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Family Factors in the Development and Management of Anxiety Disorders

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Abstract

Family variables are thought to play a key role in a wide variety of psychopathology according to many theories. Yet specific models of the development of anxiety disorders place little emphasis on general family factors despite clear evidence that anxiety runs in families. The current review examines evidence for the involvement of a number of family-related variables in the development of anxiety disorders as well as the importance of families in their management. Evidence across most areas is shown to be weak and inconsistent, with the one exception being an extensive literature on the role of parenting in the development of anxiety. There is also currently little evidence that family factors have a strong role to play in treatment of anxiety, aside from research demonstrating the value of parents and partners as noncritical supports in therapy. The promises and hints in the literature, combined with the currently inconsistent methods, suggest that considerably more research is needed to determine whether specific family factors may yet be shown to play a key role in the development and management of anxiety disorders.
Family Factors in the Development and Management of Anxiety Disorders

Family Factors in the Development and Risk for Anxiety

Family Transmission

There is little doubt that anxiety runs in families. A large number of studies has shown evidence for higher levels of anxiety and anxiety disorders among first degree relatives of people with anxiety disorders (Hettema, Neale, & Kendler, 2001). This family concordance has been shown for adults with anxiety disorders (Fyer, Mannuzza, Chapman, Martin, & Klein, 1995; Stein et al., 1998), children with anxiety disorders (Last, Hersen, Kazdin, Orvaschel, & Perrin, 1991; Lieb et al., 2000), and people high on trait anxiousness (Jardine, Martin, & Henderson, 1984).

Hettema and colleagues (2001) conducted a meta-analysis of family concordance for anxiety disorders, primarily focused on clinical populations. Based on very careful study selection criteria, they concluded that there is substantial family concordance for panic disorder, generalized anxiety disorder, obsessive compulsive disorder and the phobias (including social phobia, agoraphobia, and specific phobias). Across anxiety disorders they estimated that for a given proband with an anxiety disorder the odds of having a first degree relative with an anxiety disorder is around 4-6.

Since this review, several population level studies have supported these conclusions. For example, a data linkage study of over 20,000 people in the Danish population with a psychiatric record indicated an individual with an anxiety disorder was 6.8 times more likely to have a first degree relative with another disorder (Steinhausen, Foldager, Perto, & Munk-Jorgensen, 2009). Similar probabilities were demonstrated regardless of whether the relative was a parent, sibling or child of the proband. A similar population data linkage study in
Sweden looked at parent-child concordance (Li, Sundquist, & Sundquist, 2008). The risk of a child having an anxiety disorder if their parent had an anxiety disorder was around 2, regardless of whether the parent with anxiety was the mother or father. If both parents had an anxiety disorder, the risk increased to 5.1. Although these studies utilise clinical diagnoses that are potentially unreliable and restrict themselves to the relatively small proportion of anxiety sufferers who seek professional help, their impressive sample sizes provide clear support to the more carefully conducted earlier family studies (Hettema et al., 2001).

One of the key questions concerning family transmission is the specificity of these effects. In other words, does having a relative with an anxiety disorder increase the risk for the same specific disorder, any anxiety disorder, or any psychopathology? Fewer studies have addressed these questions, but those that have indicate a moderate degree of specificity within particular anxiety disorders, although results are not entirely consistent. One of the only studies that examined this issue across a range of anxiety disorders compared diagnoses in first degree relatives of probands with social phobia, agoraphobia, simple phobia and controls, based on DSM-III-R criteria (Fyer et al., 1995). Specific family transmission was demonstrated – probands with each of the anxiety disorders had increased odds to have relatives with the same anxiety disorder but not with other anxiety disorders. Similar specificity has been shown in a number of other studies for social phobia (Coelho, Cooper, & Murray, 2007; Low, Cui, & Merikangas, 2008; Stein et al., 1998), panic disorder (Low et al., 2008; Mendlewicz, Papadimitriou, & Wilmotte, 1993; Noyes et al., 1986), and OCD (Carter, Pollock, Suvak, & Pauls, 2004; Fyer, Lipsitz, Mannuzza, Aronowitz, & Chapman, 2005). Studies on the familial aggregation of GAD have shown less clear specificity (Beesdo, Pine, Lieb, & Wittchen, 2010; Coelho et al., 2007; Mendlewicz et al., 1993), perhaps due to the lower diagnostic reliability for this disorder.
Clearly the family aggregation of anxiety disorders reflects a strong genetic penetrance. Twin studies estimate the heritability for anxiety at around 30-40% or higher (Hettema et al., 2001). However, few twin studies estimate marked variability due to disorder-specific genes, but instead conclude that the majority of the genetic influence is general across anxiety and even related disorders (Gregory & Eley, 2007). This discrepancy between the conclusions from family and twin studies is intriguing and might point to a general heritable component to anxiety that is shaped into specific disorders by family-related variables. In contrast to such a conclusion, data from twin studies indicate little if any variance in anxiety is attributable to common environmental components (such as family factors), at least for adult anxiety disorders (Hettema et al., 2001). For child anxiety disorders, the data are less clear and several studies attribute significant variance to influence from the common environment (Eley et al., 2003; Feigon, Waldman, Levy, & Hay, 2001; Topolski et al., 1997). Further a main effect of either genetic or environmental factors is highly improbable for any disorder and it is far more likely that anxiety disorders are a product of complex gene-environment correlations and interactions, which are very hard to model from twin data (Eley & Lau, 2005). What appears to be true is that there is a strong genetic basis to anxiety disorders that is nevertheless weaker than for some disorders such as schizophrenia or bipolar disorder. There may also be some influence from shared environmental and family factors, but this is likely to be considerably weaker than for certain disorders such as externalizing disorders or substance abuse and almost certainly interacts with the child’s genetic makeup. There has also been considerably less research into environmental contributors to anxiety disorders than for many other disorders. The remainder of this paper will summarise some of the limited evidence on the association between family-related variables and anxiety disorders. Given that anxiety disorders are chronic problems that commonly begin in childhood or adolescence, family factors during childhood are likely
to have the greatest (if any) influence. Parents and other family members are also likely to be
more influential in the development of psychopathology and related behaviour during the
childhood years. Therefore the majority of relevant research and the majority of this review
will focus on family influence on childhood anxiety. However, family support and related
factors during adulthood may play a role in the maintenance of the disorder and also need to
be evaluated.

