

# The etiology of specific fears and phobias in children: A critique of the non-associative account

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## The etiology of specific fears and phobias in children: a critique of the non-associative account

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### Abstract

The non-associative account of phobic etiology assumes that a number of specific fears (e.g., fear of heights, water, spiders, strangers, and separation) have an evolutionary background and may occur in the absence of learning experiences (e.g., conditioning). By this view, these specific fears pertain to stimuli that once posed a challenge to the survival of our prehistoric ancestors. Accordingly, they would emerge spontaneously during the course of normal development and only in a minority of individuals, these specific fears would persist into adulthood. While the non-associative approach has generated interesting findings, several critical points can be raised. First, it capitalizes on negative findings, i.e., the failure to document learning experiences (e.g., conditioning, modeling) in the history of phobic children. Second, it largely ignores factors that have been found to be crucial for the acquisition of early childhood fears (e.g., the developmental level of the child, stimulus characteristics such as novelty, aversiveness, and unpredictability, and early experience with uncontrollable events). As an alternative to the non-associative account, we briefly describe a multifactorial model of childhood fears and phobias. © 2002 Elsevier Science Ltd. All rights reserved.

*Keywords:* Fears and phobias; Children; Etiology, Non-associative account

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### 1. Introduction

Specific fears are common in childhood (see e.g., King, Hamilton, & Ollendick, 1988), yet most of them are short-lived and dissipate within months (Bauer, 1976; Ferrari, 1986). However,

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in some children, specific fears persist and become invalidating in the sense that they interfere with normal functioning. In these cases, a diagnosis of specific phobia should be considered (see Diagnostic and Statistical Manual of Mental Disorders, fourth edition [DSM-IV]; American Psychiatric Association [APA], 1994).

While researchers have reached a considerable degree of consensus about the assessment and treatment of childhood specific phobias (King, Ollendick, & Murphy, 1997; Ollendick & King, 1998), their etiology remains a matter of some debate. Some researchers maintain that fears and phobias predominantly arise as a consequence of learning experiences. Others assume that these phenomena reflect innate, spontaneous reactions to evolutionary prepotent cues (see for a review, Merckelbach & De Jong, 1996). The non-associative account of phobic etiology is a typical exponent of the latter position. It assumes that fear of heights, water, spiders, strangers, separation, and so on represent evolutionary-relevant fears that occur without critical learning experiences involving these feared objects (Menzies & Clarke, 1995; Poulton & Menzies, 2002).

To be sure, the non-associative view has generated a vast amount of interesting findings that will inform the research domain of anxiety disorders. Nevertheless, some critical points can be raised. In the first section of our commentary, we will critically discuss the evidence for the non-associative account. We will argue that there are a number of alternative explanations for the emergence of childhood fears that deserve scientific evaluation before assuming that these fears are innate. In the second section, we will describe a model of childhood fears which integrates empirical material that has accumulated over the past years. In our opinion, this model provides a more useful starting point for further research than does the non-associative approach.

## **2. Commentary on the non-associative account**

### *2.1. The three pathways to fear and the non-associative account*

Rachman's (1977) influential three-pathways theory suggests that three types of discrete learning experiences play a role in the acquisition of childhood fears and phobias: (1) aversive classical conditioning, (2) modeling, and (3) negative information transmission. Retrospective research relying on child and parent report found empirical evidence suggesting that these learning experiences are, indeed, involved in the etiology of childhood fears and phobias (see for reviews, King, Gullone, & Ollendick, 1998; Merckelbach, De Jong, Muris, & Van den Hout, 1996; Muris & Merckelbach, 2001). Contrary to Rachman's theory, however, there are a number of fears, such as fear of water (King et al., 1998), for which no conditioning, modeling, or information pathways can be found in a substantial proportion of children. Apparently, these fears occur without evident learning experiences, an observation that serves as the starting point for the non-associative account.

The main premise of the non-associative account is that these fears are evolutionary-relevant. According to Menzies and Clarke (1995), they pertain to stimuli that once posed a challenge to the survival of our prehistoric ancestors. Accordingly, fear responses would occur spontaneously during the course of normal development.

