

Social anxiety disorder: A review of environmental risk factors

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Abstract: Social anxiety disorder (SAD) is a debilitating and chronic illness characterized by persistent fear of one or more social or performance situations, with a relatively high lifetime prevalence of 7% to 13% in the general population. Although the last two decades have witnessed enormous growth in the study of biological and dispositional factors underlying SAD, comparatively little attention has been directed towards environmental factors in SAD, even though there has been much ongoing work in the area. In this paper, we provide a recent review and critique of proposed environmental risk factors for SAD, focusing on traditional as well as some understudied and overlooked environmental risk factors: parenting and family environment, adverse life events, cultural and societal factors, and gender roles. We also discuss the need for research design improvements and considerations for future directions.

Introduction

Definition

Social anxiety disorder (SAD) is a debilitating and chronic illness characterized by “a marked and persistent fear of one or more social or performance situations involving exposure to unfamiliar people or possible scrutiny by others” (Furmark 2002, p 84; Schneier 2006, p 1030). Publication of the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, text revision (DSM-IV-TR) by the American Psychiatric Association (APA 2000) includes these defining symptoms amongst other criteria. SAD only became an independent diagnosis with the printing of the DSM-III (APA 1980; Hidalgo et al 2001). Earlier, the DSM-I and -II (APA 1952, 1968) grouped it with all other phobias, and to this day psychologists still refer to SAD as social phobia (SP; Furmark 2002; Hudson and Rapee 2000). Within the DSM-III-R (APA 1986) and DSM-IV (APA 1994), SAD was divided into two sub-types called generalized and non-generalized (Berman and Schneier 2004; Chavira and Stein 2005). The generalized form included fear of most social situations whereas the non-generalized, sometimes also referred to as SP, comprised fear of one or a few identifiable circumstances. Clinicians considered generalized SAD to be the more serious sub-type, with greater severity in symptomology and associated increases in functional impairment. Usually those individuals diagnosed with this sub-type have additional comorbidity and an extensive family history of SAD (Berman and Schneier 2004). At the same time that SAD became part of the DSM-III, so too did another Axis II mental illness called avoidant personality disorder (APD; Muller et al 2004). In a somewhat perplexing overlap, researchers working in this area considered APD to be a more severe form of SAD, especially the generalized sub-type. In an effort to make sense of this confusion, several researchers proposed a spectrum of social discomfort to account for these different constructs. Shyness with little impairment is located at one end, and the spectrum extends into the two sub-types of SAD, with increasing symptomology in the center, and expands into chronic APD at the other end (Muller et al 2004).

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Over the past two decades, research on SAD has accumulated. Unfortunately, the terminology has not remained static over time, a problem exacerbated by accompanying definition changes in the DSM between 1980 and 2000. Papers written over the years have included a variety of similar and related constructs including avoidant disorder, overanxious disorder, fear, shyness, neuroticism, worry, social withdrawal, social anxiety, social phobia, passive anxious, fearful social inhibition, social reticence, self-consciousness, social isolation, audience sensitivity, peer neglect, anxiety sensitivity, and behavioral inhibition (Hudson and Rapee 2000). Another complementary classification system has arisen alongside the DSM based on empirical evidence that suggests problem behaviors cluster in two broadband groups known as internalizing and externalizing disorders (Achenbach and Edelbrock 1978). While the externalizing behaviors parallel the DSM attention deficit hyperactivity disorder (ADHD), conduct disorder (CD) and oppositional defiance disorder (ODD), internalizing behaviors are analogous to DSM depression and anxiety disorders. Many times the authors of papers on internalizing problems make little to no distinction between depression and anxiety. For the purpose of the present review paper, we preserved the terminology as written in each article. However, when summarizing in our own words, we used SAD as the term of preference based on current trends (Ollendick and Hirshfeld-Becker 2002).

Prevalence, epidemiology and comorbidity

The lifetime prevalence of SAD is somewhere between 7% and 13% in Western countries (Furmark 2002). Adolescents seem to have higher rates of SAD than younger age groups, although slightly less than adults. The actual rates are hard to determine with changing research methodology and diagnostic criteria, such as using the DSM versus the International Classification of Diseases (ICD-10 1990) or the DSM III-R versus the DSM-IV (Chavira and Stein 2005). However, generally children's rates are consistent with a range between 0.6% and 3.5% that go up as the children mature into adulthood (Merikangas 2005). Additionally, there is a higher incidence of SAD in both girls and adult women, all younger adults, those less well-educated and those of lower socioeconomic status (SES; Hidalgo et al 2001). The mean age of onset for SAD occurs between early and late adolescence, although reports suggest it can begin as early as 7 or 8 years of age (Chavira and Stein 2005). Moreover, epidemiological studies have found this disorder to be the most widespread of all the anxiety disorders, and the third most common

psychiatric disorder after major depression and alcohol abuse (Hidalgo et al 2001; Schneier 2006).

SAD places individuals, both children and adults, at risk for chronic distress and impairment and differs from shyness and performance anxiety by its greater severity and pervasiveness (Beidel et al 1999; Schneier 2006). Often people diagnosed with SAD will avoid important activities, including school and work, or if they attend, they will not participate. This withdrawal results in lower achievements in vital parts of their daily lives that end in decreased occupational, academic and family function. People with SAD are also less likely to marry than those who do not have this disorder. Overall, there is a reduction in quality of life, an increase in alcohol and drug misuse and a risk of suicide (Baldwin and Buis 2004). Associated with these issues is a substantial economic burden, since people with SAD are more likely to be unemployed, absent from work or have reduced work productivity. Unfortunately, most cases go untreated given there is a relative lack of awareness of the symptoms by the general population (Furmark 2002; Schneier 2006).

Comorbidity is another important issue related to SAD. Studies suggest that lifetime comorbidities for SAD are between 69% and 81% (Fehm and Wittchen 2004). Disorders most frequently and strongly associated with SAD are other anxiety disorders, mood (depression) and substance abuse disorders. Some scientists suggest that comorbidity is an indication that categories of mental illness are too imprecisely distinguished to be useful for valid diagnosis. However, research suggests that comorbidity is not necessarily an artifact of the DSM classification system but rather a true reflection of the prevalence of mental illness (Fehm and Wittchen 2004). Data suggest that persons with comorbid SAD have considerably worse disabilities and quality of life than those with SAD alone. As well, psychologists consider comorbidity a useful tool in the search for the etiology of this mental illness. One thought is that pre-existing disorders may promote the development of SAD, or that SAD enhances the risk of a wide variety of other disorders. Accordingly, any search for the antecedents of SAD should involve investigations of comorbidity in the hopes of pursuing useful information in the understanding of, or treatment regimes for, SAD (Egger and Angold 2006).

Risk factor model

No single mechanism seems to account for the development of SAD, making it difficult to form a theoretical framework to understand this mental illness (Hidalgo et al 2001). However, taking into consideration the complexity of risk factors

believed to play a role, one particular school of thought links biological, psychological and environmental factors into a diathesis-stress paradigm that may represent the etiology of this anxiety disorder (Schmidt et al 2005). This concept features an interaction between a predisposition towards a disorder (diathesis) and environmental disturbances (stress). The greater the underlying genetic vulnerability toward a particular disorder, the less stress needed to trigger associated problem behaviors. In the last five years, a number of studies have hypothesized such a model for SAD (Hudson and Rapee 2000; Hidalgo 2001; Ollendick and Hirshfeld-Becker 2002; Manassis et al 2004; Rapee and Spence 2004; Chavira 2005; Merikangas 2005; Muris 2006a; van Brakel et al 2006). Mostly, these models describe four distinct areas of contribution to the development of SAD, including genetic and temperament factors, cognitive aspects, parent-child interactions and adverse environments, together added to societal and cultural influences. Tying these together is a developmental psychopathology perspective, which emphasizes the relation between risk and vulnerability factors interacting with protective factors in a developing individual (Muris 2006b).

Purpose

The purpose of the present paper was to provide a recent review of risk and vulnerability factors that potentially evolve from the environment. We attempted to cover the most current evidence that adds to our understanding of well-documented environmental risk factors, as well as those that have been relatively understudied in the past, but which have been implicated in the etiology of SAD. Primarily, the present review covers articles published since 2000. The paper is divided among four major sections with a recent literature review and limited critique pertaining to each: (1) parenting and family environment, (2) adverse life events, (3) cultural and society factors, and (4) gender roles. Although these sections have been delineated as discreet topics, their boundaries are somewhat indistinct. Specifically, the influence of the family is pervasive through almost all the sections. This issue comes as no surprise since it is difficult to separate the child from his environment, which in most instances involves the family. Although the first section most directly addresses family influences on the development of SAD, the remaining three sections also make reference to the family environment, and these references have been identified by subtitles within each segment.

