Jong et al., 1995). Except for the lack of difference between ontogenetic and phylogenetic fear-relevant stimuli, these results show that measures of expectancy track many of the effects of fear-relevant stimuli previously reported for conditioned autonomic responses (e.g., Öhman, 1979a). Thus, experimental contingencies and autonomic responses appear to be reflected in the participants' reportable expectancies of what is likely to happen in the situation.

However, the relationships between different measures of expectancies are complex. As previously discussed in relation to covariation biases, in contrast to data from a priori and on-line expectancies, there is an a posteriori covariation bias only for biological fear-relevant stimuli when participants are asked to make retrospective estimates of covariations between cues and outcomes after the experiment (Amin & Lovibond, 1997; Kennedy et al., 1997). Nevertheless, in spite of this dissociation, measures of a priori and on-line expectancy and a posteriori covariation biases show substantial intercorrelations (Amin & Lovibond, 1997; De Jong et al., 1995). When initial and on-line expectancies and SCRs were entered into a multiple regression analysis to predict a posteriori covariation bias, only on-line expectancy turned out to be an independent predictor (De Jong et al., 1995; but about 40% of the variance in covariation bias was left unexplained by the other measures). Thus, different expectancy measures appear to share a common variance component that may account for some of the findings, but different aspects of expectancy must obviously be taken into account to explain all of the data.

An expectancy alternative to preparedness. Davey (1995; 1997) used results on expectancy to develop a conditioning alternative to preparedness theory (Öhman et al., 1985; Seligman, 1970, 1971) that does not rely on any evolutionary explanatory framework (Davey, 1997). According to this model, the relationship between the CS and the cognitive representation of the UCS

is mediated by an outcome expectancy activated by the CS. Such expectancies are determined not only by "situational contingency information" and "emotions elicited by the CS" but also by "verbally and culturally transmitted information about the contingency" and by "existing beliefs about the CS and the contingency" (Davey, 1997, p. 305). As a result, the participants bring expectancies about a UCS likely to follow a CS with them to the experiments (a priori biases), others are determined by instructions, and yet others are developed as a function of the experimental contingencies to which the participants are exposed. Davey (1995) suggested that "identifying the factors that determine (1) the a priori expectancy bias and (2) the rate of disconfirmation of this bias should provide a predictive model of selective associations" (p. 295).

Davey (1992) further proposed that "UCS expectancies will play a major role in determining the autonomic CRs traditionally measured" (p. 37). The expectancy, however, is not sufficient to support SCRs, because the "cognitive representation of the UCS" must also be primed (e.g., by prior presentations of the stimulus). The representation of the UCS may be modified by subsequent habituation to the UCS alone but also by "UCS revaluation processes" in the absence of actual UCS presentations. Such processes include "socially/verbally transmitted information about the UCS," "interpretation of interoceptive cues," "cognitive rehearsal of the UCS," and "coping strategies which neutralize the UCS" (Davey, 1997). Such UCS revaluation processes may inoculate what appeared to be traumatic conditioning episodes ("The masked robber was merely a boy with a toy gun") or inflate what appeared to be an innocuous conditioning episode ("Falling into this river may be dangerous; there may be alligators around").

Associations between expectancies and SCR conditioning. We now turn to a discussion of research that has been used as support for expectancy theory. The pioneering study on expectancy, SCRs, and fear-relevant stimuli was reported by Dawson et al. (1986). They used a complex differential conditioning procedure to examine whether expectancy ratings and SCR were dissociable. Fear-relevant (snakes and spiders) and fear-irrelevant (flowers and mushrooms) CSs+ and CSs− were embedded among other pictures in what was presented to the participants as a visual memory experiment in which their task was to recall pictures in a specified order. Shock was included allegedly to manipulate arousal. The purpose of this paradigm was to retard the participants' discovery of the relationship between the critical CS component (which was fear relevant for one group and fear irrelevant for another) and the electrical shock UCS. Expectancy for the UCS was assessed on a trial-by-trial basis. Dawson et al. hypothesized that conditioning to the fear-relevant CS would be less dependent on expectancies than conditioning to fear-irrelevant stimuli. However, they did not observe any differential SCRs to either type of CS before the conditioning trial on which the participants gave evidence of having discovered which stimulus predicted the UCS. Furthermore, the extinction of expectancies and SCRs occurred in parallel, with no evidence of differential responding to the CS+ and the CS− after expectancy extinction, even though extinction was slower for both measures when the CSs were fear relevant. Thus, Dawson et al. were the first to report a strong association between enhanced UCS expectancy after fear-relevant CSs and enhanced SCRs to these stimuli.

The role of expectancy in SCR conditioning to fear-relevant stimuli was further analyzed by Davey (1992). In a series of experiments, he demonstrated an expectancy bias for an aversive UCS (a vibrotactile–loud tone stimulus) after fear-relevant but not fear-irrelevant CSs when expectancy was manipulated both by the presentation of actual aversive UCSs and explicit threat (without actual presentations) of such stimuli. However, the expectancy bias was not manifested in differential SCRs to fear-relevant versus fear-irrelevant CSs in the absence of actual UCS presentations, perhaps because the internal representation of the UCS was too weak and ill defined to translate into SCRs. Accordingly, a single exposure to the actual UCS, either preexperimentally (Experiment 2) or at some point during the experiment (Experiment 4), tended to transform the enhanced UCS expectancies associated with fear-relevant stimuli into differential SCRs.

In a regular differential conditioning experiment with the noise–tactile UCS (Davey, 1992, Experiment 3), the participants were explicitly instructed that no UCS would be presented during preconditioning habituation trials and during extinction. The UCS expectancy ratings showed higher expectancies for fear-relevant than for fear-irrelevant stimuli during habituation and better differentiated expectancies between CSs+ and CSs− when the stimuli were fear relevant rather than fear irrelevant during both acquisition and extinction. Skin conductance showed largely parallel findings except that there were no reliable effects of fear relevance during the first phase of the experiment (habituation). Note that this experiment showed reliable resistance to extinction both of expectancies and SCRs to fear-relevant stimuli in spite of explicit instructions that no shocks would be given during the extinction phase (cf. Hugdahl & Öhman, 1977).

Evaluating the expectancy model. The results presented by Davey (1992) are broadly consistent with his expectancy model of preparedness that was proposed in the same article and then developed in subsequent publications (e.g., Davey, 1995, 1997). According to this model, research participants enter the experiment with enhanced expectancies for aversive events to follow fear-relevant as compared with fear-irrelevant stimuli. If no aversive stimuli are presented, this expectancy bias dissipates with repeated exposure to fear-relevant stimuli. However, a single UCS presentation is sufficient to reintroduce the bias, perhaps, Davey has argued, because it reinstates a vivid representation of the UCS. Thus, the bias can be incremented or decremented as a function of reinforcement or nonreinforcement by aversive UCSs (contingent or noncontingent on a CS). These modulated expectancies, finally, are assumed to have a causal influence on SCRs.

