

## UNEARTHING THE NONASSOCIATIVE ORIGINS OF FEARS AND PHOBIAS: A REJOINDER

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**Summary** — In recent years numerous disagreements and controversies have ensued over the place of Pavlovian or associative conditioning in the etiology of specific phobias and other fear-related clinical syndromes. A major source of disagreement emerged from clinical observations suggesting that environmental aversive conditioning events could not be identified for many specific phobias. Part of the controversy can also be traced to disagreements over what constitutes a direct conditioning event and over what exactly is being conditioned in phobic acquisition. More fundamental, however, is confusion over the critical process variables involved in the conditioning etiology of human phobias and fear-related clinical syndromes. We address some of the recent controversies surrounding associative conditioning accounts of phobic onset in light of recent proposals that nonassociative factors account for the etiology of many specific phobias. The viability of the nonassociative position is questioned and alternatives are suggested that emphasize the complex and multifaceted processes involved in the etiology of specific phobias. © 1997 Elsevier Science Ltd. All rights reserved

Early behavior therapy approached the problem of phobic fear onset as one of simple Pavlovian or respondent associative conditioning. It was not long, however, before evidence mounted questioning the adequacy of respondent conditioning theory as a comprehensive account of phobic onset. A major source of dissatisfaction with associative conditioning accounts of phobia emerged from clinical observations suggesting that environmental aversive conditioning events could not be identified for a number of specific phobias (e.g., Menzies & Clarke, 1995; Rachman, 1977). According to these views no identifiable conditioning event meant no associative conditioning (Menzies, 1996). Part of the dissatisfaction can also be traced to disagreements over what constitutes a direct conditioning event and over what exactly is being conditioned in phobic acquisition. More fundamental, however, is confusion over the critical process variables involved in the conditioning etiology of human phobias and fear-related clinical syndromes. In this paper, we discuss some of the recent controversies surrounding associative conditioning accounts of phobic fear onset in light of recent proposals that nonassociative factors account for the etiology of many specific phobias.

### The Nonassociative Etiology of Specific Phobias

Borrowing from Darwinian evolutionary theory, and more recent work in modern ethology, Menzies and Clarke (1995) proposed a nonassociative model to explain the etiology of many

specific phobias. According to this view, “no direct or indirect traumatic pairing with the feared stimulus is required, and neither is negative information” to explain phobic onset (Menzies & Clarke, 1995, p. 38). Rather, they suggest that evolutionary pressures have endowed certain stimulus configurations (e.g., fear of conspecific threat in primates, separation anxiety, fear of heights, visual looming, odors, and novelty) with fear evoking functions across a variety of species including humans. The view that certain stimulus configurations may be evolutionarily predisposed to evoke fear responses is, in part, consistent with Seligman’s (1971) reformulation of associative conditioning to include the biological concept of preparedness.

According to Seligman (1971), ontogenic and phylogenetic selection helped establish a tendency to respond fearfully to stimuli that were threatening to the survival of the species over the long course of evolution. The point at which nonassociative models and preparedness differ is in explaining the etiology of specific phobias, not the tendency to respond fearfully to some classes of stimuli and not others. Preparedness includes associative learning at some point in the evolutionary history of the species and contemporarily, in the more recent ontogenic learning history of the organism. Thus, preparedness does not obviate associative learning. The preclusion of associative learning in the etiology of phobic disorders is what makes the nonassociative position unique. However, this omission may also constitute a critical shortcoming.

#### Fears and Phobias: A Dimensional View

Some of the controversy regarding etiological models of phobia arises from discordant assumptions about the basic taxonomy of fear and its manifestations. For instance, the terms fear and phobia are often used interchangeably in the anxiety literature. However, it has been argued that their distinction can be conceptually important. Fears are generally regarded as an adaptive biopsychological response to real threat or danger (Barlow, Chorpita & Turovsky, 1996; Mineka & Zinbarg, 1996). By contrast, phobias represent similar psychobiological responses in the absence of real threat or danger. Phobias subsume the extreme manifestation of a fear response to some object or event, but not all fear responses are phobias. Rather, fears and phobias are best understood as separate regions on a continuum from adaptive to nonadaptive, respectively. Understanding the dimensional nature of fear and phobia is important, especially for those interested in the etiology of specific phobias and other fear-related clinical disorders. At what point does an adaptive fear response become a nonadaptive phobic disorder? In attempting to articulate etiological mechanisms, accounts of associative learning processes (e.g., language-symbolic learning, vicarious and direct conditioning) explain how one moves from an adaptive fear response to a nonadaptive phobic disorder. By contrast, the nonassociative account has a more difficult time explaining how one moves from a predisposition to respond fearfully to certain stimuli, to the clinical manifestation of a specific phobia.

For instance, separation anxiety and fear of heights early in development are used as support for the nonassociative position (e.g., Menzies & Clarke, 1995). Both examples, however, are quite common developmentally, yet, the vast majority of the population does not go on to develop phobic disorders of heights, people, or of separation later in adulthood. These and other examples used to bolster the nonassociative argument largely ignore the complex, subtle, and often cumulative learning experiences that shape and influence subsequent behavior across time and as a function of different contexts (see Mineka & Zinbarg, 1996). Claims that phobic

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