Although a comprehensive review of all aspects thought to be involved in the etiology of SAD would be

useful, an examination of such a literature is beyond the scope of this document. For example, although modeling, social learning experiences and early learning are part of the potential environmental risk factor perspective, these issues were not addressed here. The interested reader is directed to other recent articles for this information (Muris et al 1996, 2001, 2002; Gerull and Rapee 2002; Bögels et al 2003; Muris, Bodden, et al 2003; Alden and Taylor 2004; Mineka and Zinbarg 2006; Rosnay et al 2006; Taylor and Alden 2006; Voci et al 2006; Lawson et al 2007).

Parenting and family environment

Parenting

The role that parent influence has in the development of anxiety is a complicated issue that has yet to be completely unraveled. Nonetheless, research has definitely opened a window that has increased our understanding of some factors that might be important in the development of SAD. Specifically, parenting traits such as overcontrol, lack of warmth or rejection, and overprotection are known to be associated with the etiology of this disorder (Stark et al 1990; Rapee 1997; Caster et al 1999; Hudson and Rapee 2000; Hidalgo et al 2001; Ollendick and Hirshfeld-Becker 2002; Neal and Edelman 2003; Hollenstein et al 2004; Chavira and Stein 2005). Although most of the research has focused on maternal parenting, fathers are finally being included in the research as possible contributors to child behavior problems, especially in later childhood (Rapee and Melville 1997; Greco and Morris 2002). Even the impact of siblings is starting to attract attention (Lindhout et al 2003). As researchers have pursued this issue in the last five to ten years, a complicated picture has evolved illustrating that parenting is just one possible risk factor in a multitude of other environmental factors and one that it is not specific to SAD. Moreover, work has not adequately addressed the environmental factors of cultural and ethnic determinants that might be associated with parenting behaviors. Currently, researchers are looking toward different and more comprehensive methodologies and new constructs and models to expand their present understanding of how parenting interacts with other risk factors to give rise to SAD in particular and anxiety disorders in general.

Negative rearing practices

Among the various environmental factors believed to be antecedents of anxiety disorders are those of negative parental rearing practices. The interpretation of this term as it relates to anxiety has encompassed many constructs

over the last 20 years. These include practices of control, overprotection, rejection, neglect, lack of warmth or affection, anxious parenting, insensitivity, restrictiveness, social isolation, criticism, shame tactics, behavioral rigidity and concern with the opinions of others (Stark et al 1990; Rapee 1997; Caster et al 1999; Hudson and Rapee 2000; Hidalgo et al 2001; Ollendick and Hirshfeld-Becker 2002; Neal and Edelman 2003; Hollenstein et al 2004; Chavira and Stein 2005). A variety of mechanisms may work to promote anxiety through these constructs. For instance, parental overcontrol diminishes a child's ability to explore and learn new skills independently, thereby possibly promoting anxiety in situations of perceived fear. While parental rejection fosters an insecure attachment, potentially leading to psychopathology in general, including anxiety disorders (Lindhout et al 2006). Very early research on parenting style or behaviors pointed to a connection between these perceived parental practices and the development of anxiety disorders, specifically phobic disorders (Arrindell et al 1983; Arrindell et al 1989). To capture evidence for this association, socially phobic and agoraphobic adult in- and out-patients completed retrospective questionnaires on perceived parenting behaviors, which revealed their parents to be overprotective, rejecting and lacking in social warmth. This relation was far stronger for patients diagnosed with SP than agoraphobia. Rapee (1997), who summarized much of the early literature on the role of childrearing practices as an antecedent to anxiety disorders, agreed with their results by detecting a general trend in the literature, despite some variable data, that rejection and overcontrol by parents might be positively associated with later anxiety disorders. Rapee's own work confirmed this finding when he collected retrospective information on rearing practices from both socially anxious adult participants and their mothers. His research showed that parental overcontrol and rejection were significantly related to children's anxiety symptoms (Rapee and Melville 1997). Rapee et al also proposed that parental overcontrol might specifically play a role in the onset and maintenance of social anxiety, and parental rejection correlates more specifically with child depression. To further advance the understanding of this relation, another group chose to study a population of normal 9–12 year olds instead of adults with SAD (Muris and Merckelbach 1998; Grüner et al 1999). Once more, the results showed anxiety symptoms to be positively associated in general with parental rejection, overcontrol and anxious rearing, but not lack of emotional warmth. Thus, early social relationships between the child and parent are most likely essential to a child's appropriate emotional development.

Fathers and paternal influences

Research into the familial influences on child psychopathology has primarily focused on the anxious child and mother (Rapee and Melville 1997). Often the father's contribution to parenting is encapsulated in the 'parent' response, implying both father and mother have identical parenting styles. In the hopes of ameliorating this oversight in the investigation of 'parenting' style or behavior, the association of father behavior with child social anxiety has been also investigated (Greco and Morris 2002). Children aged 8–14 completed two questionnaires: one including items detecting SP, and the other on perceived parental style. Afterwards, the father and child collaborated on a challenging task while under observation. The data suggested fathers were more controlling with socially anxious children during the collaborative task, but no more rejecting than fathers of non-socially anxious children. This pattern did not translate into differences in the children's perception of their fathers' rearing styles. Ratings of fathers from both the high and low socially anxious groups were not significantly different. While pointing to the limitations of their research that might make their results unreliable, the authors felt including fathers in psychopathology research was important to future investigations into SAD, especially since it is probable that mothers and fathers make unique and individual contributions to the family environment.

Siblings

Little work has been conducted on the part that siblings play in the development of SAD. One recent study has examined the role of sibling relationships in anxiety disorders in general (Lindhout et al 2003). These researchers were interested in determining if anxiety-disordered children differed from non-disordered children in perceived affection or hostility from a sibling and perceived differential treatment from their parents. Using semi-structured diagnostic interviews and child self-report measures, the clinical population of anxious children perceived themselves being treated differently by their parents. Since they did not differ from the controls in perceived affection or hostility from a sibling, Lindhout and colleagues hypothesized that siblings probably did not contribute to the development of anxiety disorders in children except by an indirect route: the anxious children probably used their siblings as the standard against which they compared themselves. This study bears repeating in that the investigation should have assessed perceptions of differential parental treatment from a number of points of view, not just from that of the anxious child. Other informants could provide a clearer picture of actual versus perceived

relationships. Despite this, the sibling impact on anxiety development is most likely very small and probably only important in extreme cases of negative sibling interactions.

Culture

Many of the studies to date on perceived parental rearing behaviors and their potential role in the etiology of anxiety disorders have occurred in Western countries using predominantly Caucasian participants. One group questioned the limitations of this research by asking whether standardized questionnaires for anxiety symptoms translated into reliable instruments for the detection of anxiety in different cultures and in ethnicities within a single culture. This group also wondered whether there might be a cultural difference in children's anxiety levels due to a disparity in parental rearing behaviors based on different cultural and ethnic groups (Muris, Loxton et al 2005). Muris, Loxton et al (2005) investigated DSM-defined anxiety symptoms in white, colored and black youths from South Africa using the reliable and validated questionnaires from Western countries called the Screen for Child Anxiety Related Emotional Disorders (SCARED) and the EMBU (an acronym for 'my memories of my upbringing' in Swedish). The results obtained showed several interesting cultural risk factors associated with anxiety development.

First, the psychometric properties of the SCARED and EMBU matched up well to those obtained in Western countries. Second, the colored and black youths displayed higher levels of anxiety compared to the white youths. Third, the perceived parental rearing behaviors in all groups were significantly associated with rejection, overprotection and anxious rearing, in line with Western findings. However, in South Africa an additional factor came into play. SES wholly explained the difference in perceived parental overprotection between white and colored or black youths, which suggested overprotective colored or black parents were responding to the deprivation, violence and poverty of their living conditions. In response to their data, the authors suggested that their research directed attention toward implementing early intervention programs in the communities of colored or black South Africans with the goal of preventing the development of internalizing problems. This type of research also broadens our knowledge of anxiety disorders to encompass the international arena.