Davey's (1992, 1995) strong thesis is that this model is able to account for most of the data generated in the context of human Pavlovian conditioning to fear-relevant stimuli without any need to invoke an evolutionary explanatory framework. The central thesis of this model is that cognition (i.e., expectancies about the UCS) play a major role in mediating SCRs (both assertions are obviously contrary to the position being articulated in this article). At the level of these strong assertions, however, there are several problems with Davey's model.

First, his expectancy model has difficulty explaining some of the effects observed in his own and others' experiments. For example, Davey argues that the frequently observed failures of expectancies to translate into SCRs (even in his own experiments) may be due to the participants having either a vague and ill-defined, or a

decayed, internal representation of the UCS. This post hoc argument, however, does not appear testable without an independent measure of the participants' internal representation of the UCS. In fact, a substantial part of the alleged explanatory power of the theory can be attributed to the vagueness of the UCS representation construct. For example, in several of the examples invoked by Davey (1997), it is unclear whether the revaluation concerns the UCS per se, the fear response (whether the CR or the unconditioned response), or even the CS. Thus, transforming the observable, manipulable UCS into an internal representation with unclear referents in the external world undermines one of the most important virtues of conditioning theory (i.e., its anchoring in observable events in the external world, observable behavior, or identifiable events within the body in interoceptive conditioning).

Second, and perhaps more important, Davey's expectancy model cannot explain other key findings in the literature that do support the preparedness model being presented here. In an important experiment, Schell et al. (1991) reported an experimental dissociation between expectancy and SCRs that is very difficult for expectancy theory to handle. They exposed research participants to pictures of snakes, spiders, flowers, and mushrooms and conditioned different groups either to fear-relevant (snakes or spiders as CS+ or CS−) or fear-irrelevant (flowers or mushrooms as CS+ or CS−) stimuli with either a short (0.5 s) or a long (8 s) CS–UCS interval. After this conditioning session, the participants were brought back to the laboratory either 1 or 6 months later. At this second session, the participants' memory of the first session was first tested by asking them to recall the events and to indicate which of the stimuli had been followed by shock. This was followed by a single reinforced CS+ presentation and nonreinforced presentations of the other stimuli before an extended extinction series was given. Larger differential SCRs in participants conditioned to fear-relevant than in those conditioned to fear-irrelevant stimuli were evident for all extinction trials in groups conditioned with both interstimulus intervals and for both the retention intervals even though participants conditioned to fear-relevant and fear-irrelevant stimuli reached expectancy extinction at about the same point in time. Moreover, when SCR differentiation after expectancy extinction was examined, participants conditioned to fear-relevant stimuli with the short CS–UCS interval continued to show reliably larger SCRs to the CS+ than to the CS− in the absence of any corresponding differential expectancies. Such a dissociation was not evident for participants conditioned to fear-irrelevant stimuli.

An additional set of findings that is not addressed by the expectancy model is the apparent differences in response patterns that are conditioned to fear-relevant and fear-irrelevant stimuli. As reviewed in a previous section, a heart rate acceleration associated with startle potentiation is conditioned to fear-relevant stimuli, whereas the conditioned heart rate response to fear-irrelevant stimuli is primarily a deceleration (E. W. Cook et al., 1986; Dimberg, 1987). Similarly, in the study by Dimberg (1987), an angry face induced conditioned enhancement of activity in the corrugator muscle of the face, suggesting that a negative emotion had been conditioned. No such effect was observed for a happy CS+. Furthermore, enhanced fear ratings were observed only if the CS+ was fear relevant.

Finally, Davey's (1995) underlying assumption that expectancies as cognitive phenomena provide an alternative to evolutionarily shaped mechanisms is questionable. To pit expectancy against evolutionary mechanisms involves a category error, because cognition, just like fear conditioning, is a process that is shaped and constrained by evolution. From the evolutionary perspective, cognition is just another type of mechanism guaranteeing regular and adaptive relationships between the organism and its environment. The rapid expansion of cortical tissue with hominid evolution offered new tools for natural selection to use for keeping individuals away from potentially deadly contexts. These new opportunities to fine-tune defenses in anticipation of danger, however, did not make the old defense systems inoperative. Cognitive mechanisms such as expectancy biases to fear-relevant stimuli should be viewed as supplementary to, rather than as replacing, the conditioning mechanisms that are at the focus of the present article. As shown in the previous section on the automaticity of fear, the assumption of expectancy theory that cognition always determines emotional responding is problematic. With fear, the reverse relationship appears to be more valid (Robinson, 1998). The emotional response comes first, and cognitive processes come in later, when the defense response is under way (see, e.g., LeDoux, 1996; Öhman, Flykt, & Lundqvist, 2000). Rather than playing a main role in emotional activation, as assumed in traditional cognitive theories of emotion (e.g., Lazarus, 1991), a more likely role of cognition in fear is to rationalize the emotion. From this perspective, the cognitive biases discussed by Davey (1992, 1995, 1997) do not determine but are more likely to be consequences of fear responding. When phobics overestimate the danger of the phobic stimulus (e.g., Taylor & Rachman, 1994; Telch, Valentine, & Bolte, 1994; Thorpe & Salkovskis, 1995), this is more likely to be an effect of the phobic fear (sometimes an attempt to justify it) rather than one of its determinants. Cognitive mechanisms help us understand a dangerous situation, and expectancy biases can be viewed as tools to prompt more early and general avoidance of potential threats than that occasioned by conditioned threat cues. For example, inferences of dangers could be made and avoidance actions taken on the basis of verbal warnings in the absence of explicit cues signaling danger.

Encapsulation of Fear: Concluding Discussion

Many types of expectancies are activated in experiments with fear-relevant stimuli, and they show interesting relations to many experimental parameters. However, expectancies per se remain insufficient to provide a convincing theoretical account of the range of results reported from such experiments. Instead, the frequent dissociations between autonomic responses and expectancies suggest that expectancies are more consequences than causes of fear responding, as would be expected if fear is controlled by an encapsulated module. Thus, we take the evidence discussed in this section, particularly when considered in conjunction with the research discussed in the previous section on automaticity, as strong evidence for the relative independence of cognition and activation of the fear module.

