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# The development of anxiety disorders in childhood: an integrative review

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We present an integrative review of the development of child anxiety, drawing on a number of strands of research. Family aggregation and genetic studies indicate raised vulnerability to anxiety in offspring of adults with the disorder (e.g. the temperamental style of behavioural inhibition, or information processing biases). Environmental factors are also important; these include adverse life events and exposure to negative information or modelling. Parents are likely to be key, although not unique, sources of such influences, particularly if they are anxious themselves. Some parenting behaviours associated with child anxiety, such as overprotection, may be elicited by child characteristics, especially in the context of parental anxiety, and these may serve to maintain child disorder. Emerging evidence emphasizes the importance of taking the nature of child and parental anxiety into account, of constructing assessments and interventions that are both disorder specific, and of considering bidirectional influences.

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## Introduction

Anxiety disorders in childhood and adolescence are common and disabling (Costello *et al.* 2004). They often run a chronic course and are associated with the development of other disorders, such as depression, conduct disorder or attention deficit disorder (Bittner *et al.* 2007). There have been several strands of research into the development of child anxiety. First, research has concerned vulnerability factors, especially biological characteristics (genetic, temperamental), and information processing styles. Second, research has concerned environmental influences that may increase risk for disorder, either alone or in combination with some vulnerability factor. These include adverse life events and learning routes, such as modelling and information transfer. This review considers these vulnerability and environmental factors, paying particular attention to the role of parents, who are likely to be key sources of at least some of these influences. Notably, recent research shows the importance for understanding the development and maintenance of child anxiety of integrating evidence concerning child vulnerability with that on parenting, and highlights the potential bidirectionality of child and parental influences. The review concludes with a schematic

representation of this integration and a brief discussion of directions for future research and treatment.

## Vulnerability factors

### *Family aggregation and genetics*

The familiarity of anxiety disorders found in adults (Hettema *et al.* 2001) has also been found in inter-generational studies of family aggregation. Thus, children whose parents have anxiety disorders exhibit an increase of anxiety disorders over the base rate themselves (Weissman *et al.* 1984; Turner *et al.* 1987; Biederman *et al.* 1991; Warner *et al.* 1995; Mancini *et al.* 1996). Similarly, parents of children with anxiety disorders have a raised rate of anxiety disorders compared to control parents (Last *et al.* 1987, 1991; Cooper *et al.* 2006), with the elevation of risk largely confined to mothers (Cooper *et al.* 2006).

The extent to which aggregation is accounted for by genetic transmission is uncertain because research has seldom involved samples of children with actual anxiety disorder, but instead has reported genetic effects on anxiety symptoms or certain endophenotypes, such as behavioural inhibition (discussed later). These studies consistently reveal a genetic influence of moderate magnitude (Gregory & Eley, 2007), but one that varies depending on the child's sex (e.g. Eaves *et al.* 1997; Feigon *et al.* 2001) and, possibly, on age (e.g. Feigon *et al.* 2001). The magnitude of effects is also influenced by the information source: genetic influences

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are more significant, and shared environment less so, where the child anxiety is parent rated, and the reverse is the case where self-rated (Thapar & McGuffin, 1995; Eaves *et al.* 1997). The extent to which studies have implicated shared and non-shared environmental factors has also varied as a function of the form of anxiety, although findings are inconsistent (Silove *et al.* 1995; Eaves *et al.* 1997; Legrand *et al.* 1999; Feigon *et al.* 2001; Eley *et al.* 2003; Ehringer *et al.* 2006).

Most of the search for a genetic substrate to child anxiety has centred around a functional polymorphism in the promoter region of the serotonin [5-hydroxytryptamine (5-HT)] transporter (5-HTT) gene. A meta-analysis of studies involving adults found that the short allele was associated with a predisposition to anxiety and negative emotionality (Munafo *et al.* 2003); and it has also been found to be associated with increased amygdala activation to presentation of fearful faces (Hariri *et al.* 2002). Studies involving children have produced a more mixed picture. Thus, in one study no relationship was found between the 5-HTT gene and the fearful temperamental style of behavioural inhibition (Schmidt *et al.* 2002); and whereas shyness was found to be associated with the long form of the 5-HTT gene in one study (Arbelle *et al.* 2003), in another it was associated with the homozygous short-short 5-HTT allele (Battaglia *et al.* 2005). This confused picture may partly be a function of looking for simple associations, when gene-pathology relationships are in fact moderated by other factors. Thus, some evidence suggests that 5-HTT status interacts with environmental factors to confer risk for the development of depression (Caspi *et al.* 2003; Kaufman *et al.* 2004). The same seems to be true for social anxiety: Fox *et al.* (2005) found that, controlling for temperamental status at age 4 years, the presence of the short 5-HTT allele significantly raised the risk of both objectively assessed behavioural inhibition and maternal reports of child shyness in 7-year-olds, but only where there had been reduced levels of social support. The significance of interactions of this sort is taken up below.

