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Non-associative fear acquisition: a review of the evidence from retrospective and longitudinal research

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Abstract

It is axiomatic that the capacity to experience fear is adaptive, enabling rapid and energetic response to imminent threat or danger. Despite the generally accepted utility of functional fear, the nature of maladaptive fear remains controversial. There is still no consensus about how specific fears and phobias are acquired and modulated. Two major schools of thought are apparent: those suggesting dysfunctional fear arises largely as the result of associative-conditioning processes versus those who favour more biologically based etiological explanations. In this regard, the non-associative model of fear acquisition postulates the existence of a limited number of innate, evolutionary-relevant fears, while emphasising conditioning modes of onset for evolutionary-neutral fears. Recent retrospective and longitudinal studies have tested predictions from the non-associative model. In general, findings support non-associative hypotheses and are difficult to reconcile with neo-conditioning explanations of fear acquisition. These data suggest that four pathways to fear may provide the most parsimonious theory of fear etiology. The theoretical and practical implications of adding a fourth, non-associative path to Rachman's (Behav. Res. Ther. (1977) 15, 375–387) three 'associative' pathways are discussed. Unresolved issues requiring further investigation are considered.

1. Overview and introduction

During the last 30 years, non-associative accounts of fear acquisition have begun to attract writers from a variety of theoretical backgrounds (e.g. Bowlby, 1973; Clarke & Jackson, 1983; Marks, 1969, 1987; Menzies & Clarke, 1995a; Rachman, 1978). Non-associative models assert that most members of a species will show fear to a set of biologically relevant stimuli from early

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encounters, given normal maturational processes and normal background experiences (Menzies & Clarke, 1995a). That is, non-associative accounts suggest that some fears may appear without any relevant associative learning experiences, either direct (pairings of feared objects with stimuli producing pain or distress), or indirect (vicarious conditioning or information/instruction). In general, these models propose that Darwinian natural selection has favoured individuals who displayed some level of fear on their first encounter with a dangerous object or situation, rather than individuals who readily acquire fears given appropriate learning experiences, as suggested by Seligman (1971).

As Menzies and Clarke (1995a) point out, the notion that fear can arise without associative learning involving the fear stimulus is not a new one. As early as 1877, Charles Darwin had proposed that such fears might arise from natural selection within a given species. After observing his 2-year-old son's fear of large zoo animals Darwin asked, "May we not suspect that … fears of children, which are quite independent of experience, are the inherited effects of real dangers … during savage times" (Darwin, 1877, cited in Marks, 1987, p. 112). A variety of authors have echoed Darwin's thoughts since that time. Notably, in the 1970s, Rachman described a model of fear consistent with the Darwinian notion, and very similar to that subsequently dubbed the "non-associative model" by Menzies and Clarke (1993a, 1995). Rachman (1978, p. 255) entertained the possibility that:

> the predisposition to develop the most common fears is innate and universal, or nearly so, and that what we learn is how to overcome our existing predispositions. In large part, we learn to stop responding fearfully to predisposed or prepared stimuli.

Rachman (1978, p. 255) went on to suggest that fears of the dark and strangers represent clear examples of such fears:

> It is less a matter of acquiring fears of the dark and of strangers than of developing the necessary competence and courage to deal effectively with the existing predispositions or actual fears. Over a period of years, our fearful predispositions are weakened and shaped by habituation experiences.

The notion that the role of the environment is to abate biologically relevant fears, rather than account for their emergence, is at the heart of the non-associative position of Menzies and Clarke (1995a). Like Darwin and Rachman, these authors suggest that direct or indirect associative learning will not be required for fear to emerge to a variety of biologically relevant stimuli. Unlike Darwin and Rachman, however, Menzies and Clarke (1993a, 1995) do not suggest that the emergence of fear is independent of all experience. Rather, certain background or maturational experiences may be required for fear to arise. For example, fear of heights in human infants appears to require a brief period of self-produced locomotion for its emergence (Bertenthal, Campos, & Barrett, 1984). Similarly, fear of moving objects seen in young chicks appears to vary with the level of illumination that occurred while in the egg (Dimond, 1966; Salzen, 1979). In essence then, biologically relevant fears are better said to "reflect maturation under genetic control *during interaction with the environment*" (Marks, 1987, p. 109, italics added). However, the critical point in the non-associative position, as presented by Menzies and Clarke (1995a), is that aversive

associative learning producing associations between the relevant stimulus and negative outcomes is not necessary for fear to arise. On this point, there is agreement between the positions of Rachman, Menzies, Poulton and others (e.g. Gray & McNaughton, 2000).

The fears that Menzies and Clarke (1993a, 1995) propose as potentially non-associative have the following features: (1) the feared object/situation/activity must represent a long-standing danger to the species; (2) fear and avoidance of the object/stimulus/activity must have increased reproductive opportunities, presumably by extending life, in our ancestors; and (3) fear and avoidance of the object/situation/activity is partly under genetic control. Each of these assumptions is, of course, difficult to test. Evidence for the non-associative model has therefore tended to come from findings, in both retrospective and longitudinal cohort studies, that suggest that relevant associative learning has not occurred in the histories of phobic individuals. In addition, specific predictions regarding failures to overcome 'innate' fears have recently been tested. Menzies and Clarke (1995a) propose two explanations for the failure of some individuals to habituate to pre-potent stimuli. These are: (1) insufficient opportunity for exposure at critical points in development; and (2) individual differences in the rate or speed at which habituation takes place. This paper reviews evidence that fears and phobias may emerge in the absence of direct or indirect conditioning, and will demonstrate how a non-associative model can provide a more satisfactory account of fear phenomena than conditioning theory.

2. Why a non-associative model?

Contiguity (contemporaneous pairing of a CS and a UCS) is no longer the sine qua non of conditioning theory. Research has clearly demonstrated that it is learning about contingency or the predictive value of a particular stimulus that is critical for fear acquisition in the laboratory (Mackintosh, 1983; Rescorla 1980, 1988). This recognition has helped negate many of the earlier criticisms of 'traditional' conditioning models (Rachman, 1998). However, it has also spawned neoconditioning theories that lack parsimony. That is, "In theory, almost any stimulus or past stimulus or event can become a signal for fear, but in practice, people are found to have comparatively few fears" (Rachman, 1990a, p. 11).