Family Demographics

Perhaps surprisingly, there is little evidence that family demographic factors including
family size, composition, birth order, or living circumstances are strongly associated with
anxiety. Several large epidemiological studies have failed to report consistent relationships
between diagnoses of anxiety disorders in childhood and demographic factors such as these
(Canino et al., 2004; Ford, Goodman, & Meltzer, 2004; Lewinsohn, Hops, Roberts, Seeley, &
Andrews, 1993). The only exception is data that suggest a possible relationship between low
socioeconomic status and the existence of anxiety in childhood, although results have not
been completely consistent (Cronk, Slutske, Madden, Bucholz, & Heath, 2004; Ford et al.,
2004; Medina-Mora et al., 2005; Xue, Leventhal, Brooks-Gunn, & Earls, 2005). Support for
this relationship has also been shown in one longitudinal study that demonstrated that low
socioeconomic status during childhood assessed by maternal and paternal employment level
was a significant predictor of a diagnosis of generalised anxiety disorder in adulthood
(Moffitt et al., 2007).

As mentioned, there is little evidence that other demographic factors increase risk for
anxiety. Consistent evidence indicates that adults with anxiety, especially social phobia, are
less likely to be married or in a regular relationship than non-disordered populations (Hunt,
Issakidis, & Andrews, 2002; Lampe, Slade, Issakidis, & Andrews, 2003; Magee, Eaton,
Wittchen, McGonagle, & Kessler, 1996), but this is more likely a consequence than a cause of the disorder. Of course it is possible that lack of a romantic relationship that occurs as a consequence of social anxiety may also maintain or exacerbate the disorder, but this possibility has not been evaluated.

**Inter-Parental conflict, violence, and relationship satisfaction**

A wealth of research has shown that parent relationship factors such as marital distress and separation, interpersonal violence, and conflict are associated with child psychopathology (Hudson, 2005). The majority of this research has focused on child externalising problems, with considerably less interest in internalising difficulties as an outcome. In general, parent relationship difficulties, especially interparental conflict and violence have shown a slightly stronger relationship with externalising than internalising symptoms in offspring (Buehler, Anthony, Krishnakumar, & Stone, 1997; Hudson, 2005). Where internalising has been assessed, it has commonly been measured as a single score that combines anxiety and depression and there have been few studies that have included clear and distinct measures of anxiety. Nevertheless, the few studies that have assessed anxiety indicate modest relationships between parent relationship difficulties and anxiety in offspring.

In a small study of 35 adolescents (aged 11 to 15 years) whose parents divorced, those whose parents continued to engage in post-divorce conflict reported higher rates of anxiety and withdrawal than those whose divorce did not involve high conflict (Long, Slater, Forehand, & Fauber, 1988). Mixed results were shown in a cross-sectional study of 5 and 6 year old children whose mothers completed measures of marital quality and their child's anxiety (Peleg-Popko & Dar, 2001). Marital quality was negatively associated with two specific fears but was not a significant predictor of social anxiety. One of the more thorough
studies was part of the Christchurch longitudinal cohort (Fergusson & Horwood, 1998). Over 1200 young people at age 18 years completed retrospective reports of interparental violence during their childhood and were also carefully assessed for psychosocial outcomes and a number of relevant demographic and family functioning variables. Adolescent-perceived violence displayed by both mother and father toward their partner was significantly associated with anxiety disorders in the adolescent, however, after controlling for significant covariates, only paternal violence significantly predicted anxiety disorders.