Familiality is therefore strong in child anxiety disorder, although the evidence for significant heritability is meagre, and the genetic substrate is poorly understood.

### **Behavioural inhibition (BI)**

The term 'behavioural inhibition' (BI) has been used by Kagan (1989) and others to refer to a child temperamental pattern of responding characterized by fearfulness, reticence or restraint when faced with unfamiliar people or situations. In longitudinal research, around 50% children have been found to maintain their early BI classification through childhood (Kagan

*et al.* 1988; Kagan, 1994; Turner *et al.* 1996) and adolescence (Kagan *et al.* 2007), with those classified as inhibited in infancy tending to show greater continuity than those who were uninhibited. Strong associations have been found between BI in children and anxiety disorders in parents (e.g. Rosenbaum *et al.* 1991; Rickman & Davidson, 1994), and between anxiety disorders in parents and BI in children (e.g. Rosenbaum *et al.* 1988, 2000; Biederman *et al.* 2001a). Furthermore, in longitudinal research, early BI has been found to predict later frank anxiety disorder (Turner *et al.* 1996), especially of the social form (Hayward *et al.* 1998; Schwartz *et al.* 1999; Biederman *et al.* 2001a). However, despite this clear association between BI and anxiety disorder, it remains the case that only a proportion of those who are inhibited do go on to develop an anxiety disorder, and it is important to consider what factors might account for continuities and discontinuities (see below).

### **Information processing**

It has been suggested that vulnerability to the development of anxiety may be reflected in biased information processing, in both attentional bias in relation to threat and biases in interpretation of ambiguous material (Rapee, 2001; Hudson & Rapee, 2004; Rapee & Spence, 2004). Attentional biases have certainly been identified in older anxious children (see review by Hadwin *et al.* 2006), although in younger children the evidence is less consistent (Martin & Jones, 1995; Kindt *et al.* 1997). However, these studies have been cross-sectional, with already anxious subjects, and therefore cannot elucidate whether these biases represent vulnerability. One recent prospective study of infants of socially anxious mothers did find evidence for sensitivity to fearful faces, consistent with the possibility of a fundamental cognitive vulnerability (Creswell *et al.* 2008c); however, further prospective enquiry is required to establish the reliability of this finding and its longer-term clinical significance.

The evidence for interpretation biases in child anxiety is strong and consistent (Hadwin *et al.* 2006); in the main, however, the studies have also been cross-sectional, and thus their role as vulnerability factors is not clear. Two prospective studies do provide some indication that interpretation bias may predate anxiety: Warren *et al.* (2000) found preschool-age children's negative story endings predicted anxiety 1 year later, controlling for earlier anxiety. Similarly, Creswell (2004) found children's negative interpretations of ambiguous stimuli predicted increased anxiety symptoms following transition to secondary school.

Although the evidence for information processing biases predating the onset of anxiety is scarce,

identifying children vulnerable because of particular cognitions is a promising line of enquiry that could open up possibilities for preventive intervention.

## Environmental influences

### *Life events and difficulties*

Investigations of the role of adverse life events in the aetiology of childhood anxiety disorders have been influenced by the associative account, which suggests that fears and phobias are classically conditioned anxiety responses; that is, they develop from the paired association of a neutral stimulus with a traumatic event. Studies of the role of distinct, traumatic events have generally lacked control groups and been retrospective and therefore subject to 'plaintive bias' (Kendler *et al.* 2000); and although many people with a phobia do attribute onset to specific events (e.g. Ollendick & King, 1991; Muris *et al.* 2002), many do not (e.g. Menzies & Clarke, 1995). Similarly, many individuals experience a trauma but do not develop an anxiety disorder (e.g. Rachman, 1990). To account for such variability, contemporary learning accounts incorporate the role of pre-existing individual vulnerabilities, previous learning experiences and stressors, in addition to post-stressor experience (e.g. Ollendick, 1979; Beck, 1986; Barlow, 1988; Rapee, 2001; Muris *et al.* 2002).