As well as struggling to explain the non-random distribution of common fears (e.g. Rachman 1990a,b, 1998; Merckelbach, de Jong, Muris, & van den Hout, 1996), modern conditioning models still have difficulty accounting for the emergence of fear in circumstances in which direct or indirect conditioning events cannot be identified. While a number of mechanisms have been proposed to account for 'spontaneous' fear (e.g. see Davey 1992a, 1995; Mineka & Zinbarg, 1996; Merckelbach et al., 1996), these are often difficult to demonstrate outside the laboratory. Historically, the non-random distribution of fear and the spontaneous emergence of fear have been the greatest challenges confronting conditioning accounts of fear acquisition. However, there now appears to be an even greater threat to their validity. Recent data have demonstrated that fearful individuals had significantly *less* relevant direct traumatic experiences than those without fear (Poulton, Davies, Menzies, Langley, & Silva, 1998). These findings are in the opposite direction to that predicted by conditioning theory, but are consistent with predictions from the non-associative model and other evolutionary-based accounts of psychiatric disorder (e.g. Marks & Nesse, 1994; Nesse & Williams, 1994).

We aim to demonstrate how a non-associative perspective can contribute to the understanding of: (i) the non-random distribution of fears; (ii) failures to recall direct or indirect conditioning events in fear onset; and (iii) the apparently paradoxical finding that people with high levels of fear report less direct traumatic events than those without such fear. Our ultimate goal is to convince readers that not only do sufficient data exist to support a non-associative pathway to fear (cf. McNally, 1995; Merckelbach et al., 1996; Davey, 1995), but that *four* pathways to fear provides the most comprehensive and parsimonious theoretical account of fear etiology.

2.1. Retrospective reports

Recent research into the non-associative model that has focussed on retrospective reports from analogue and clinical phobic cases has been predominantly carried out by Menzies and his colleagues at the University of Sydney. Prior to their work, origins of phobia research was dominated by the use of Ost and Hugdahl's (1981) phobic origins questionnaire (POQ), an instrument that restricts possible onset models to Rachman's (1977) three associative-learning pathways (i.e. direct conditioning, vicarious conditioning, information/instruction). Because there were no questions aimed at non-associative possibilities, it is difficult to evaluate the relevance of much of the data collected with the POQ. What has been claimed by several authors is that the POQ generally leads to an overestimate of the frequency of conditioning events since: (1) it does not identify the prior affective neutrality of the CS; (2) it does not seek to identify an independent UCS in supposed conditioning events; (3) it appears to equate any traumatic incident with conditioning; and (4) as stated above it does not include items to detect non-associative modes of onset (see Menzies & Clarke, 1994; Menzies, Kirkby, & Harris, 1998; Kirkby, Menzies, Daniels, & Smith, 1995; Menzies, 1996). In a recent examination of the convergent validity of the POQ, Menzies et al. (1998) showed that the POQ was consistently associated with a much greater likelihood of classifying the origin of fear reactions to direct conditioning pathways than alternative instruments. These authors recommended against the continued use of the instrument, and little attention is therefore paid to POQ-based data in the present paper.

Importantly, viable alternatives to the POQ are available. In 1993, Menzies and Clarke investigated the acquisition of fear of heights in an undergraduate student sample using a new origins instrument, simply titled the origins questionnaire (OQ). This 16 page instrument was designed to avoid the methodological pitfalls of earlier retrospective questionnaires (see Menzies et al., 1998) and to give a comprehensive picture of an individual's history in relation to the phobic object or situation prior to the onset of their concerns. The instrument has established inter-rater reliability and convergent validity (Menzies & Clarke, 1993a).

In the first report using the OQ, the origins of height fear in an undergraduate sample were investigated. Height-fearful and non-fearful groups were formed on the basis of extreme scores to the heights item on the FSS-III (Wolpe & Lang, 1964). Subjects were then assessed with a battery of measures including: (1) the OQ; (2) the acrophobia questionnaire (AQ; Cohen, 1977); (3) global assessment of severity; (4) self-rating of severity; and (5) an origins interview. Results obtained questioned the significance of simple associative-learning events in the acquisition of height phobia. Only 18% of fearful subjects were classified as directly conditioned cases. No differences between groups were found in either the proportion of subjects who knew others who

were fearful of heights, the proportion of subjects who had experienced relevant associative-learning events, or in the ages at which these events had occurred.

In a subsequent study with a clinical sample, Menzies and Clarke (1995b) sought to replicate these findings with a group of acrophobic sufferers who had sought treatment. This replication was particularly important given Rachman's (1977) hypothesis regarding the possible relationship between onset and response patterns, where he speculated that severe clinical phobias may be more likely to result from direct, traumatic conditioning episodes. All of the measures used in the earlier study were administered to 148 subjects who sought treatment at a height phobia clinic. In addition, a behavioural test that allowed the collection of physiological data was included. Menzies and Clarke (1995b) found that more people with acrophobia claimed that their fear had always been present, or had arisen from a non-associative traumatic event, than were classified as directly conditioned cases. In fact, non-associative categories accounted for 56% of subjects, compared to only 11% of subjects who were classified as cases of direct conditioning. Despite the inclusion of physiological and avoidance data, no relationships between onset, severity and individual response patterns could be found. Again, no differences between groups were found in the proportion of subjects who knew others who were fearful of heights, the proportion of subjects who had experienced relevant associative-learning events, or in the ages at which these events had occurred.

Retrospective investigations of the origins of spider fear/phobia have also failed to identify associative-learning events in the histories of subjects. Excluding reports relying on the POQ, four studies have examined the origins of spider fear. In the first report, Kleinkneckt (1982) found no cases of direct conditioning. Using the OQ, Jones and Menzies (1995) replicated this finding, adding that they could also not identify any cases of vicarious learning or information/instruction in their sample of spider fearful students. Davey (1992b) identified only one student in his sample of over 100 for whom an arbitrary aversive UCS was paired with spiders at onset. Finally, using the OQ, Kirkby et al. (1995) found direct conditioning in only 6% of their spider phobic patients, with a further 9% accounted for by indirect associative learning pathways. This compared to 45% of the sample who, consistent with the non-associative model, claimed to have always been fearful. In total, across the 228 subjects in these four studies, only 3 subjects were found to have experienced a relevant direct conditioning event.