If inter-parental distress and aggression is shown to predict anxiety, one of the key questions is what may mediate these relationships. There has been little research to address this issue. In a cross-sectional study of 267 families, the relationship between parents' depression and children's internalising symptoms shared common variance with the extent to which parents reported fear, sadness, or lacked problem solving during interparental conflict (Du Rocher Schudlich & Cummings, 2003). The authors argued that marital conflict styles mediate the relationship between parent dysphoria and child internalising. However, it is also possible that parent emotionality may share a genetic predisposition to child anxiousness that is independently associated with conflict styles. Along similar lines a temperamental predisposition to anxiety might be reflected in research that has shown that the appraisals made by children during interparental conflict may influence experienced symptoms (Hudson, 2005). For example, Dadds and colleague (Dadds, Atkinson, Turner, Blums, & Lendich, 1999) have shown that children reporting self blame and high perceived threat during parental conflict are more likely to express internalising than externalising symptoms. Similar results, particularly linking perceived threat during the conflict with symptoms of anxiety, have been reported by others (Grych, Fincham, Jouriles, & McDonald, 2000; Jouriles, Spiller, Stephens, McDonald, & Swank, 2000).
These associations demonstrated in cross-sectional data have been supported by a few longitudinal studies. For example, parental separation or divorce early in a child's life (prior to grade 6) has been shown to predict internalising symptoms by age 15 years (Lansford et al., 2006). Similarly, in a small study of 37 families, marital satisfaction and wellbeing reported by parents and observed parent harmony and discrepancy when the child was a year old predicted teacher's reports of the child's anxiety at age 4 years (McHale & Rasmussen, 1998). Finally in a very long term follow-up, offspring (aged between 6 and 23 years) of parents either suffering major depressive disorder or no disorder were assessed for psychopathology at 10 and 20 years after initial assessment (Nomura, Wickramaratne, Warner, Mufson, & Weissman, 2002; Pilowsky, Wickramaratne, Nomura, & Weissman, 2006). Poor marital adjustment reported by parents at baseline predicted anxiety disorders in their offspring 10 years later but only for parents who had no psychiatric disorders. These results were not replicated 20 years later and no effects on anxiety were found by actual parent divorce.

The results in the preceding section indicate a small and inconsistent influence of parental divorce and dissatisfaction on anxiety. It is possible that these results actually reflect the interparental conflict and violence that are often associated with marital dissatisfaction and divorce. Along these lines a study of 682 families demonstrated that family violence witnessed by the child at age six was associated with internalising symptoms, however when child internalising two years earlier was statistically controlled, these effects mostly disappeared (Litrownik, Newton, Hunter, English, & Everson, 2003). A carefully conducted study by Jekielek (1998) among 1,640 children aged 6-14 years whose parents provided data over the preceding four years showed that both parental divorce and interparental conflict predicted later child anxiety. However, it was parental conflict that demonstrated the strongest and most consistent prediction of later anxiety. Children from conflicted families
whose parents divorced more than two years earlier, showed relatively low levels of anxiety. In contrast, children whose parents did not divorce and were high in conflict showed the highest levels of anxiety. Importantly, these data suggest that (understandably) living in a situation of high conflict can result in concurrent (state) anxiousness among children, but does not necessarily mean that it will produce enduring (trait) anxiety.

**Family Quality**

Measures of family quality and distress as predictors of psychopathology are likely to be non-specific variables, likely reflecting a variety of factors including inter-parental distress, stressful environments and life events, and the perceptions and temperament of the index person. Nevertheless, the quality of the family environment has been viewed as potentially influential in the development of some forms of psychopathology (Hudson & Rapee, 2005), although it has not typically been emphasised in most specific models of the development of anxiety disorders (Chorpita & Barlow, 1998; Hudson & Rapee, 2004). Perhaps for this reason, few empirical studies have evaluated the general family environment and its specific relationship with anxiety.

A number of studies that have examined the overall association between anxiety and broad questionnaire measures of family environment have demonstrated inconsistent results. Some studies have reported poorer overall family environments in adults or children with anxiety disorders than in control groups (Warner, Mufson, & Weissman, 1995) while other studies have failed to support this association (Beidel, Silverman, & Hammond-Laurence, 1996; Knappe et al., 2009). One small study demonstrated a significant difference between children with anxiety disorders and controls on one subscale of the Family Environment Scale (expressiveness) but not on another (control)(Suveg, Zeman, Flannery-Schroeder, & Cassano, 2005). In a cross-sectional study of maternal reports of their young child's anxiety
(aged 5-6 years), greater family rigidity was related only to a fear of strangers and not to other fears (Peleg-Popko & Dar, 2001). Interestingly in this study, greater family cohesion was related positively to higher levels of fearfulness and social anxiety. The authors related this finding to research indicating greater overprotection in families of anxious children (see below). In a larger population study of over 3,000 young people who were followed for 10 years, poor family environment was significantly associated with generalised anxiety disorder but not with other anxiety disorders (Beesdo et al., 2010).