In our view, the non-associative account of childhood fears suffers from several shortcomings. To begin with, its advocates (e.g., Poulton & Menzies, 2002) claim that some fears (e.g., fear of

water) consist of evolutionary-relevant and, therefore, non-associative responses, while other fears (e.g., driving fears) are evolutionary-neutral responses that require associative learning. But how do they know? One could easily formulate a plausible evolutionary scenario for, say, driving fears. Consider, as another example, fear of insects. At first sight, this fear appears to be a prototypical case of an evolutionary-relevant fear. Meanwhile, historians remind us of the fact that it was only after the discovery around 1900 of the insect role in disease transmission that people's attitude towards insects changed dramatically. In the words of Riley (1986; p. 844): "These discoveries, communicated to health authorities and concerned lay people often in lurid cries about the dangers of arthropod vectors, evidently provoked a sudden change in attitudes toward certain insects, especially the common fly. Whereas people had previously shown an attitude of friendly tolerance to insects, specialists suddenly advised treating them as dangerous pests". The fear inducing power of negative information is also nicely illustrated by the recent work of Field and colleagues (Field, Argyris, & Knowles, in press). These authors showed that providing children with negative information about fictitious animals is enough to elicit fear beliefs about these creatures. Findings such as these are largely ignored by the non-associative account.

A second point concerns the genetic implications of the non-associative view. The assumption that a significant number of important specific phobias reflect innate response tendencies implies, almost by definition, that there should be a substantial genetic contribution to the category of specific phobias. However, that is not what behavioural-genetic studies have generally found. For example, Kendler, Neale, Kessler, Heath, and Eaves (1992) concluded from their twin data that relative to other anxiety disorders, specific phobias have the lowest heritabilities, but the highest specific environmental influences. This led the authors to conclude "that in the simple phobias, pathogenic environmental experiences are usually highly specific (e.g., being locked in a dark closet, bitten by a snake, nearly falling out of a window)" (Kendler et al., 1992; p. 280). This conclusion is, of course, difficult to reconcile with the non-associative view.

A third point has to do with the way in which advocates of the non-associative view treat the data that were obtained with the various versions of the Phobic Origin Questionnaire (POQ). The POQ intends to gather information about the learning history of phobic patients. Poulton and Menzies (this issue) are quite right when they point out that retrospective self-report instruments like the POQ are subject to all kinds of limitations. Yet, these limitations do not justify Poulton and Menzies' cavalier-like ignorance of all POQ studies. For one thing, by focussing on children's onset experiences and their verification through parental reports, the methodology of some more recent POQ studies has been vastly improved. These studies show that a significant number of spider and dog phobic children report associative onset experiences that are confirmed by their parents (e.g., Kheriaty, Kleinknecht, & Hyman, 1999; Merckelbach, Muris, & Schouten, 1996; Merckelbach & Muris, 1997; Muris, Merckelbach, & Collaris, 1997). Together with laboratory research on learning processes involved in fear (e.g., Malloy & Lewis, 1988; Forsyth and Eifert, 1996), these findings suggest that Rachman's (1991) three-pathways-to-fear model still is a good starting point for understanding the etiology of phobias. Again, this conclusion does not accord with the non-associative view.

Our fourth and final point concerns the empirical basis of the non-associative view. In our opinion, its empirical basis is meagre because it largely consists of the observation that Rachman's three pathways to fear do *not* occur in the history of some fearful or phobic children. Here, the non-associative view runs the risk of becoming tautological: if you find that phobic subjects fail

to report associative onset experiences you may call their fear evolutionary-relevant. At the same time, evolutionary relevance is derived from the fact that subjects cannot recall onset experiences.

## *2.2. Development and predictability as determinants of early fears*

As to the development of early childhood fears, it is a well-documented fact that they only appear after children have reached a certain maturational stage (see Marks, 1987). For example, experimental research employing the so-called visual cliff procedure has shown that fear of heights critically depends on children's locomotor development (Bertenthal, Campos, & Barrett, 1984). Similarly, fear of separation only occurs after children have developed object permanence for faces (Kagan, Kearsley, & Zelazo, 1975). When trying to understand why these developmental changes are associated with fear, one has to keep in mind young children's situation. They are constantly exposed to new stimuli and situations, yet they have little experience, physical strength, and coordination.