All of the retrospective studies described above are, of course, subject to errors inherent in research using the retrospective method. That is, subjects may have simply forgotten critical events (or critical aspects of the events), and this may have biased the results against conditioning explanations. This is particularly the case since the subjects in all of these reports were adults who were being asked to recall long-distant events, often in early childhood. In an attempt to overcome this latter problem, Menzies and Clarke (1993b) investigated the origins of a clinical group of child water phobics, aged 3–8 years. This was an important study, since it was the first to investigate the origins of any clinical group of child phobics, and allowed for parental verification of supposed learning events. Parents were asked to identify the most influential factor in the onset of their child's fear from a list of commonly identified events in pilot investigations of the etiology of water fear. Only one of 50 parents questioned could recall a classical conditioning episode at the onset of their child's phobia. Thirteen parents identified possible vicarious learning events and none attributed onset to information/instruction. Consistent with the non-associative position, however, 28 of the 50 parents claimed that their child had always been afraid. That is, 56% of parents

claimed that their child had displayed fear of water *from their first contact with the stimulus*. More recently, Graham and Gaffan (1997) have replicated and extended these findings. In a small sample of non-swimming children with current fears of water, 77% of parents claimed that their child's fear had been present on the first contact. None of the parents attributed their child's fear to any associative-learning pathway. Graham and Gaffan (1997, p. 107) concluded, in words that echo Rachman's (1978) speculations about the role of life-experience in the emergence of fear:

> Overall, our findings favour the view of Menzies and Clarke (1993b) that fear of water arises with no or minimal learning, perhaps enhanced by a heritable propensity towards fears in the unknown and danger categories. *The role of children's experience is usually to prevent or diminish the strength of fear, rather than to instigate it*. (italics added).

While the data from these studies are clearly supportive of the non-associative position, Menzies and colleagues have recently acknowledged that, in general, the origins instruments used in retrospective studies have not done justice to all neo-conditioning explanations of the emergence of fear. For example, though clearly an advance on the POQ, Menzies and Clarke's (1993a) OQ does not include items that assess critical constructs of neo-conditioning approaches. Like previous origins instruments, the OQ searches for contiguous pairings of the phobic stimulus with an independent UCS, or for observational or informational transmissions. Davey (1997), and others, have pointed out that contemporary conditioning accounts do not depend on the occurrence of aversive CS–UCS pairings. As stated earlier, based on Rescorla's notion of contingency rather than Pavlov's contiguity, conditioning acquisitions are said to depend on an individual developing an expectancy that a given CS will be followed by an aversive event. Such learning may, or may not, be preceded by direct pairings of the CS and the UCS. This has been demonstrated many times in the laboratory (e.g. White & Davey, 1989). Further, even when an aversive CS–UCS pairing does occur, Davey (1997) and Mineka and Zinbarg (1996) and others have argued that a variety of factors can account for whether or not a given subject will acquire fear from the pairing. These factors include, but are not limited to: (1) the general neuroticism or trait anxiety level of the individual; (2) the individual's existing beliefs about the contingency being acquired and the threat of the CS; (3) the amount of previous non-noxious exposure to the CS experienced by the individual; (4) the level of fear and pain experienced during the event, and; (5) the extent to which the individual subsequently revalues the UCS owing to experiences with the UCS alone, cognitive rehearsal of the UCS, new information about the UCS and the like.

All of these neo-conditioning factors may explain the failure of researchers to find differences in the frequency of associative-learning events between fearful and non-fearful groups. Acknowledging this, Menzies and Parker (2001) recently attempted a comprehensive evaluation of neoconditioning possibilities in the acquisition of height fear. Modifying the OQ, items were included to assess the relevance of latent inhibition, UCS inflation/revaluation, prior fear levels, prior expectancies of harm, and fear and pain levels experienced during supposed learning events. Undergraduate height-fearful students (54) completed the OQ-II, while 54 matched controls completed a modified version (OQM-II) that examined their prior experiences with heights. In general, few differences between groups were found. First, height-fearful and control groups did not differ on trait anxiety as measured by the state-trait anxiety inventory (Form Y-2) (Spielberger, 1983). This is important, since many theorists have argued that the failure of control subjects to acquire

fear from similar traumatic encounters to those experienced by phobics may be due to general temperamental differences between control and phobic individuals (Mineka & Zinbarg, 1996). Second, height-fearful and control subjects did not differ on the frequency of negative encounters with heights, the age at which these events had occurred, prior fear levels, prior expectancies of harm, or reports of UCS inflation/revaluation procedures. However, in a finding directly opposite to that expected from a conditioning account, the mean fear and pain scores reported by subjects who had experienced direct conditioning events were significantly higher in the non-fearful group than in the height-fearful group.

This latter finding is particularly difficult to reconcile with a conditioning model of fear. Why is it that individuals receiving minor injury/fear/distress would go on to develop intense fear reactions and those receiving significantly greater pain/distress would not? As stated above, the height-fearful group did not have higher levels of trait anxiety than the non-fearful group. Similarly, height-fearful subjects did not report subsequent events or information that had produced a growth of their fear through UCS inflation/revaluation. While inconsistent with associative learning positions, these findings are consistent with the non-associative model of fear. According to the non-associative position described earlier, the biologically relevant developmental fears serve to protect the individual by discouraging full engagement with the stimulus *from the earliest possible encounters*. Those individuals with stronger fear responses from infancy will be best protected from the dangers associated with the stimulus across the lifecourse, and may go on to display fear/phobia in adulthood despite a history characterised by less dangerous/painful encounters with the stimulus. Such individuals will have been less likely to fully engage with play/climbing equipment in the home and school yard, generally climbing to lower heights and placing themselves in fewer risky situations (see Menzies and Parker, 2001). While, like all children, they may have their share of falls, these are likely to be less painful/severe and produce less distress.

2.2. Prospective research: the Dunedin findings

As stated earlier, while clearly supportive of the non-associative position, the retrospective data summarised above are subject to a variety of errors inherent in research using the retrospective method. For this reason, recent longitudinal studies by Poulton and his colleagues have led to a better understanding of how fears emerge across development. They have tested a number of hypotheses from the non-associative model among individuals enrolled in the Dunedin Multidisciplinary Health and Development Study (hereafter the Dunedin Study). Because of the rarity and explanatory power of large prospective reports, the Dunedin study will be described in considerable detail.

The Dunedin study is a longitudinal investigation of the health, development and behaviour of a cohort of births between April 1, 1972 and March 31, 1973, in Dunedin, a provincial capital city of 120,000 on New Zealand's South Island. Perinatal data were obtained at delivery, and when the children were later traced for follow-up at age three, 1037 (91% of the eligible births, 52% males) participated in the assessment, forming the base of the sample for longitudinal study (Silva & Stanton, 1996). The sample is representative of the social class distribution in the general population of similar age in New Zealand's South Island, and cross-national comparisons of social problem indicators lend some confidence for the generalisability of the findings from the Dunedin

study to other industrialised countries. For example, prevalence rates of psychiatric disorders such as major depression (16.8%), specific phobias (8.4%), antisocial personality (3.2%) and alcohol dependence (9.8%) in the Dunedin sample at age 21 (Newman, Moffitt, Caspi, Magdol, Silva, & Stanton, 1996) match closely the rates at this age period from the two nationally representative US surveys (Robins & Regier, 1991; Kessler, McGonagle, Zhao et al., 1994).

