Chapter 10

Social Anxiety in Children and Adolescents: Biological, Developmental, and Social Considerations

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Much has changed since Victorian times, when psychology was dominated by the concept of *precocity,* in which children and adolescents were conceptualized as being merely miniature versions of adults. The implications of this conceptualization were both numerous and profound, not the least of which was that it fostered an ignorance of the relationship between ontogeny and pathology. Over time, this precocial view was eventually abandoned in favor of one with a greater appreciation for lifespan developmental processes. Most child and adolescent practitioners today receive clinical training steeped in a developmental perspective (Cicchetti & Cohen, 2006; Cicchetti & Toth, 2009; Ollendick & Hirshfeld-Becker, 2002). In this chapter, we discuss the phenomenon of social anxiety in children and adolescents. We adopt a biopsychosocial model of pathology, where case formulation is approached with a thorough appreciation for the various individual factors at play within the context of larger family, academic and cultural systems (Adler, 2009; Engel, 1977). We propose pediatric social anxiety is a product of the interaction among various biological, developmental, and social factors and begin with a thorough review of these systems. Next, we propose a model of social anxiety that outlines the reciprocal interactions between youth and these biopsychosocial systems. Finally, we describe an evidence-based therapeutic intervention for pediatric social phobia and conclude with recommendations for clinicians who work with this unique and often highly impaired clinical population.

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HISTORY AND MORPHOLOGY OF SOCIAL ANXIETY DISORDER (SOCIAL PHOBIA)

In DSM-III (American Psychiatric Association, 1980), social phobia first appeared as a distinct diagnosis in adults. For children, a separate category was introduced and termed avoidant disorder of childhood and adolescence (AVD). AVD was initially described only as a chronic and excessive withdrawal from others, significant enough to interfere with peer relationships and failing to identify any subtypes of social fears other than this generalized form. Unfortunately, there was considerable overlap between the criteria of AVD and social phobia. The differential diagnosis was hampered further by the presence of a third diagnosis, overanxious disorder in childhood and adolescence (OAD), which allowed for social fears but itself overlapped considerably with generalized anxiety disorder. The revised DSM-III-R (1987) did not preclude the diagnosis of social phobia in child and adolescent populations, but it did not specifically reference them in the criteria either, leading many clinicians to refrain from assigning this diagnosis to youth (Stein, Chavira, & Jang, 2001). With growing recognition that many cases of AVD and OAD overlapped with other disorders (Beidel, 1991; Francis, Last, & Strauss, 1992), DSM-IV (American Psychiatric Association, 1994) revised the differential diagnosis criterion to include youth if the symptoms were present and stable for six months. Kendall & Warman (1997) reported only 18% of their clinic sample met DSM-III-R criteria for social phobia, whereas 40% of that same sample met DSM-IV criteria following the revision in diagnostic criteria.

Under the DSM-IV-TR (American Psychiatric Association, 2000), the criteria for social phobia (social anxiety disorder) with children and adolescents were largely similar to those for adults, with certain exceptions. For children, a marked and persistent social/performance fear of negative evaluation was required with peers, as opposed to merely with adults, and emotional reactions such as freezing, crying, or acting out via tantrums qualified as anxious behavioral responses to feared social situations. Finally, children were permitted to display limited insight and it was not necessary for them to recognize the severity of their anxiety as being excessive or unreasonable to meet diagnostic criteria.

Many of the changes to the social anxiety disorder definition in DSM-5 are largely superficial, though importantly one alteration in particular appears to work against purported developmental considerations. With regard to children and adolescents, the most substantial DSM-5 alteration to SAD criteria concerns the addition of a "performance-only" specifier, and concomitant removal of the "generalized" specifier. This change is counter to the empirically supported distinction between generalized and non-generalized SAD in children and adolescents, and replaces this distinction with a specifier of limited clinical utility for the classification of youth with SAD (Chou, Cornaccio, Cooper-Vince, Crum, & Comer, under review). Specifically, despite

evidence demonstrating meaningful differences between youth with generalized and nongeneralized SAD (e.g., Burstein, He, & Katten, 2011; Hofmann et al., 1999), concerns had been raised about the applicability of this DSM-IV-TR distinction (Bögels et al., 2010). A small body of research supported the use of a performance-only specifier, but these investigations focused almost solely on adults (e.g., Blöte, Kint, Miers, & Westenberg, 2009; Faravelli et al., 2000). Kessler, Stein, & Berglund, 1998; Knappe and colleagues (2011) found evidence of diagnostic utility for the performance-only specifier in a younger population, with about one third of youth and young adults exhibiting solely performance-related social fears, but the inclusion of emerging adults up to 24 years of age interfered with clear developmental interpretations. To address this need, Kerns, Comer, Pincus, and Hofmann (2013) compared the relative clinical utilities of a generalized and speaking/performance-only specifier in a sample of anxious youth. Kerns and colleagues found almost twice the number of youth qualified for generalized (64.2%) versus nongeneralized (35.8%) SAD, and youth in the generalized cluster exhibited greater clinical severity and comorbidity rates with depressive disorders, as well as higher levels of depressive symptoms. Further, although 93.6% of youth endorsed speaking/performance symptoms, none qualified for performance-*only* SAD. This observed limited relevance and utility of distinguishing performance-only fears in treatment-seeking children and adolescents with SAD is consistent with epidemiologic data examining children in the general population. Specifically, among U.S. adolescents in the general population, less than 1% of children with SAD exhibit performance-only fears (Burstein, He, Katten, et al., 2011; Merikangas, et al., 2007), whereas over half exhibit generalized fears (Burstein et al., 2011).