Examination of more specific family environment factors has been less common. In one longitudinal study of around 1,000 adolescents in grades 7-11 at baseline, the adolescent's perception of arguments and conflict in the family at baseline predicted a small proportion of variance in the adolescent's symptoms of anxiety (as well as depression and physical symptoms) a year later (Mechanic & Hansell, 1989). Unfortunately the assessment of conflict was based on a single item that was not well operationalised and was not rated independently of the adolescent and the assessment of anxiety was based on a four-item measure developed by the authors. In a sample of 149 African-American children aged 6 years at baseline child-reported (but not parent-reported) symptoms of the child's anxiety six years later were significantly predicted by a very broad measure of family environment that contained items tapping marital distress, negative life events, and parental psychopathology (Grover, Ginsburg, & Ialongo, 2005). In contrast to these results, two very long-term studies of depressed parents (reported above) failed to demonstrate significant prediction of symptoms of anxiety in offspring at 10 and 20 years by a measure of family cohesion (Nomura et al., 2002; Pilowsky et al., 2006).

There appears to be little evidence that a negative or conflicted family environment is a major or specific predictor of anxiety. Admittedly however, this issue has not been well researched to date. Measures have been inconsistent and well controlled longitudinal studies
are very rare. The influence of general family environment on the maintenance of anxiety in adults has not been investigated.

Physical and sexual abuse

Abuse of a child is most likely to be perpetrated by a family member and hence it is included here within an examination of family influences on anxiety (although it is recognized that not all child abuse reflects family influence). However, few studies of the effects of abuse on later anxiety distinguish between intra- and extra-familial abuse and hence the evidence with respect to abuse specifically as a family factor is not entirely clear.

Several large, well-controlled studies have assessed childhood sexual abuse and later disorders and have controlled for background family adversity variables (Fergusson, Boden, & Horwood, 2008; Kendler et al., 2000; Nelson et al., 2002). Anxiety disorders have generally been assessed using structured diagnostic interviews while childhood sexual abuse was assessed using retrospective self report. After statistically controlling for family adversity and environment factors, most studies have demonstrated significant relationships between childhood sexual abuse and a variety of anxiety disorders. The associations are more consistently significant when the abuse involved genital contact and especially intercourse. Importantly however, sexual abuse is shown to be a risk factor for a variety of forms of psychopathology and in general it seems that the odds of experiencing an anxiety disorder following childhood sexual abuse is somewhat less than the odds of experiencing a number of other disorders (Fergusson et al., 2008; Kendler et al., 2000). In one study, childhood sexual (and physical) abuse was not significantly related to "pure" anxiety and was only related to anxiety when it was comorbid with depression (Levitan, Rector, Sheldon, & Goering, 2003). Thus, although childhood sexual abuse appears to be a risk factor for anxiety, it perhaps plays a less significant role in this disorder than in several others.
Considerably less research has addressed the importance of physical abuse in the development of anxiety disorders and the results in general have been less consistent. One study of 682 families showed that children's internalising symptoms at age 6 were predicted by parent verbal aggression, even after controlling for the child's level of internalising two years earlier (Litrownik et al., 2003). In contrast, "minor" violence was not a significant predictor of the child's internalising at age 6 once symptoms at age 4 were statistically controlled. In one longitudinal study of 375 participants, physical abuse was assessed at ages 15 and 18 and psychopathology was assessed when participants were aged 21 (Silverman, Reinherz, & Giaconia, 1996). For both males and females, experience of physical abuse before age 18 increased the risk for PTSD at age 21 but did not increase the risk for the other anxiety disorders assessed (simple and social phobia). In a larger study over 1,200 young people in New Zealand were assessed on retrospective reports of physical and sexual abuse at ages 18 and 21 and were assessed on a variety of forms of psychopathology at age 25 (Fergusson et al., 2008). A large number of relevant covariates including family adversity, IQ, parent education and SES were also assessed. The results showed a moderate and highly significant relationship between childhood physical abuse and later anxiety disorders. However, this effect disappeared when the covariates, including sexual abuse were controlled. In contrast, the relationship between childhood sexual abuse and anxiety disorders remained significant even after controlling for covariates, including physical abuse.