General assessment procedures. Study members have been assessed with a diverse array of psychological, medical and sociological measures with high rates of participation at the following ages: 3 years (n=1037); 5 years (n=991); 7 years (n=955); 11 years (n=925); 13 years (n=850); 15 years (n=976); 18 years (n=993); 21 years (n=992) and most recently (1998–1999) at age 26 years when 980 study members were assessed. This is equivalent to 96.2% of the original cohort who were still alive (n=1019). By age 26, approximately 40% still lived in or near their birth city, Dunedin. The rest have primarily emigrated as young adults to other areas of New Zealand and Australia, and a minority are now living in the United Kingdom, North America, and Asia.

At each assessment phase (numbered to reflect the chronological age of the participants), Dunedin Study members are invited to spend a day at the Research Unit for private interviews and examinations. On average there have been eight assessment modules in each phase: for example, modules within one phase might include delinquency, dental examination, mental health, injuries, partner relationships, physical examination, labour force experiences, and respiratory functioning. Each of the various research modules is administered in different rooms by different interviewers who are blind to the study members' responses to other modules. This practice is designed to diminish the artefactual inflation of effects sizes by shared method variance that afflicts studies using measures collected on the same questionnaire, or in the same interview. The modules are presented in counter-balanced order across study members, to prevent precedence effects or fatigue effects. We believe that these features of data collection at the research Unit contribute to inferential power.

Assessment of fear and phobia in the Dunedin study. In late childhood (i.e. age 11), study members were interviewed using version XIII-III of the diagnostic interview schedule, child version (DISC-C; Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982), which contains a number of items clustered by disorder and based on *DSM-III* criteria (see Anderson, Williams, McGee, & Silva, 1987). This interview contained questions about a number of 'simple' fears. From age 18 onward, study members have been administered the adult mental health interview consisting of items from the version of the diagnostic interview schedule current at the time (e.g. Robins, Helzer, Cottler, & Goldring, 1989; Robins, Cottler, Bucholz, & Compton, 1995). Only items which give *DSM III-R or DSM-IV* classifications, including specific fear and phobias, were administered in the interviews (see Feehan, McGee, Nada Raja, & Williams, 1994 and Newman et al., 1996 for more information about psychometrics and reliability of these interviews in this sample). Details about how fear measures were constructed at different ages are provided in the original reports. The independent variables differ in each study and will be briefly described in the following review of findings. Again, the reader is referred to the original reports for further information about individual measures.

The first study of the Dunedin cohort investigating fear acquisition examined the relation between conditioning experiences (operationalised as caries level) at age 5 and age 15 and self-reported dental fear at age 18 (Poulton et al., 1997). Findings indicated that caries experience at age 5 was not related to the development of dental fear in late adolescence. In contrast, dental

caries experience up to age 15 was significantly and specifically related to dental fear at age 18. Study members with greater caries experience at age 15 were almost five times more likely to report dental fear, but not other fears, at age 18. This finding suggests that poor dental health in middle-late childhood and early adolescence (requiring more extensive dental work with increased likelihood for aversive conditioning), was causally related to the onset of dental fear at age 18.

The key role played by putative conditioning processes in both early and late (>18 years) onset dental fear has recently been confirmed (Poulton, Waldie, Thomson, & Locker, 2001c). These data also revealed the contribution of personality characteristics to the development of early but *not* late onset dental fear (cf. Mineka & Zinbarg, 1996). While consistent with conditioning theories, both sets of findings are also predicted by the non-associative model which posits that onset of evolutionary-neutral fears such as dental or driving fear should be related to conditioning events.

The next study in this series was designed to examine the relation between traumatic events and the development of an evolutionary-relevant fear. We chose heights because earlier retrospective reports (Menzies & Clarke, 1993a, 1995b) supported predictions from the non-associative model and because height fear appeared to be the prototypical evolutionary-relevant fear. We used data from assessments conducted at ages 3, 5, 7, 9, 11 and 18 to examine the relation between serious falls before age 9 and height fear at age 11 and 18 years (Poulton et al., 1998). Fall categories included: fall on or from stairs, steps, ladders or scaffolding; fall from or out of a building or other structure (e.g. balcony, bridge, windows, roofs); fall into a hole or other surface opening (e.g. pit, quarry, shaft, tank, well); and fall from playground equipment, cliff, tree or bank. This information was obtained as part of a more general injury questionnaire completed by parents at ages 3, 5, 7 and 9. It was structured so that a description of the injury was first elicited, followed by circumstances of injury and the treatment sought. Where possible, details regarding the nature of the injury were verified by reference to Public Hospital inpatient and radiology files. Only serious falls resulting in a fracture, dislocation, laceration or intracranial injury were included in the study.

Despite falls being the most common accident occurring to children up to 9 years (Gafford, Silva, & Langley, 1996), no positive relation between these putative aversive events and height fear at age 11 or phobia at age 18 was found. In contrast, falls resulting in serious injury between 5 and 9 years occurred with greater frequency in those *without* a fear at age 18. Most importantly, *no* individual who had a height phobia at age 18 had a history of a serious fall before the age of nine. The similarity of these findings to those obtained in the retrospective study of Menzies and Parker (2001) is striking. In both reports, serious levels of pain/injury or distress caused by falls were found in subjects *without* height fear. That is, obvious conditioning experiences did not produce fear, seriously damaging the relevance of associative learning models of height fear.

This report (Poulton et al., 1998) also provides the first support for Marks and Nesse's (1994) concept of 'hypophobia'. Marks and Nesse (1994, p. 254) argue that the absence of 'normal' levels of developmental fears represents a serious disorder that places the individual at increased risk of injury or death. They state:

> Anxiety, too, is beneficial … too little anxiety leads to behaviour that makes us more likely to fall off a cliff … people with too little anxiety do not come to psychiatrists complaining of deficient fear, so their disorders, the 'hypophobias', still await formal description.

In perhaps the first clear evidence for hypophobia, we showed that the absence of adult height fear was associated with an elevated history of serious falls in childhood. It is suggested that children without sufficient fear are likely to fall when in high, vulnerable positions. Further, directly opposed to a conditioning position, such children do not go on to develop height fear in adulthood. Instead, they continue with less fear than those individuals who have never fallen (Poulton et al., 1998).

