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Fears born *and* bred: toward a more inclusive theory of fear acquisition

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Abstract

Alleged differences between associative and non-associative perspectives are sometimes more apparent than real. The non-associative model describes a pathway to fear that is complementary to associative pathways. It does not seek to usurp conditioning models as applied to evolutionary-neutral fear. We discuss vexing definitional issues surrounding what qualifies as a conditioning event and what characterises the non-associative pathway. Genetic findings are shown to be consistent with the non-associative model of fear. Following discussion of the relation between stress-diathesis models and the non-associative position we conclude by urging a developmental, life-course approach to the understanding of fear acquisition.

1. Clarification of the non-associative position

Some commentators appear to have interpreted the non-associative model of fear acquisition as rejecting associative learning models/processes. This is not the case. As we state at the very beginning of our review "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" (Abstract).

We do not dispute the relevance of conditioning explanations for understanding the acquisition of evolutionary-neutral fears/phobias nor do we suggest that such events never influence acquisition of evolutionary-relevant fears. Rather, we argue that for these innate fears, conditioning processes are not necessary for the acquisition of fear in the majority of cases. Thus, we accept

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the validity of neo-conditioning accounts of fear acquisition, as well as the importance of personality and experiential factors in the development of certain types of fear. The important difference, however, lies in our view that the non-associative pathway is the primary pathway to fear for evolutionary-relevant fears, with conditioning pathways the primary route for evolutionary-neutral fears. Consistent with recent suggestions (Poulton, Waldie, Menzies, Craske, & Silva, 2001; Poulton, Milne, Craske, & Menzies, 2001), Marks also alludes to the potential interactive nature of both processes when he states "we are not dealing with an either/or issue, but rather with a continuum of situations according to the ease with which they can become the target of fear. The question is not black or white, whether a given fear is associative or not, but rather *how much* association (learning) is needed to evoke a particular kind of fear. The less aversive pairing needed to establish that fear, the more innate it is. It is a *relative* non-association." (Marks, 2002). Nonetheless, while accepting that interactive processes may apply in some cases, we maintain that for a number of fears/individuals, no aversive pairings are required whatsoever.

The confusion about what the non-associative model entails is most evident when Mineka and Ohman conclude "..thus we do not take the non-associative account as a viable alternative to conditioning (and Rachman's three pathways) theory as a primary explanation of phobias (Rachman, 1978). At most it may be a fourth pathway to fear, albeit a problematic one from several standpoints". While disagreeing with the "problematic" assertion (see below), it was always our position that the non-associative pathway is best viewed as an additional 4th pathway to Rachmans original three pathways model (see Poulton et al., 2001a, Poulton & Menzies, 2002 this special issue).

In one sense then we are advocating a more inclusive theory of fear acquisition. To reiterate, the non-associative model does not deny the relevance of conditioning models for some fears. On the contrary, it stipulates the relevance of both associative and non-associative processes, but makes specific predictions about relevance based on the evolutionary salience of the particular fear.

2. What counts as a conditioning event?

Several authors (McNally, 2002; Mineka & Ohman, 2002) stress the need to clarify the issue of what constitutes a conditioning event. McNally puts it succintly, "Some suggest that sudden terror in the presence of the to be feared stimuli should be considered as conditioning events that explain the subsequent phobia. This formulation, of course, begs the question of why the person experienced terror in the first place…Because there is nothing akin to a US to explain this … fear, Poulton and Menzies rightly interpret these data as inconsistent with a conditioning account. Yet inexplicable terror in the presence of neutral stimuli is what gets counted as a conditioning event by conditioning theorists!".

In contrast, Mineka and Ohman suggest that "researchers in this area who follow the admonitions of Menzies and Clarke (1995a); (see also Poulton & Menzies) to adhere to their old strict textbook definitions of classical conditioning are clearly not aware of current developments in learning that render some of these definitions outmoded". In particular, they question the exclusion from conditioning categories those cases in which phobics cannot recall a specific UCS that triggered fear. Mineka and Ohman argue that such a practise excludes the possibility of interocep-

tive conditioning, thus inflating those cases categorised (falsely) into non-associative categories. Thus, Mineka and Ohman claim that "when subjects can only recall a fear/panic response (but no identifiable UCS)", one should classify them as associative cases. But where would such a practise leave the definition of conditioning? If one were to classify all such cases as necessarily associative in nature then the possibility of non-associative acquisition is eliminated entirely.