Taken as a group, both the prevalence and impact of anxiety disorders are profound. Comprising over one-third of the total, anxiety disorders are the most common psychiatric illnesses in the United States (Kessler, Berglund et al., 2005). Greenberg and colleagues (1999) conducted an economic impact study and concluded that the annual cost of anxiety disorders in the United States during the 1990s was approximately $42 billion, or roughly one-third of the total health budget and representing over $1500 per individual. Anxiety disorders are also the most common disorders observed in child and adolescent populations (Beesdo, Knappe, & Pine, 2009; Costello, Egger, & Angold, 2004; Merikangas, He et al., 2010). It is well established that social phobia most commonly begins in late childhood or adolescence, typically around 13 years of age, although rarely following age 25 (Beesdo et al., 2007; Kessler, Berglund et al., 2005; Wittchen & Fehm, 2003). Estimates of the prevalence rate for social phobia vary somewhat in the literature based upon the nature of assessment and geographic location of participant catchment. It is also noteworthy to mention that the substantial diagnostic changes that occurred between DSM-III and DSM-IV limit the degree to which prevalence rates can be compared and the reader is cautioned to be mindful of which diagnostic criteria were used when considering published estimations (for example, see Wittchen & Fehm, 2003).

Traditionally, the lifetime prevalence rate for social phobia has been estimated to be somewhere between 5 and 15% (Comer & Olfson, 2010; Heimberg, Stein, Hiripi, & Kessler, 2000). Kessler, Chiu, Demler and Walters (2005) reported the adult prevalence of social phobia to be about 6.8%. Cox and colleagues (2009) studied a data set of over forty thousand individuals from the National Epidemiologic Survey on Alcohol and Related Conditions and found the lifetime prevalence for generalized social phobia to be 2.8%. The pattern is similar for child and adolescent populations. Costello and colleagues (1996) reported a one-year prevalence of social phobia of 13% for children and adolescents ages 9 to 17. Estimates of the prevalence of pediatric social phobia in the United States have ranged from 5% to 10%, with an average of around 7% (Fichter, Kohlboeck, Quadflieg, Wyschkon, & Esser, 2009; Schneier, 2006). Beesdo and colleagues (2009) conducted a comprehensive review of the reported prevalence rates of social phobia and other anxiety disorders in children and adolescents and concurred with a prevalence estimate of 7%. More recent epidemiologic data suggests that 9% of the US adolescent population has suffered from SAD at some point in their lifetime (Burstein, He, Kattan, Albano, Avenevoli, & Merikangas, 2011). Common for most anxiety disorders, a gender difference is also clearly observed with social phobia by adolescence; the prevalence rate for females is nearly double that of males, although there is a developmental effect whereby the difference generally increases with increasing age (Beesdo et al., 2009; Craske, 2003; Pine, Cohen, Gurley, Brook, & Ma, 1998; Wittchen, Nelson, & Lachner, 1998).

BIOLOGICAL FACTORS

Youth, particularly adolescence, is a period marked by elevated activity across numerous biological processes (Cicchetti & Curtis, 2006; Ojeda, Lomniczi, Sandau, & Matagne, 2010). Pediatric clinical syndromes are best approached with acknowledgment of those maturational changes under way for children at varying points of development. In-depth discussion of the biological basis for social anxiety disorder is beyond the scope of this chapter and available elsewhere in this volume. Therefore, we have restricted our discussion to those specific biological contributors to social anxiety that are most influential for youth.

Genetic Influence

Clinicians have long suspected there is a genetic component to social anxiety, but the evidence has traditionally varied in breadth and scope. Differences in estimates are due to various factors, such as the nature of the dependent variable studied (e.g., clinical anxiety disorder versus subclinical anxiety symptoms) or source of information (Gregory & Eley, 2007; Murray, Creswell, & Cooper, 2009), but the magnitude of genetic influence upon anxiety in general is believed to be moderate (Gregory & Eley, 2007). Evidence has clearly demonstrated a higher base rate of anxiety disorders in the children of

parents who themselves have anxiety disorders, as well as vice versa (Biederman et al., 1991; Cooper et al., 2006; Last et al., 1987; Last et al., 1991; Turner et al., 1987; Warner et al., 1995; Weissman et al., 1984). Studies also have demonstrated the aggregation of social phobia specifically among family members, particularly the generalized subtype (Fyer, Mannuzza, Chapman, Liebowitz, & Klein, 1993; Fyer, Mannuzza, Chapman, Martin, & Klein, 1995; Mancini, van Ameringen, Szatmari, Fugere, & Boyle, 1996; Mannuzza, Schneier, Chapman, Liebowitz, Klein, & Fyer, 1995; Reich & Yates, 1988; Stein et al., 1998; Aktar, Majdandžić, de Vente, & Bögels, 2014), as well as relationships between parent and child social anxiety symptom severity (Schreier & Heinrichs, 2010). Moreover, recent evidence suggests that genetic factors—such as the serotonin transporter promoter polymorphism and marker rs6330 in Nerve Growth Factor gene— may even contribute to poorer response to cognitive-behavioral therapy among anxious youth (Hudson, Lester, Lewis et al., 2013).

Twin studies are helpful in teasing out the differential contribution of genetic and environmental influences upon social behavior and/or psychiatric disorders (Gregory & Eley, 2007). Unfortunately, however, there are few twin studies examining social anxiety specifically and more research is warranted. Kendler and associates (1992) examined twins identified from the Virginia twin registry and found a 24.4% concordance rate of social phobia for monozygotic twins and a 15.3% concordance rate for dizygotic twins, yielding a heritability index (h^2) of about 30% for social phobia. However, this study has significant limitations in that it included only female twin pairs and utilized DSM-III diagnostic criteria. Ogliari et al. (2006) conducted a twin study focusing upon 378 Italian twin pairs ranging from late childhood through adolescence (ages 8–17) and reported an h^2 of around 0.6 for self-reported symptoms of social anxiety.

Behavioral genetic twin studies have classified three main influences upon behavior (Gregory & Eley, 2007) – genetic influences (inherited from family members), shared environmental influences (non-genetic environmental factors often shared with immediate family members such as parenting style, living environment, diet, etc.), and non-shared environmental influences (non-genetic environmental factors usually not shared with immediate family members, such as illness history, specific friendships, trauma history, etc.). Eley and colleagues (2003) employed both phenotypic and genetic approaches in an examination of over four thousand British preschool twin pairs. With regards to parental report of a subclinical shyness/inhibition temperamental trait, they found high levels of genetic influence accounting for around two-thirds of the variance, and one-third due to non-shared environmental factors. In a subsequent study, Eley and colleagues (2008) conducted a multivariate analysis of anxiety phenotypes in a sample of young people that included those who met diagnostic criteria for social phobia by age six (as assessed by semi-structured clinical interview). They found non-shared environmental factors to be the only significant influence upon social phobia (79%), with only small non-significant influences from genetic (14%) and shared environmental factors (10%).