Overall, the current research seems to indicate that sexual abuse during childhood does increase the risk for anxiety disorders but this effect is not specific to anxiety and sexual abuse appears to be less strongly linked with anxiety disorder than with other forms of psychopathology. Physical abuse appears to show an even less specific link with anxiety disorders and seems to increase anxiety only as part of a general increase in psychopathology and distress. Whether abuse leaves an enduring influence on anxiety disorders years after it
has ended has not received much attention and there is little data on whether anxiety disorders in adulthood (aside from PTSD) can be maintained by an ongoing abusive relationship.

**Parenting styles**

Of the various family relationship factors, childrearing or parenting styles have been by far the most extensively studied with respect to anxiety disorders. Almost by definition, parenting styles are only likely to be influential during childhood and not later in development. A wealth of empirical studies, using both retrospective questionnaire and direct observational methods, have shown that the parent-child interactions of people with anxiety disorders is different to that of non-anxious controls (McLeod, Wood, & Weisz, 2007; Rapee, 1997; van der Bruggen, Stams, & Bögels, 2008). The majority of this research has suffered a number of methodological limitations including small sample sizes, retrospective reports, and inconsistent operationalisation of parenting constructs. Nevertheless, the sheer volume of consistent findings, provides a reasonable degree of confidence in the results. Few studies have compared anxious populations with other forms of psychopathology. Those that have, often failed to discriminate disordered groups on their associated parenting, suggesting that certain parenting styles may be associated with a variety of forms of psychopathology (Rapee, 1997). However, as stated, the lack of consistency of assessing parenting constructs, especially in the earlier research, may contribute to the lack of specificity. In an early review of the parenting literature it was argued that the bulk of evidence suggested that parental overprotection and control may be more consistently associated with the anxiety disorders while parental rejection and lack of warmth may be more strongly associated with depression (Rapee, 1997). Hence, theoretical models have particularly highlighted parental overprotection as a key factor in the development of anxiety (Chorpita & Barlow, 1998;
Consistent with these suggestions, a recent longitudinal study of over 3,000 adolescents/young adults (aged 14-24 years at baseline), demonstrated that anxiety disorders were significantly predicted by baseline reports of parent overprotection, but not rejection or lack of warmth, while mood disorders were predicted by rejection and lack of warmth, but not by overprotection (Beesdo et al., 2010).

While the majority of research has utilised cross-sectional methodology, a few longitudinal studies have begun to emerge (Edwards, Rapee, & Kennedy, 2010; Rapee, 2009; Rubin, Burgess, & Hastings, 2002). Most of these studies have been conducted with very young (preschool-aged) children. Results have variously supported two different directions of relationship - overprotection at time 1 predicting anxiety at a later time and anxiety at time 1 predicting overprotection at a later point. These results are consistent with most theoretical models that predict a reciprocal relationship between parent overprotection and offspring anxiety (Hudson & Rapee, 2004; Rubin et al., 2009). One of the few studies that has supported the reciprocal relationship hypothesis within the one dataset assessed parents of over 600 children aged around 4 years at baseline and reassessed them one year later (Edwards et al., 2010). According to mothers' reports, baseline overprotection predicted child anxiety one year later while child anxiety predicted maternal overprotection a year later. In contrast paternal data supported the relationship only in the direction from overprotection to later anxiety.

Some pilot research has recently begun to demonstrate that overprotective parental behaviours may actually cause more anxious behaviour. In the first of these studies, 26 nonclinical children (aged 7-13 years) were asked to present two brief speeches (de Wilde & Rapee, 2008). Mothers of the children were present while their child prepared the first speech and were randomly allocated to either act in a highly protective and controlling fashion or in
a minimally involved but supportive fashion. When delivering the second speech that children prepared entirely independently, children whose mothers had previously acted in a more protective fashion demonstrated more overt signs of anxiousness. This study has recently been replicated using a very similar method (Thirlwall & Creswell, 2010). In this latter study maternal control interacted with child anxiety in that children high in trait anxiety whose mothers were controlling demonstrated the highest levels of anxiety in response to the speech.

Hence, current data appear to be relatively consistent in demonstrating an influence of parental overprotection on anxiety. However, demonstration of a causal association is still relatively untested and requires considerably more work. In addition, data from twin studies showing inconsistent support for the role of shared environment in anxiety suggests that any parental influence is likely to work through interaction with the child’s genes. Evidence for such gene-environment interactions is still sparse. Although other parenting styles such as criticism or lack of warmth may also play a role in anxiety, their influence on anxiety specifically has been less consistently established.