In essence, Mineka and Ohman's position would have us adopt a non-falsifiable, all-encompassing view of the associative learning of fear. What type of onset event would not be associative according to these authors? For example, consider the case of a man walking down the street who becomes distressed and experiences palpitations, for the first time, at the sight of a snake on a path some 50 yards ahead of him. No fear was apparent before sighting the snake and fear appeared to quickly arise upon seeing the animal. The individual is not aware of any other cause of the fear, and in fact directly attributes it to seeing the snake. The fear and palpitations are terminated as soon as the individual turns and leaves the scene. This is the first event in which the individual ever recalls a snake fear being evident. Now according to the practise recommended by Mineka and Ohman such an event must be classified as a conditioning onset since it is possible that the event is an example of interoceptive conditioning, with an internal (unrecognised) UCS accounting for the fear.

While we agree that such an explanation is possible, we do not believe that it is a parsimonious explanation in the present case. There is no evidence that the individual has had a spontaneous panic attack (since too few symptoms were experienced and the fear arose and was terminated by the presence and absence of a particular stimulus). There is no evidence of any other interoceptive cause of fear in this case. There is direct evidence (according to the testimony of the individual) that an external stimulus (i.e. the snake) caused the fear to arise. Since a single stimulus can hardly serve as both the CS (i.e. neutral stimulus) and the US (i.e. fear-provoking stimulus) in the one event, how can this experience possibly be regarded as an associative learning onset? Yet this is the classification that would arise from the adoption of Mineka and Ohman's recommendations.

We strongly urge against the adoption of such a non-falsifiable position. The most recent version of the Origins Questionnaire (OQ) (see further Menzies & Parker, 2001) allows for the collection of information about spontaneous panic, UCS inflation/revaluation experiences and other constructs that may play a role in the associative learning of fear from a neo-conditioning perspective. We are not adhering to "old strict textbook definitions of classical conditioning" and we dispute Mineka and Ohman's inference that we are "clearly not aware of current developments in learning". Equally, however, we will not adopt such an all-encompasing definition of conditioning that allows any event involving fear to be classified as associative learning. We strongly urge clinical researchers to attempt to take on board contemporary developments in learning theory derived from laboratory research, and examine their relevance in clinical cases. This is precisely what we have done in our most recent examination of height fear. To restate the findings of Menzies and Parker (2001), 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.

3. Genetic findings and the non-associative model

Several commentators (Davey, 2002; Kleinknecht, 2002; Mineka & Ohman, 2002; Muris, Merckelbach, de Jong & Ollendick, 2002) interpret extant genetic findings as inconsistent with the non-associative model. Kendler, Neale, Kessler, Health, and Eaves (1992) are often cited to support this position (e.g., Kleinknecht, 2002; Muris et al., 2002; Merckelbach & de Jong, 1997). However, in our review we pointed out that Kendler, Karowski, and Prescott (1999) have recently revisited this issue using a more reliable assessment. They concluded "Our results are inconsistent with two major aetiological theories of phobia acquisition: social learning and classical conditioning….By contrast, our results are supportive of an inherited phobia proneness model… which suggests that through natural selection, man has evolved an inherited predisposition to form phobic reactions to certain stimuli" (p. 550).

Nonetheless, one issue continues to create confusion. In general, reported fear heritabilities range from .50 to .60, suggesting a role for environmental influence. How does this reconcile with the non-associative model? First, the non-associative model predicts exactly this, in that the theory posits that lack of exposure to fear eliciting material early in life (Menzies & Clarke, 1995a; Poulton et al., 2001a) and/or fear dishabituation against a background of non-specific stress (Menzies & Clarke, 1995a; Poulton, Waldie, Craske, Menzies, & McGee, 2000) can prevent habituation or lead to re-emergence of a previously habituated fear. Both are clearly environmental processes. Second, as stated previously, evolutionary-relevant fears need not be exclusively acquired via the non-associative pathway, despite the hypothesised primacy of this pathway. Third, population group heritabilities of .50 conceal individual heritability scores that in theory could range from 0 to 1. This is particularly relevant when we consider how fears have been typically grouped in genetic studies. That is, evolutionary-relevant (e.g., blood-injury) and evolutionary-neutral (e.g., dental) fears have been grouped together (e.g. Kendler et al., 1999), thus potentially obscuring heritability differences between different classes of fear.