We sought to establish the generalisability of the findings from the height fear study, this time using a different evolutionary-relevant fear, namely fear of water. We assessed the relation between water exposure, water skills and water trauma between ages 3 and 9, and the development of water fear or phobia in late adolescence (Poulton, Menzies, Craske, Langley, & Silva, 1999). No relation was found between swimming experiences (e.g. swimming ability, being rescued from water) up to the age of nine and water fear at age 18. A similar finding was obtained for these childhood swimming variables and water phobia at age 18, with the exception that Study members less able to immerse themselves in water with confidence at age 9 were more likely to report water phobia at age 18. Again, these findings were consistent with a non-associative account of fear acquisition, and with the results from the earlier retrospective studies of children with water fear (Menzies & Clarke, 1993b; Graham & Gaffan, 1997).

However, both these prospective studies focussed on the role of *direct* conditioning events in fear development. Neither examined two other major associative pathways to fear, namely vicarious learning/modeling and transmission of information. Thus, it remains possible that height and water fear may have developed via either of these less direct, conditioning routes. Accordingly, we sought to conduct a more comprehensive test of the theory by comparing three associative pathways (see Rachman, 1977) against the non-associative account in the development of separation anxiety assessed at age 3, 11 and 18 years (Poulton, Milne, Craske, & Menzies, 2001a). Separation experiences were classified as: (1) direct conditioning (e.g. the number and duration of separations from mother; days of overnight hospitalisation); (2) modeling (e.g. mothers self-reported fear, teacher ratings of mothers separation anxiety at the start of schooling); and (3) transmission of information (i.e. parents threatening separation as a form of punishment). Finally, because low socio-economic status has been suggested to be a risk factor for separation anxiety (Bird, Gould, Yager, Staghezza, & Canino, 1989), we controlled for socioeconomic status in all analyses.

Conditioning events (separations from mother, hospitalisations) were not related to behavioural ratings of separation anxiety at age 3. Vicarious learning (modeling) in middle childhood (age 9 years) was the conditioning variable most strongly related to self-reported separation anxiety at age 11, but accounted for only 1.8% of the variance in separation anxiety symptoms. Separation experiences (hospitalisations) before the age of 9 were found to be negatively correlated with self-reported separation anxiety at age 18. That is, more overnight hospital stays in childhood were related to *less* separation anxiety in late adolescence. However, none of these conditioning correlates remained significant predictors of separation anxiety in adjusted regression models. In general, the findings were consistent with predictions from the non-associative theory of separation fear acquisition (e.g. Bowlby, 1973; Clarke & Jackson, 1983; Marks, 1987). That vicarious learning processes appeared to modulate, albeit to a minor degree, the expression of separation anxiety during mid-late childhood suggests that there may be critical periods during which some individuals are susceptible to the interactive effects of both associative and non-associative fear processes.

In sum, results from five prospective studies (three with different evolutionary-relevant fears and two using the same evolutionary-neutral fear) were consistent with predictions from the non-associative model of fear acquisition. Notwithstanding, several commentators continue to voice misgivings about the validity of the non-associative account because it predicts more fear than is actually observed. According to Merckelbach and de Jong (1997, p. 336):

> the non-associative account leaves unexplained why not all people suffer from specific phobias. Menzies and Clarke (1995a,b) argue that developmental fears take a chronic course in poor habituators, but this is begging the question. Why exactly do some individuals habituate rapidly and others poorly to prepotent fear stimuli? The non-associative account provides no direct answer to this question.

This is not correct. Specifically, it has been hypothesised that poor habituators and those who do not have the opportunity for safe exposure can be expected to retain their childhood fear which, if persistent, may result in specific phobias in adulthood (Clarke & Jackson, 1983). It has also been hypothesised that non-specific stress may lead to the return of previously habituated fear (Menzies & Clarke, 1995a; also see Jacobs & Nadel, 1985).

Two prospective studies have been conducted to test non-associative explanations of why a minority of individuals do not grow out of developmental fears. In a controlled test of the fear dishabituation hypothesis, non-specific stress levels in mid-adolescence (age 15) were compared among individuals with an evolutionary-relevant fear (height), an evolutionary-neutral (dental) fear and those without fear (Poulton, Waldie, Craske, Menzies, & McGee, 2000). Only individuals who had reported an onset of height or dental fear or phobia *between* age 11 and 18 years were selected. That is, at age 11 they did not report fear but did so seven years later at age 18. In this context, height fear dishabituation was assumed to occur between 11 and 18 years because of findings and theory suggesting that all members of our cohort were likely to have been afraid of heights in infancy following self-produced locomotion (e.g. Gibson & Walk, 1960; Marks, 1987; Menzies & Clarke, 1993a; Nesse & Abelson, 1995).

The results confirmed our hypothesis. Dishabituation of height fear and phobia was observed for study members reporting high levels of non-specific stress at age 15. Further, and as predicted by the non-associative model, this finding was specific to height fear. That is, those reporting dental fear at age 18 did not differ from the no-fear group in their experience of non-specific stress. Interestingly, stress of at least moderate intensity was related to self-report of height fear whereas only very high stress levels were related to height phobia (Poulton et al., 2000).

To test if individuals with height fear and phobia at age 11 and 18 years had less exposure to height stimuli early in life compared to no-fear controls, we compared these groups on their frequency of exposure (at ages 3 and 5) to eight activities selected to represent common outdoor childhood activities (Silva, 1980). These included: (1) “climbed trees, fences”; (2) “played on swings, bars”; (3) “swum, paddled in a pool”; (4) “dug holes”; (5) “rode on trike or similar”; (6) “played in sandpit”; (7) “played with a ball”; and (8) “went out in a car”. We found support for the hypothesis that individuals with height fear at age 11 and 18 would have less exposure (and presumably less opportunity for habituation) to height stimuli up to the age of three. Importantly, differential exposure was specific to height stimuli (Poulton, Waldie, Menzies, Craske, & Silva, 2001b).