Research design improvements

Over the last decade, researchers have attempted to strengthen the relation between parental rearing and anxiety by improving experimental design. One change was the use of observational

techniques in the laboratory to monitor parent-child dyads, in addition to the usual self-report questionnaires. They also started to incorporate information from more than one source. In one such study, mothers and their clinically anxious children were observed and coded for their interaction on two fronts, general negativity (rejection) and involvement (overcontrol) behaviors, during two difficult cognitive tasks (Hudson and Rapee 2001). The children filled out questionnaires to detect chronic anxiety, and the mothers completed questionnaires about their child's behavior and their own depressive and anxious symptomology. The data came back with strong indications that mothers of anxious children were more involved, intrusive, and more negative than mothers of non-anxious children. A subsequent paper reported on the parenting style used with siblings of these clinically anxious children, with the purpose of differentiating between a general style of parenting and a specific response to an anxious child's needs (Hudson and Rapee 2002). Results revealed that mothers and fathers were equally overinvolved and intrusive during the execution of a complicated puzzle task with their anxious child and the sibling of that child, suggesting that this parenting trait might not be specific to anxiety development.

Another change in research design was to look more closely at specific parental rearing constructs associated with anxiety. One inquiry, for example, focused on the construct of control (Aunola and Nurmi 2005). The investigators followed children for a two-year period to assess their internalizing (and externalizing) behaviors as they related to parenting style. Information was collected through questionnaires, one asking children about their problem behavior and another questioning parents about their parenting styles of affection (warmth), behavioral (limit setting on actions) and psychological (guilt inducing or love withdrawal) control. The results showed that high levels of maternal psychological control and high affection predicted increases in the levels of internalizing problems, in this case depression only. These results were unexpected because internalizing problems are normally associated with lack of warmth. Among a variety of speculations, the authors believed part of the inconsistency in results was due to the notion that a single parenting style was not responsible for problem behavior, but rather a combination of 'sub' styles was influencing the child's development. The authors suggested that future studies would help clarify the understanding of parenting styles and internalizing disorders.

Research directions

Traditionally, research has correlated parenting practices with anxiety in three domains, acceptance or rejection,

control or overprotection, and modeling of anxious behaviors. However, in an assessment of empirical evidence on this subject, a research group suggested that many studies had mixed results and limited evidence (Wood et al 2003). Wood et al reasoned that traditional evidence lay in correlations of single main effects (such as overcontrol) with anxiety, where one main effect did not account for most of the variability in anxiety symptoms. To fill in this gap, they encouraged scientists to adopt a new contemporary model that reflected what they conceived as a complex, multi-determined process where parenting potentially played multiple roles. This process embraced the concepts of multifinality, where a single factor leads to multiple outcomes, or equifinality, where multiple factors interact to reach a common outcome (Ollendick and Hirshfeld-Becker 2002).

Experimental directions have also been altered in recent years by redefining parental rearing constructs within the dynamics of family interactions. In contrast to the traditional research of correlating an identified risk factor with the development of anxiety disorders. Parental overcontrol is one such construct that has been reformed; research has explored the issue of the mediating and moderating role of the perceived locus of control in family functioning and its correlation with anxiety symptoms (Ballash et al 2006). The mediating role is one of an intervening agent that indirectly causes problem behavior, while the moderating role is one that reduces or prevents extreme behaviors. In this study, researchers asked university students to fill out questionnaires on several dimensions of family functioning (general functioning, affective involvement, behavioral control and communication); their perceived control over anxiety symptoms, emotional responses and external problems; and the severity of their anxiety symptoms. Ballash and colleagues (2006) found support for a model where perceived control acts as a mediator, but not a moderator, between family functioning and anxiety symptoms in young adults. Although the sequential relation between locus of control, family functioning, and anxiety symptoms is still unclear, there is reason to believe that further investigation of perceived control within the complex family setting may shed light on one of the many mechanisms involved in development of anxiety disorders, including SAD.

Summary

The research community has successfully correlated parenting as one small, but integral part of the mechanism in developing SAD and other anxiety disorders. In particular, parenting attributes such as overcontrol that result in less

child autonomy, and to a lesser extent lack of warmth or rejection resulting in insecure attachment, are the identified traits. Researchers are also beginning to realize that parenting is not the exclusive domain of mothers and are now including fathers or partners in their studies. In addition, the literature is expanding rapidly to acknowledge that parenting factors alone cannot account specifically for this disorder and others. The research community is beginning to recognize that a complex and multifarious route involving many environmental factors, including parenting traits, is probably the root cause of why some people develop SAD.

Parental psychopathology

Evidence suggests that one reason for the variance in the prevalence of SAD is due to shared environmental risk factors such as parenting and parental psychopathology (Ollendick and Hirshfeld-Becker 2002; Rapee and Spence 2004). Although the role of parenting has emerged as a small but significant risk factor, less research has focused on parent psychopathology. This lack of attention may be due, in part, to the difficulty in separating out genetic contributions from environmental impact. Despite this, there is some evidence that parents with specific disorders contribute significantly to an increased rate of many childhood and adolescence disorders, potentially even within a particular developmental pathway (Stanger et al 1999; Henin et al 2005; Burstein et al 2006). Some studies consider the role played by each parent to be unique and related to child age effects and specific parent mental health problems (Connel and Goodman 2002). Notwithstanding some contradictory evidence, there is also extant research to suggest that well-known parenting behaviors, such as overcontrol, mediate the relation between parent psychopathology and child problem behavior (Whaley et al 1999; Lieb et al 2000; Bögels et al 2001; Spence et al 2002; Lindhout et al 2006). Further research also speculates on the potential directionality of this relation, where the child's temperament dictates the style of parenting and not the parent psychopathology (Moore et al 2004). The few studies investigating SAD have uncovered significant correlations between mother and child psychopathology (Bruch 1989; Lieb et al 2000; Bögels et al 2001). Although some schools of thought suggest that certain parenting styles like overcontrol are part of the process, others hypothesize that alternative family rearing aspects such as a chaotic environment may be more important. Despite some uncertainty about the mechanism of transfer, SAD parents seem to have significantly more SAD children than is explained by any genetic contribution.

Psychopathology in general

Children whose parents have a history of psychopathology have higher rates of internalizing (and externalizing) problems than those whose parents do not have this history (Connell and Goodman 2002). Consequently, researchers consider parents with psychopathology to be a potential risk factor for the development of behavioral problems in children. Although genetic factors of many individual psychopathologies show relatively constant rates of transmission from parent to child through family and twin studies (30%–35% for anxiety disorders; McClure et al 2001), they do not completely account for the varying rates in behavior problems. Researchers believe that there are other psychosocial factors at play in these circumstances. For example, parents suffering from mental illness may engage in a different parenting style because of their psychopathology. Some evidence for this notion was found in a study comparing three groups of children aged 2 through 18: children referred to mental health services, children not referred, and children with cocaine and opiate dependent parents (Stanger et al 1999). Using the age appropriate Child Behavior Checklist (CBCL) questionnaires completed by an adult fulfilling the parent role and, controlling for age, gender, informant, ethnicity and SES, children referred to mental health services had the highest scores for internalizing (and externalizing) problems. Children raised by drug abusers had significantly more psychopathology than the control group of non-referred children. Stanger et al (1999) concluded that children raised by a parent with psychopathology appeared to have greater overall risk for behavior problems than the non-clinical population. Further evidence of parent psychopathology increasing the risk of problem behavior in offspring came from another research laboratory studying parents with bipolar disorder (Henin et al 2005). Using the DSM-IV age-based clinical interview to obtain diagnostic information, they assessed children aged 4–18 for behavioral problems. Children of parents with bipolar disorder had elevated rates of psychopathology, including internalizing (and externalizing) problems, and significantly more impaired global assessment functioning (GAF) scores. As well, the researchers noticed a developmental course of psychopathology starting with the onset of ADHD, ODD, anxiety disorders and depression in early to middle childhood, followed by bipolar disorder, obsessive compulsive disorder (OCD), panic disorder, agoraphobia and substance abuse disorder in adolescence. These findings provided support for the hypothesis that children of parents with psychopathology, are at a significantly increased risk of developing a wide range of severe psychiatric disorders.

In one other study exploring the non-genetic link between parent and child psychopathology, experimenters looked at the risk of internalizing problems in children of substance-abusing parents, paying particular attention to their parenting role (Burstein et al 2006). Using standardized self-report questionnaires, parents answered queries about their own anxiety and depression symptoms, their parenting behaviors, and their child's behavior. Results were as follows: mothers reported higher levels of internalizing problems than did fathers; negative parenting did mediate the relation between a parent's internalizing problems and a child's affective problems but not anxiety problems; high parent involvement did not have an influence on this relation; negative parenting did not mediate the relation between a parent's externalizing problems and a child's internalizing problems; and finally, high parent involvement did moderate the relation between parent externalizing problems and child internalizing problems. The results of the study provided evidence for a complex interaction between parent psychopathology, negative parenting, high parent involvement, and child problem behavior. This pattern in turn supported a vulnerability model for the development of psychopathology that considers the differential impact of environmental factors on the development of a particular anxiety disorder. If further research could tease apart these differential impacts and their complex interactions, clinicians might be able to identify vulnerable populations and implement prevention and treatment programs targeted to their express needs.