Modeling

Following conditioning theories of anxiety, it has long been assumed that vicarious learning is one of the key paths to the acquisition of fears (Field, 2006; Merckelbach, de Jong, Muris, & van den Hout, 1996; Mineka & Zinbarg, 2006). Given the assumed influence of families in the development and maintenance of child anxiety, it has been something of a truism that child anxieties may be influenced through modeling of their parents’ fears. However, demonstration of this mechanism has been extremely difficult and evidence is limited. This is especially true given that the importance of modeling independent of shared genes has not been demonstrated.
The potential for vicarious influence has been demonstrated in animal research that has shown that monkeys can learn to fear previously neutral stimuli after observing a partner react fearfully to that object (Cook & Mineka, 1989). In humans, developmental research has shown that infants will reflect their mothers’ fearful reactions to novel objects (Hornik Parritz, Mangelsdorf, & Gunnar, 1992; Mumme & Fernald, 1996). In a further development of this paradigm it has been shown that very young children (aged under two years) can learn to fear and avoid a novel object following a single fearful reaction to that object from their mother (Dubi, Rapee, Emerton, & Schniering, 2008; Gerull & Rapee, 2002). Of more direct relevance to typical anxiety disorders, this modeled acquisition of fear has been shown to apply to fearful reactions to strangers (de Rosnay, Cooper, Tsigaras, & Murray, 2006). Interestingly, in this latter study, modeling interacted with the child’s temperament such that fear of strangers was most strongly demonstrated in those children who had seen their mother reacting fearfully and were characterized as having an inhibited temperament.

Of course, this evidence does not demonstrate that such learning actually occurs in anxious families. A closer approximation to this link is found in the evidence described earlier that parents of anxious children are more likely to also be anxious. Thus it is likely that anxious parents will demonstrate overt fearful reactions more often than other parents. In support of this suggestion, mothers of anxious children self report a greater tendency to express anxiety and fearfulness in front of their child (Muris, Steerneman, Merckelbach, & Meesters, 1996). In two related studies, Murray and colleagues observed socially anxious and non-anxious mothers and their children (10 weeks and 10 months) during natural interactions with a stranger (Murray, Cooper, Creswell, Schofield, & Sack, 2007; Murray et al., 2008). In both studies fearfulness in response to the stranger increased among the children of socially anxious mothers and this development was associated with the extent to which the mothers expressed anxiety and social avoidance in front of the infant.
From a slightly different perspective, a growing body of research has shown that older children are able to learn to fear a novel stimulus following overt, verbal expression about its dangerous qualities (Muris & Field, 2010). As with the research on modeling, the assumption is that anxious parents will express more threat and danger when describing objects and experiences and this, in turn, will lead to fear acquisition among their children. Some research has shown that biases toward threatening interpretations shown by anxious children are also shown by their mothers (Creswell, Schniering, & Rapee, 2005; Gifford, Reynolds, Bell, & Wilson, 2008). However, it is very possible that this relationship simply reflects the heightened anxiety that is shared between anxious children and their mothers. At least one study has shown that anxious children expect that their mothers will interpret ambiguous material in a threat-consistent manner (Lester, Seal, Nightingale, & Field, 2010).

There is growing evidence that verbal and/or behavioural expression of anxiety by a parent can increase anxiousness among their children, but whether this mechanism accounts for a cause in the development of anxiety disorder is still far from demonstrated. It might also be tempting to speculate that similar expressions by say, romantic partners, might help to maintain anxiety in adulthood, but hypotheses like this have not yet been tested.

**The Influence of Family Factors on the Treatment of Anxiety**

**Family variables as treatment predictors**

Evaluation of variables that predict treatment outcome frequently results in inconsistent and non-significant results for a variety of reasons including the fact that moderation requires large effect sizes and that treatment studies are not designed to test for prediction. Consistent with this "truism", studies of the treatment of anxiety have rarely identified family predictors of outcome that emerge in more than one study.
Among research into the management of child anxiety, several studies have indicated that various measures of parent emotional distress (either maternal or paternal anxiety and/or depression) predict worse outcome (Berman, Weems, Silverman, & Kurtines, 2000; Bodden et al., 2008; Cooper, Gallop, Willetts, & Creswell, 2008; Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008; Rapee, 2000). While parental emotionality may influence the family environment in a number of ways, it is very likely that these effects simply reflect a greater personality or genetic loading assessed by these measures (Rapee, Schniering, & Hudson, 2009).

Perhaps surprisingly, measures of the parent relationship have rarely been included in treatment studies for child anxiety and when they have, they have failed to significantly predict outcome. In a study of over 100 clinically anxious children treated with a variety of broadly cognitive behavioural strategies, Berman and colleagues failed to find that the quality of the parents' relationship predicted treatment response (Berman et al., 2000).

Similarly, the overall family environment has also rarely been assessed in studies of treatment outcome for childhood anxiety. In two studies of the treatment of OCD in which family environment was assessed, results were contradictory. In one study family environment (based on the McMaster Family Assessment Device) reported by both the mother and father significantly predicted improvement 12 months after treatment (Barrett, Farrell, Dadds, & Boulter, 2005) however, in another study, reports of family environment (based on a single item developed by the authors) did not significantly predict treatment response (Wever & Rey, 1997).