The hypothesis that some individuals are simply poor habituators who never grow out of their developmental fear (Menzies & Clarke, 1993a, 1995a; also see Rachman, 1978, p. 255) was tested among individuals with height and dental fear at age 11 and height and dental phobia at age 18. Because arousal plays an influential role in habituation (Rachman, 1990b; Lader & Wing, 1966; Lader & Mathews, 1968; Watts, 1979) we chose a measure of stress reactivity as a proxy for habituation. The measure was taken from the Multidimensional Personality Questionnaire (MPQ; Tellegen 1982, 1985). The dental fear group was included to provide a more stringent test (i.e. specificity) of the poor habituation hypothesis. Findings indicated that individuals with height phobia at age 18 were more stress reactive (i.e. poorer habituators) than those with dental fear or study members who did not report any fears. This finding applied only to phobia (as opposed to mild fear) and was specific to the stress reactivity scale insofar as comparisons using the MPQ negative emotionality superfactor failed to reveal group differences (Poulton et al., 2001b). Thus, the data provided support for the non-associative hypothesis as applied to height phobia only — poor habituation as measured by stress reactivity was not related to the sub-clinical variant of height fear.

3. Evaluating the evidence from the Dunedin study

The Dunedin study is a unique resource for the study of fear development for several reasons. First, it is a prospective-longitudinal study in which information is obtained about the same persons as they are assessed repeatedly over time. In the absence of prospective studies, researchers have been forced to rely upon retrospective studies requiring people to recall detailed information about events occurring many years earlier. Unfortunately, most people are inefficient and inaccurate processors of information about their past. There is little agreement between how individuals recollect themselves and what is known about them from historical data sources (Henry, Moffitt, Caspi, Langley, & Silva, 1994; Rutter, Maughan, Pickles, & Simonoff, 1998). The Dunedin study, in which people were followed up and assessed repeatedly in real time, helps overcome this problem. Second, because the Dunedin study is a developmental-longitudinal study of a representative population sample, it can provide an unbiased understanding of associations between variables. Most importantly, it avoids distortions in associations between variables that are common in volunteer samples or in selected samples of various kinds (Berkson, 1946; Newman, Moffitt, Caspi, & Silva, 1998). Third, from the beginning, the Dunedin study has had a multidisciplinary focus that has resulted in the availability of a wide array of developmental data. This has enabled evaluation of the relation between apparently unrelated areas of functioning that have recently emerged as relevant to fear acquisition.

To recap, findings from the two dental fear papers supported the role of conditioning events in the development of dental fear. From the non-associative perspective, this finding is expected. The differences observed between early versus late onset dental fear (Poulton et al., 2001c) provide support for Mineka and Zinbarg's (1996) Stress-in-dynamic context anxiety model in which they suggest that fear onset can be influenced by aspects of personality. Our findings suggest that age of onset might be an important moderator of this relation for evolutionary-neutral fear. Findings from the height fear paper are clearly consistent with the non-associative model and the apparent protective effect of fear seen in the height fear group is antithetical to predictions from

associative-conditioning models. In this sense then, the non-associative model provides explanation where others cannot (see Merckelbach & de Jong, 1997). Further, it is notable that the findings provided no support for latent inhibition effects. Together, the findings from this study, and those recently reported by Menzies and Parker (2001), represent a serious challenge to the validity of conditioning models, at least with regard to the development of height fear — the prototype of evolutionary-relevant fear.

There are several possible interpretations of the data from the study of water fear (Poulton et al., 1999). First, consistent with the non-associative model of fear acquisition, the findings suggest that water fear is innate and can manifest without being paired with aversive experiences. However, it remains possible that children had aversive experiences with water without their parents knowledge or that water may have been connected with aversive experiences in the years between ages 9 and 18. Other interpretations of the data can be considered. For example, it has been argued that conditioned associations may develop insidiously, resulting from a series of experiences rather than a single traumatic event. (e.g. Kleinkneckt, 1994). However, if this were the case, we would have expected those with multiple experiences to report more negative incidents with water, not less. The failure to find any difference in the frequency of water trauma in the fearful and non-fearful groups does not support a multiple trauma onset account. It is also possible that previous experience with the to-be-feared stimulus served as a latent inhibitor of the expression of fear. However, water fearful and non-fearful study members did not differ in the age at which they had learnt to swim, the amount of water exposure (i.e. the number of times swimming in the previous year), or the age at which water trauma first occurred, rendering the latent inhibition hypothesis an unlikely explanation for the findings.

The study of the etiology of separation anxiety was particularly important because it compared three conditioning pathways against predictions from the non-associative model. Additionally, this study was presumably able to control for incubation or sensitisation effects hypothesised to influence fear acquisition (e.g. Davey, 1997; Mineka & Zinbarg, 1996). This was assumed because the same individuals were interviewed repeatedly over 18 years and the full range (mild to extreme) of putative conditioning events were assessed. Another strength of the study relates to the measures used to assess separation anxiety. At age 3, we created a composite index of separation anxiety based on behavioural observations made by two raters (one medical doctor and one psychometrist) who were trained to rate separation anxiety and fear of strangers on different scales according to predefined protocols (see the American Collaborative Study Behavioral Protocols, 1970). This was important because fear of strangers differs from distress at separation from the caretaker. The two fears have been found to summate if the child is separated from its caretaker in the presence of a stranger, and correlate slightly (Sroufe, 1974). Many tests for one of these two fears have compounded both situations (Marks, 1987). At age 11 and 18, as for earlier studies, we used standardised, structured diagnostic interviews (DISC and DIS, respectively) to elicit self-reported information about distress experienced in a number of separation situations.

This study is noteworthy for another reason. Although the findings were consistent with a non-associative model of fear acquisition, they also hinted at the possibility of an interaction between conditioning and non-associative processes during specific developmental epochs (i.e. ages 7–9 years).

Several strengths of the fear dishabituation study (Poulton et al., 2000) are apparent: first, it provided evidence for specificity of dishabituation to height fear and secondly, it used an age-

relevant stress measure based on the individuals perception of the stress (i.e. "how bad" it was for the individual). This was important because it is the individual's perception of threat or difficulty associated with adverse life events that determines the level of stress experienced, not events per se (e.g. Paykel, 1983; Thoits, 1983). Further, the stressors were genuinely non-specific (e.g. not having done homework on time, arguing with your friends or breaking up with them) and therefore could not be viewed as height-related traumas leading to conditioning of height fear at age 18. Of course, it remains possible that height fear may have had an onset between age 11 and 15, prior to the assessment of stress levels. This may indicate a role for conditioning events(s), vicarious learning or the like in the genesis of height fear rather than the influence of non-specific stress. However, this argument would apply equally to dental fear for which no relation with non-specific stress was observed. One might also argue that the onset of height fear may have caused stress rather than vice versa but this seems unlikely judging by the nature of the stressors most commonly reported at age 15.