Father psychopathology

Research in the last several decades has focused primarily on the maternal influence in the development of child problem behavior. Although there is evidence to document the father's impact on normal child behavior, researchers have either disagreed on their contribution to psychopathology or neglected it (Connell and Goodman 2002). To ameliorate this omission, a meta-analysis assessed the association between mother and father psychopathology and the presence of internalizing (and externalizing) disorders in children (Connell and Goodman 2002). Examination of the assembled data showed that internalizing problems in children were associated with psychopathology in the mother, while externalizing problems linked with psychopathology in both parents. The analysis also alluded to a potential age effect where psychopathology in fathers became more salient later in the children's development. Moreover, specific parent mental health problems were predictors of risk for the child namely internalizing problems in children closely related to maternal

versus paternal depression. The researchers postulated that these gender differences might reflect the differences in prevalence rates for mental health disorders; clinicians diagnose women more frequently with depression and anxiety, and men with alcoholism and antisocial personality disorder (ASPD). Since exposure to different psychopathologies is likely to be unequal for each child in the family environment, risk may be associated with these gender specific prevalence rates. Overall, Connel and Goodman offered convincing evidence that the risk for psychopathology in children is associated with both the mother and father. If their analysis is extended to SAD and other anxiety disorders, future research on potential environmental risk factors would be more persuasive if it included the father's contribution, especially during later child development, or perhaps, in early development with the father as the primary caregiver. It would not be surprising to find that fathers make a minor, but nonetheless important, contribution to the array of risk factors implicated in the development of SAD.

Parent anxiety disorders

In the last ten years, research on anxiety disorders has started to compile evidence to suggest an association between anxiety-disordered parents, their parenting style and child anxiety problems. Lately, one group of researchers has speculated that if parenting styles were part of the mechanism for transmission of anxiety disorders, then overcontrolling and less warm parenting styles would be more prevalent amongst anxiety-disordered parents (Lindhout et al 2006). Through psychiatric assessment and a self-report childrearing questionnaire, Lindhout et al (2006) found anxiety-disordered mothers and fathers to be significantly less nurturing (less warm) and more restrictive (overcontrolling) in their rearing style than non-disordered parents. Child-report results concurred that anxiety-disordered parents were overcontrolling. Although there was no comparison group of parents with psychopathology to assess whether these parenting traits were specific to anxiety-disordered parents, the study did suggest a potential mechanism for transgenerational transmission of anxiety disorders. Other groups have taken this association a step further by exploring the role of parenting in the relation between parent and child anxiety disorders (Whaley et al 1999; Lieb et al 2000; Bögels et al 2001; Spence et al 2002). Based on a multiple informant approach, which improved the reliability of reported rearing behaviors, mothers' anxiety was tied more closely to their children's anxiety than that of the fathers' anxiety (Bögels and van Melick 2004). In addition, the data strongly linked the parental rearing practice of

overprotection with child and parent anxiety levels. Bögels and van Melick correlated paternal overprotection with child anxiety, and maternal overprotection with mother anxiety. McClure et al (2001) reported contradictory results. They did not find evidence to support parenting behavior as a mediator in the relation between maternal and child anxiety disorders. Despite this disagreement, they did find that maternal, but not paternal, anxiety disorders significantly predicted the presence of anxiety disorders in children. However, correlational analysis showed maternal anxiety disorders did not predict levels of psychological control in parenting behaviors. On closer inspection, the contradictory results likely arose from their use of a single informant, the child, to report on parenting behavior. The study by Bögels et al (2004) had three informants on rearing dimensions: the mother, father and child. This multiple informant approach appeared statistically more reliable, giving added weight to their suggestion of the potential mediating role that parenting factors play in the transmission of anxiety from parent to child.

Another group altered their direction of inquiry to examine the interactive behaviors between mother and child to see if they were associated with either the mother's or the child's anxiety, or a combination of both their anxieties (Moore et al 2004). In this experiment, results from clinical interviews and observations of mother-child dyad interactions indicated anxious and non-anxious mothers of anxious children were less warm, more controlling, and more likely to catastrophize. The authors speculated on the directionality of the mother-child interaction given these results: parent anxiety might not be driving an overprotective parenting style that then fosters child anxiety, but rather an anxious temperament in a child might be shaping a parent's behavior to be protective.

Parent social phobia

Literature exploring the connection between parent mental illness and child SAD is scarce, although there is some evidence that adult social phobics recall their parents isolating them from outside social experiences, stressing the importance of others' opinions, and limiting family socializing both in and out of the home (Bruch 1989). Whether this parent behavior translates as parent psychopathology, specifically SAD, is uncertain. Two recent studies have tried to address this question. One study assessed the relation between SP, parental psychopathology, parenting style (rejection, emotional warmth and overprotection), and family function (problem solving, communication, roles, behavioral control, affective responsiveness and involvement) in a community population of adolescents (Lieb et al 2000).

Researchers gathered most of the information from youths through structured interviews for diagnostic assessment and through questionnaires for parenting style and family functioning. They diagnosed parents through independent interviews. Data supported a strong association between parental psychopathology, particularly SP and depression, and SP in offspring. Parenting style, but not family function, was also associated with the development of SP in the adolescents. These results also supported a multiple familial risk factor model in the development of SP, where parental SP might be one small but important component. Another study published results on a similar investigation into the relation between SP, parental rearing practices, and parent psychopathology (Bögels et al 2001). In comparison to the first study, this research found little support for a link between parenting behaviors of rejection (less warmth) and social fears in children. Although mother-overcontrol as perceived by the child predicted child SAD, the SAD child did not differ from the control group in amount of exposure to overcontrolled parenting. Poor family sociability as judged by the child and mother, however, significantly predicted SAD in the child. Furthermore, data revealed maternal SAD strongly predicted child SAD. However, most of the traditional rearing behaviors associated with child psychopathology were not relevant to the development of SAD in this case study. The authors concluded that it was only the child's perception that they had interpreted, and perhaps other untapped family rearing practices could be involved in the etiology of SAD, such as underprotection or a chaotic family environment. Taken together, these two studies proposed parent psychopathology as a partial risk factor for the development of SAD, but they left the mechanism of transfer ambiguous. In fact, it may well prove difficult for research to delineate a proportion of environmental risk to parent psychopathology, as it is inextricably interwoven with parenting and other shared and interacting environmental factors.

Summary

There seems to be little doubt that parents with psychopathology influence their children's emotional and mental development in a manner different from those parents who have no mental health issues. What appear to be uncertain, however, are both the relative significance of this potential risk factor and the mechanism of transmission. Our understanding remains limited, but research has established several connections. Anxious parents are more likely to have anxious children, and mediation of this relation could be through specific parenting behaviors like overcontrol. The

psychopathology of fathers and mothers likely contributes uniquely to their child's psychopathology since a difference exists in the prevalence of disorders between the genders. In addition, a father's influence usually begins in late childhood and early adolescence. Finally, investigations of SAD show SAD parents have significantly more SAD children than can be attributed to genetics alone. Accordingly, any thought of improving the future of a child with SAD must include interventions at the parental level where parents receive both the support they may need for their own problems and advice regarding healthy child emotional development.

Attachment

One family factor that likely contributes to the development of anxiety disorders is attachment. This concept refers to the type of enduring relationship that is established between children and their primary caregiver in the first year of life, as measured through a well-known laboratory assessment called the Strange Situation (Ainsworth et al 1978). Originally, attachment theorists identified three types of relationships through this laboratory procedure: the secure, insecure-resistant (ambivalent), and insecure-avoidant. Later on, a fourth was recognized and called the insecure-disorganized. Within the context of the family environment, researchers consider attachment significant to the pathogenesis of childhood anxiety disorders; they suggest that insecure relationships, as opposed to secure, predict the development of anxiety disorders (Bögels and Brechman-Toussaint 2006; Dadds and Roth 2001, cited in Muris 2006b). Although longitudinal research has inferred that insecurely attached children more regularly display anxiety disorders (Warren et al 1997, cited in Muris 2006b), this literature search did not find any work that established a connection between insecurely attached children and the development of SAD. Consequently, although the quality of the parent-child bond may influence a child's social anxiety, there seems to be no research to substantiate the specificity of this association.