In contrast to these findings, some research with adults has shown that the extent to which relatives are perceived as critical or hostile predicts worse response to treatment (Chambless & Steketee, 1999; Renshaw, Chambless, & Steketee, 2003; Steketee, Lam, Chambless, Rodebaugh, & McCullough, 2007). For example, in one such study, adults who
were being treated for their OCD or agoraphobia completed weekly ratings of the extent to which they perceived and were upset by criticism and hostility from their relative (mostly spouses or parents) (Steketee et al., 2007). Both weekly levels of anxiety and the extent of distress in response to exposure were directly predicted by the extent to which participants were upset by their partner’s criticism and this latter variable was in turn predicted by the extent of perceived criticism. Of course it is unclear from this study whether perceived criticism reflected actual criticism. However, in an earlier study, Chambless and Steketee (1999) showed that relatives’ hostility (as measured by the Camberwell Family Interview) negatively predicted treatment outcome for agoraphobia and OCD. Perceived criticism also predicted outcome, even after controlling for actual criticism. Thus it appears that both the actual degree of hostility expressed by the relative and the extent to which the patient themselves perceives criticism may influence response to treatment.

Given the wealth of research described above into parenting factors in child anxiety, it is surprising that little research has examined whether the parent-child relationship predicts outcome. However, one study of adults with OCD showed that family accommodation, that is the extent to which families adapt to and allow obsessive compulsive behaviours (a construct that is related to overprotection), predicted worse outcome (Amir, Freshman, & Foa, 2000). Similar research has also demonstrated that family accommodation to obsessions and compulsions is a negative predictor of treatment outcome in children with OCD (Merlo, Lehmkuhl, Geffken, & Storch, 2009; Storch et al., 2010). From the opposite perspective, Chambless and Steketee (1999) concluded that relatives’ specific criticism of avoidant behaviours in the absence of hostility toward the patient themselves, could actually be motivating and increase treatment response.

Hence there is evidence that several factors reflecting family, parent and partner relationships and behaviours can influence treatment outcome for anxiety disorders in both
adults and children. However, research is still very limited and a large range of questions remain to be addressed and replicated. The field would benefit from a theoretical overview of why and how family variables would be expected to affect treatment response. It is also unlikely that many of these factors will be specific to treatment of anxiety, but evaluation of specificity of these effects has not been conducted.

**Importance of including families in treatment for child anxiety**

A vexed question in the area of treatment of childhood anxiety is whether there is benefit in including parents in the treatment (Creswell & Cartwright-Hatton, 2007; Rapee et al., 2009). Overall reviews and meta-analyses have indicated that programs in which parents of anxious children are incorporated into the treatment result in no greater efficacy than programs targeting the child alone (In-Albon & Schneider, 2006; James, Soler, & Weatherall, 2006). However, these reviews do not typically consider differences in age of the child and in fact there are few studies that either include a sufficient age range or have sufficient power to detect relevant differences (Creswell & Cartwright-Hatton, 2009; Rapee et al, 2009). In a careful review of the literature, Creswell and Cartwright-Hatton (2007) concluded that there are consistent hints in the literature that including parents in treatment for anxious children does provide a greater benefit, but that effects are small.

Of more importance to a review of family factors in anxiety, is whether it is possible to improve treatment effects by specifically targeting theoretically important family variables. The studies that have evaluated the benefit of including parents in treatment have utilised a wide variety of methods of involvement and few have tried to specifically target key family variables. One exception is a trial in which a very brief treatment for parental anxiety was added to standard treatment for the child's anxiety (Cobham, Dadds, & Spence, 1998). Results were somewhat mixed, with only one measure (child anxiety diagnoses at post-
treatment) showing stronger effects for children of anxious parents who received the parent treatment and most measures failing to show differences between treatments. These results were more consistent in a recent larger study that included a slightly longer and more intensive parent anxiety management component (Hudson et al., 2009). Despite the greater focus on parent anxiety in this latter study, the results again failed to demonstrate larger effects on child anxiety when parent anxiety was also treated. Treatments that address other aspects of family functioning such as parent depression, marital functioning, family quality, and so on have not been conducted in the child anxiety area. One exception is a focus on parental overprotection, which is included in some programs (e.g. Cool Kids; Rapee et al., 2006), however, to date there have been no controlled studies that have compared effects of treatment for anxious children that includes or does not include a focus on parental overprotection.

Clinically there appear to be several advantages to including and incorporating parents into treatment for child anxiety. Expert clinicians in the field would mostly argue that including parents provides the greatest benefits for practical reasons such as helping to ensure homework compliance and assist with real-world generalization and that these factors are likely to be of greater importance for younger children. The empirical evidence to date is contradictory and this may be largely due to the fact that studies have generally not identified specific parent roles in therapy (such as homework compliance) and have not sufficiently examined the influence of age.