With regard to the findings about reduced exposure to fearful stimuli, there are several possible reasons why those with height fear at age 11 and at age 18 had reduced exposure to swings and bars earlier in life. These include: (1) limited access to such equipment; (2) limited interest of the child in such outdoor activities; (3) limited parental interest in such outdoor activities; (4) high levels of 'innate', non-associative fear of heights resulting in avoidance of such equipment; and (5) a history of early falls producing fear of such equipment. However, what was critical in this study was that, in support of the non-associative model, height-fearful study members *did have* less exposure and *did* display fear at a later age. This finding alone supports the 'insufficient opportunity to habituate' hypothesis of the non-associative model. Of course, if the reduced exposure was due to a history of relevant falls, the non-associative model is not needed to explain either avoidance or fear of height stimuli at age 11 and 18. However, this possibility seems unlikely since Poulton et al. (1998) have previously shown that height fear in adolescence was associated with a reduced (rather than increased) history of childhood falls.

The combined results of the exposure and habituation studies are consistent with the non-associative model that predicts that some people do not overcome their fear of heights because of limited opportunities to do so (non-exposure) or because a small percentage of individuals are simply at the high end of the normal distribution in terms of arousal or reactivity and have difficulties overcoming their highly reactive dispositions. The question remains as to why some individuals have limited exposure to height stimuli or are poor habituators. It seems likely that familial and/or genetic influences are important in this regard (Marks, 1969).

Overall, the data reviewed suggest that an expansion of Rachman's (1977) three pathways of fear acquisition to include a fourth, non-associative pathway is warranted. This may result in a more comprehensive theory of fear acquisition and generate a number of interesting possibilities.

4. Four pathways — implications for theory

Acceptance of the fourth pathway as a possible route to fear would help to address the "associative-learning bias" described by Menzies and Clarke (1994). These authors argued that most retrospective studies of the origins of phobias have imposed preliminary constraints upon their possible results by limiting pathway options to various conditioning-based alternatives. For example, in

one of the earliest retrospective studies of the etiology of human fear, Rimm, Janda, Lancaster, Nahl, and Dittmar (1977) revealed their leaning to the conditioning account early in their paper when they stated that "learning plays a necessary role in the acquisition of phobias" (p. 231). This bias is further evident in the four categories typically chosen for the classification of phobic onsets: (1) direct experience akin to classical conditioning; (2) vicarious experience; (3) verbal instruction; and (4) inability to recall a pertinent experience. The first three categories are learning-based explanations and even the fourth category, as Menzies and Clarke (1994) point out, suggests a bias to the learning model since it implies a failure to recall, rather than an absence of an assumed traumatic experience involving the feared stimulus. These categories simply do not cover all possibilities.

Adding the non-associative pathway to the three conditioning pathways described by Rachman (1977) should help integrate diverse findings from a variety of disciplines (behavioural, biological, epidemiological, evolutionary medicine). It also provides for a more parsimonious theory of fear acquisition that is consistent with clinical and epidemiological findings using retrospective and prospective designs and with neuropsychological, animal and pharmacological data (Gray & McNaughton, 2000) and recent genetic findings (e.g. Kendler, Karkowski, & Prescott, 1999). Further, and recalling the explanatory weaknesses plaguing conditioning theory, it would be possible to argue that the non-random distribution of fears are best explained by the failure to unlearn (either via lack of safe exposure or poor habituation) innate fears and that the inability to recall conditioning experiences is to be expected. Obviously, an individual cannot be expected to recall events that have never occurred. Until recently, this possibility has been extremely difficult to test. However, the longitudinal sampling design used in the Dunedin study has helped to largely overcome this problem with recall, and suggests that some form of rapprochement between environmental-conditioning and biological perspectives is required.

This is an ambitious proposal and it will only be realised if the limits of each pathway (or mechanism) are clearly established, while at the same time acknowledging that fear acquisition is a dynamic and complex process (e.g. Mineka & Zinbarg, 1996; Pynoos, Steinberg, & Piacentini, 1999; Poulton et al., 2001a). Accepting that fear is primarily either learned or innate (Valentine, 1930; Gray, 1987) may also have implications for how fear is classified in official nomenclatures and classification systems. Currently, DSM-IV (APA, 1994), groups specific fears into four types: animal, natural environment, blood-injection-injury and situational. While DSM-IV states that "feared objects or situations tend to involve things that may actually represent a threat or have represented a threat at some time in the course of human evolution" (p. 408), the fears are not grouped accordingly. For example, dental and injection fear are grouped with blood-injury fear, yet the non-associative theory predicts these fears will be distinct in terms of etiology, and possibly patterns of comorbidity (e.g. Poulton, Thomson, Brown, & Silva, 1998). The importance of this issue is well illustrated by recent studies seeking to validate the DSM-IV system of fear classification (e.g. Curtis, Magee, Eaton, Wittchen, & Kessler, 1998), and by studies investigating the role of specific fear or phobia as a risk factor for the later development of other psychiatric disorders (e.g. Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998; Kessler, Nelson, McGonagle, Liu, Swartz & Blazer, 1996; Kessler, Grum, Warner, Nelson, Schulenberg, & Anthony, 1997; Davey, Menzies, & Gallardo, 1997).

The distinction between different etiological paths also has important implications for studies investigating the genetic basis of fear disorders (e.g. Eley, 1997; Flint, 1997; Smoller & Tsuang,

1998). Based on the data presented, the relative genetic and environmental contributions underlying fear phenotypes can be expected to vary. Further, explaining fear outcomes will ultimately depend on understanding gene-environment gene–environment interactions, and these can be expected to differ markedly depending on the type of fear studied and what aspect of the 'environment' is under investigation. For example, non-specific stress influences the re-emergence of evolutionary-relevant fear but not evolutionary-neutral fear (Poulton et al., 2000), early safe exposure to specific environmental stimuli can strongly determine liability to evolutionary-relevant fear but appears less important for other fears, and the influence of personality (vulnerability) traits on fear acquisition differs according to their evolutionary relevance and age of onset (Poulton et al., 2001c).

5. Implications for practice

The primary prevention implications that flow from the non-associative model are clear. Early intervention is likely to be of benefit for non-associative fears (e.g. Dadds et al., 1999) particularly in school settings. This applies equally to fear of heights and water as to school refusal and separation anxiety. The latter may be especially important as separation anxiety has been viewed as both a specific risk factor for the development of panic disorder-agoraphobia (e.g. Klein, 1980; Silove et al., 1995; Silove, Manicavasager, Curtis, & Blaszczynski, 1996) and as a non-specific vulnerability for the development anxiety disorders, depression and/or somatic symptoms (e.g. Lipsitz et al., 1994; Furukawa et al., 1999; Waldron, 1976).