Attachment and anxiety

A number of studies have examined the relation between attachment style and internalizing behavior problems and have verified a connection between insecure attachment and later disorders (Green and Goldwyn 2002). The authors of these papers also identified research that explicitly associated the ambivalent attachment style with social withdrawal in middle childhood (Renken et al 1989, cited in Green and Goldwyn 2002) and anxiety in late adolescence (Warren et al

1997, cited in Green and Goldwyn 2002). More predictive than these types of attachment, however, was the disorganized type, which linked with higher overall psychopathology by late adolescence, including internalizing disorders (Lyons-Ruth 1996, cited in Green and Goldwyn 2002). Although it is unclear how the insecure attachment factor contributes to SAD, it appears to provide an unmistakable marker for risk of, and vulnerability for, later psychopathologies.

Attachment and parenting styles or behaviors

Although attachment style is almost certainly a marker for later psychopathologies, the lack of specificity for any one disorder has prompted new research to investigate its association with other familial factors. Adverse parental rearing is one factor in particular thought to play a role in the etiology of internalizing (and externalizing) problems of children. In a recent study, researchers examined the link between attachment style, perceived parental rearing behaviors and psychopathology in non-clinical children aged 9–12 (Roelofs et al 2006). The children completed three different questionnaires: one measuring anxiety and depression symptomology, another determining perceived parental rearing behavior, and the last one assessing attachment style. Data analysis revealed that perceived parental rearing behaviors to be significantly associated with internalizing (and externalizing) disorders, while attachment style seemed to play a smaller role in these two groups of problem behavior. Also noteworthy was the gender specific finding that negative parenting factors associated with fathers had a greater impact on symptoms in boys versus girls, and these same parenting factors in mothers affected the girls more profoundly. These results from Roelofs et al (2006) confirmed and strengthened the relation found in previous studies where perceived negative parental rearing and insecure attachment style were found to account for a unique part of the variance in internalizing (and externalizing) symptoms (Muris et al 2000; Muris, Meesters et al 2003).

Summary

Studies on attachment and anxiety consistently show an association between insecure attachment and development of anxiety. However, the lack of specificity in this relation to anxiety disorders makes it difficult to unravel the distinctive contribution of insecure attachment bonds. Current research has attempted to introduce other potential family risk factors into the equation with the hopes of better understanding the complexity of family interactions. Correlational data suggest

that insecure attachment style and negative parental rearing behaviors are each uniquely significant to the severity of internalizing (and externalizing) symptoms, but that their individual contributions are still difficult to tabulate. Thus, at present, there seems to be no research linking insecure attachments specifically to the etiology of SAD.

Adverse life events

Pre- and perinatal

Scant research is evidenced in the extant literature regarding the influence of pre- and perinatal adverse life conditions and their involvement in the pathogenesis of anxiety disorders. Neither is there mention of this topic in the most up to date reviews discussing possible developmental profiles of SAD (Ollendick and Hirshfeld-Becker 2002; Neal and Edelman 2003; Rapee and Spence 2004). This void is surprising given that starting from conception, the mother and fetus share the same environment. The research is just now starting to explore this area of investigation with data supporting tentative connections between pre- and perinatal maternal stress and later child anxiety disorders, including SAD (O'Keane and Scott 2005; Phillips et al 2005). To explain these results, O'Keane and Scott (2005) have postulated a new neurobiological model. Some groups are even looking at whether psychotropic medication during pregnancy might ameliorate the deleterious effects of maternal psychopathology on the fetus during pregnancy. Although a relatively new area of investigation, researchers have hypothesized that the pre- and perinatal environment may well affect the fetus-child and possibly comprise one of the many possible environmental risk factors involved in the development of SAD.

Maternal stress and anxiety

Some research has focused on the possibility that environmental risk factors may start acting on the fetus and newborn as antecedents of anxiety disorders and other psychopathologies. Studies at first did not identify any obstetric complications as possible precursors for later mood disorders, however, when the cluster of complications (viral or bacterial infections, septicaemia, birth complications and maternal psychopathology, and stress) were teased apart, a different picture surfaced (O'Keane and Scott 2005). Investigations started to focus on pre- and peri-natal maternal stress and child anxiety. One prospective study collected data on early maternal stressors at discrete points over time from pre- and post-natal to five years of age, and found them significantly predictive of anxiety disorders in later adolescence; they

included maternal prenatal stress, multiple maternal partner changes, economic hardship, maternal and partner deviance, childhood illness, and maternal stressful life events (Phillips et al 2005). These data confirmed earlier work by Allen et al (1998) who found retrospective reports on similar pre- and peri-natal events predicted the future occurrence of anxiety disorders in adolescent offspring, especially infant illness in the first 12 months of post-natal life and, poor maternal obstetrical history involving miscarriages and stillbirths thought to be associated with maternal anxiety and stress. In another report, researchers looked at maternal stress and anxiety during pregnancy as having possible long-term effects on behavioral and emotional problems in children (O'Connor et al 2002). Based on maternal reports, they found a connection between antenatal maternal anxiety and the manifestation of these problems in children at age 4. Even when these researchers controlled for socioeconomic status, postnatal maternal depression, and other obstetrical risks, antenatal psychosocial stress and anxiety still significantly predicted behavioral and emotional problems in the child.

Mother-fetus neurobiological model

With emerging consensus that maternal psychosocial stress during pregnancy is one probable antecedent of later anxiety disorders in children, a new neurobiological model has arisen to explain these results (O'Keane and Scott 2005). The model hypothesizes that maternal stress can permanently change the developing hypothalamic-pituitary-adrenal axis (HPA) of the fetus. During periods of stress, the mother responds biologically with high levels of cortisol that likely cross the placenta. In the fetus, cortisol potentially acts to inhibit intra-uterine growth, initiate early birth, and alter the glucocorticoid receptors in the brain. With this alternation, the belief is that the HPA is set constantly on 'high' resulting in a constant endocrine stress response theorized to correspond with the associated increase in negative emotional behavior of the child.

Psychotropic medications during pregnancy

Corresponding to the idea that untreated anxiety and mood disorders in the mother may expose the fetus to unfavorable conditions, there is interest in whether administering psychotropic medications during pregnancy harms the fetus. One recent article described results on the association between prenatal exposure to psychotropic medications and internalizing behavior in children aged 4 and 5 (Misri et al 2006). The study made use of parent and teacher reports, as well as observations of mother and child interactions, to assess

levels of internalizing behaviors in those prenatally exposed to selective serotonin reuptake inhibitors (SSRI). Exposure to SSRIs was not associated with internalizing behaviors at 4 years of age. However, increased symptoms of maternal anxiety and depression did link to internalizing behaviors in the children. The authors suggested further research to resolve whether maternal psychiatric disorders, the medications, or both, affected the child's outcome.

Summary

Although a recent review of vulnerability factors for anxiety disorders maintains that there is no evidence that pre- or perinatal factors play a role in the etiology of anxiety disorders (Merikangas 2005), a closer look at the scientific literature suggests that, in fact, there may be evidence to refute this contention. The work reviewed above connects antenatal maternal stress and anxiety to later child internalizing behaviors. Although this research is small in scope and needs careful design to avoid confounding factors such as perinatal parenting influences (Barlow 2002), further longitudinal studies could combine genetic, prenatal and postnatal experiences to strengthen this connection. As well, it is conceivable that further research will support the new neurological model of prenatal stress that may be the genesis of many anxiety disorders and other psychopathologies. The concept of multifinality dovetails nicely with this line of research; the etiological factor of maternal stress and anxiety may well lead to several psychopathologic outcomes, depending on the person and his or her context. As this area of study expands, it may prove to be very important information in the treatment regime of anxiety disorders such as SAD. In theory, interventions at the very beginning of life could obviate the need for them later in childhood or adulthood.

Traumatic events

There are environmental risk factors for SAD identified as stressful life events because they place increased pressure on the developing child and potentially result in adverse outcomes. Some of these events fall within the purview of 'typical' family functioning such as divorce, death, illness, natural disasters, changing schools, and academic failure. Other aversive events are labelled as part of 'normal' modes of functioning but they are not really, such as bullying, familial violence, sexual and physical abuse. A number of studies have looked into these traumatic events and linked them consistently with the development of anxiety disorders and SAD in particular. Since traumatic events are well-documented for inducing stress and subsequent anxiety, these correlations

are not surprising. A more perplexing question is why only some children develop anxiety since life is by nature aversive. Although research has yet to determine causal roots for SAD, there are some tantalizing hints in the newly developed concept of risk and resilient factors whereby protective factors successfully aid a child in coping with anxiety provoking situations (Manassis et al 2004; Muris 2006b).