Neural Mechanisms of Fear Generation and Fear Learning

In previous sections of this article, we have reviewed large sets of results indicating that fear conditioning to fear-relevant CSs shows the characteristics of selectivity, automaticity, and encap-

sulation that one would expect for an evolved behavioral module of fear learning. An evolved module is likely to be mediated by specific neural circuitry. This circuit, furthermore, would be expected to be distinct from neural circuits controlling other types of emotion or associative learning, and it would be expected to have a location in the brain suggesting an ancient evolutionary heritage. In particular, this system would be expected to be located, not in the recently evolved neocortex, but rather in parts of the brain that are shared with much more primitive organisms than humans, such as structures in the limbic system (LeDoux, 1993b). Knowledge of a fear system in the brain that appears to meet these requirements has expanded rapidly during recent years. However, as we elaborate later, it should be explicitly acknowledged from the outset that this research has used what in humans would be called *neutral* or *fear-irrelevant* rather than *fear-relevant* stimuli (see Davis, 1992; Fanselow, 1994; LeDoux, 1996; Rosen & Schulkin, 1998; Whalen, 1998, for reviews of this literature). Nevertheless, the neural circuit that emerges from this research is helpful in understanding several aspects of the literature that we have reviewed.

Neural Circuitry of Fear: Organization of Fear Responding

The amygdala, a collection of neural nuclei in front of the hippocampus at the tip of the temporal lobe, appears to be a central neural structure for the control of fear. For example, the classical observation of pronounced fearlessness of monkeys with ablation of the temporal lobe (Klüver & Bucy, 1939) can be attributed to lesions of the amygdala (Weiskrantz, 1956). Furthermore, electrical stimulation of the amygdala produces fear-typical behavior (e.g., freezing and autonomic responses) in many animals (Applegate, Kapp, Underwood, & McNall, 1983), including humans (Gloor, 1992). Thus, any of the overt manifestations of fear are controlled from the amygdala, particularly from its central nucleus (Davis, 1992).

Davis and coworkers have used potentiation of the startle reflex for probing the fear network in the rat brain (see Davis, 1992, 1997, for reviews). Startle potentiation, measured as whole body startle, has been readily observed to conditioned fear stimuli in rats (e.g., Berg & Davis, 1984; Kurtz & Siegel, 1966; see Davis, 1992, for a review) and, measured as eyeblink amplitude, in humans (e.g., Hamm & Vaitl, 1996). Further, startle reflex amplitude in human participants is reliably enhanced when startle probes (acoustic stimuli with fast onsets) are presented against a background of negatively evaluated (Vrana, Spence, & Lang, 1988) or feared (Globisch et al., 1999; Hamm et al., 1997) visual stimuli. Thus, startle potentiation, measured as the difference in startle amplitude between stimuli presented in neutral and fear-inducing contexts, appears to provide a convenient index of conditioned fear and anticipatory anxiety in both rats and humans; furthermore, it provides a relatively direct reflection of activation of the amygdalar fear circuit, particularly the central nucleus of the amygdala (e.g., Davis, 1996; Lang et al., 1997).

Efferent fibers from the central nucleus project via the stria terminalis and the ventral amygdalofugal pathway to various regions in the diencephalon and brain stem that are involved in the expression of a wide range of fear-related behavior (LeDoux, 1987; Smith & DeVito, 1984). Thus, electrical stimulation of the central nucleus not only enhances startle amplitude but also produces the type of cardiovascular changes (i.e., elevated blood pressure) seen in fear episodes (Rosen & Davis, 1988). Such autonomic changes appear to be mediated by input from the ventral amygdalofugal pathway to the lateral hypothalamus, because conditioned cardiovascular changes (increase in heart rate and blood pressure, or fear bradycardia in rabbits) are blocked by lesions in this area (Kapp, Wilson, Pascoe, Supple, & Whalen, 1990; LeDoux, 1993a; LeDoux, Iwata, Cicchetti, & Reis, 1988; Smith, DeVito, & Astley, 1990). This pathway, furthermore, appears to be independent of the pathway controlling behavioral fear such as freezing, which is mediated by fibers from the amygdala to the ventral periaqueductal gray in the midbrain (Fanselow, 1994; LeDoux et al., 1988). The central nucleus also regulates the state of arousal of the neocortex, as revealed by electroencephalograph desynchronization (Kapp et al., 1992). This amygdalar–cortical link may be a primary route for the amygdala to influence attentional biases for fear stimuli (Mathews & MacLeod, 1994; Öhman, Flykt, & Esteves, in press; Öhman et al., 2001) as well as other cognitive processes (LeDoux, 1992).

Afference to the Amygdala

Sensory information reaches the amygdala from cortical regions subserving both uni- and polymodal sensory information processing (LeDoux, 1987). Unimodal, fully processed visual information is conveyed to the amygdala from the anterior inferotemporal cortex (area TE), primarily to the lateral nucleus of the amygdala, whereas unimodal auditory information may be conveyed from the rostral portion of the superior temporal gyrus (Amaral, Price, Pitkänen, & Carmichael, 1992). The perirhinal cortex may provide an important link in relaying sensory information to the amygdala because lesions here disrupt conditioned fear responses both to auditory and visual CSs (Davis & Lee, 1998).

However, the amygdala, particularly its lateral nucleus, also receives less processed input from parts of the thalamus whose nuclei serve as relay stations for peripheral sensory information destined to the primary sensory areas of the cortex. This is most clearly established for auditory stimuli via the medial geniculate body (Amaral et al., 1992; LeDoux, 1996), but a similar input to the amygdala may exist for thalamic nuclei involved in visual information processing, such as the pulvinar nucleus (LeDoux, 1987; Morris et al., 1999).

This subcortical thalamic input to the amygdala plays a crucial role in emotional activation and emotional learning (LeDoux, 1996). Using a fear conditioning paradigm with auditory CSs and footshock as the UCS and autonomic (increases in arterial blood pressure) as well as somatic (behavioral freezing) responses as fear indices, LeDoux and coworkers (see reviews by LeDoux, 1992, 1996) traced the neural circuitry that activated the amygdala by emotional stimuli. Their findings demonstrated that the auditory cortex was not a necessary way station for information reaching the amygdala to elicit the fear response. Thus, both autonomic and somatic indices controlled by the CS were unaffected by complete lesioning of the primary auditory cortex. By anatomical techniques, LeDoux and colleagues demonstrated that there are direct axonal connections from the medial geniculate body to the lateral nucleus of the amygdala (LeDoux, Farb, & Ruggiero, 1990; LeDoux, Ruggiero, & Reis, 1985; LeDoux, Sakaguchi, & Reis, 1984). Confirming the critical role of the amygdala, lesioning its

lateral nucleus led to loss of conditioned blood pressure increases and conditioned freezing responses (LeDoux, Chicchetti, Xagoraris, & Romanski, 1990). Thus, the lateral nucleus of the amygdala appears to serve as a critical sensory relay, in which affective characteristics of various input are analyzed and evaluated and subcortically mediated sensory information is integrated with input from the cortex (LeDoux, 1992, 1996). Indeed, "thalamic inputs to the amygdala allow sensory signals to activate it either before or simultaneous with the arrival of signals at the cortical level, and may therefore play an important role in preconscious and precognitive emotional processing" (LeDoux, 1992, p. 192).