Complicating matters, we now understand genetic influence can be a dynamic process (Bird, 2007; Kendler, Gardner, & Lichtenstein, 2008). Even with a stable genetic *architecture*, genetic *influence* can wax and wane over an individual's lifespan in response to environmental or developmental influences such as puberty (Eaves, Long, & Heath, 1986; Whitelaw & Whitelaw, 2006). In their seminal meta-analysis, Bergen and colleagues (2007) alert us to the importance of maintaining a developmental perspective when considering heritability. For example, the heritability of a phenotype observed in early childhood may be different to the heritability of that same phenotype observed in adolescence or young adulthood. Consider for a moment that young children typically passively receive the genetic contribution and environmental control of their parents. Teens and young adults, however, take a more active approach and begin to seek out reinforcing environments based more upon their own unique genotypic expression, creating a stronger interaction between genetics and environment as they both shape and are shaped by the settings they encounter. Results of the Bergen et al. meta-analysis revealed an age-related increase in heritability (h^2 ranging from approximately 0.10 at age 10 to 0.60 by age 25) with anxiety symptoms demonstrating the highest effect size for per-year increase of any domain assessed (+0.030, $t = 4.22$, $p = 0.0056$).

Brain/Cognitive Development

One of the more exciting areas of research in recent years belongs to an emerging field attempting to integrate the findings of social cognitive neuroscience with those of clinical developmental psychopathology (Cicchetti & Curtis, 2006; Yeates et al., 2007). Where neuroscience is the interdisciplinary study of those underlying brain systems associated with human thoughts, emotions, and behaviors (Kandel, Schwartz, & Jessell, 2000), social cognitive neuroscience specifically focuses upon the brain–behavior relationship as it pertains to an individual's social functioning (Brothers, 1990; Moss & Damasio, 2001). Social cognitive neuroscience models drew heavily upon observations of children who suffered traumatic brain injuries. After it was observed that many of the interpersonal functioning deficits displayed following damage to the brain were similar to those characteristic of some clinical syndromes, the call came for greater integration between neuroscience and developmental psychopathology. For example, in much the same way that a neuropsychologist may capitalize upon an extensive knowledge of neural systems to help clarify a decline in school functioning into more specific terms of deficits in processing speed versus working memory versus attention (each with their own implications for intervention), the social cognitive neuroscience model may strive to clarify a decline in social functioning in terms of deficits of areas such as interpersonal problem-solving versus social communication versus emotion regulation. Today, there is a growing elucidatory research base identifying the development of brain structures and systems that play a part in the emergence of social

competence during childhood and adolescence (see Adolphs, 2001; Benuzzi et al., 2007). Further findings will undoubtedly provide insight into the role of neurobiological factors affecting social anxiety; however, progress is hindered to the extent that adaptive social functioning as a construct has proven difficult to define (c.f., Bukowski, Rubin, & Parker, 2001; Cavell, 1990; Rubin, Bukowski, & Parker, 2006).

Although a thorough discussion of all of the brain systems involved in social competence is beyond the scope of this chapter, three warrant mentioning in terms of their relationship to both social development and social anxiety.

Amygdala

The amygdala is an almond-shaped structure located within the anterior portion of the temporal lobes, comprising a component of the limbic system and known to play a part in controlling emotion, motivation, and memory. It plays a clear role in the processing of both social cues and emotional expression and is involved with the sympathetic nervous system's response to anxiety, leading many to identify the amygdala as playing a role in social anxiety (Birbaumer et al., 1998; Etkin & Wager, 2007; Rosen & Schulkin, 1998; Shin & Liberzon, 2010). Normally, the amygdala responds to facial cues with an increase in activity as perceived social threat increases (Morris et al., 1996). Individuals with social phobia have been shown to demonstrate an attentional bias for negative social cues (e.g., Alden & Wallace, 1995). As would be expected, research has shown a correlation between the degree of amygdala activity and severity of social anxiety when presented with social threat cues. This has been demonstrated both in individuals diagnosed with social phobia (Phan, Fitzgerald, Nathan & Tancer, 2006), as well as those not yet meeting diagnostic criteria but high in behavioral inhibition, a risk factor for a future social phobia diagnosis (Pérez-Edgar et al., 2007). Further, individuals with increased social anxiety have been shown to display social skills deficits (Beidel & Turner, 1998; Miers, Blöte, de Rooij, Bokhorst, Westenberg, 2013) and damage to the amygdala typically leads to profound deficits in social functioning, such as difficulty with emotional processing, problem-solving, and the ability to discern the emotional nuance of facial expressions or body language (Ammerlaan, Hendriks, Colon, & Kessels, 2008; Cristinzio, N'Diaye, Seeck, Vuilleumier, & Sander, 2010; Scott et al., 1997; Shaw et al., 2004; see also Corden, Critchley, Skuse, & Dolan, 2006). Finally, individuals and non-human primates with damaged or surgically removed amagdalae often demonstrate restricted emotional expression in social contexts, decreased display of pro-social skills such as maintaining adequate eye contact, and occasionally (although not always) increased social withdrawal, escape and avoidance behaviors in response to the presence of others (Mori & Yamadori, 1989; Ozmen, Erdogan, Duvenci, Ozyurt, & Ozkara, 2004; Spezio, Huang, Castelli, & Adolphs, 2007). Avoidance is viewed as a maintaining factor in social anxiety, with exposure-based interventions

that limit avoidance shown to yield the largest effect sizes in studies examining different treatment approaches for social anxiety (Gould, Buckminster, Pollack, Otto, & Yap, 1997; Hope, Heimberg, & Bruch, 1995).