Adverse conditions

Conditioning through traumatic events is a mechanism implicated in the acquisition of fear responses (Pavlov 1928; see also Rosen and Schulkin 1998, for a review). Studies focused on this area of research have looked for associations between adverse life events and anxiety symptoms. Tiet et al (2001) collected from their sample of youth, the self-perceived adverse events and psychiatric disorders from the previous year. Overall, they found that groups of psychiatric disorders were more closely associated with some adverse life events than with others. Concerning SAD in particular, they saw a strong association between the arrival of a new stepparent and the development of an overanxious disorder (now subsumed under generalized anxiety disorder in the DSM-IV; APA 1994) in girls, and changing schools with socially phobic boys. Grover et al (2005) looked more closely into a longitudinal association between general anxiety and trauma in first grade children, and when they reached seventh grade. Using multiple informants, they found that those who had experienced losses through death and separation, academic failure and a more negative family environment showed a greater degree of anxiety at both age levels. Data also revealed that the greater the number of total risk factors, the higher the level of anxiety. In an attempt to connect the vicarious learning of fear with SAD specifically, Bandelow et al (2004) collected retrospective reports from adults on their separation from parents, parental marital discord, sexual abuse, familial violence, and childhood illness. All these events linked with higher rates of SAD, with separation experiences having the highest correlation. In addition, there was no evidence suggesting that any age between 0 and 15 years was particularly sensitive to any one risk factor. In another study, Chartier et al (2001) detected a gender difference in response to adverse life events and the development of SAD, where females were more likely to report sexual abuse and boys more prone to report contact with the juvenile justice system. This same research also pointed to a potential difference in the role of risk factors between the subtypes of SP, where the adversities reported were more strongly associated with complex (generalized)

versus talking-only (non-generalized) SP disorders. As a result, through the data collected, it is becoming evident that many adverse life events have some role to play in the etiology of SAD, although the precise understanding of this association is still unclear.

Sexual and physical abuse

Some groups have targeted their research toward looking at particular aversive life events, such as sexual and physical abuse, as antecedents of SAD to understand better the possible environmental causes of this disorder. After controlling for demographic and family background variables, Freerick and Snow (2005) showed that childhood sexual abuse explained a small but significant amount of women's SAD for avoidance and social distress. Higher scores arose when the women's abuse included actual or attempted intercourse, occurred early in life, frequently, and involved psychological pressure. This result confirmed previous studies that linked early sexual abuse and SP, particularly in women (Magee 1999; Dinwiddie et al 2000). Research also correlated physical abuse with later development of phobia anxiety disorders, such as SAD (Magee 1999). However, this relation was considerably less significant, and results connected panic disorder more closely with physical abuse (Stein et al 1996; Safren et al 2002). What has become increasingly clear through these investigations is that neither sexual nor physical abuse works in isolation from other factors. Developmental trajectories of anxiety disorders, such as SAD, are not necessarily predetermined in the presence of sexual and physical abuse when resilience effects come into play. Furthermore, an interaction between these risk and resilience factors appears to change the developmental outcome of children exposed to various adverse life events. One prospective study gathered information on both risk and resilience factors on children from kindergarten until grade 8 (Lansford, Malone, Stevens et al 2006). The results showed resilience factors of unilateral parental decision-making, reduced early stress, diminished adolescence stress, and an enhanced adolescence hostile attribution moderated the connection between early physical abuse and internalizing behaviors. This work directs us to consider additional information on environmental resilience factors of SAD, and not just risk factors, to have a better understanding of the pathogenesis of this anxiety disorder.

Peer relationships, teasing and bullying

Another area of investigation into specific risk factors of SAD is that of peer relationships, teasing and bullying. Severe and traumatic bullying appears to be a likely determinant for

anxiety, especially social anxiety given the negative social interaction between the bully and victim. Several research groups of late have looked into a possible connection. Gladstone et al (2006) studied an adult population from a mood disorder clinic where the participants were assessed using structured clinical questionnaires through self-report and interviews. In addition to assessing their moods in the present day and retrospectively, researchers also looked for a history of childhood bullying and other adverse environmental or personality correlates. Those correlates that particularly associated with reports of bullying were parental overcontrol, illness or disability, and the likelihood of having an early inhibited temperament. Independent of other childhood risk factors, the study also found a strong relation between depression with comorbid anxiety, including SP, and childhood bullying. Others have looked into the connection between bullying and the development of SAD because the defining criterion of this disorder is fear of social situations in which embarrassment or humiliation occurs (Neal and Edelman 2003). Teasing and bullying interactions clearly represent such a situation. McCabe et al (2003) found a connection between anxiety disorders and a history of teasing in childhood and adolescence, and this relation was significantly higher for those diagnosed with SP than those with OCD or panic disorder, with or without agoraphobia. To avoid confounding their results, McCabe et al (2003) separated the participants into four distinct anxiety subgroups, with three of them lacking comorbid SP, before analysis of the data. Their results were consistent with previous research that linked childhood teasing with social anxiety in adulthood through retrospective reports (Roth et al 2002). As well, other work correlated high school adolescent social anxiety with poor peer acceptance (La Greca and Lopez 1998) where higher levels of social anxiety linked strongly with poorer social functioning and fewer friendships, particularly in girls. Thus, it appears that peer relations, bullying and teasing may play some role in the etiology of SAD. However, it is uncertain whether this connection is causal or how it integrates in the environmental etiological model of SAD.

Marital discord

Finally, research has focused on the idea that parent divorce and separation may influence the trajectories of internalizing behavior development. Serious family conflict, which arises out of these particular situations, may affect children's adjustment over time (Spence et al 2002). Lansford, Malone, Castellino et al (2006) investigated this behavioral adjustment or outcome in children starting in kindergarten and

continuing through to grade 10. Subdividing the children into two groups based on whether their parents were divorced, they collected teacher and mother reports on child behavior each year. Results suggested that early parent divorce was associated with the development of internalizing problems, whereas later parent divorce corresponded to poorer grades in school. These data replicated previous work on behavior adjustment to divorce in between fourth and sixth grade (Wood et al 2004). Multiple informant behavior ratings in the work of Wood et al (2004) showed that those children whose parents divorced had significant and ongoing adjustment difficulties that translated into internalizing (and externalizing) behaviors, as compared to those who lived with their married parents. In addition, they also found that depressive or withdrawn parenting seemed to play a role in the child's adjustment problems, and that the influence of this parenting trait diminished as the child transitioned from preadolescence to early adolescence. The impact of marital quality was further studied in a sample of non-clinical 5 and 6 year olds (Peleg-Popko and Dar 2001). The mothers of these children completed questionnaires on marital quality, family adaptability and cohesion, and child fears and social anxiety. The findings suggested that rigid, fused families or low quality marriages (marital discord) might be risk factors for high levels of fears and social anxiety in children. Although all of the results above did not specifically connect marital discord or divorce with the development of SAD, negative environments often arise in divorced households and may well be a contributing risk factor in the trajectories of SAD and other anxiety disorders.

Summary

In review, there appear to be a number of different traumatic events that may be part of the conditioning response for SAD. Events that have been recently studied and thought to contribute to the environmental etiology are losses such as death or separation, negative family environment or marital discord, family violence, sexual and physical abuse, childhood illness and bullying. Although there has yet to be a causal association established, the current research does point to and aid in developing possible interventions that could alter the developmental course of SAD (Chavira and Stein 2005).

Societal and cultural factors

Socioeconomic status (SES)

Little mention of SES is discussed as a potential risk factor in some of the most current reviews on SAD (Ollendick and

Hirshfeld-Becker 2002; Neal and Edelmann 2003; Chavira and Stein 2005). Yet, the consensus in the literature is that rates of anxiety disorders are greater for those with a SES disadvantage (Merikangas 2005). This potential risk factor for SAD plays out even more dramatically in developing countries (Vorcaro et al 2004; Muris, Loxton et al 2005). Research suggests that the burden of extreme poverty seems to affect social functioning quite strongly. Therefore, it is important to keep sociodemographic variables such as SES in mind when investigating possible environmental antecedents of SAD, especially in nations with extreme deprivation.