Mechanisms of Learning in the Amygdala

More recent work from LeDoux's laboratory has investigated the cellular mechanism of fear learning in the amygdala and further confirms the central role of this structure for learned fear. For example, Rogan and LeDoux (1995) demonstrated that high-frequency stimulation of the medial geniculate body produced long-term potentiation (a marker of learning at the cellular level; see, e.g., Bliss & Collingridge, 1993; Malenka & Nicoll, 1999) of neurons in the lateral amygdala that transmit auditory information about the CS. In a subsequent study, Rogan, Stäubli, and LeDoux (1997) showed that electrical field potentials from this population of neurons were modified during the standard aversive auditory Pavlovian conditioning procedure used by LeDoux (e.g., 1996). Thus, as conditioned fear became apparent at the behavioral level, neural responses in these neurons increased, only to decrease in extinction as the behavioral response waned. These changes in electrical activity in neurons of the lateral nucleus were specifically related to the forming of associations between the CS and the UCS, because such changes were not observed in animals given unpaired presentations of the CS and the UCS. Thus, it appears that associative emotional learning can be tied to neuronal changes in the lateral nucleus of the amygdala.

In an updated review of fear conditioning and the amygdala, Fendt and Fanselow (1999; see also Fanselow & LeDoux, 1999) concluded that amygdalar lesions abolish conditioned fear as indexed by a range of fear indicators, that the amygdala receives convergent information from the CS and the UCS, that pharmacological blockade of neural plasticity in the amygdala abolishes fear learning, and that there is evoked neural activity in the amygdala that changes after Pavlovian conditioning. In their view, therefore, "the inescapable conclusion is that the amygdala is a crucial structure for the learning of fear" (Fendt & Fanselow, 1999, p. 749).

Conditioned Fear and the Amygdala of the Human Brain

Several studies have now confirmed the role of the amygdala in human fear conditioning. In one recent experiment, Morris, Öhman, and Dolan (1998) used PET to directly test the hypothesis that fear conditioning is centered on the amygdala in the human brain, using angry faces as CSs. In a conditioning session that immediately preceded PET scanning, participants were presented with pictures of four different male faces, two of which were angry and two neutral. One of the angry faces was paired with an aversive noise and thus served as a CS+. The second angry face served as a CS−. Booster conditioning trials were presented in between masked extinction trials with short intertrial intervals (5-s) that were presented during PET scans. There were two different masking conditions for extinction trials. One of them involved the CS+ and the CS− as target stimuli, each presented for 30 ms and immediately and consistently followed by one of the two neutral faces, presented for 45 ms as masks. In this condition, therefore, the CSs were unreportable. For the second masking condition, the order of the targets and the masks was reversed. The neutral faces were presented for 30 ms on extinction trials and were immediately followed by the CS+ or the CS− presented for 45 ms. Thus, in this condition, the CSs could be clearly seen by the participant. SCR data confirmed reliable differential responding to the CS+ and CS− in both the masked and the nonmasked extinction series during the PET scans. This design allowed a stringent test of the hypothesis that human fear conditioning resides in the amygdala. By contrasting images of regional cerebral blood flow produced by the masked CS+ (angry face masked by a neutral face) and the masked CS− (different angry face masked by a different neutral face), the difference would reveal locations in the brain of the fear response conditioned to the CS+ elicited by an unreportable fear-relevant stimulus. A similar contrast of the nonmasked CS+ and the CS− would reveal locations in the brain of a fear response elicited by a consciously elicited stimulus.

Confirming the hypothesis of amygdalar involvement, the overall contrasts between the CSs+ and the CSs− showed significant differential activations specifically in the amygdala regions. This effect, however, showed a strong and unanticipated interaction with brain laterality. For the masked CSs, only the right amygdala was activated (see Figure 6); for nonmasked CSs, reliable activation was observed only in the left amygdala. These effects were not perfect mirror images of each other. For reportable stimuli, the activation was somewhat superior and posterior to that seen with nonreportable stimuli, even though both resided within the amygdalar complex. In the present context, the important finding is that the human amygdala was specifically activated by conditioned fear stimuli, whereas the laterality effect of nonreportable versus reportable stimuli must be left for further research to elucidate. Because this experiment included only fear-relevant CSs, it does not allow firm conclusions about the importance of fear relevance for the obtained results. However, given that SCR conditioning survives backward masking only provided that the CSs are fear relevant (Esteves, Dimberg, & Öhman, 1994; Öhman & Soares, 1993; Parra et al., 1997; Soares & Öhman, 1993a, 1993b), similar effects would appear unlikely had a condition with fear-irrelevant CSs been included.

In a follow-up study on the same experiment, Morris, Öhman, and Dolan (1999) examined whether the nonconscious activation of the amygdala by nonreportable stimuli was occasioned through a subcortical route, as could be expected from LeDoux's (1996) model. Thus, they examined the neural connectivity between the amygdala and other brain regions when the amygdala was activated by masked stimuli. The specific hypothesis was based on the literature on blind sight (Weiskrantz, 1986, 1997). According to this literature, the remaining but nonconscious visual capacity sometimes seen in patients with damage to the primary visual cortices may be mediated by a parallel visual route through the superior colliculus and pulvinar nucleus of the thalamus, which is served by large, rapidly conducting neurons. Morris et al. (1999) argued that this route may be less susceptible to masking than the