Secondary prevention or treatment implications also exist, especially as the success rates of fear treatments are less than perfect (Craske, 1999). That is, clinicians should avoid insisting that associative learning events occurring in the patient's past have lead to their current problem. Insisting that the patient is wrong in claiming to have always been afraid, or insisting that they have simply forgotten supposed conditioning events, is likely to be counter productive in therapy (Menzies & Clarke, 1995a). When patient and therapist models of etiology are diametrically opposed, compliance in therapy is likely to be low (Clarke & Wardman, 1985). The non-associative model also suggests that some treatments will be more appropriate for certain fears than others. For example, if evolutionary-relevant fears like height fear or separation anxiety result from a failure to unlearn or overcome biological programming (i.e. failures of habituation) then repeated, graduated exposure should be the treatment of choice. Not surprisingly, cognitive treatments have been shown to lead to poor outcomes for such fears (Menzies & Clarke, 1995a). In contrast, evolutionary-neutral fears like those of the dentist can be influenced by personality traits and conditioning experiences (i.e. the development of threat expectancies), and may therefore benefit from a combination of cognitive and behavioural treatments (e.g. Locker, Liddell, Dempster, & Shapiro, 1999; Poulton et al., 2001c).

6. Where to from here?

Future research should firstly attempt to test the limits of the non-associative model. Can Darwinian models extend to other anxiety disorders, for example, social anxiety disorder (nee social

phobia) as suggested by some (e.g. Ohman, 1986; Ohman, Dimberg, & Ost, 1985), or psychiatric disorders in general (Nesse, 1999), or is it only applicable to specific fear?

The public health implications of predictions of the non-associative model also deserve more attention. For example, the consequences of hypoanxiety or hypophobia should be investigated further. This should be done using designs that are ecologically valid and less vulnerable to the vagaries of retrospective recall. For example, based on the height fear findings, we would predict that people with blood-injury fear would have a history of fewer physical injuries than those without such a fear. Further exploration of the relation between 'hypophobia', fearlessness and courage (Rachman, 1990b) might also provide new insights into the nature of resiliency and adaptation in the face of threat or danger.

A non-associative model accepts that evolutionary-relevant fears can be acquired via conditioning processes but maintains that this would be the exception rather than the rule. That is, on average, evolutionary-relevant fears do not require conditioning events to appear, whereas evolutionary-neutral fears do so. This relation may be assymetrical and, if so, the four pathways might be viewed hierarchically, with the non-associative as primary. The potential validity and utility of such a hierarchical system can only be determined by future empirical work.

A number of inconsistent findings remain to resolved. For example, Taylor and Deane (1999) interpreted the low rate of conditioning events reported in their driving fear study as supporting a non-associative model. However, fear of driving in a car is generally regarded as an evolutionary-neutral fear and should be most strongly related to conditioning events. This may still be true, as a follow-up report by the Taylor and her colleagues (Taylor, Deane, & Podd, 1999) demonstrated considerable one-year instability in their subjects' attributions about mode of fear onset. A prospective test operationalising Rachman's (1977) three conditioning pathways (direct trauma, modelling and information) in the development of driving fear would help clarify this issue. Nonetheless, these findings serve as a timely reminder of the limitations of the retrospective method in fear acquisition research (also see Kendler et al., 1999).

A closer examination of the evidence supporting various conditioning mechanisms is also warranted. For example, the mechanism most commonly proposed to account for failures to develop fear following trauma is latent inhibition (e.g. Rachman, 1990a,b; Mineka & Zinbarg, 1996; Merckelbach et al., 1996; Davey, 1992a; Marks, 1987). That is, previous benign exposure (perhaps over a long duration) to a CS (e.g. dogs as a family pet) will protect against fear development when finally associated with an aversive UCS (e.g. dog bite). However, while some experimental and retrospective data support latent inhibition (Davey, 1989; Doogan & Thomas, 1992; de Jongh, Muris, Ter Horst, & Duyx, 1995), recent prospective data do not (e.g. Poulton et al., 1998a,b, 1999). That is, people experiencing traumas with height and water at a later age were no less likely to develop fears than those experiencing these events earlier in life. In fact, quite the opposite was found in the case of height fear.

The potential benefits of adopting a developmental approach to issues of fear acquisition are emphasised by findings from the prospective studies. Information is now required about what factors influence the emergence and maintenance of fear early in life. Exploring the interaction between habituation (the capacity for which is assumed to be normally distributed in the population, e.g. Kagan, Reznick, & Snidman, 1988) and the determinants of insufficient exposure to evolutionary-relevant stimuli in infancy (i.e. failures to unlearn) might be a useful starting point.

Finally, we must acknowledge that although the findings from retrospective and prospective

human studies reviewed in the present paper are consistent with the non-associative model of fear acquisition, a number of laboratory findings are not. For example, in the fascinating series of primate studies conducted by Mineka and her colleagues, monkeys were not found to be scared of snakes upon first exposure, but quickly learned to fear these reptiles following observation of fearful models. These findings appear to be most consistent with a preparedness explanation of fear acquisition (Seligman, 1971; Mineka & Zinbarg, 1996). Of course, other laboratory findings are more consistent with the non-associative model of fear development. For example, it is hard to imagine stronger support for a non-associative account of height fear than that obtained in laboratory work with the visual cliff by Gibson and Walk (1960). It must be remembered that no chick, lamb or goat tested in this research program *ever* stepped onto the glass on the 'deep' side, *even* at one day of age. In addition, previous falls have been found to be unrelated and unnecessary for avoidance of the visual cliff in human infants (cf. Menzies & Clarke, 1995a). While a comprehensive review of laboratory findings is beyond the scope and purpose of the present paper, we believe that the retrospective and prospective data presented provides sufficient evidence that fear may arise without the involvement of oversize associative-learning processes.

By advocating what some have described as a 'minimalist' position (e.g. Gray & McNaughton, 2000) we take the view that specific fears can be acquired via either associative or non-associative means. The non-associative perspective simply predicts the most likely etiology based on the survival relevance of the particular stimulus. Nonetheless, our data also provide some evidence for an interaction between associative and non-associative paths during specific developmental periods for at least one evolutionary-relevant fear (Poulton et al., 2001a). Together, these findings are consistent with both the preparedness effects as reported by Cook and Mineka (1989, 1990) as well as with uniquely spontaneous and conditioned fear. In this regard, the most useful contribution of the non-associative model might be to establish an important and necessary anchor point on a fear acquisition continuum. The single most challenging task ahead is to develop a fear taxonomy that takes account of fear acquisition via multiple, interacting pathways.

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