Developed versus developing countries

The relative effect of SES in developed as opposed to developing countries shows interesting differences. Research in Australia, for example, suggests poverty in the first five years of life influences the development of high internalizing problems when there is associated maternal depression (Bor et al 1997, cited in Spence et al 2002). However, a different study found poverty to be considerably predictive of later high anxiety (and depressive) symptoms in adolescence after controlling for marital discord and maternal psychopathology (Spence et al 2002). Although these two studies were not quite in line with one another, Spence and colleagues' evidence partially corroborated earlier findings that SP was associated with socioeconomic circumstances when other psychiatric disorders were not at play (Schneier et al 1992, cited in Vorcaro et al 2004). More concerning is the effect of SES in developing countries where socioeconomic conditions are of vital importance to everyday survival. A recent paper on this topic publicized the prevalence of SP and its associated factors in a Brazilian community (Vorcaro et al 2004). Using community samples, the researchers collected information through interviews on a variety of variables including sociodemographic characteristics and health problems. Data analysis revealed a high prevalence of SP, similar or greater than that observed in developed countries, and an associated poorer health status. Additionally, there was a very strong link between SES and SP, which strikingly exposed the major social inequalities of the impoverished Brazilian community. A replication of this result occurred in a South African study, where colored or black youth developed SAD in connection with parenting styles associated entirely with SES (Muris, Loxton et al 2005). The strength of these results makes it imperative to consider SES as one probable risk factor in the etiology of SAD, most especially in developing nations and as part of a global perspective on this anxiety disorder.

Summary

It appears SES is one of many possible antecedents in the development of SAD. Its potential mechanism, however, is not very clear. In developed countries, research suggests SES may or may not be associated with other psychopathology, and it is conceivable that poverty could be either a cause or the result of psychopathology. This potentially reciprocal relation needs further elucidation. In developing countries, the association is much stronger, and treatment programs that address poverty may ameliorate some of the worst psychopathology, and particularly SAD. Thus, the relative impact of SES seems to depend on context. Further research in this area could help expand our understanding of SAD in a global framework.

Culture and society

Culture is described as the customary beliefs, the set of collective attitudes, values, and practices, or the characteristic features of everyday life that are shared by people in the same place or time. By virtue of this definition, conventions espoused by a culture shape the society. In turn, the society and all of its social rules likely influence emotional development, but the mechanism is obscure. Research investigating these social norms in different countries found that they correlate to different prevalence rates of social anxiety (Heinrichs et al 2006). As well, the construct of social anxiety seemed to be culturally defined (Kleinknecht et al 1997). When confining a study of anxiety disorder symptoms inside one country, researchers also revealed differences in prevalence due to ethnicity (Muris, Loxton et al 2005; Vendlinski et al 2006). Another laboratory decided to study anxiety as it correlated to different birth cohorts as groups representing social trends within a country and found differences between the generations (Twenge 2000). All of these studies make it clear that any discussions on the antecedents of anxiety, and SAD more specifically, should include cultural and societal norms as potential contributors.

Cultural norms

At present, cross-cultural studies divide the world into two groups categorized as collectivistic and individualistic cultures (Hofstede 1984, cited in Heinrichs et al 2006). Collectivistic societies are those whose people pursue harmony within a group to the virtual exclusion of their own individual needs. Individualistic societies embrace individual feelings and thoughts that may supersede the needs of the group. Accordingly, more rules are thought to guide social behavior in collectivist societies to support and protect the group

identify than in individualistic cultures (Heinrichs et al 2006). Heinrichs and colleagues (2006) assessed whether these perceived social norms shaped the level of social anxiety in their respective cultures. Eight countries participated in a cross-cultural collaboration with three registering as collectivistic and five as individualistic. Participants responded to vignettes based on societal norms across cultures and completed questionnaires assessing levels of social anxiety and fear of blushing. When commenting on cultural norms within their own societies, data showed that collectivistic participants displayed more acceptance of socially reticent and withdrawn behavior than did individualistic participants. In contrast, when asked about their personal perspectives, participants from both individualistic and collectivistic countries were equally accepting of these same behaviors. Collectivistic contributors also reported higher levels of SAD and more blushing. In conclusion, the correlation between cultural acceptance of withdrawn behavior and greater levels of SAD in collectivistic nations provided initial evidence that cultural norms were associated with the development of this disorder.

Another study also investigated the impact of cultural factors on SAD. In this case, experimenters examined two different culturally defined forms of social anxiety, the DSM-IV SAD in the United States and *Taijin Kyofusho* (TKS, heightened concern over offending others through behavior or appearance) in Japan (Kleinknecht et al 1997). Factor analysis of SAD, TKS, and self-designation as independent or interdependent elucidated a different set of predictors for these two culturally defined forms of social anxiety but also revealed some correlations between high scores of TKS and SAD. The authors hypothesized that culture-mediated the expression of SAD and suggested that both forms existed in each country. Thus, a clearer picture has evolved focusing on cultural specific types of anxiety that may have core anxiety characteristics in common. Future work should focus on identifying core versus culture specific symptoms to clarify the role that culture plays as a potential risk factor in the development of SAD.

Ethnicity

Within the boundaries of many nations today, various religious, linguistic, or cultural groups coexist. These different groups are subject to the same social and legal structures instituted by a country, but often their identities remain separate and intact. It is alongside this factor, ethnicity, that a recent paper published results on DSM-defined anxiety symptoms and perceived parental rearing in South Africa

(Muris, Loxton et al 2005). As mentioned above in the parenting section on culture, investigators found significant differences in anxiety across different ethnic groups. Colored or black youth displayed appreciably higher anxiety levels than white youth, and this pattern was associated with perceived parental rearing behaviors. The authors also coupled the parenting behaviors with the previous South African Apartheid regime, where being part of the colored or black ethnic group dictated cruel treatment at the hands of the ruling white party. Although being part of one ethnic group linked the youth to higher levels of anxiety, adverse life conditions may also have been additionally involved in the development of these symptoms. Other research looked at a more targeted role for ethnicity in internalizing disorders. Vendlinski et al (2006) contended that ethnic differences play a potential part in moderating the connection between poor family functioning (marital conflict and lack of warmth) and anxiety. Through interviews and questionnaires, this group discovered that the African American background reduced the strength of the association between poor family functioning and internalizing symptoms, whereas the European American background increased this association. When they analyzed the results without regard to ethnicity, family functioning was not associated with internalizing problems in seven out of eight tests. The authors proposed that this finding partially due to ethnicity being crucial to the understanding of family functioning and a child's adjustment. Although it is unclear how these factors are interacting, there is evidence that part of the mechanism for SAD development may involve ethnicity as a mediator.

Birth cohorts

In the field of psychology, scientists generally acknowledge that people from one generation to another think and behave differently. Research reflects this ideology in experimental design by undertaking cross-sectional studies in which age or birth cohort separates participants into groups. Because the environment is constantly changing, research tries to capture how these differences affect human behavior. Twenge (2000) was interested in how this played out in the areas of anxiety and neuroticism. He studied birth cohorts between the years of 1952 and 1993, and collected data from child and college age studies over this period. His two meta-analyses found that Americans today have significantly higher levels of anxiety, and the average child of the 1980s had substantially more anxiety than the child psychiatric patient of the 1950s. This growth in anxiety over time correlated with increases in measures of environmental dangers

and decreases in recorded social connectedness. Twenge argued that the potential impact of the larger sociocultural environment on psychopathology was important beyond the boundaries of individuals, their families and genetics. Although his data included information on all anxiety disorders, it was indicative of trends within the spectrum of SAD and pointed to possible predictors for this disorder.

Summary

The literature does not reflect potential cultural and social antecedents of SAD well, even though the few studies published to date on this issue indicate that cultural impacts are important to etiological discussions. Data that shows different prevalence rates are associated with different social norms, and social anxiety concepts appear culturally defined. Research in this area also suggests that core symptoms are likely between collectivistic and individualistic societies, but each society has unique and identifiable anxiety characteristics that are culture specific. Future investigations warrant a broader focus to include a more global perspective on anxiety and understanding of the development of SAD. In this way, clinical practice can target treatment to ethnically or culturally specific populations, especially when one population is at a disadvantage.

Gender roles

Gender differences

Reports consistently place females as having higher rates of SAD than males by a ratio of approximately 3:2 (Hidalgo et al 2001; Rapee and Spence 2004). In rare cases the ratio is equal between the sexes, but varying methodologies could account for these results (Degonda and Angst 1992, cited in Hidalgo et al 2001). In spite of the disparity between the genders, there has been little investigation into why there is a difference. To remedy this oversight, several groups have looked at male and female gender orientation and discovered an appreciably higher proportion of anxiety symptoms associated with feminine traits (Ginsburg and Silverman 2000; Muris, Meesters et al 2005; Palapattu et al 2006). The authors proposed a gender role theory to explain sex differences in severity of anxiety symptoms. Another team investigating this same topic found that family adversity affected the sexes differently in the onset of SAD (DeWit et al 2005). De Wit et al (2005) suggested that gender was a moderator of the effects of childhood family adversity thought to increase the risk of SAD. Although it is not altogether apparent how gender interacts in all situations to give identifiable risks in the development of SAD, initial

proposals suggest several psychosocial explanations such as gender socialization. Although none is yet verifiable, it does emphasize the importance of including gender and gender socialization in any examination of the etiology of SAD.