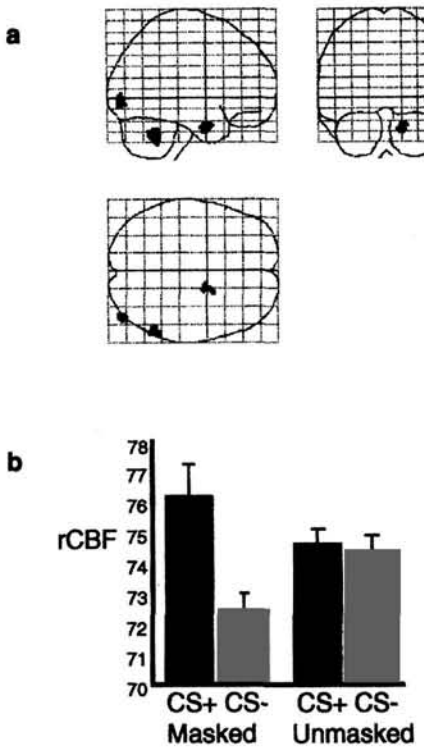

Figure 6. (a) A statistical parametric map showing the contrast between masked presentation of an angry face conditioned stimulus (CS) previously conditioned (CS+) by an aversive noise unconditioned stimulus and another angry face previously presented without any noise (CS−). The masks were neutral faces. Significant regions are displayed in orthogonal views of a transparent standardized brain image. The significantly activated region ($p < .01$; corrected for the volume of brain analyzed) in the medial temporal lobe lies within the boundary of the right amygdalar complex. In addition to the predicted effects for the amygdala, there were significant differential activation in the right cerebellum and the right inferior occipital gyrus. (b) Adjusted regional cerebral blood flow (rCBF) in ml/dl*min^{-1} at the maximally activated voxel in the right amygdala ($x = 18$, $y = -2$, and $z = -28$) during stimulation with the masked and unmasked CS+ and CS−. Bars represent 2 *SE*s. Note that the right amygdala differentiated only between the masked stimuli. The left amygdala ($x = -16$, $y = -8$, and $z = -14$), on the other hand, differentiated only between the unmasked stimuli ($p < .02$; not shown in figure). From "A Subcortical Pathway to the Right Amygdala Mediating 'Unseen' Fear," by J. S. Morris, A. Öhman, and R. J. Dolan, 1999, *Proceedings of the National Academy of Sciences, 96,* p. 1681. Copyright 1999 by The National Academy of Sciences. Adapted by permission.

classical visual pathway through the lateral geniculate. In support of this hypothesis, Morris et al. (1999) reported that activation of the (right) amygdala by masked stimuli could be reliably predicted from activation in the superior colliculus and the pulvinar, whereas such a relationship was not obvious when the (left) amygdala was activated by nonmasked stimuli. The research by Morris et al. (1998, 1999) is important in demonstrating that a similar system to that previously delineated in the rodent brain (see review by, e.g., LeDoux, 1996) for fear conditioning appears to be operating in the brains of humans.

In a neuropsychological study, LaBar, LeDoux, Spencer, and Phelps (1995) examined 22 patients who had been treated by unilateral removal of the amygdala as a means of controlling epileptic seizures. They reported impaired SCR conditioning in patients as compared with normal controls in both a simple auditory differential conditioning paradigm with aversive noise as the UCS and in a more complex conditional discrimination task in which an auditory CS signaled the UCS depending on simultaneously presented visual stimuli. However, the patients showed comparable UCR magnitudes to those of controls, and the overwhelming majority of them were able correctly to report the CS–UCS contingency, showing that conscious awareness of a CS–UCS contingency is not sufficient for SCR conditioning without an intact amygdala.

In an extension of this work, LaBar, Gatenby, Gore, LeDoux, and Phelps (1998) also studied changes in the response of the human amygdala by means of functional magnetic resonance imaging (fMRI) during differential Pavlovian conditioning in normal human participants. As predicted, they observed enhanced activation of the amygdala to the CS+ compared to the CS− when conditioning started. This activation, however, reached a peak and then declined, virtually to disappear by the end of acquisition training. When the UCS was omitted at the start of extinction, the amygdalar response to the CS+ reappeared, only later to disappear again. In fact, similar changes across fear conditioning and extinction trials were reported from cellular recording in the lateral amygdala (Quirk, Armony, & LeDoux, 1997). Büchel, Morris, Dolan, and Friston (1998) confirmed the report of LaBar et al. (1998) that the activation of the amygdala (measured by event-related fMRI) to CS+ observed early in conditioning training subsided as training proceeded. Rather than tracking the regular and gradual increment of a conditioned association, these changes are similar to what is typically observed when autonomic responses are followed during conditioning (see review by Öhman, 1983). Indeed, several studies have reported strong correlations between activation of the amygdala (measured by PET or fMRI) and SCR during aversive conditioning (Büchel et al., 1998; Fredrikson et al., 1998; Furmark, Fischer, Wik, Larsson, & Fredrikson, 1997). Much like the decrementing autonomic responses during conditioning, therefore, activation of at least the lateral amygdala may reflect the initial surge of processing of the CS–UCS contingency early in conditioning that subsides as a representation of the contingency is formed (see Öhman, 1979b, 1983; Whalen, 1998).

Recent human data (Büchel et al., 1998; LaBar et al., 1998), therefore, introduce complications in identifying the amygdala as the exclusive site of conditioned fear, because activation of this structure appears most pervasive early in training, later to subside. Partly this can be accounted for by realizing that the amygdala is not a homogenous structure but is composed of several distinct nuclei. Thus, when the role of the amygdala in fear is the topic of discussion, there is reason to be cautious in presuming a unified function for the whole structure. The significance-evaluating function of the amygdala may primarily pertain to its lateral nucleus (LeDoux, 1996), which also appears to be the site for the formation of associations during aversive conditioning (Rogan et al., 1998). The basolateral nucleus may assist in such processing (Fanselow & LeDoux, 1999; Kapp et al., 1992; LeDoux, 1993b), even though this nucleus may also be critical for the influence of the amygdala on explicit memory (Cahill & McGaugh, 1998). The central nucleus, finally, appears to be the efferent interface, because it

controls the various peripheral manifestations of fear (Davis, 1992) as well as the arousal dynamics within the brain during fear (Kapp et al., 1992). This detailed localization of different processes within the amygdala as determined by animal studies is too fine-grained to be captured by human-imaging methodology (Whalen, 1998), which therefore may reflect a combination or weighting of activity in the different nuclei. The take-home message of this complexity is that our knowledge of the details of the amygdalar fear circuit still is relatively rudimentary, even though important progress has been made during recent years.

The Specificity of the Circuit for Fear Conditioning: Concluding Discussion

It is an important part of the evolutionary argument that the neural circuit we have described is specifically dedicated to fear and fear learning. In general, there are several different circuits in the brain that are related to different forms of learning. With regard to Pavlovian conditioning, for example, there is one circuit that appears to be specific to the acquisition of conditioned skeletal responses, such as eyeblink or nictitating membrane responses. Animal data (Thompson, 1990, 1992) as well as human brain lesion (Daum et al., 1993) and imaging (Logan & Grafton, 1995) studies concur in giving the cerebellum a central position in this circuit (with no role for the amygdala), which sets it apart from the fear conditioning circuit.

Further support for the critical and selective involvement of the amygdala in aversive human conditioning can be recruited from studies of brain-damaged patients. In a systematic case study, Bechara et al. (1995) reported that a patient with specific bilateral lesions of the amygdala did not acquire conditioned SCRs in separate visual and auditory multiple-cue aversive conditioning paradigms, even though she showed normal SCRs to the aversive sound used as the UCS. However, she did acquire factual knowledge about the conditioning contingency in the sense that she could recall the nature of the CSs and their relationships to the UCS (see also LaBar et al., 1995). A patient with bilateral hippocampal damage, on the other hand, acquired differential conditioned SCRs but failed to provide evidence of learning the contingency at the cognitive level. Thus, these data suggest a double dissociation between emotional and cognitive learning, with impeded emotional but intact cognitive learning after amygdalar lesions and the opposite effects after hippocampal lesions. These human data support the idea that the amygdalar circuit is specifically dedicated to fear learning, whereas the hippocampus is important for conscious cognitive learning.