When specifically considering child populations, one must be mindful of the interaction between the amygdala's development and the emergence of social competence and social anxiety. Development of the amygdala continues for about two years following birth and this maturation both affects the social behavior of the infant and is affected by the reciprocal social stimulation the child receives in return (Joseph, 1999). Accordingly, infants demonstrate increasingly pro-social behavior (Hasselmo, Rolls & Baylis, 1989; Yakovlev & Lecours, 1967) in the early stages of the amygdala's maturation. This not only demonstrates newly acquired social skills, but also serves to solicit additional adult social interaction, furthering the development of the amygdala and fine-tuning the child's emerging social competence. Infants raised in socially impoverished environments often display delays in neural development within the amygdala and subsequent deficits in social functioning later in life (Jacobson, 1986; Tranel & Hyman, 1990).

Developmentally speaking, it behooves infants to interact with as wide a social network as possible during the early stages of amygdala maturation in order to capitalize on the bidirectional influence of context upon development. We would not expect to see a volitional restriction of socialization until such time as maturation has progressed sufficiently so as to not be hindered by the lack of social stimuli. In fact, this is exactly what is observed. Stranger anxiety (i.e., a developmentally appropriate behavioral response to unfamiliar adults marked by distress and behavioral withdrawal) typically peaks around nine months of age (Ainsworth, Blehar, Waters, & Wall, 1978; Bronson, 1974; Schaffer, 1966; Waters et al., 1975). This emergence corresponds directly with more advanced stages of amygdala development in which the infant has already developed an improved ability to discern between different faces, genders, emotions and subtle social cues. In fact, phases of infant socio-emotional development have been shown to correlate with differential maturation rates of the amygdala and supporting systems, in turn influencing infant approach-avoidant behavior and the formation of loving attachments with caregivers in corresponding fashion (Joseph, 1999).

Prefrontal Cortex

Along with the amygdala, the prefrontal cortex is another key component of the neural circuit involved with the processing of social threat (Adolphs, 2001; Blair, Morris, Frith, Perrett, & Dolan, 1999; Gallagher & Frith, 2003; Gross & Hen, 2004; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Haxby, Hofmann, & Gobbini, 2002; Monk et al., 2003). In simplistic terms, the amygdala is believed to be involved with the identification of the emotional quality of a stimulus, whereas the prefrontal cortex is believed to be involved with activating the higher cognitive processes necessary for regulating emotion and choosing a

subsequent course of reaction to the stimulus (Nelson, Leibenluft, McClure, & Pine, 2005). The prefrontal cortex is assumed to provide the cognitive resources to boost the efficiency of those behaviors commonly referred to as representing "executive functioning" (Amunts et al., 2004; Aron, Robbins, & Poldrack, 2004; Buchsbaum, Greer, Chang, & Berman, 2005; Curtis & D'Esposito, 2003; Duncan, 2001; Langenecker, Nielson, & Rao, 2004). As such, this area's role in the processing of social behavior becomes clear when you consider social competence requires an adaptive management of attentional skills, problem-solving and a host of additional executive functioning skills governed by the prefrontal cortex (White, Helfinstein, Reeb-Sutherland, Degnan, & Fox, 2009).

As with the amygdala, evidence of the relationship between the prefrontal cortex and anxiety comes in the form of data demonstrating differences in the activation patterns correlated with subjective social anxiety (Sripada et al., 2009; Tillfors et al., 2001; Tillfors, Furmark, Marteinsdottir, & Fredrikson, 2002). Monk et al. (2006) conducted an experiment in which functional magnetic resonance imaging captured the brain activity of adolescents as they completed a task measuring attention while presented with angry and neutral faces. Adolescents with generalized social phobia showed greater activation in the ventrolateral area of the prefrontal cortex, compared to non-anxious adolescents as they were spontaneously demonstrating a significantly different degree of attentional avoidance of angry faces. Damage to the prefrontal cortex often results in disruptions in behavior required for competent social functioning. Mah, Arnold, and Grafman (2004) compared the ability of adults with prefrontal cortex lesions with control subjects on a social perception task and found an association between damage to this brain area and poorer social perception ability.

A distinguishing characteristic of the prefrontal cortex is its protracted developmental timeline. Many regions of the prefrontal cortex follow a nonlinear developmental trajectory and do not reach physical maturity until the early adulthood period of the late teens to early twenties (Casey, Giedd, & Thomas, 2000; Conklin, Luciana, Hooper, & Yarger, 2007; Giedd et al., 1999; Sowell et al., 2003; Sowell, Thompson, Holmes, Jernigan, & Toga; 1999; Toga, Thompson, & Sowell, 2006). In the frontal lobes, the peak in gray matter volume (the neural material comprising cell bodies and their dendrites) coincides with the onset of puberty, followed by an increase in the volume of white matter (the neural material comprising the myelinated connecting nerve fibers) in the prefrontal cortex thereafter and throughout the mid-twenties (Barnea-Goraly et al., 2005; Giedd et al., 1996; Giedd et al., 1999; Reiss, Abrams, Singer, Ross, & Denckla, 1996; Sowell, Thompson, Tessner, & Toga, 2001; Sowell et al., 2003; Toga et al., 2006). The period from late adolescence throughout the third decade of life is marked by synaptic fine-tuning within the prefrontal cortex through a process of increased axonal myelination and synaptic pruning to allow for improved communication between inter-related brain areas (Durston et al., 2001; Giedd, 2004; Huttenlocher, 1979; Huttenlocher, De Courten, Garey, & Van Der Loos, 1983; Reiss et al., 1996; Sowell et al., 2001; Yakovlev

& Lecours, 1967). This neurodevelopmental process of improving communication between inter-related brain areas in late adolescence and early adulthood has implications for the experience of social anxiety as described below.