With regard to more general life events, some prospective studies provide evidence consistent with their causal role. The study of Goodyer *et al.* (1988) found that, compared to non-anxious children, those with anxiety experienced more negative events in the 12-month period preceding onset of disorder. Similarly, Phillips *et al.* (2005) found difficulties experienced in the first 5 years (e.g. a mother's change of partner) to predict anxiety disorder in adolescence, even controlling for current adversity. Nevertheless, studies also show reciprocal influences: thus, Swearingen & Cohen (1985) found that child distress (anxiety or depression) predicted occurrence of subsequent adverse events, including those objectively rated as 'uncontrollable'. Research over a longer time-scale by Kim *et al.* (2003) similarly showed that, although life events increased the risk of internalizing problems, the latter then predicted further negative experiences.

Some studies suggest it is important to consider the type of event experienced. Eley & Stevenson (2000) found, consistent with adult data (Finlay-Jones & Brown, 1981), that trait anxiety in school-aged children was specifically associated with threat events; and Shanahan *et al.* (2008) found that living in a dangerous neighbourhood and parental drug use were specific risks for child anxiety disorder.

The processes whereby the experience of negative life events increases risk for disorder are likely to be multi-faceted. Early exposure to adversity may affect the child's developing physiological systems [e.g. the hypothalamic-pituitary-adrenal (HPA) axis (Phillips *et al.* 2005)] and the development of child cognitions, particularly concerning threat and control (Chorpita & Barlow, 1998); and its effects may be mediated by parental perceptions of child vulnerability and consequent alterations in parenting practice (Creswell *et al.* 2008c). Finally, the association may be accounted for by some third variable (e.g. parental psychopathology).

### *Modelling and information transfer*

In addition to the role of traumatic events, learning accounts have emphasized two further routes for the development of anxiety. The first concerns the child's observation of others' anxiety, and is generally referred to as a modelling or a vicarious acquisition process. The second concerns communication to the child of information regarding the threatening properties of the environment, usually termed informational acquisition (for reviews see Bandura, 1986; Rachman, 1990; King *et al.* 1998; Muris *et al.* 2002; Field, 2006; Mineka & Zinbarg, 2006; Fisak & Grills-Tauchel, 2007). In the course of naturally occurring social interactions, these two processes are likely to overlap, but for clarity we distinguish them here.

### *Modelling*

In the modelling account of the development of anxiety, animal experiments, most notably those of Mineka and colleagues with rhesus monkeys, clearly show how persistent fears may develop following observation of others' fearful responses (Cook & Mineka, 1987), with the extent of fearfulness varying as a function of the degree of fear modelled (Mineka, 1985). Notably, these studies show the effect to be restricted to stimuli for which a fearful response is evolutionarily advantageous, where there is a 'preparedness' for fear (e.g. for snakes but not flowers; Mineka, 1988). Individual differences in susceptibility are also found, with modelling effects attenuated by previous experience of mastery or control (Mineka *et al.* 1986).

Evidence from human studies confirms the importance of these processes. Clinical accounts of World War II experiences document, for example, how the display of fear by mothers during air raids determined whether children expressed similar fears (Lewis, 1942). Retrospective studies by adults with a range of phobias document modelling by others as having been a significant influence, particularly for animal phobias,

blood phobia, social phobia and panic (see reviews by Mineka & Zinbarg, 2006; Fisak & Grills-Taquechel, 2007). Mothers' own reports of their manifest expression of fearfulness have also been found to be associated with child specific fears (Muris *et al.* 1996) and post-traumatic stress disorder (PTSD) symptoms (McFarlane, 1987).