On balance, even though the results reviewed in this section must be regarded as less than conclusive, they still are consistent with the notion that there is an amygdalar circuit that is specifically dedicated to fear learning. Note, however, that the studies in both rats and humans examining the specificity issue have not used what in humans would be considered as fear-relevant CSs. Although Morris et al. (1998) used angry faces as CSs, they did not include a comparison conditioning between angry and happy faces that would have allowed conclusions about the effect of fear relevance of the CSs on conditioning in the amygdala. Thus, there is, in fact, little empirical basis for arguing that the amygdalar circuit is specifically dedicated to evolutionarily facilitated fear conditioning because the animal studies typically have used simple, evolutionarily nonprepared sensory stimuli such as sound or lights as CSs (although, as we argue later, there is reason to believe that they may operate like fear-relevant stimuli in the context of fear conditioning paradigms used in rats). Nevertheless, as we see shortly, the circuit provides good explanations for many of the phenomena observed in human conditioning studies using fear-relevant stimuli. To anticipate, we attempt to accommodate the amygdala-centered fear circuit with the fear module concept by speculating that evolutionarily fear-relevant stimuli have privileged access to the amygdalar fear circuit.

Different Levels of Learning in Human Conditioning

According to our review, the amygdala appears to be the crucial neural structure for the fear module. First, it plays a crucial role in activating fear in humans as well as in animals. Second, activating the amygdala appears not to require conscious perception of the stimulus. Third, nonconscious activation of the amygdala appears to be achieved through a neural route that does not involve the cortex. Fourth, there is reason to assume that the neural circuitry centered on the amygdala described here is specific to fear. Fifth, however, as we have seen, there are virtually no data on the amygdalar circuitry that address a central concern of the fear module concept: that fear conditioning is selectively facilitated to the kinds of evolutionarily fear-relevant stimuli discussed in this article.

Neural Circuitry and the Fear Module

Except for the last point (to which we return), the neural circuit we have described appears to do a good job in explaining the behavioral characteristics of the fear module. Because it can be rapidly activated from incompletely processed stimuli through the thalamus–amygdala link, the amygdalar fear circuit accounts for the automaticity of the module. Once the relevant thalamic (and midbrain) structures encounter critical stimulus features, information is conveyed to the amygdala that then starts to recruit the fear response. This provides the basic explanation for the surprising efficacy of masked, nonreportable fear-relevant stimuli to activate fear responses (see reviews by Öhman, 1996; Öhman, Flykt, & Lundqvist, 2000). Because the thalamus–amygdala link is critical for fear learning (LeDoux, 1996), it also explains why human autonomic responses can be conditioned to masked, nonreportable fear-relevant stimuli (Esteves, Parra et al., 1994; Öhman & Soares, 1998), just as emotional responses can be conditioned in animals even after lesions of the relevant sensory cortex (LeDoux et al., 1984).

We have also argued that the fear module is encapsulated in the sense that it is impenetrable, particularly to conscious cognitive control. Accordingly, fear activation and fear learning in the amygdala are readily dissociable from declarative acquisition of information via the hippocampus (Bechara et al., 1995), even though the amygdala may enhance hippocampal memorization of events (Cahill & McGaugh, 1998). Furthermore, LeDoux (1996) argued that the fear circuit has widespread effects on complex cognition, whereas the amygdalar circuit is relatively immune to conscious cognitive control. Accordingly, verbally induced changes in expectancies have little effect on autonomic fear responding for

fear-relevant stimuli (e.g., Hugdahl & Öhman, 1977; Soares & Öhman, 1993b).

However, while accounting for automaticity and encapsulation, it is less definite what can be said about the fear circuit regarding the selectivity of the activating input. LeDoux (1996) voiced a speculation:

> Perhaps neurons in the amygdala that process prepared stimuli have some prewired but normally impotent connections to other cells that control emotional responses. The trauma might only have to mildly massage these pathways rather than create from scratch novel synaptic assemblages between the input and output neurons of the amygdala. (p. 254)

This statement can be expressed more formally in terms of the computational model of fear conditioning described by Armony and LeDoux (2000; Armony, Servan-Schreiber, Cohen, & LeDoux, 1997). The architecture of this model involves modules mapped on the anatomical structures of fear learning delineated in LeDoux's (1996) work, and their interconnections represent the anatomical connectivity observed in the brain. Learning involves changes in the weights of excitatory connections according to a Hebbian learning rule. To interpret prepared connections in terms of this model, one would merely assume that some of the connections between units representing features of certain CS–UCS combinations for specific fear-relevant CSs and aversive UCSs rather than being given random weights in the beginning of simulation would start with positive weights, thus more rapidly gaining strength in the competition to control the next layer in the model. This proposition not only provides an interpretation of phylogenetic effects on fear conditioning but also implies that that similar effects may result from ontogenetic experience. Given extensive and consistent experience (literature, lore, media) with particular stimuli (e.g., guns) in aversive contexts, the weights connecting them to fear may have gained weight to an extent that makes them function like evolutionary prepared associations.

Two Levels of Learning in Human Conditioning

One way to reconcile the human work on preparedness with the animal-based work that established the amygdalar fear circuitry is to assume that there are two levels of learning in humans exposed to a Pavlovian conditioning contingency. Such a notion was pioneered by Gregory Razran and was included in his views on evolution and learning (Razran, 1971). He argued that higher levels of learning (e.g., cognitive and contingency learning) emerged later in both ontogeny and phylogeny relative to lower levels of learning (e.g., habituation, sensitization, and basic classical conditioning seen even in invertebrates). In the present context, the levels-of-learning idea would imply that humans independently learn a cognitive contingency as well as an emotional response when subjected to joint presentations of a CS and an aversive UCS (i.e., neither level mediates the other). The level of emotional learning reflects the outcome of operations of the fear module, but the cognitive level of learning more likely reflects the outcome of operations of circuits centered on the hippocampus (e.g., Bechara et al., 1995). Activating the fear module in humans would result in the type of conditioning that is typically observed in animals, because the conditions used in animal research, for example, more intense UCSs, are likely to result in more genuine emotional learning. In fact, one could argue that most animal fear learning, at least as studied in the laboratory, is prepared in the sense that it depends on the fear module. The plausibility of this idea is supported when one considers the laboratory paradigms used to study fear. Animal experiments typically involve exposure to pain in uncontrollable, often highly novel and unnatural situations, the real danger of which must put stringent requirements on the risk-assessment device of rats (Blanchard & Blanchard, 1988). Given these circumstances, all defense systems the animal has in its repertoire should be primed, and the amygdala should assist in transferring fear from the UCS to the CS eventually to support escape and avoidance. Simultaneously, the cognitive associative apparatus centered on the hippocampus is prompted to focus on picking up whatever informative relations there are between cues and consequences (Cahill & McGaugh, 1998).