Amygdala-Prefrontal Cortex Connectivity

Rather than simply being two neural structures similar in function, there is evidence that the amygdala and prefrontal cortex work in conjunction as part of a more complex emotion regulation circuit (Adolphs, 2003; Baxter, Parker, Lindner, Izquierdo, & Murray, 2000; Gross & Hen, 2004; Hariri et al., 2003; McClure et al., 2007) with implications for both social functioning in general as well as the development of affective disorders like social anxiety disorder (Drevets, 2003; Mayberg, 1997, 2007; Pezawas et al., 2005; Rauch, Shin, & Phelps, 2006). In simplistic terms, the prefrontal cortex is believed to develop into a top-down modulator of the amygdala's emotional reactivity to threat or fear-inducing stimuli (Casey, Getz, & Galvan, 2009; Hare et al., 2008; Tottenham et al., 2009). This executive control of amygdala activation by the prefrontal cortex in response to threatening situations facilitates the flexibility of attentional and behavioral processes presumed to be necessary for not only successful social functioning (Blair, 2004; Blair & Cipolotti 2000; Hariri et al., 2003; Kringelbach & Rolls, 2003) but also the process of habituation (Hugdahl & Nordby, 1994; Wright et al., 2001).

As described earlier, the prefrontal cortex has a protracted developmental timeline that continues well into the second or third decade of life. During adolescence, changes occur at the cellular level that involve the generation of new synapses and a subsequent synaptic pruning that Nelson, Rubin and Fox (2005) described as a *use or lose* process in which neural connections are strengthened through an individual's experience (Huttenlocher, 1979; Huttenlocher et al., 1983). In the adolescent prefrontal cortex, gray matter density decreases, whereas white matter increases due to increased myelination, especially the myelination of those fibers connecting this brain region with others (Casey, Galvan, & Hare, 2005; Yakovlev & Lecours, 1967). Total brain volume changes little during this period (Blakemore & Choudhury, 2006; Choudhury, Blakemore, & Charman, 2006) and these changes in gray and white matter density are believed to represent a process of synaptic reorganization (Paus, 2005).

This reorganization enables greater executive control of amygdala activation by the prefrontal cortex (Yurgelun-Todd, 2007; Rubia et al., 2000; Rubia et al., 2006; Tamm, Menon, & Reiss, 2002). By adolescence, the amygdala has reached maturity whereas the prefrontal cortex has not. Increases in sex hormones occurring with puberty profoundly affect the amygdala (Nelson, Leibenluft et al., 2005). Brain imaging studies have shown that, relative to children and adults, adolescents demonstrate more pronounced amygdala activation when processing emotional information (Ernst et al., 2005; Galvan et al., 2006; Kuhnen & Knutson, 2005; Matthews et al., 2004; Monk et al., 2003; Montague & Berns, 2002). Further, amygdala activation differences can reliably differentiate

adults with and without social anxiety (Birbaumer et al., 1998; Lorerbaum et al., 2004; Phan et al., 2006; Stein, Goldin, Sareen, Zorrilla, & Brown, 2002; Straube, Kolassa, Glauer, Mentzel, & Miltner, 2004; Tillfors et al., 2001; Veit et al., 2002). In the absence of top-down regulation from the prefrontal cortex, adolescents are more prone to the interference that comes from the emotional properties of social stimuli (Nelson, Leibenluft et al., 2005). Monk and colleagues (2006) noted that increases in prefrontal cortex activation were correlated with decreased subjective anxiety symptoms for those with generalized anxiety disorder. In a related study, Monk and colleagues (2003) asked adolescents and adults to vary their attention between emotional and non-emotional aspects of a social stimulus and found that only adults demonstrated prefrontal cortex activation in response to this experimental demand. Thus, pubescent adolescents (who demonstrate more amygdala activity in response to social threat and find it difficult to disengage attention from that social threat due to the immaturity of the prefrontal cortex) appear to have a neurobiological vulnerability during this period. They can easily become preoccupied with the perception that they are being negatively evaluated by others and social anxiety concerns increase as a result (Rapee & Heimberg, 1997).

Stress Response and Subjective Experiences of Psychophysiological Arousal

Youth with social anxiety also show an oversensitivity to perceived physiological arousal, relative to their non-affected peers. In a study by Schmitz and colleagues (2012), pre-adolescents (ages 10–12) with high levels of social anxiety, perceived themselves as more autonomically aroused during public speaking situations than children with low social anxiety, despite comparable heart rates during the task. Those with high social anxiety also exhibited increased worry after receiving feedback about signs of autonomic arousal, whereas children with low social anxiety did not. It appears this tendency among youth with social anxiety to overestimate physiological arousal, and to exhibit a hypersensitivity to others' perceptions regarding arousal, persists through adolescence. For example, whereas 13–17-year-old youth with high social anxiety rated themselves as higher in negative affect, lower in positive affect, and higher in physiological hyper-arousal than youth with low social anxiety, objective measures of heart rate variability did not suggest objective differences between these groups (Anderson, Veed, Inderbitzen-Nolan, & Hansen, 2010).

Puberty

Puberty refers to the developmental transition period marked by a cascade of biological changes resulting in sexual and physical maturation (Forbes & Dahl, 2010; Buck Louis et al., 2008). Complicating the direct effects puberty can have on biology, pubertal onset also overlaps with a crucial transitional period in which children progress through adolescence and eventually young

adulthood. A healthy understanding of both the physiological changes as well as the shift in social demands that occur during this development period must be considered when conceptualizing social anxiety in the adolescent population. There is some evidence suggesting puberty may be a factor, increasing the risk for many affective disorders (Angold, 2003; Angold, Costello, Erkanli, & Worthman, 1999; Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Angold, Worthman, & Costello, 2003; Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Hayward, 2003). Adolescence often witnesses the onset of new, or an increase in severity of pre-existing, anxiety symptoms (Burstein et al., 2011; Reardon, Leen-Feldner & Hayward, 2009; Kessler, Avenevoli, et al., 2012; Wittchen, Stein, & Kessler, 1999; Zgourides & Warren, 1988). This is likely related in no small part to the fact that increases in hormone levels during puberty directly affect the response to stress in the body and brain (Chrousos, Torpy, & Gold, 1998; Spear, 2000). Also, there is growing evidence that early pubertal development may be an additional risk factor for anxiety (Hayward et al., 1997; Zehr, Culbert, Sisk, & Klump, 2007).