The question is why most new stimuli only elicit a brief orientation reaction, whereas some (e.g., heights, separation) provoke fear. Gray's (1982; see Gray & McNaughton, 1996) theory is relevant in this respect. According to Gray, fear can best be viewed as the output of a subcortical circuit dubbed the Behavioural Inhibition System (BIS). In order to predict events, the BIS constantly compares new information from the outside world to what is already stored in memory. As soon as the individual is confronted with aversive, novel, and/or unpredictable stimuli, the BIS is activated and fear arises. It is assumed that stimuli that are characterized by higher levels of aversiveness, novelty, and/or unpredictability will elicit greater BIS activity, and hence higher levels of fear. There are also good reasons to believe that there are temperamental differences between people in BIS functioning.

## *2.3. The role of control in the early environment*

While it is plausible that Rachman's three pathways contribute to fear acquisition in children of all ages (King et al., 1998), other types of learning experiences may also be relevant. Germane to this issue is the work by Chorpita and Barlow (1998). These authors suggest that early experiences with uncontrollable events may be thought of as a primary pathway to the development of fear and anxiety in that such experiences may foster an increased likelihood to process events as not within one's control (i.e., a psychological vulnerability). Children reared with an increased sense of control have relatively greater access to information that predicts the possibility of avoiding negative consequences. Conversely, children experiencing diminished control over events during development have predominantly stored information predicting that nothing can be done to prevent negative outcome.

## *2.4. Conclusion*

The non-associative account capitalizes on the observation that learning experiences such as conditioning, modeling, and/or negative information do not occur in the history of some fearful or phobic children. In the final analysis, this account lacks subtlety in that it ignores a number of factors that seem to be crucial for the acquisition of early childhood fears, viz. developmental

transitions of children, stimulus characteristics such as novelty, aversiveness, and unpredictability, and early experiences with uncontrollable events.

### **3. An alternative framework: the multifactorial model**

We recently described a framework that is better able to structure current knowledge on the etiology of childhood specific phobias than the non-associative account (Muris & Merckelbach, 2001). Briefly, our multifactorial framework rests on the following observations: (1) The majority of children display normal developmental fears that decrease with the passage of time; (2) A minority of the children have a genetic vulnerability factor that predisposes them to develop maladaptive fears; (3) This genetic vulnerability manifests itself in certain behavioural patterns; (4) Environmental factors interact with normal developmental fears and genetically linked behavioural patterns to produce extremely persistent fears that culminate in specific phobias; and (5) Once a specific phobia exists, it is maintained by cognitive biases. Below, we will briefly address each of these points.

#### *3.1. Normal developmental fears*

Research has shown that mild fears are fairly common among children (see for a review, Craske, 1997) and follow a predictable course: in infancy, children become fearful of stimuli in their immediate environment, but as the child matures these fears begin to incorporate anticipatory events and stimuli of an imaginary or abstract nature (Gullone, 2000). It is assumed that this developmental pattern of fears reflects everyday experiences and is to an important extent mediated by children's cognitive capacities (Marks, 1987). Thus, these fears should be viewed as normal phenomena.

#### *3.2. Genetics*

Behavioural-genetic studies indicate that genetic transmission contributes to the etiology of specific fears and phobias. Stevenson, Batten, and Cherner (1992) compared the frequency of self-reported fears in monozygotic and dizygotic twin pairs with ages ranging between 8 and 16 years. The authors found that a twin's level of fearfulness could be predicted from the co-twin's score. Furthermore, the frequency of fear was more similar in monozygotic than in dizygotic twin pairs. Finally, whereas heritability was significant for general fearfulness, examination of specific fear factors revealed that only some fears had a significant heritability (e.g., fears of small animals).

In a recent review of behavioural-genetic studies, Taylor (1998) distinguished two types of genetic influences that may contribute to phobic etiology: a general genetic factor would act as a vulnerability factor to a wide range of phobic fears, whereas specific genetic factors would only predispose to certain types of fears.

### 3.2.1. *Behavioural inhibition.*

Taylor (1998) points out that the general genetic component may constitute the biological substrate of what is normally referred to as trait anxiety, neuroticism, or negative affectivity. One obvious behavioural approximation of this general trait is behavioural inhibition. Behavioural inhibition refers to the tendency of some children to interrupt ongoing behaviour and to react with distress and withdrawal when confronted with unfamiliar people or situations. Behavioural inhibition is thought to be a stable and inherited response disposition that characterizes approximately 10 to 15% of the children (e.g., Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984). Cross-sectional and longitudinal studies conducted by Biederman and colleagues (1990, 1993) strongly suggest that this disposition serves as a risk factor for anxiety disorders. These studies showed that preschool children identified as behavioural inhibited are more likely to have anxiety disorders including specific phobias compared to control children. This became even more prominent at a 3-year follow-up: in the cohort of children who were initially identified as behaviourally inhibited, the rates of specific phobias and other anxiety disorders had increased markedly. Thus, behavioural inhibition seems to be a vulnerability factor for a broad range of anxiety disorders, among which specific phobias (see for a review, Biederman, Rosenbaum, Chaloff, & Kagan, 1995).