More recently, researchers have directly observed the effect of parental modelling on child fearful responses. Thus, Gerull & Rapee (2002) showed, in a non-anxious sample, how maternal modelled fear and disgust to potentially fear-provoking objects led to the expression of fearful and avoidant responses in 15- to 20-month-olds. Similarly, de Rosnay *et al.* (2006) showed that maternal modelling of anxious *versus* non-anxious social responses to a stranger determined infants' subsequent expression of fear to the same unfamiliar adults. This process was recently examined longitudinally in an anxious population. Mothers with social phobia, and non-anxious control group mothers, conversed with a stranger while their infant watched, and the infant's response to the stranger was rated. Maternal expressed anxiety at 10 months, which was, as expected, more prominent in the social phobia group, predicted increased infant avoidance of the stranger at 14 months (Murray *et al.* 2008).

In sum, there is strong laboratory evidence that modelling plays an important causal role in the intergenerational transmission of anxiety, although the manner in which this process operates naturalistically remains to be elucidated.

#### *Information acquisition*

The role of providing information conveying threat in the development of child fears and avoidance has been examined in a series of experimental studies with normal populations of 6- to 13-year-old children by Field and colleagues (Field & Lawson, 2003; Field *et al.* 2003; Lawson *et al.* 2007). Information that was either positive, negative or neutral was presented to children concerning novel non-social stimuli (e.g. fictitious animals) and various social situations. There were consistent findings of the impact of negative information on child fear and avoidance, and also evidence for the persistence of effects over several months. Moreover, the nature of information delivery, its source, and also previous experience all influenced the impact, suggesting that relatively complex processes are involved.

Learning accounts of the development of child fears and anxiety have consistently identified parents as playing a particularly important role through instruction and information transfer (Hadwin *et al.* 2006). Indeed, a large body of research with normal populations has shown how, through tuition and

spontaneous conversations, parents both consciously and unconsciously transmit evaluative cognitions to their child (Fivush, 1991; Nelson, 1993; Denham *et al.* 1994), with systematic associations being found between the nature of parental discourse and children's own cognitive and socio-emotional functioning (Dunn *et al.* 1991; Fivush & Vasudeva, 2002). Observational studies of anxious populations provide some evidence for these processes in the transmission of disorder, with distinctive features of parent-child conversations being identified. Thus, in one study by Whaley *et al.* (1999) and Moore *et al.* (2004), anxious parents were found to make more catastrophizing comments to their children than non-anxious parents. Suveg *et al.* (2005) found that parents of anxious children were less likely to refer to positive emotions than were parents of non-anxious children. Barrett and colleagues found that, following parent-child discussion, anxious children became more likely to choose avoidant solutions to hypothetical threats (Barrett *et al.* 1996; Dadds & Barrett, 1996).

Experimental studies indicate that information transfer is relevant to the transmission of anxiety, and observational evidence is consistent with this process in intergenerational transmission.

#### *Other parental influences: warmth and control*

In addition to parents' modelling of anxious responses, and their provision of information to the child that may directly induce fear and anxiety, a principal focus for research on the role of parent-child relationships in the development of child anxiety has been parental lack of warmth or rejection, and parental overcontrol. Lack of parental warmth has been considered important because it may lead to the child's believing that the environment is fundamentally hostile and threatening, and to a sense of low self-worth and competence (Parker, 1983; Bogels & Tarrier, 2004). With regard to parental overcontrol, a parent's excessive regulation of the child's activities and routines, and their discouragement of independence, is likely to promote a limited sense of competence and mastery, and may serve to reinforce child avoidance of challenge (Parker, 1983; Chorpita & Barlow, 1998). Three reviews of parenting (Wood *et al.* 2003; DiBartolo & Helt, 2007; McLeod *et al.* 2007) conclude that the evidence for parental lack of warmth being associated with child anxiety is inconsistent, whereas that for parental overcontrol is stronger. One of these reviews (McLeod *et al.* 2007), a quantitative meta-analysis, is notable in having examined the role of methodological factors (e.g. informant, assessment method), whether child anxiety diagnoses or else anxious/fearful symptoms were assessed, and subdimensions of the two

parenting constructs. This review showed that effects were strongest where direct observations were made of parenting in contrast to questionnaire or interview assessment, and for diagnosed child anxiety rather than symptoms. With regard to parenting dimensions, overall effect sizes for rejection and control, across all types of study, were only 0.20 (i.e. accounting for 4% variance in child outcome) and 0.25 (6% variance) respectively. However, subdimensions were important to consider; thus, although the effect size for warmth was just 0.06, effect sizes for overinvolvement, aversiveness and withdrawal were 0.22–0.23, and that for autonomy granting was 0.42.