Gender roles

The concept of gender role is the degree to which a person demonstrates the traits, behaviors and attitudes consistent with a stereotypical female or male role. Those persons expressing fearfulness and anxiety are in line with the accepted behavior of the feminine gender role, while those who do not are displaying the socially appropriate masculine gender role. The gender role theory is one that embraces the idea that society socializes girls and boys differently to display these gender specific roles. This theory, then, potentially explains why we expect girls to be generally more fearful than boys. Several studies have investigated this phenomenon. The first to examine gender role orientation and anxiety in children assessed them between the ages of 6 and 11 for their self-reported masculinity and femininity traits, and anxiety (Ginsburg and Silverman 2000). As expected, the data supported a relation between gender role and fearfulness in children with anxiety disorders. More specifically, those with higher levels of masculinity showed lower overall fearfulness: however, levels of femininity did not correlate to anxiety. A different research group released results that augmented this preliminary, but partial, support for the gender role theory (Muris, Meesters et al 2005). They examined non-clinical referred children between the ages of 10 and 13 and found that femininity was positively, and masculinity negatively, associated with fear and anxiety. Criticism of this work, however, contended that masculinity was a substitute for self-esteem since both represented traditional masculine traits such as confidence and assertiveness (Ohannessian et al 1999, cited in Palapattu et al 2006). Additionally, Ohannessian et al (1999) proposed that any study assessing masculinity was really measuring self-esteem. In an attempt to clarify this argument, another group instituted further work to examine the relation between gender role orientation, self-esteem, and anxiety symptoms (Palapattu et al 2006). Palapattu and colleagues' data supported the gender role theory as an explanation for a higher incidence of anxiety symptoms in girls than factors of biological gender and self-esteem. Even so, self-esteem played a significant moderating role between femininity and anxiety. Hence, evidence appears to support the gender role theory of sex differences in anxiety. Whether this translates into explanations for gender differences in the prevalence of SAD is uncertain; however, it does lead to much supposition

and future research. In addition, these findings also have important implications for the gender specific socialization of children as it relates to treatment regimes for SAD or other anxiety disorders.

Gender responses to family adversity

Speculation also revolves around whether the gender of the child moderates or mediates the effect of family adversity risk factors for the onset of SAD. In several studies, gender based interactions were associated with sexual abuse and prediction of SAD, where significantly more female victims were likely to develop this disorder than males (Magee 1999; Dinwiddie et al 2000; Chartier et al 2001; Freerick and Snow 2005). Another study found gender composition of the parent-child dyad linked negative parenting factors to the development of internalizing problems; fathers had a greater impact on symptoms in boys as opposed to girls, and the opposite was true for the mothers (Roelofs et al 2006, see attachment and parenting section above). DeWit and his colleagues examined the gender differential in the onset of SAD and its moderating role on the indicators of childhood family adversities that potentially increase the risk of developing SAD (DeWit et al 2005). First, data indicated gender differences in the prevalence of SAD sub-types. Females exceeded the number of males with the generalized sub-type across all ages, while females only outstripped males in probability of developing the non-generalized subtype after 12 years of age. Second, increased risk of developing SAD was strongly associated with family adversity by gender. Males were twice as likely to develop both sub-types of SAD, or only the non-generalized sub-type, if they had grown up without a close and confiding relationship. Females were one and half times more likely to develop both sub-types of SAD if they experienced the effects of marital conflict growing up. As well, girls had an increased probability of developing generalized SAD if they reported physical abuse by their father. Lastly, females were twice as likely to develop non-generalized SAD if their mother suffered from mental illness, especially bipolar disorder. The authors emphasized the importance of considering gender differences in the effects of family adversity as a result of SAD sub-types. However, DeWit and colleagues also pointed to the limitations of their potentially biased retrospective data and stressed the need to broaden future research directions to include a prospective approach that demarcates variables of gender, severity of symptoms, and sub-types of SAD. Regardless of these contentions, gender differences in SAD raised by these results indicate that future treatment should target gender specific outcomes.

Summary

Evidence has long supported higher prevalence rates of SAD in females versus males. More recently, gender differences have been associated with negative child-parent interactions that likely result in SAD. However, in other than a few experiments, little research has focused specifically on sex differences in the onset and development of this disorder. More often, the gender differences studied are a by-product of other research goals, where the researchers analyze all the variables multifactorially and find correlations. Data derived from experiments designed explicitly to target gender links with SAD, perhaps investigating gender socialization parameters, might be more informative. Even though research is limited, what has become apparent is that female gender orientation is a potential risk factor for SAD. Furthermore, adverse life events that possibly promote the development of SAD are gender specific; girls respond negatively to martial conflict, maternal mental illness, and physical abuse, while boys react poorly to the lack of a close and confiding relationship. It would be of practical interest to delineate these differences more precisely to clarify the gender patterns that are involved in the pathogenesis of SAD.

Discussion

Limitations, implications, and future directions

What is evident from this review of the recent extant literature on SAD is that the research methodology used over the last 20 years is unsystematic and needs standardization (Cartwright-Hatton 2006). The terminology is inconsistent making it difficult to compare studies or concepts. In many cases, there are no data for comparison because little research has been undertaken in the area of SAD. Furthermore, the SAD research community is presenting ideas that have yet to mature, especially since new information and concepts are frequently similar but often obdurately distinct as well. With the sizeable number of reviews on the potential etiology of SAD published in the last five years, the research community appears poised to focus ideas and refine research directions. Here are some thoughts for this endeavor.

Research should include proper controls to eliminate confounds or independent factors; longitudinal studies to assess cause-effect relations; prospective self-reports from a variety of informants; increased use of and uniformity in observational procedures and questionnaires; investigations into differences in anxiety across age groups into old age; and further purposeful international collaborations in the

effort to maximize the pool of participants and minimize costs. Longitudinal studies in particular could help elucidate a developmental pathway for SAD by following the trajectory of individuals over a significant period of time, especially over development. This direction would enhance our understanding of the complexity of interacting risk and resilience factors from birth or conception to adulthood, thereby helping to pinpoint actual causes and outcomes of this disorder. In addition, it would be extremely useful to access information from many informants on many fronts since this could eliminate some of the bias in response and provide a broader perspective on the disorder. No matter what approach is taken, further study is needed to illuminate the bigger picture of the involvement of environmental risk factors in the development of SAD.

In general, no single study or literature review from the last five years seems completely authoritative on the etiology of possible environmental risk factors of SAD. The strongest data to date are not sufficiently strong enough that other groups are not supplanting it with alternative data. There were contradictions and discrepancies in the research presented, which is most likely due to the relatively small amount of research done in this specific area, preventing the SAD community from reaching an assured consensus. As well, teasing apart the relative importance of *all* possible risk factors, be they genetic, cognitive, or environmental within a developmental perspective, is turning out to be a very complex scientific process. The particulars of the multimodal and integrative diathesis-stress model, hypothesized as the best fit so far, are persistently recondite. Moreover, although SAD research has weighed out the nuances of each risk and resilience factor, the members of the medical community must exercise caution in how they apply this information to treatment regimes. Gene-environment interactions have only been studied in relation to SAD in a very limited fashion. Without an understanding of the dynamic interactions between these two intimately connected spheres, we cannot hope to fully understand the etiology of SAD. Beyond this overarching lack of recent research on SAD, there are many factors of interest that could be explored further. These include, but are certainly not limited to, the presence of physical defects, pervasive cultural role models as portrayed in the global media, and perhaps most importantly, Internet communication as an insulating influence. Ultimately, there is no doubt that this work will provide proper guidelines for interventions that will prevent the disorder or, more realistically, improve the quality of life for those who suffer from SAD.

Conclusion

Our review of the environmental risk factors of SAD indicates that there are four general areas currently being studied. Parenting and the family environment are by far the best researched, and results show a connection between parental overcontrol and parental psychopathology with childhood SAD. Second, adverse life events such as sexual abuse, negative peer relationships and marital discord are also found to contribute to the etiology of this disorder. Third, SES and different cultural values, as seen between individualistic and collectivist societies or between different ethnicities, are also tied to SAD development. Last, consistently higher rates of female versus male SAD have been traced to the concept of gender roles and to gender differences in child-parent interactions. Although these connections have been established, there must be some caution practiced in the interpretation of these results, given the paucity of research in most areas except for parenting. Despite the obvious limitations in the science of SAD alluded to in the previous paragraphs, research is making great strides towards understanding the pathogenesis of this mental disorder. As a result, the research points to a multi-faceted process of environmental risk and resilience factors that are interrelated symbiotically in a developmental pathway to social anxiety disorder.

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