Importance of including partners in treatment for adult anxiety

A number of studies have examined the value of including partners into treatments for adult anxiety. Much of this work has focused on agoraphobia following early suggestions that marital discord was an important factor in the development of agoraphobia (see Emmelkamp
& Gerlsma, 1994). Some parallel work has also focused on OCD. In general, studies that have examined the inclusion of partners into treatment for adult anxiety have demonstrated mixed results (see Emmelkamp & Gerlsma, 1994; Renshaw, Steketee, & Chambless, 2005; Steketee & Shapiro, 1995 for reviews). However, as for the literature on the inclusion of families in treatment for child anxiety reviewed above, simply including or not including a partner in treatment may not be the key issue. Rather, it may be more important to look at how those partners are included and whether a focus on specific skills and aspects of the couple relationship might enhance outcomes (Renshaw et al., 2005; Steketee & Shapiro, 1995). As an example, several recent studies have shown that the extent to which patients perceive criticism from their partner in addition to the extent of actual hostility and negative attributions made by the partner can both reduce the efficacy of exposure treatments for anxiety (Renshaw et al., 2003; Renshaw, Chambless, & Steketee, 2006; Steketee et al., 2007). Thus it may be that programs that specifically target partner criticism and hostility as well as teaching the patient to cope better with that hostility may produce improved outcomes.

**Summary and Conclusions**

Research into the role of family factors in both the etiology and treatment of anxiety disorders has been surprisingly limited. Surprising because family factors lie at the basis of many theories of psychopathology and have been shown to play a key role in several forms of disorder. But it appears that family factors may not play as large a role in the development or treatment of anxiety disorders as they do in other forms of psychopathology.

Few demographic factors appear to be related to the development of anxiety disorders. Of the factors that have been assessed, some evidence suggests that low socioeconomic status may predict later anxiety, but even this variable does not account for marked variance in later anxiety. General family relationships and overall quality of family
life have not demonstrated marked or significant relationships with the development or the
treatment of anxiety disorders. Of course this lack of effect may partly be due to the poor
operationalisation of family quality and the wide variety and weak psychometric properties of
many of the measures. Future research that focuses more specifically on detailed and clearly
operationalised aspects of family quality may yet demonstrate some relationships. One
variable that has been carefully researched and has demonstrated some contribution to
variance in anxiety disorders is childhood sexual abuse. It is interesting to note however, that
even such a theoretically logical variable (given its elicitation of unpredictable threat) appears
to account for considerably less variance in the development of anxiety disorders than in
several other forms of psychopathology.

The most extensive research has linked anxiety disorders with parent-child
interactions and especially with parent overprotection. A wealth of research has demonstrated
an association between parent overprotection and anxiety in offspring, although concluding a
causal association is still far from clear. Nevertheless, some longitudinal and experimental
studies are beginning to indicate a possible causal role of parent overprotection in later
anxiety in addition to a reciprocal elicitation of overprotection by child anxiety. Future
longitudinal studies that more carefully assess parent overprotection early in life may begin to
demonstrate some interesting effects.

Given the importance of families in the children’s development, it is surprising that
research has failed to indicate a strong influence of family involvement or relationships on
treatment outcomes for anxious children. Some evidence clearly suggests that including
parents in the treatment of anxious children may be beneficial, but this is more likely for very
young children and is not associated with very strong or marked effects. There are key issues
that remain to be addressed, such as importance of specifically targeting parent
overprotection within treatment, but it is likely that the key role that parents may play in
treatment for child anxiety is to enhance treatment adherence and real world generalization. Similarly, evidence has been mixed about the value of including partners in treatment for adult anxiety, but more specific measurement has indicated that addressing perceived hostility and criticism may produce benefits. Here as well, it is likely that a key mediator is the added adherence to treatment provided by a supportive and constructive partner. Clearly if the couple relationship is poor, this is likely to interfere with enhancement of treatment adherence and work on the couple’s relationship is likely to be beneficial. Thus, clinical benefits may accrue through specific, targeted attention to the couple interaction and ways in which the partner can non-judgmentally and non-critically encourage the client to participate in therapy.

The field, particularly with respect to intervention, is lacking a comprehensive conceptual model that outlines the ways in which parent, partner, and family variables might influence the development, maintenance and modification of anxiety disorders at different stages of development. Such a model would help to organise and focus empirical research efforts. Avoidance and threat appraisals lie at the heart of anxiety and so it is likely that if family factors have a role to play, it would be through their influence on these core processes. For example, it may not be that partner criticism broadly is important for treatment outcome but more specifically criticism aimed at attempts to engage in new approach behaviours. Similarly, sexual abuse per se may be less influential in the development of anxiety than sexual abuse by certain perpetrators at certain times and in certain ways, that inflates estimates of threat and vulnerability. There are clearly a vast number of questions that remain to be addressed before the role of families in the onset and management of anxiety disorders can be understood.
References