By contrast, for participants in any typical human fear conditioning experiment, the situation is entirely different. They know that they are participating in kind of a make-believe situation that never is allowed to become dangerous. Typically they are asked to decide the level of aversiveness of the stimuli themselves and know they can terminate the experiment at any time. These procedural details required in human-conditioning experiments impart a sense of perceived control that is known, even in rats, to dramatically reduce fear conditioning (e.g., Mineka, Cook, & Miller, 1984). With arbitrary CS–UCS contingencies and the less aversive UCSs typically used in human research, conditioning would usually engage only the cognitive level. Thus, the participants would simply learn that the CS predicts the UCS without involvement of significant emotionality. The difference between conditioning at the cognitive level in humans from that seen in rats is that the human cognitive level is much more advanced, incorporating linguistically formulated narratives, beliefs, and a priori expectancies. On the other hand, what evolutionarily fear-relevant stimuli may do in this situation is to provide a shortcut to the fear module and thus assure that the basic level of emotional learning is accessed, even granted the low level of fear involved. It would also be expected that more direct and real traumatic conditioning events such as often occur in everyday life would also engage both the emotional and the cognitive conditioning levels.

Invoking a two-level account of human conditioning challenges the wide acceptance of the notion that human autonomic conditioning can be fully accounted for in terms of expectancies (Dawson, 1973; Dawson & Schell, 1985; Lovibond, 1993). However, it is not without precedent. Mandel and Bridger (1973), for example, argued that independent cognitive and emotional levels are activated during aversive human conditioning on the basis of their findings from a series of experiments (Bridger & Mandel, 1964, 1965; Mandel & Bridger, 1967).

Moreover, several lines of evidence that we have reviewed in this article, in fact, contribute to resurrect the levels-of-learning notion, particularly when fear-relevant stimuli are used (see Öhman, Hamm, & Hugdahl, 2000). One line of evidence concerns the findings that different types of responses appear to be conditioned to fear-relevant and fear-irrelevant stimuli. As we have already seen, E. W. Cook et al. (1986) reported that pairing fear-relevant stimuli (snakes and spiders) with aversive events (aversive noises or electric shocks) resulted in a reliable conditioned heart rate acceleration that peaked at about 3 s after CS onset, with the subsequent secondary deceleration hardly reaching

below the level achieved by the CS−. This was in marked contrast to conditioning to neutral stimuli, in which the secondary deceleration was clearly the dominant event indexing conditioning. Similarly, Dimberg (1987) found that angry faces paired with shock resulted not only in conditioned SCRs and increased fear ratings but also in an acceleratory heart rate response and in a conditioned increase in activity in the corrugator muscle controlling the frowning eyebrow.

In a second line of evidence, Hodes, Cook, and Lang (1985) used an individual-differences approach and examined the relationships between heart rate responses during preconditioning presentations of snakes and spiders (later to be used as the CS+ and the CS−) and differential heart rate responses during subsequent conditioning and extinction. Participants who tended to show acceleratory heart rate responses before conditioning also showed acceleratory differential conditioning to the CS+ and the CS− during acquisition. Furthermore, such participants also showed larger resistance to extinction of the conditioned SCR and rated the slides as more aversive after conditioning than before. Thus, according to changes in ratings of the emotional valence of the CSs from before to after conditioning, these participants appeared to have modified their emotional response to the CS+, as would be expected if their learning incorporated emotional conditioning. Participants showing conditioned heart rate decelerations, on the other hand, showed rapid SCR extinction and no conditioned changes in the valence of the stimuli. For them, therefore, the conditioning contingency appeared to result merely in expectancy changes.

In an additional line of evidence supporting the levels-of-learning hypothesis, Hamm and Vaitl (1996) reported reliable SCR conditioning (collapsed over pictorial CSs with affectively neutral, positive, and negative content) with both aversive and nonaversive UCSs (electrical shock and a reaction time task, respectively), with minimal differences in the CR as a function of the UCS condition. Furthermore, in agreement with previous findings (see reviews by Dawson & Schell, 1985; Öhman, 1983), only participants who correctly could identify which stimuli served as the CS+ showed reliable SCR conditioning regardless of which UCS was used. However, Hamm and Vaitl (1996) also assessed the eyeblink startle reflex to probe stimuli presented during the CSs and during the intertrial intervals. Their data showed that startle magnitudes to probes presented during the CS− did not differ from those presented during the intertrial interval but those to probes presented during the CS+ were clearly enhanced, provided that the UCS was an aversive event. With the nonaversive reaction time task serving as the UCS, no startle enhancement to the CS+ was observed. Furthermore, with startle potentiation as the dependent variable, there was no relationship between contingency awareness and conditioning with the aversive UCS. Rather, conditioning was as obvious in participants failing consciously to pick up the contingency as in those correctly reporting the contingency, showing that contingency awareness was not mediating the emotional level of fear conditioning. Moreover, analyses of covariations between the different measures of conditioning in Hamm and Vaitl's data showed an interesting pattern. Participants showing conditioned heart rate accelerations also showed conditioned startle potentiation, whereas those showing conditioned heart rate decelerations did not show any enhanced startle to probes presented during the CS+ (regardless of their contingency awareness). Thus, from these data, it appears that startle potentiation and heart rate acceleration index the conditioning of an emotional response, presumably with a neural origin in the amygdala. Conditioned SCRs and heart rate decelerations, on the other hand, appear at least partially to be related to consciously accessible expectancies of the UCS to follow the CS, with a minimal role for aversiveness and little modification of the perceived valence of the CS+ (Hodes et al., 1985).

A final line of evidence supporting the idea that human conditioning involves two levels of learning concerns the results on conditioning and extinction to masked stimuli that we reviewed in detail in previous sections of this article. Backward masking can be viewed as a way of short-circuiting the cognitive contingency level of learning. Nevertheless, as shown in several studies, not only can SCRs be elicited to masked fear-relevant CS+s (e.g., Esteves, Dimberg, & Öhman, 1994; Öhman & Soares, 1993), but they can also be conditioned to masked fear-relevant stimuli presented in a Pavlovian contingency with an aversive UCS (Esteves, Parra, et al., 1994; Öhman & Soares, 1998). Thus, emotional learning to fear-relevant stimuli apparently can occur even when the contingency learning level is short-circuited by backward masking. When there is evidence of contingency learning, as revealed by expectancy ratings (Katkin et al., in press; Öhman & Soares, 1998), it can most likely be interpreted as secondary to the perception of autonomic feedback from the CR to the masked contingency (Katkin et al., in press).

