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The etiology of social anxiety disorder: An evidence-based model

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ABSTRACT

The current paper presents an update to the model of social anxiety disorder (social phobia) published by Rapee and Spence (2004). It evaluates the research over the intervening 11 years and advances the original model in response to the empirical evidence. We review the recent literature regarding the impact of genetic and biological influences, temperament, cognitive factors, peer relationships, parenting, adverse life events and cultural variables upon the development of SAD. The paper draws together recent literature demonstrating the complex interplay between these variables, and highlights the many etiological pathways. While acknowledging the considerable progress in the empirical literature, the significant gaps in knowledge are noted, particularly the need for further longitudinal research to clarify causal pathways, and moderating and mediating effects. The resulting model will be valuable in informing the design of more effective treatment and preventive interventions for SAD and will provide a useful platform to guide future research directions.

1. Introduction

In 2004, we published a paper that presented a model of the etiology of social phobia based on a review of the empirical evidence at that time (Rapee & Spence, 2004). The aim of the present paper is to evaluate research over the intervening 11 years and consider its support for the model or whether modifications are needed. We will also examine implications for the prevention and treatment of social phobia, which is now referred to as social anxiety disorder (SAD). Given that the onset of SAD for the great majority of cases occurs during childhood or adolescence (Jin et al., 2005), the focus of this paper is primarily upon evidence relating to young people rather than adults.

1.1. Symptoms and diagnostic criteria for social anxiety disorder

Since 2004, there have been only minor changes to diagnostic classification systems for SAD, with the key elements of both DSM and ICD criteria remaining relatively consistent. According to DSM-5 (American Psychiatric Association, 2013), the core defining features of SAD include fear or anxiety in social situations in which the individual is exposed to possible scrutiny by others and a fear of acting in a way that will be negatively evaluated by others (either resulting from the individual's own behavior or from showing anxiety symptoms such as blushing, trembling or sweating). Where possible, the social situations are avoided or are endured with intense anxiety. DSM-5 further specifies that the fear must be out of proportion to the actual threat, that it has persisted for longer than 6-months, is not due to the physiological effects of a substance, another mental disorder, or a medical condition, and that it leads to clinically significant distress or impairment in important areas of social functioning. For a DSM-5 diagnosis in young people, the anxiety must include interactions involving peers, and may be expressed by crying, tantrums, freezing, clinging, shrinking or failing to speak in social situations. Typically, feared social situations for children include speaking in front of the class, asking a teacher a question, performing in front of others, meeting new people, joining in conversations, asking for help in shops or at school, and going to parties or social events where peers are present (Beidel & Turner, 2007; Rao et al., 2007).

1.2. Epidemiology of SAD in children and adolescents

Recent studies have informed our knowledge of the epidemiology of SAD which is relatively common amongst children and adolescents (Burstein et al., 2011; Lawrence et al., 2015). Prevalence rates in community samples are generally found to increase from

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childhood through adolescence (Beesdo et al., 2007; Burstein et al., 2011; Canino et al., 2004; Lawrence et al., 2015), although no such increase was found in a large UK epidemiological study (Ford, Goodman, & Meltzer, 2003). Adult prevalence also appears to differ markedly between countries, with lower reported rates in East Asian countries than elsewhere (Brockveld, Perini, & Rapee, 2014). In our previous paper, we noted that social anxiety is expressed along a severity continuum, with many young people reporting high symptoms of social anxiety without necessarily reaching the threshold for a clinical diagnosis. Recent studies confirm the relatively high prevalence of sub-clinical social anxiety symptoms (eg. Knappe et al., 2011). We also noted that the distinction between high social anxiety and a clinical diagnosis of SAD depends on more than just severity, being influenced by the impact of social functioning and a range of cultural factors (see below). Studies suggest that SAD during childhood and adolescence tends to persist if left untreated (Beesdo-Baum et al., 2012; Burstein et al., 2011; Kessler et al., 2012), with onset prior to 11 years of age increasing the risk of persistence into adulthood (Abidin, 1992; Beesdo et al., 2007; Wittchen & Fehm, 2003). In fact, SAD is one of the more chronic and persistent of the mental disorders across the lifespan although it is more likely to have a waxing and waning course than a stable one (Beesdo-Baum et al., 2012; Bruce et al., 2005).