Although research interest in parenting outlined above has largely focused on closely observed parent–child interactions and the assessment of proximal parenting processes, it is important to note that wider parenting practices are also likely to be implicated in the development of child anxiety. Thus, parents' decisions concerning, for example, the use of day-care, and family socialization and recreational activities, all stand to enhance or else limit child exposure to potential challenges beyond the home, and thereby the opportunity to develop coping skills (see reviews by Chorpita & Barlow, 1998; Bogels & Brechman-Toussaint, 2006).

In sum, a range of parenting practices have been considered as relevant to child anxiety; of these, lack of autonomy granting seems to be particularly important at the micro-level of observed parent–child interactions and also wider, more general parenting practices.

#### *Further issues in parenting*

Two further issues regarding parenting effects are important to consider, namely the direction of effects and the specificity of effects.

##### *Direction of effects*

Although certain parenting practices have been found to be more common in the context of anxiety, the question arises of whether these are (i) a function of the parents who, as the family aggregation studies show, are themselves frequently anxious, or (ii) generated as a response to having an anxious child, or else (iii) result from some interaction between parent and child characteristics. This question can be addressed only in studies that include assessment of both parent and child. Such a design has seldom been used. Moore *et al.* (2004) observed parent–child interactions in four groups: anxious and non-anxious mothers with either anxious or non-anxious children. They found both maternal warmth and autonomy granting to show a main effect of child diagnosis, with no effect

of the mother's anxiety status. For 'catastrophizing', although there was a main effect of maternal diagnosis, within the group of non-anxious mothers those with anxious children were significantly more likely to show this behaviour than those of children who did not have anxiety disorder. This suggests that child influences on parenting are as important to consider as parental characteristics.

Studies using experimental manipulations, longitudinal designs, cross-over between parents and children, or those including sibling comparisons, are also potentially informative regarding the question of the contributions of parent and child characteristics to parenting quality. The influence on parenting of the parents' prior experience of their child is apparent from the study of Dumas & LaFreniere (1993), who found that mothers of anxious children interacted more negatively than mothers of other groups of children (e.g. competent or aggressive) when with their own child, but more positively when with another anxious child, suggesting that their capacity to behave positively was intact but was compromised by the history of the relationship with their child. The studies of anxious children by Hudson & Rapee (2002) and Barrett *et al.* (2005), which used sibling controls, found that mothers behaved similarly towards their two children, and were more overinvolved/controlling with both of them than were comparison mothers with their non-anxious children. However, although these findings were interpreted as showing parenting to be more a function of maternal rather than child characteristics, it should be noted that the anxious children's siblings often had anxiety symptoms themselves (Hudson & Rapee, 2002), and did not differ in their behaviour from the anxious child (Barrett *et al.* 2005), leaving open the possibility of child effects being important. Thus, although sibling studies are useful in disentangling the relative contributions of different individuals, it is important that full account is taken of participant characteristics.

Several studies, typically with young children or infants, have taken child temperament or BI into account when examining parenting. It seems that, among anxious, but not non-anxious, parents, infant or child inhibition may provoke parenting styles that contribute further to child anxiety. For example, Hirshfeld *et al.* (1997) found that maternal criticism of the child was a function of an interaction between child inhibition and maternal anxiety status; that is, anxious mothers of inhibited children were likely to be critical (65% were so), whereas those with non-inhibited children were not (18%). Among non-anxious mothers, by contrast, child inhibition was unrelated to maternal criticism. The study of Murray *et al.* (2008) produced similar findings: mothers with

social phobia showed low levels of encouragement to their infants to engage with a stranger *only* if their infant was behaviourally inhibited, whereas among non-anxious mothers, the opposite was true. This suggests that, whereas non-anxious mothers respond to their inhibited child by facilitating the child's positive engagement with potential challenges, anxious parents respond to child inhibition by encouraging their child to avoid potential threat.