It warrants mentioning here that the timing of the onset of puberty is somewhat dynamic and can be influenced by such lifestyle factors as stress, diet, and exercise (Adams, 1981; Dick, Rose, Pulkkinen, & Kaprio, 2001; Gluckman & Hanson, 2006; Paikoff & Brooks-Gunn, 1991). Perhaps as a result, the onset of puberty is currently occurring earlier than was documented forty to fifty years ago (American Psychiatric Association, 2002; Anderson, Dallal, & Must, 2003; Bellis, Downing, & Ashton, 2006; Bodzsar & Susanne, 1998). Although there is some variability due to cultural factors, generally speaking, pubertal onset is now typically observed between the ages of 10–12 in males and 12–13 in females (Anderson & Must, 2005; Whincup, Gilg, Odoki, Taylor, & Cook, 2001; Herman-Giddens, 2006). One unfortunate result of the decline in average age of pubertal onset is a widening of the gap between physical and social development during adolescence. There are growing concerns the physical maturity ushered in by puberty may now occur prior to requisite emotional development and a call has been raised to address the paucity of social support covering this gap (Dahl & Spear, 2004; Ge, Brody, Conger, & Simons, 2006; Ge, Conger, & Elder, 1996; Gluckman & Hanson, 2006). Research has demonstrated a correlation between earlier pubertal onset and a host of social and emotional risk factors in general. For example, Downing and Bellis (2009) conducted a survey in the United Kingdom and found self-reported earlier onset of puberty was a predictor of drug and alcohol abuse prior to 14 years and sexual experience including unprotected sex prior to 16 years for both sexes (see also Costello, Sung, Worthman, & Angold, 2007; Felson, & Haynie, 2002). However, the timing of the onset of puberty may also have direct implications for social anxiety.

Research has demonstrated the age of pubertal onset can affect adolescents' subjective social anxiety and self-esteem. Early maturing girls and late maturing boys tend to report lower self-esteem than those whose pubertal development more closely approximates the mean (Berk & Shanker, 2006). This is often

presumed to be a result of the negative attention received as a result of outward appearance (e.g., questioned machismo of less physically developed boys versus unwanted romantic overtures towards more physically developed girls) and warrants further attention, given that peer rejection and taunting have been associated with the development of social anxiety (La Greca & Harrison 2005). Currently few studies exist examining the relationship between pubertal development and social anxiety specifically. Deardorff and colleagues (2007) studied social anxiety symptoms in adolescents and found a relationship between symptoms and pubertal status, but within the context of an interaction with gender. Advanced pubertal development was associated with increased social anxiety, but only for females. However, this study was limited by the inclusion of only a restricted range of pubertal development (nine and a half through 11 years of age), its use of self-report measures and the examination of predominantly subclinical social anxiety symptoms as measured by a questionnaire. Blumenthal and colleagues (2009) also studied the relationship between pubertal status and social anxiety symptoms, but additionally sought to explore moderating factors. They found early pubertal development plus interpersonal problems with peers combined to predict higher social anxiety symptoms. This study employed a larger range of the pubertal cohort (adolescents aged 10–17), but was similarly restricted by the reliance upon self-report measures and examination of subclinical symptoms via a single questionnaire. Further research in this area is still needed.

The biological process of puberty results from hormonal changes triggered by the brain that effect the gonads, which in turn produce hormones that bring about maturational changes in other parts of the body (Buck Louis et al., 2008). As summarized in Forbes and Dahl (2010), gonadotropin-releasing hormone from the hypothalamus triggers pituitary changes in the production of luteinizing hormone and follicle-stimulating hormone. This in turn triggers maturation of the gonads and the production of the sex hormones (estradiol and testosterone). Germaine to our discussion of social anxiety, it is worth noting the neural areas that comprise the amygdala–prefrontal cortex emotion regulation circuit mentioned above are highly influenced by these gonadal steroids and their presence contributes to the structural and functional changes observed in that brain area during puberty (Giedd, Castellanos, Rajapakse, Vaituzis, & Rapoport, 1997; McEwen, 2001; Nelson, Leibenluft et al., 2005; Osterlund & Hurd, 2001; Romeo, Richardson, & Sisk, 2002; Stevens, 2002). Hormone levels have also been shown to exert influence over various aspects of social behavior, such as those related to approach-avoidance (Hull et al., 1999; Insel, 1997; Pfaff, Frohlich, & Morgan, 2002; Winslow & Insel, 2004) and thus suggest puberty may be a critical period for the development of patterns of adaptive adult social functioning (Flemming & Corter, 1995; Nelson, Leibenluft et al., 2005). This could in part help explain why the prevalence of anxiety disorders rises significantly during adolescence (Pine et al., 1998; Pine, Cohen, Johnson, & Brook, 2002) and why those with pubertal onset of social

phobia often experience persistence of clinically significant symptoms well into adulthood (Öst, 1987; Turner & Beidel, 1989).

Gonadal sex hormones coupled with an increase in secretion of adrenal androgens result in the development of secondary sex characteristics, which are typically the most conspicuous changes adolescents face during puberty (Forbes & Dahl, 2010). Leary (1995) identified personal appearance as "perhaps the most apparent nonverbal channel of self-expression" (p. 25). Those who view their body image less favorably tend also to report elevated levels of social anxiety (Hart, Leary, & Rejeski, 1989), as is the case for those whose specific fear is related to others having a negative perception of their physical appearance (Leary & Kowalski, 1993). This concern is not entirely unfounded as attractive individuals are perceived as being more socially skilled than less attractive individuals (Feingold, 1992). Unfortunately, puberty brings with it a host of rapid physical changes for teens to view as undesirable, from growth spurts and a redistribution of body fat to cracking voices and new body hair.