1.3. Comorbidity and association with adverse consequences

SAD in youth is comorbid with a significant number of mental health problems, particularly other anxiety disorders and depression, and with substance use in older adolescents (Beesdo-Baum et al., 2012; Burstein et al., 2011; Wittchen, Stein, & Kessler, 1999). There is insufficient evidence, as yet, to determine whether SAD is a cause or effect of such comorbid problems, or whether these patterns of comorbidity reflect common underlying causal factors. However, longitudinal studies are beginning to examine these complex relationships. For example, Beesdo et al. (2007) demonstrated that SAD during adolescence significantly increased the risk for depression during early adulthood, although this effect was not found by Buckner et al. (2008). Similarly, SAD in adolescence has been shown to increase the risk of future alcohol use (eg. Black et al., 2015) as well as cannabis and alcohol dependence in early adulthood (Buckner et al., 2008), although this relationship may be specific to females (Buckner & Turner, 2009). Furthermore, although depression predicted future substance use in the study by Buckner et al. (2008), this effect was explained by its association with SAD, suggesting that SAD in particular may increase young people's risk for future substance abuse. In contrast to these patterns, an early study failed to show that adolescent social phobia predicted any adult disorders other than adult social phobia (Pine, Cohen, Gurley, Brook, & Ma, 1998). Clearly the heterotypic continuity of SAD from adolescence is complex and more longitudinal research is needed to cast light on specific patterns, as well as on the mechanisms by which early SAD may continue or may contribute to development of other mental health problems.

2. Summarizing the Rapee and Spence (2004) model

The Rapee and Spence (2004) model was designed to consider factors that might describe how high levels of social anxiety emerge across early development. It began from the assumption that social anxiety exists along a continuum of intensity and severity. Although a diagnosis of SAD is more likely be given toward the upper end of the continuum, the actual clinical diagnosis requires that the symptoms have a marked impact and significantly interfere with the individual's life. Thus the model argued that a clinical diagnosis of SAD will be affected by a range of risk factors that influence the life interference value of symptoms including age, gender, and cultural "fit". The model outlined the role of various risk and protective factors that were proposed to influence the shift along the continuum of social anxiety until young people reached the point at which the severity and duration of symptoms, along with their impact are sufficient to warrant a clinical diagnosis of SAD.

We noted that causal and maintaining factors for SAD may not be the same and that different risk factors may be more or less influential at different ages. Risk factors were predicted to interact, with some factors combining to increase the impact upon the development of SAD, and others having a protective effect that may buffer the impact of a particular risk factor. We noted that different pathways and combinations of factors can result in SAD (the principle of equifinality) and that any one risk factor can result in various outcomes, not just SAD (the principle of multifinality). Furthermore, it was acknowledged that risk factors can be transactional and reciprocal, with young people influencing their own social outcomes which, in turn, impact upon the young person. These points regarding etiology remain relevant today, and we will examine more recent evidence that clarifies these issues.

While acknowledging the considerable limitations in the research up to 2004, we were able to draw together sufficient evidence to propose a series of interacting risk and protective factors that influenced the development and maintenance of SAD. There was emerging evidence of a complex interplay between intra-individual factors (genetic, biological processes, cognitive processes and social skills) and environmental factors (such as parental influences, aversive social experiences, and negative life events) in the etiology of SAD. We proposed that cultural factors can influence both the form and characteristics of social anxiety as well as its life impact. However, there remained a major task for researchers to tease apart these interrelationships and to determine their specific mechanisms of action. A strong need for longitudinal research to clarify cause and effect relationships was noted, to identify what is proximal versus distal in terms of impact on SAD and to inform us about mediators/mechanisms of action, and moderating/interacting effects. In the intervening years, many of these issues have received further attention although, as will become evident in this paper, many unanswered questions remain.

The past decade has seen a rapid expansion of research into the etiology and maintenance of SAD in young people. Although there are still significant gaps in the evidence, there have been some exciting studies that inform our understanding of this area. Since 2004 there have been several papers that have reviewed specific literature relating to the development and maintenance of SAD in children and/or adolescents (e.g., Detweiler et al., 2014; Kimbrel, 2008; Knappe, Sasagawa, & Creswell, 2015; Ollendick & Benoit, 2012; Ollendick, Benoit, & Grills-Taquechel, 2014) and we acknowledge their significant contribution to our understanding of this area. There have also been substantial increases in our knowledge of the development and maintenance of SAD in adults (See Wong & Rapee, in press, June 2016 for a review).

3. Developments in the research 2004–2015: intrinsic factors

3.1. Genetics of childhood SAD

There is substantial evidence that SAD has strong familial links - children of parents with SAD have significantly increased risk of experiencing the disorder, and parents of children with SAD are also more likely to experience the problem (See Elizabeth et al., 2006 for a review). Studies suggest that these familial