4. What counts as a non-associative fear?

A related issue to the definition of conditioning events is the definition of non-associative fear. Several commentators (Davey, 2002; McNally, 2002; Muris et al., 2002) have pointed out that attempting to reconstruct the evolutionary pressures that may have led to fear in our ancestors is surely an arbitrary, subjective undertaking. This point is made most strongly by Muris et al. who examine, for example, the case of insect fear in some detail. However, we have never argued that one can define a fear as non-associative simply because of an arbitrary Darwinian argument. In essence, we believe that a given fear should meet several criteria before it can be regarded as non-associative in nature. Ideally, these criteria would include: (1) that a plausible, evolutionary account can be developed for the fear; (2) that human retrospective evidence with clinical cases suggests that associative learning is not required for the fear to develop; (3) that human prospective evidence suggests that associative learning is not required for the fear; (4) that the fear is found in other primates and a variety of other species for whom it would have had similar adaptive advantages; (5) that a substantial genetic contribution to the fear can be found.

In the case of height fear/phobia, for example, we would argue that strong support consistent

with the first four criteria above has been presented, and that the absence of support for the fifth criterion reflects insufficient data at this point. As we have argued, height fear occurs in non-human primates and other land-dwelling animals, often on the first day of life. Retrospective and prospective human data from all studies of height fear and phobia to date strongly suggest that associative learning is not required for height fear acquisition. It appears to serve a protective purpose for the species in that adult height phobics have been shown to have experienced less severe injuries from falls across their life-course than non-fearful controls. Neo-conditioning constructs such as latent inhibition, prior fear levels, prior expectancies of harm, or reports of UCS inflation/revaluation procedures do not appear to have any explanatory power in the emergence of height fear. Finally, its adaptive value to land-dwelling animals, from a Darwinian point of view, is surely obvious and has been described by psychiatrists, developmental and clinical psychologists, biologists, ethologists and others. Given all of the above, we are surprised that Muris et al continue to describe the scientific basis of the non-associative model as "meagre".

5. Methods and mechanisms

Several commentators (Mineka & Ohman, 2002; Muris et al., 2002) suggest that we may have 'stacked the deck' in favour of the non-associative position. That is "When trying to prove something has not happened…measures that are too insensitive to register an event can provide what falsely is interpreted as support for the hypothesis that no event was present. Thus one may conclude that there is no evidence of triggering events or conditioning episodes in the life history of a phobic for the simple reason that the method one uses to assess such events lacks the power to pick them up". In this regard we draw attention to an important advantage of the longitudinal research study design. In these studies we have measured putative conditioning events (of varying degrees of severity or intensity) early in the life-course then followed up individuals at a later date to ascertain fear. Thus, the occurrence of potential "conditioning epsiodes" are not in doubt. We accept that minor events may be overlooked and where parent report is relied upon at young ages, that events may occur without parental awareness. However, because stress-diathesis principles are at the core of many neo-conditioning models (e.g. Mineka & Zinbarg, 1996; Muris et al., 2002) and these predict that (on average) the more severe the event, the greater likelihood of developing a fear, it seems unlikely that the omission of some very mild conditioning events would bias against finding a relationship between conditioning events and fear outcomes if one in fact exists.

Despite this, Mineka and Ohman complain that "…in their studies of height fear, Poulton, Davies, Menzies, Langley, and Silva (1998) operationalized exposure to a UCS as having a severe fall from a height (i.e. only those leading to a fracture, dislocation, laceration or intracranial injury). This is an overly restrictive definition because even relatively minor falls often create serious distress in children". In this study we chose to include only the most severe and consequential falls because we believed that these were the most likely to provide evidence in support of the conditioning model. When all falls were included in a re-analysis, the same results reported in Poulton et al. (1998) were obtained, with the exception that the frequency of serious *and* non-serious falls occurring between ages 5 and 9 years were no longer significantly lower among age 18 height fearfuls compared to the control group. For this analysis "All falls" comprised falls "on

or from stairs or steps; on or from ladder or scaffolding; from or out of building or other structure (balconey, bridge, window); into well; into storm drain or manhole; into hole or opening in surface (quarry, shaft, tank, pit); from playground equipment; from chair or bed; from toy (toy bike, scooter, skateboard); from one level to another; on same level from slipping, tripping, or stumbling; fall on same level from collision, pushing, shoving, by or with other person". The number of falls resulting in serious injury between birth and 5 years was n=60 (n=6.8% of the total cohort), all falls during the same period was n=153 (17.2%). Between age 5 and 9 year falls resulting in serious injury were n=62 (6.0%), versus all falls n=146 (19.0%).