As normal as the physical changes that accompany puberty may be, adolescents adjust to them with varying degrees of acceptance and occasionally normal physiological changes become the impetus for social evaluative concerns. One common example is acne vulgaris, the onset of which most commonly corresponds with the andrenarche phase of puberty when the production of adrenal androgens fosters the development of secondary sex characteristics (Kilkenny, Merlin, Plunkett, & Marks, 1998; Stewart, 1992; Yamamoto & Ito, 1992). School-aged children with acne are often teased (Mallon et al., 1999). Loney, Arnold, and Grafman (2008) studied social anxiety resulting from acne and found it correlated negatively with self-esteem and positively with avoidance of social activities such as sports. Thomas (2004) summarized this predicament perfectly, "when teens and young adults have a need to look their best, they frequently have acne, which makes them feel and look their worst." (p. 3). Puberty is a time of heightened preoccupation with body image (Harter, 1999; Lunde, Frisén, & Hwang, 2007). Generally speaking, adolescent males try to bulk up, while on the other hand adolescent girls generally try to slim down (Muris, Meesters, van de Bloom, & Mayer, 2005). Whereas eating disorders are rarely seen in prepubertal children (Zehr, Culbert, Sisk, & Klump, 2007), evidence suggests a strong relationship between pubertal body transformation and increased body image dissatisfaction and the onset of eating disordered behavior, particularly among females (Cotrufo, Cella, Cremato, & Labella, 2007; Hayward et al., 1997; Tremblay & Lariviere, 2009).

Given that adolescence is a time of heightened self-consciousness (Elkind & Bowen, 1979; Simmons, Rosenberg, & Rosenberg, 1973), it is not surprising that we also see a jump in the prevalence of social anxiety concerns at this developmental period (Costello et al., 2002; Essau et al., 1999; Graziano, DeGiovanni, & Garcia, 1979; King, 1993; Ollendick, King, & Frary, 1989; Pine et al., 1998; Steinberg, 2005; Sumter, Bokhorst, & Westenberg, 2009). According to the self-presentation model of social phobia (Schlenker &

Leary, 1982), social anxiety is conceptualized as the experience that results when individuals are motivated to make a desired impression on others, but begin to conclude they are not or cannot (see also Goffmann, 1959; Leary, 1995; Schlenker, 1980). As teens progress through puberty, they can experience all the key theoretical ingredients of this model of social anxiety. An increased motivation to make a desired impression (in the form of heightened teen self-consciousness and changing social demands) combines with a perceived failure to manage a favorable impression (in the form of distorted perceptions of body image and/or undesirable pubertal changes to physical appearance) and social anxiety results.

DEVELOPMENTAL FACTORS

Attachment

Immediately upon birth, infants become instant members of a profoundly salient social relationship—the one experienced with a caregiver, most typically a parent. It has been hypothesized that a special bond called attachment results between the infant and caregiver based upon the daily interaction that occurs over the first year of life (Bowlby, 1978). Attachment theory posits the quality of this bond in childhood may serve as a contributing factor to the development of future social anxiety problems (Brumariu & Kerns, 2008; Vertue, 2003). For infants, there is inherent evolutionary survival value in the attempt to maintain close proximity to caregivers during times of threat or stress (Prior & Glaser, 2006). According to attachment theory, early caregiver availability and emotional responsiveness during attempts to maintain close proximity lead the infant to develop standards and expectations about social relationships in general, which in turn affect emotional and behavioral responses the child then displays in social situations with novel individuals (Bretherton & Munholland, 1999; Mercer, 2006; Schneider, Atkinson, & Tardif, 2001).

Four distinct infant attachment styles have been identified: secure, avoidant, ambivalent/resistant, and disorganized (Ainsworth, 1989; Ainsworth et al., 1978; Bailey, Moran, Pederson, & Bento, 2007; Madigan, Moran, & Pederson, 2006; Main & Hesse, 1990; Main & Solomon, 1986). A secure attachment style is said to develop in response to a caregiver who is reliably available to effectively soothe the child during times of stress. The remaining three represent insecure attachment styles, said to develop in response to a pattern of inconsistent, unavailable or maladaptive caregiver responding. Securely attached infants are presumed to develop into individuals who have learned how to rely upon others as calming influences and feel secure enough to explore socially, whereas insecurely attached infants learn others are unreliable, which can amplify the fear response during times of stress and foster the development of avoidance behaviors or maladaptive coping (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1973; Cassidy & Berlin, 1994).

Lending support to this presumption, children with insecure attachments are viewed as less socially competent and are less well liked than those who are securely attached (Cohn, 1990; DeMulder et al., 2000). Clark and Symons (2009) found a secure attachment style to be correlated not only with a child's positive self-esteem, but also to more positive attributions of the social behavior of others. Raikes and Thompson (2008) found a relationship between secure attachment style and increased social problem-solving skills and decreased loneliness. Conversely, La Greca and Lopez (1998) found that adolescents who were high in social anxiety also reported a lower degree of social functioning that would appear to be reminiscent of an insecure attachment style, only with peers instead of caregivers (e.g., lower levels of peer acceptance, social support, intimacy, and companionship). Warren and colleagues (1997) studied adolescents whose attachment styles had been assessed as one-year-olds. Although some children with secure attachment styles went on to develop anxiety disorders as teens, those with insecure attachment styles (especially ambivalent/resistant) doubled the risk of doing so. Finally, Irons and Gilbert (2005) found a secure attachment style predictive of low social anxiety and social submissiveness, whereas insecure attachment predicted the exact opposite relationship, leading the authors to speculate that an insecure attachment style fostered a preoccupation with social competition, the potential for negative social outcomes, and the need to avoid those outcomes—all key ingredients of social anxiety (Barlow, 2002; Beck, Emery, & Greenberg, 1985; Clark & Wells, 1995; Schlenker & Leary, 1982).