### 3.2.2. *Disgust sensitivity.*

Disgust sensitivity is considered to be a personality trait that can be reliably measured with self-report scales that contains items such as “It bothers me to see someone in a restaurant eating messy food with his fingers” (Haidt, McCauley, & Rozin, 1994). There is now good evidence to suggest that disgust sensitivity is involved in the etiology of specific fears and phobias (see Muris, Merckelbach, Schmidt, & Tierney, 1999), in particular certain types of animal phobias (De Jong & Muris, *in press*). For example, De Jong, Andrea, and Muris (1997) assessed fear of spiders, disgust sensitivity, and spider’s disgust-evoking status in spider phobic children who applied for treatment, in non-phobic children, and in the parents of both groups of children. Phobic children were tested before and after behavioural treatment. The findings demonstrated that disgust is an important feature of spider phobia. Compared to control children, spider phobic children exhibited higher levels of disgust sensitivity and considered spiders *per se* as more disgusting. Furthermore, after treatment, reduction in spider fear was paralleled by a steep decline in spiders’ disgust-evoking status. Finally, mothers of spider phobic children attributed a high disgust-evoking status to spiders. Although modeling experiences may also be involved, one could interpret the latter finding as support for a genetic factor that is involved in the familial transmission of disgust sensitivity and, in its wake, animal phobia. Thus, following Taylor’s (1998) taxonomy, disgust sensitivity can be conceptualized as a specific genetic factor.

### 3.3. *Environmental influences*

Twin studies indicate that genetic factors play a significant, but modest role in the etiology of specific phobias (e.g., Kendler et al., 1992). Apparently, then, environmental factors determine whether genetically transmitted vulnerabilities culminate in specific phobias. Apart from discrete learning experiences (e.g., conditioning, modeling, negative information, and experiences of uncontrollability), a number of general environmental factors contribute to specific childhood phobias. Negative life events and parental rearing styles constitute such general factors.

Several studies have found an increased incidence of negative life events such as parental divorce or death of significant family members in clinically anxious children (e.g., Kashani et al., 1990). However, it is unlikely that these life events per se are responsible for the emergence of specific phobias or other anxiety disorders. Spence and Dadds (1996) argue that the negative impact of aversive life events critically depends on factors that exacerbate (e.g., behavioural inhibition) or buffer (e.g., social support or effective coping style) their effects.

Parental rearing styles may also promote the development of high fear and anxiety levels. Evidence for this comes from studies that relied on direct observation of current parent-child interactions. Following such an approach, Dadds, Barrett, and Rapee (1996) noted that parents of anxious children often encourage their children to adopt avoidant coping strategies. Similarly, a study by Muris, Steerneman, Merckelbach, and Meesters (1996) demonstrated that specific fears reported by children are a function of the extent to which their mothers express their own fears in the presence of the children. Furthermore, studies employing questionnaires that measure children's perceptions of parental rearing behaviours suggest that anxious rearing, parental control, and rejection all account for a small, but nontrivial proportion of children's fear and anxiety symptoms (Muris & Merckelbach, 1998; Grüner, Muris, & Merckelbach, 1999).

### *3.4. Cognitive biases*

The influential two-stage model of Mowrer (1960) suggests that avoidance behaviour is responsible for the maintenance of phobic fear. More precisely, avoidance would minimize direct and prolonged contact with the fear-provoking stimulus, and, hence, the phobic child would not have the opportunity to learn that it is in fact harmless. While the role of avoidance behaviour in the maintenance of phobias seems self-evident, there are also a number of cognitive biases that promote continuation of phobic fear.