Mineka and Ohman also criticise our use of a proxy (caries level) to operationalise dental conditioning history in our paper that supported a role of conditioning events in the development of dental fear (Poulton, Thomson, Davies, Kruger, Brown, & Silva, 1997). We are well aware of the potential weaknesses of this approach and were careful to describe this measure as a proxy in the original paper. Notwithstanding this potential weakness (which should have worked to reduce the likelihood of detecting any association), we reported an Odds Ratio of 4.78 (95%CI 1.72 to 13.29) that was specific to dental fear (that is, the Odds Ratio for non-dental specific fears was 0.81 (95%CI 0.56 to 1.18)) thereby *supporting* a role of putative conditioning events up to the age of 15 in the development of dental fear by age 18.

More generally, it is unclear to us why non-associative response options (e.g., always been this way) used in the retrospective studies by Menzies and colleagues are viewed as less valid than reports of associative learning. Indeed, as pointed out by McNally in the context of his recovered memory studies, there can be legitimate concerns about reports of conditioning events, "people may "recall" conditioning events that are unlikely to have occurred", a phenomenon described elsewhere as "effort after meaning" (Brown & Harris, 1978).

Mineka and Ohman also suggest that we do not "explain the transition from developmental fears to phobias" (Abstract). In their view fear dishabituation associated with non-specific stress, lack of exposure to relevant fear stimuli and poor habituation, all mechanisms we have suggested that maybe involved in this transition, "fail to address why some develop phobias — which are far more intense and disabling than most developmental fears". Instead they suggest "…that specific negative experiences are likely to play a role in this transition from developmental fears to phobias for the *small percent* of phobias that may originate from developmental fears". We accept that environmental experience can play a role as noted above (i.e. stress dishabituation, lack of exposure) and that there may indeed be other processes beyond the three we have tested that are involved in the transition from fear to phobia.

However, we were unclear to what a "small percent" refers and therefore thought it worthwhile to directly examine the strength of the association between childhood height fear (at age 11) and adult height phobia (age 26) in the Dunedin longitudinal study. Height fear at age 11 was defined as previously reported (Poulton et al., 1998). To ensure that our outcome group were truly phobic, we adopted a particularly stringent definition based on DSM-IV criteria (American Psychiatric Association, 1994). Specifically, to be defined as height phobic an individual had to report a fear of heights that was (i) unreasonable or much greater than they thought it should have been; (ii) they were very upset with themselves for having this fear; (iii) they tried hard to avoid being in their feared situation; (iv) when they had to be in the feared situation, it almost always made them extremely nervous or panicky; and (v) they would become nervous and anxious right away. As might be expected a relatively small number (n=13, 1.3%) met these strict criteria for height

phobia at age 26 years of whom n=10 also had data available about height fear at age 11. Among this group we found that height fear at age 11 significantly elevated the risk of height phobia 15 years later (OR =19.19, 95%CI: 5.00−73.69). In terms of attributable risk, 40% of those with strictly defined height phobia at age 26 also reported height fear at age 11. Thus, far from being a "small percent" almost half of those with height phobia at age 26 were fearful of heights as children.

Some response to Mineka and Ohman's interpretation of the Menzies series of retrospective studies (e.g., Menzies & Clarke, 1993a,b, 1995b) is also needed. These authors state that "the evidence cited in studies of height and water fear/phobia at best suggests that a nonassociative pathway is relevant in only a minority of cases". Mineka and Ohman seem to argue that associative pathways were more commonly reported in these studies than non-associative pathways. However, they manage to sustain such a proposition only after relabelling "non-conditioning traumatic events" as a conditioning pathway. This is consistent with their position that the absence of any evidence for an independent cause of distress (i.e. a UCS) need not worry the researcher searching for associative learning events. We have already dealt with this proposition above. In our view, simply ignoring the need to establish an alternative basis for the fear in the supposed learning event renders conditioning accounts non-falsifiable. For the record, we refute Mineka and Ohman's presentation of the Menzies retrospective data. Put simply, in each and every retrospective study of Menzies and his colleagues, non-associative modes of onset have been more frequently reported than associative pathways. Any suggestion to the contrary is simply reinventing this data.