Thus, we believe that for humans, biologically fear-relevant stimuli in a mildly aversive context provide a sufficient condition to access the basic level of emotional conditioning. One would also expect that other conditions such as, for instance, using intense UCSs or having a generally fear-conducive conditioning situation, may also engage this level. For example, in humans, inducing a temporary suspension of breathing by injection of a muscle-relaxing drug produced a strong and lasting conditioned fear of arbitrary fear-irrelevant stimuli paired with the drug-induced state (Campbell, Sanderson, & Laverty, 1964). Similarly, strongly fear-associated stimuli, such as directed guns (Hugdahl & Johnsen, 1989), could promote involvement of the fear module when serving as CS for aversive stimuli. For arbitrary CSs in mildly aversive situations, however, only the cognitive contingency level would be activated.

Effect of the Fear Module on Cognition

According to the view developed in this article, the cognitive and the emotional levels are independent and dissociable (Bechara et al., 1995) but nevertheless influence each other. Rather than presuming that various cognitive factors determine fear responding (e.g., Davey, 1997), we propose that they are often effects of such responses. The human proclivity for retrospectively making sense of experiences, using the left-hemisphere interpretive system (Gazzaniga, 1985, 1995), may take the automatically activated fear response as a prompt to provide justification of the fear behavior, just like the verbal left hemisphere is pressed to explain actions and reactions activated by stimuli exclusively presented to the mute right hemisphere in split-brain patients (Gazzaniga, 1995; LeDoux, 1996). From this perspective, it is not surprising at all that phobics judge their phobic stimuli as more dangerous than do nonphobics (Menzies & Clarke, 1995a; Thorpe & Salkovskis,

1995), inflate the likelihood of aversive events to follow phobic stimuli (e.g., Diamond et al., 1995), or retrospectively overestimate the contingency between phobic stimuli and aversive events (e.g., Tomarken et al., 1989).

However, the proposition that the causal link goes from emotion to cognition should not be taken to imply that cognitions are unimportant in phobias. The neural node of the fear network, the amygdala, is reciprocally connected to areas of the frontal lobe that serve to regulate emotion (Davidson, Putnam, & Larson, 2000). In the context of phobia, cognitions may be viewed as evolutionarily shaped mechanisms to assure that fearful individuals keep avoiding threatening situations. Thus, cognitions are important in maintaining phobic behavior. To include cognitive phenomena (e.g., expectancies or beliefs) as targets for therapeutic intervention, therefore, is clearly warranted. However, for the eventual success of the treatment effort, we surmise that it is mandatory that phobics sooner or later confront their phobic stimuli to extinguish the automatic activation of the fear module.

Conclusion

We have described an evolved module for fear learning and fear elicitation, the characteristics of which can be summarized in a number of broad generalizations:

1. In human and nonhuman primates, the fear module is activated by specific fear-relevant stimuli, particularly those for which an evolutionary origin appears likely. It is preferentially activated in aversive contexts by stimuli related to recurring survival threats during the evolution of mammals, which animals easily learned as signals for danger. Thus, the fear-relevant stimuli readily enter selective associations with aversive events, as attested by extensive research both on humans and monkeys. Evolutionarily fear-relevant stimuli often become the object of human phobias and thus help explain the selectivity of phobias with regard to the objects or situations that evoke them.

2. In humans, the fear module is automatically activated by fear-relevant stimuli, with no need for conscious access of the stimulus before a response is elicited. This is shown by studies using backward-masking techniques to present fear-relevant stimuli outside of awareness for human research participants. Such studies demonstrate not only that fearful participants, and participants with experimentally conditioned CRs, show enhanced autonomic responding to masked fear-relevant stimuli but also that fear can be conditioned to evolutionarily fear-relevant stimuli even though presented masked from conscious recognition.

3. The fear module is encapsulated in the sense that it is impenetrable to conscious cognitive control. If activated by an effective fear stimulus, the resulting fear runs its course and is very hard to control by cognitive means. For example, once the fear of a snake phobic is activated, it cannot be aborted by the realization that the snake in fact is innocuous.

4. The fear module reflects the operation of dedicated neural circuitry for fear evocation and fear conditioning centered in the amygdala. The amygdala receives more or less completely processed input from many areas of the brain, including the thalamus and the cortex, and it controls emotional output via hypothalamic and brain stem nuclei. The characteristics of the neural circuit are well mapped to the characteristics of the fear module.

5. The difference between aversive conditioning to fear-irrelevant and evolutionarily fear-relevant stimuli in humans is that typically only the latter access the fear module. It is assumed that evolutionarily fear-relevant stimuli are sufficient to activate the fear module if the situation is at least mildly aversive. Thus, with fear-relevant CSs+, the resulting CRs typically show the characteristics of the fear module (selectivity, automaticity, and encapsulation), whereas with fear-irrelevant CSs+, the resulting CRs are more closely tied to participants' expectancies about the situation. However, under certain conditions (such as those usually used to study fear conditioning with rats, i.e., a generally aversive and potentially threatening situation or relatively high-intensity UCSs), the fear module may also be activated by fear-irrelevant stimuli.

Taken at face value, these generalizations justify the conclusion that the fear module represents an evolved adaptation, particularly if its obvious functionality and conservation across phyla are considered. As such, it helps to integrate diverse findings on fear from many domains, such as animal learning, human conditioning, and covariation judgments, as well as clinical finding on fears and phobias. Nevertheless, it should also be remembered that there are many uncertainties in the databases that are relevant for the propositions in the module. In particular, the uncertainty is considerable if each data set is judged by itself. It is only when the converging impact of several lines of research is considered that the support appears strong. For example, it is the converging evidence from monkey vicarious conditioning, human conditioning, and human illusory correlation studies that justifies the conclusion that the fear module is relatively selective in the input that it accepts. If this conclusion is combined with data on automaticity and encapsulation, the confidence in the fear module concept is further strengthened. However, there are several places in which one would want more, and more conclusive, data. In particular, studies using brain imaging technology to assess changes in regional cerebral blood flow during conditioning to fear-relevant and fear-irrelevant stimuli are necessary to tie the fear circuit in the brain more definitely to the fear module concept. Thus, it is clear that the fear module concept not only integrates large domains of results but also sets an agenda for research on fear that eventually may help to connect phenomena of fear to articulated bodies of knowledge both in psychology and neuroscience. This research certainly will illuminate mechanisms of emotion that go beyond fear itself, but it may also improve our understanding of the clinical aspects of fear and anxiety that plague people.

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