#### *3.4.1. Attentional bias.*

A large number of studies have documented that phobics display hyperattention toward potentially threatening material. A frequently employed technique for demonstrating this so-called attentional bias is the emotional Stroop task. In this task, subjects are required to name the colour in which words are printed while ignoring the meaning of these words. A consistent finding in Stroop studies with, for example, spider phobics is that their colour naming of threatening words (e.g., web) is slower than that of neutral words (e.g., car). This would be due to the fact that phobics automatically direct their attention to the content of the threatening words, which in turn interferes with their main task, i.e., colour naming (Watts, McKenna, Sharrock, & Trezise, 1986). There is some evidence that attentional bias also occurs in fearful children. For example, in their Stroop experiment, Martin, Horder, and Jones (1992) found that spider fearful children exhibit retarded colour naming latencies when confronted with phobic words. Similarly, using a dot-probe paradigm, Vasey and colleagues (Vasey, Daleiden, Williams, & Brown, 1995; Vasey, El-Hag, & Daleiden, 1996) showed that clinically anxious children and children high in test anxiety are faster to react to a probe if it is preceded by a threatening rather than a neutral word. This differential reaction was not evident for the control children in the Vasey et al. studies and, therefore, is suggestive of heightened selective attention towards threatening stimuli.

### 3.4.2. Covariation bias.

The experimental demonstration of covariation bias in phobias is straightforward. Phobic and normal subjects are shown a series of slides consisting fear-relevant (e.g., spiders) and neutral (e.g., flowers) pictures. Slide offset is followed by one of three outcomes, namely an aversive shock, a tone, or nothing. Fear-relevant and neutral pictures are equally often followed by each of the outcomes. After the series of slides, subjects are asked to estimate the contingencies between slides and outcomes (e.g., “Given that you saw a spider picture, on what percentage of those trials was the spider followed by a shock?”). Under these experimental conditions, phobic subjects systematically overestimate the contingency between phobic stimuli and aversive outcomes (Tomarken, Sutton, & Mineka, 1995). Interestingly, De Jong, Van den Hout, and Merckelbach (1995) found that residual covariation bias in treated spider phobics is predictive of relapse. That is, the stronger the post-treatment overestimation of the contingency between spider pictures and aversive shock, the higher spider fear levels at 2-years follow-up. Thus, phobics have a tendency to attribute aversive experiences to the phobic object and this, in turn, will sustain their phobic fear. It is plausible to assume that covariation biases also occur in fearful and phobic children (Daleiden & Vasey, 1997).

### 3.5. Conclusion

Over the past decade, our knowledge of factors that are involved in the etiology of childhood specific phobias has increased considerably. The framework described above attempts to structure this knowledge and integrates evidence from various sources. It emphasizes a multifaceted etiology of childhood specific phobias and is based on the assumption that there is a continuity between normal developmental fears and childhood specific phobias. However, whereas childhood fears are common but transitory phenomena in most children, there is a small subgroup of children in whom these fears tend to radicalize due to a genetic vulnerability. This genetic vulnerability may manifest itself in certain behavioural patterns (e.g., behavioural inhibition and disgust sensitivity). Learning experiences (conditioning, modeling, negative information, and experiences of uncontrollability) interact with normal developmental fears and genetically based behavioural patterns to produce extremely persistent fears that ultimately take the form of a specific phobia (or another anxiety disorder; see Craske, 1997). Once a specific phobia exists, it is maintained by cognitive mechanisms such as attentional bias and covariation bias.

## 4. Discussion

Our model differs radically from the non-associative account of phobic etiology. The basic assumption of this account seems to be that discrete learning experiences play a marginal role in the etiology of certain types of specific phobias. According to the non-associative account, these phobias reflect innate and spontaneous reactions to evolutionary prepotent cues. From a scientific point of view, such an explanation is not very satisfactory. After all, science is about causal associations and so, one would like to know where “spontaneous” fears and phobias originate from (see also Forsyth & Chorpita, 1997).

In our opinion, etiological models of specific phobias cannot do without the explanatory power

of learning experiences and concepts like disgust sensitivity and behavioural inhibition. We do not claim that our model has the merits of a scientific model in the strict sense of the word. Most importantly, the precise dynamics between the various factors that figure in the model are far from clear. For example, do learning experiences only contribute to a radicalization of developmental fears during a critical period (e.g., when a certain developmental fear is at its maximum)? Or is it the case that learning experiences in combination with genetically based behavioural patterns reinstate developmental fears that disappeared during a previous phase? These issues require longitudinal prospective studies that include various parameters. However, compared to the non-associative view, our model better approaches a consensus among researchers about the complexity of the etiological antecedents involved in specific phobias.

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