In fact, longitudinal evidence suggests that infant or child BI is likely to be a predictor of subsequent child anxiety only when accompanied by problematic parenting styles. Thus, in the study of Murray *et al.* (2008), the development of avoidance of strangers by inhibited infants of mothers with social phobia was accounted for by their mothers' failure to encourage infant social responsiveness. Similarly, Rubin *et al.* (2002) found that children who were inhibited at age 2 years only became socially reticent by 4 years if their mothers had initially been controlling (intrusive or overprotective) or derisive towards their child. Warren & Simmens (2005) also found, in a large epidemiological sample, that infants rated as having a difficult temperament at 6 months were only likely to develop symptoms of anxiety and depression at 2 years if their mother was insensitive. Aside from such proximal parenting influences, wider socialization experiences also seem to influence the trajectory of early inhibition. Both Furman *et al.* (1979) and Fox *et al.* (2001) found that the development of infants who showed early vulnerability for inhibited or withdrawn behaviour was positive if the child experienced opportunities for socializing with others (e.g. through day-care).

Thus, although neither BI nor parenting factors have emerged as strongly associated with child anxiety when considered singly, their co-occurrence does seem to be of considerable aetiological significance; in particular, parents who are themselves already anxious are likely to respond to child vulnerability with parenting practices that may further reinforce the child's difficulties.

#### *Specificity of parenting effects*

Two questions arise regarding specificity of effects: first, whether particular parenting characteristics are uniquely associated with child anxiety disorders, rather than with more general child psychopathology; and second, whether some parenting characteristics are specifically related to subtypes of both parent and child anxiety disorder. In general, research on risks for childhood anxiety has not addressed these issues, and studies comparing the parenting of anxious children with that of children with other disorders have

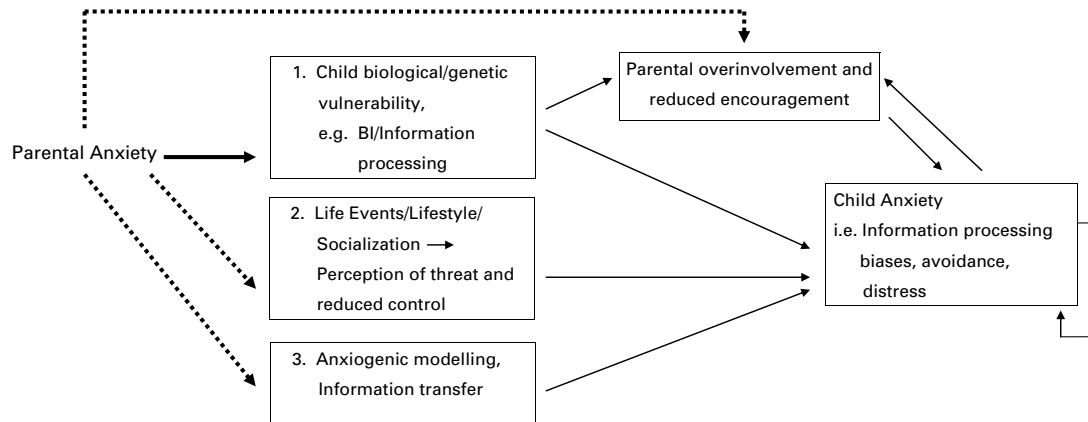
produced inconsistent findings. For example, compared to mothers of aggressive children, those of anxious children have been found to be both more negative (Dumas & LaFreniere, 1993; Dumas *et al.* 1995), less negative (Stubbe *et al.* 1993), and indistinguishable in terms of negativity (Hudson & Rapee, 2001).

Given the extent of co-morbidity between childhood disorders, it is particularly important to take different child symptom profiles into account when addressing the issue of specificity. Both the Virginia Twin Study (Kendler *et al.* 2000) and the Great Smoky Mountain epidemiological study (Shanahan *et al.* 2008) are notable for having used multiple informants, and for having carefully controlled for co-morbid disorders. The former showed cold, overprotective and authoritarian parenting all to contribute to offspring anxiety, over and above their impact on other child diagnoses, whereas the latter suggested overintrusive parenting may be specifically associated with anxiety disorders rather than conduct disorder, oppositional defiant disorder (ODD) and depression.