6. Retrospective recall

Kleinkneckt emphasises the validity problems associated with retrospective accounts of fear and phobia onset. He cautions "…without corroboration, retrospective accounts should be viewed at best as suggestive and hypothesis generating rather than hypothesis supporting or disconfirming". We support this "moderate" position. In contrast Mineka and Ohman adopt a more extreme position. In their own words "The second major problem we see in studies supporting the non-associative view (although to a considerable degree the associative viewpoint as well) concerns the unreliability of retrospective recall. …This problem is acknowledged by Poulton and Menzies and by authors of many of the studies they review, but it is our belief that all these investigators seriously underestimate the degree of these problems, generally only playing lip service to them in their papers". They cite a number of papers to support the case for unreliability of retrospective recall over both short and longer time periods. While space precludes a detailed critique of all these papers, a brief comment about several references is warranted.

Monroe (1982) used a self-report life events (PERI) scale and acknowledged a number of alternative explanations for his findings including sensitisation or measurement reactivity effects occurring as a result of his study design. He concluded "A better, albeit more difficult, procedure may be to employ a structured interview; such an approach may increase reliability by helping to clarify various items, aiding in obtaining more accurate time estimates, increasing subject involvement, and so on" (p. 610). Interestingly, research on memory for life events using semi-structured interviews conducted around the same time indicated little or no fall off in the reporting

of severe events in a 12-month recall period (Brown & Harris, 1982). The importance of the type of methodology used to collect information about past events is aptly demonstrated by these two studies. It is further reinforced by Rutter, Maughan, Pickles, and Simonoff (1998) whose review of the literature (e.g., Henry, Moffitt, Caspi, Langley, & Silva, 1994; Zoccolillo, Pickles, Quinton, & Rutter, 1992; Holmshaw & Simonoff, 1995) on the accuracy of retrospective recall was reassuringly positive, especially when discrete events are enquired about and interviewer-based personalised timing approaches are adopted (e.g. Caspi et al., 1996). They summarise "Probably, recall is best when the phenomena can be assessed in terms of clearly definable events … rather than of mood states or behaviours that rely on judgments about severity in relation to other peoples behaviour" (p. 230). The study by Kendler et al. (1999) indicated moderate reliability of short-term diagnoses but only modest reliability of lifetime diagnoses. The vagaries of lifetime psychiatric diagnoses have been well reviewed elsewhere (see Simon & VonKorff, 1995). Importantly, however, in the Kendler et al. study a proportion of the unreliability in diagnoses was attributable to the unreliability of *interviewer* assessments of what constituted a phobia — not respondent memory problems.

Mineka and Ohman then question the supposed superiority of longitudinal data, "…especially unless corroborated by parental report". This is a puzzling statement given that in general our longitudinal study data derives from multiple sources (including self-, parents, teachers, significant others and official records (e.g. hospital) (see Silva & Stanton, 1996), with data sources detailed in each of the original reports. For example, in the study of height fear acquisition (Poulton et al., 1998), falls resulting in injury between birth and age 9 years were reported by parents (a reliable source for this type of data, e.g., Pless & Pless, 1995) and then checked against official hospital records where possible (e.g., Langley & Silva, 1985; Langley, Silva & Williams, 1988).

7. Vulnerability to fear and experiential factors (stress diathesis models)

A number of commentators (Davey, 2002; Mineka & Ohman, 2002; Muris et al., 2002) emphasise that certain vulnerablities or experiential factors can influence the likelihood of fear acquisition (e.g., disgust sensitivity, temperament/personality, locus of control, predictability, behavioural inhibition). We agree that some of these factors can influence fear acquisition, especially in the case of evolutionary-neutral fears as we ourselves recently reported (Poulton et al., 2001c). However, the amount and quality of the evidence in support of some diatheses is less than might be supposed. For example, Muris et al cite work by Kagan and his colleagues (e.g. Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984) to support behavioural inhibition as a risk factor for the development of later anxiety and fear. Yet more recent findings from Kagan's group do not support this. For example, "Because no high reactive infant became a consistently uninhibited child, it is more accurate to write that a high reactive temperament constrains the probability of the child becoming consistently uninhibited, rather than to claim that high reactivity determines the development of an inhibited or anxious profile." (Kagan & Snidman, 1999 p. 1540).