Little evidence exists concerning specificity in the effects of parenting within the anxiety disorders. Indeed, a notable feature of research into parenting effects is that different anxiety disorders in both child and parent populations have generally been grouped together. Furthermore, the conditions for assessing parent-child relationships have often been non-specific, obscuring parental and child difficulties that might be elucidated only in contexts that are disorder relevant. Two recent studies have sought to address these issues. Murray *et al.* (2007) found that, compared to non-anxious control mothers, those with social phobia, but not those with generalized anxiety disorder (GAD), showed parenting difficulties in the context of a mild social challenge (i.e. interacting with an unfamiliar person) and their infants were similarly specifically affected. Wood *et al.* (2006) compared parenting in groups where children had either separation anxiety disorder (SAD) or other anxiety disorders (social phobia or GAD), in a task designed specifically to elucidate parental overprotection because child dependency on the parents promoted by this behaviour was hypothesized to be a specific risk for SAD. In line with predictions, overprotection was significantly correlated with SAD but not with other disorders.

Both the Smoky Mountain and the Virginia twin studies also examined the question of specificities in parenting within the anxiety disorders. Harsh discipline was associated with GAD in the Smoky Mountain study, as was the similar dimension of authoritative parenting in the Virginia study. However, in the latter overprotection was also important; in the Smoky Mountain study, no other parenting dimension





**Fig. 1.** Pathways to child anxiety. —→, Parental anxiety accounts for the factor; - - -→, Parental anxiety raises the risk of the factor. BI, Behavioural inhibition.

contributed significantly to social phobia or child SAD, over and above their contribution to other child disorders, whereas in the Virginia study, all three parenting dimensions assessed made significant contributions to phobias (including social phobia), independently of effects on other disorders.

In sum, although few studies have addressed the issue of specificity, a body of evidence is beginning to emerge implicating particular styles of parenting in particular forms of child anxiety.

### Summary and directions for future research

Taken together, the evidence reviewed here suggests an interplay between several factors in the development of child anxiety, as illustrated in Fig. 1.

First, family aggregation and genetic studies indicate raised vulnerability to anxiety in offspring of adults with the disorder. Such vulnerability may be manifest in terms of behavioural inhibition, and it is possible that particular information processing biases also represent genetically mediated vulnerability. Parental anxiety may also place offspring at increased risk for disorder through exposure to negative life events, which may directly increase the risk of child anxiety, or do so through diminishing the child's sense of control. In addition, anxious parents may, as a function of their own disorder, place limits on their children's wider experiences, thereby restricting their opportunities to encounter and tackle challenging circumstances. Research on parenting suggests that certain parenting styles associated with child anxiety occur directly as a function of parental anxiety disorder, most notably behaviourally expressed anxiety and anxious cognitions such as catastrophizing and overattribution of threat, and these may serve to promote child anxiety through learning processes of modelling and information transfer. Although the role

of parental anxiety in conferring risk to the child has been emphasized, it is important to note that child anxiety can occur in the absence of parental disorder, and that risk factors (e.g. the occurrence of adverse events, provision of negative information) may impact on the child independently of parental behaviour. Furthermore, some parenting dimensions associated with child disorder seem to be provoked by particular child characteristics, such as BI or, indeed, child anxiety itself. These include behaviours such as overinvolvement or overprotection, lack of encouragement or autonomy promotion, and negative or aversive behaviour. Notably, such behaviours are especially likely to arise in parents who are themselves prone to anxiety. These parenting styles, initiated by the child's behaviour, also play a role in maintaining child inhibition and anxiety. This being so, treatment research manipulating different parenting dimensions stands to make a significant contribution to the understanding of mechanisms influencing the maintenance of child disorder, in addition to the improvement of child outcome. Finally, little research has so far directly addressed the question of the specificity of parenting styles relevant to different child anxiety disorders; nevertheless, emerging evidence of specificity of effects suggests that it will be important in future studies to control adequately for the nature of both child and parent anxiety, and to design observational frames, coding schemes and interventions that are relevant to the disorder in question.

The proposed model has potential implications for the prevention and clinical management of child anxiety disorders. Although individual vulnerability factors (e.g. BI) may not be strong predictors of later anxiety disorder, identifying children who experience a combination of vulnerability factors (e.g. high BI and 'anxiogenic' parenting) may allow effective targeting of preventive interventions. Furthermore, the model

identifies key parenting practices that are likely to require specific intervention (e.g. reduced encouragement). The presence of high parental anxiety is likely to be a barrier to optimal child treatment outcome (e.g. Cobham et al. 1998; Creswell et al. 2008c); however, whether it is necessary to address parental anxiety itself or whether it is sufficient to deal only with the parenting consequences of that anxiety requires further empirical investigation.

### Declaration of Interest

None.

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