Muris et al also describe Gray's behavioural inhibition system (BIS) as consistent with vulnerability to childhood fear. However, their interpretation of the theory seems at odds with that of the theorists (e.g. Gray & McNaughton, 2000). According to Muris et al. (this Special Issue) "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 characterised by higher levels of aversiveness, novelty, and/or unpredictability will elicit greater BIS activity, and hence higher levels of fear." Yet according to Gray and McNaughton (2000, pp. 303–314), fear is located in the flight-fight system and not in the BIS which is associated with anxiety. Anxiety occurs in response to conflict between goals (i.e. approach avoidance conflict). According to Gray and McNaughton, heights and separation for example, are assumed to be innate fear stimuli, and hence, via uncertainty and conflict, anxiety stimuli. Specific phobias are assumed to be innate fear stimuli (usually uncompounded with conflict as they are localised and can be easily avoided). Gray and McNaughton (2000) therefore view them as innately eliciting a subliminal appropriate fear reaction. Finally, specific phobias are not sensitive to anxiolytic drugs and so are not outputs of the BIS (N. McNaughton, personal communication, 20th December, 2000).

Mineka and Ohman place a great deal of emphasis on stress-diathesis processes (e.g., see the "stress-in-a-dynamic-context-model", Mineka & Zinbarg, 1996). They explain "In all the psychological disorders that we can think of where stress plays a role in precipitating onset (ranging from schizophrenia to major depression to post-traumatic stress disorder), it is well known that stress only has this effect in people with certain biological or experiential vulnerabilities" (this Special Issue). As stated previously, from the non-associative perspective, "stressors" are not necessary for certain fears to develop. However, neither does the non-associative model exclude the possibility that such diatheses play a role in the development of some cases of evolutionary-relevant fear. Perhaps a more interesting proposition is the role that temperament might play in unlearning innate fears (see Rachman, 2002, this Special Issue). For example, is a child with an impulsive, aggressive temperament less likely to maintain height fear or separation anxiety than an inhibited or well-adjusted child? (cf. Moffitt, Caspi, Harrington, & Milne, 2001).

While accepting a potential role for such vulnerability and/or experiential factors in fear acquisition, a number of questions remain unanswered. For example, where does disgust sensitivity come from? Personality and temperament research shows moderate heritabilities (e.g., Tellegen, Lykken, Bouchard, Wilcox, Segal, & Rich, 1988; Goldsmith & Lemery, 2000). Do these factors exert their influence via genetic mechanisms (i.e., in terms of capacity for habituation) or environmental routes? Why do some individuals have little experience of control (mediated presumably via exposure) over their environment? It remains possible that non-associative mechanisms (e.g. fear dishabituation associated with non-specific stress, limited exposure to fear relevant stimuli early in life; poor habituation capacity) may be implicated in these processes.

8. Summary

We are not as pessimistic as some commentators about the value of retrospective and prospective approaches for increasing our understanding of the fear acquisition process. Pointedly, McNally reminds us of the need for ecologically valid research due to difficulties generalising from the laboratory to real world settings (McNally, 2001). We urge the adoption of a developmental, life-course perspective to fear etiology that uses multiple-source data wherever possible. Mapping the interaction of multiple influences along the life-course is the greatest challenge. In this regard "critical periods" (e.g., Kuh & Ben-Shlomo, 1997), and processes such as reactive, evocative and proactive interaction, and heterotypic and homotypic continuity (e.g., Caspi & Mof-

fitt, 1995;) are likely to be relevant in the pursuit of a deeper understanding of how fear is "learned and unlearned" (see Rachman, 1978, pp 254–256). The point made by Rachman in the Introduction to this Special Issue is also noteworthy in this regard, "Acceptance of the existence of non-associative fears would lead to a revival of interest in how and when children learn to not-fear". The non-associative model specifies that learning-environmental processes are relevant to the understanding of fears — both born and bred. Consistent with this, we urge a more inclusive view of fear acquisition, however one that nevertheless retains parsimony and is capable of generating refutable predictions. An expanded, four-pathway model that takes account of the modifying/mediating influence of a variety of background factors appears to be the best model currently available.

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