Temperament

Hippocrates, the father of modern medicine, proposed a theory in the fourth century BC that emotions had a predominantly physiological basis and differences were due specifically to fluctuations in body fluids called "humors." In the second century AD, the Roman physician Galen built upon Hippocratic humor theory and proposed four classic "temperamental" character styles—melancholic, phlegmatic, sanguine, and choleric. Today, the idea of an innate, constitutionally based character style persists, although considerable controversy exists (Buss & Plomin, 1984; Rothbart & Derryberry, 1981; Rothbart & Posner, 1985; Seifer & Sameroff, 1986; Strelau, 1983; Thomas & Chess, 1977). Although presumed to have a physiological basis, specific biological processes associated with temperament have been difficult to identify and study (Kagan, 2001). Further, there is disagreement regarding the classification and number of distinct temperamental styles (cf., Goldsmith & Campos, 1982; Kagan, Reznick, & Snidman 1988; Rothbart, 2004; Thomas, Chess, & Birch, 1968). Temperament is generally defined as unlearned, constitutionally based individual differences in both presentation style and ability to regulate emotion, attention, and behavior (Rothbart & Bates, 2006; Rothbart, Ellis, & Posner, 2004). Simply put, developmental clinicians view temperament as a predisposing dispositional factor that can either help or hinder a child's adaptation to their environmental

setting (Clark & Watson, 1999; Rothbart & Bates, 2006). A considerable research base has been devoted to the degree to which temperament serves as a risk factor for future psychopathology, either directly or indirectly through elicited changes to parental caregiving (Barron & Earls, 1984; Betts, Gullone, & Allen, 2009; Cutrona & Troutman, 1986; Essex, Klein, Slattery, Goldsmith, & Kalin, 2010; Kagan, Reznick, & Snidman 1988; Lerner, Castellino, Patterson, Villaruel, & McKinney, 1995; Putnam, Sanson, & Rothbart, 2002). For example, Thompson, Connell, and Bridges (1988) found a fearful temperamental style had both a direct and indirect influence on children's social interaction. Eisenberg and colleagues (2001; 2005) examined a temperamental style termed negative emotionality (Rothbart & Bates, 2006) and found its components contribute differently to internalizing versus externalizing problems, with internalizers more prone to fear and shyness (see also Bates, Pettit, Dodge, & Ridge, 1998; Leve, Kim, & Pears, 2005). A temperamental style marked by high negative emotionality plus physiological over-arousal has been linked to anxiety problems later in life (Brown, Chorpita, & Barlow, 1998; Chorpita & Daleiden, 2002; Lonigan, Carey, & Finch, 1994; Watson, Clark, & Carey, 1988).

A considerable amount of attention has been applied to the study of behavioral inhibition, an anxious temperamental style marked by exaggerated physiological responding (Schmidt & Fox 1998; Schmidt, Fox, Schulkin, & Gold, 1999; Schmidt, Fox, Sternberg et al., 1999), attentional hypervigilance (Perez-Edgar & Fox, 2005) and an avoidant behavioral style in unfamiliar situations (Kagan et al., 1988). Several studies have identified an association between behavioral inhibition with an increased risk for social phobia later in life (Biederman et al., 2001; Caspi, Moffitt, Newman, & Silva, 1996; Chronis-Tuscano et al., 2009; Hayward, Killen, Kraemer, & Taylor, 1998; Hirshfeld et al., 1992; Muris, Merckelbach, Wessele, & Van de Ven, 1999; Muris, Merckelbach, Schmidt, Gadet, & Bogie, 2001; Reznick, Hegeman, Kaufman, Woods, & Jacobs, 1992; Schwartz, Snidman, & Kagan, 1999). In one study, 61% of children identified as being behaviorally inhibited at age two had social anxiety when evaluated at age 13 (compared with only 27% of those identified as being uninhibited), and this relationship was specific for generalized social anxiety but not other forms of anxiety (Kagan, 1989; Schwartz et al., 1999). Similarly, Hirshfeld-Becker and colleagues (2007) conducted a five-year follow-up of children assessed for temperament and also found behavioral inhibition significantly predicting new onset of social phobia in middle childhood, without observing an association with this temperamental style and any other anxiety disorders.

In addition to behavioral inhibition in early childhood, research has identified several other temperamental and behavioral styles that may influence the development of social anxiety, including the expression of dysregulated fear. Toddlers with a dysregulated fear profile respond to low-threat situations with disproportionately high levels of fear, failing to calibrate fear levels to actual danger, and show social wariness during preschool and early kindergarten

(Buss, 2011). In a follow-up study conducted by Buss and colleagues (2013), dysregulated fear at age two predicted anxious behavior during laboratory interactions with both unfamiliar peers and adults at age five. This profile also emerged as a strong predictor of future impairment, as children with greater dysregulated fear at age two were four times more likely to show high levels of social anxiety symptoms in kindergarten.

Anxious-solitary behaviors comprise yet another temperamental construct that may relate to social anxiety. Compared to controls, Gazelle, Workman, and Allan (2010) found that third and fourth graders identified by peers as anxious-solitary were at greater risk for developing social anxiety disorder over time, as well as other anxiety disorders. Children fitting this description are verbally and behaviorally inhibited, even among familiar peers, are hesitant to socialize, and frequently engage in "solitary on-looking behavior." For more discussion of temperament and social anxiety, please refer to the chapter by Kagan (Chapter 12) for a more detailed review.

SOCIAL FACTORS

Parenting Style

Social learning theory (Bandura, 1977) posits learning through the observations of others. Not surprisingly, research has established a link between parenting style and internalizing problems in youth (Cooper-Vince, Pincus, & Comer, in press; McLeod, Weisz, & Wood, 2007; Wood, McLeod, Sigman, Hwang, & Chu, 2003). Parents not only can serve as powerful models of social behavior for children, but also have a unique opportunity to shape their children's behavior over the course of years through parent–child interactions. Non-anxious toddlers have been shown to display anxious and avoidant behavior to a stranger after observing their mothers react fearfully (de Rosnay et al., 2006; Gerull & Rapee, 2002). Murray et al. (2008) further demonstrated how mothers' anxious interactions with a stranger displayed in front of their 10-month old children continued to predict the toddlers' avoidant responding at 14 months.

In addition to maladaptive behavioral responses, cognitive biases and catastrophic interpretations of threat are also believed to be subject to parental influence and an important factor in the development of childhood anxiety (Barrett, Rapee, Dadds, & Ryan, 1996; Dix, Ruble, Grusec, & Nixon, 1986; Joiner & Wagner, 1996). The parents of anxious youth often maintain pessimistic expectations about their children's functioning in various domains (Cobham, Dadds, & Spence, 1998; Kortlander, Kendall, & Panichelli-Mindel, 1997). Within this context anxious children begin to demonstrate threat biases consistent with those observed with anxious adults (Hadwin, Garner, & Perez-Olivas, 2006; Muris et al., 2009), in that ambiguous situations are generally perceived as being more threatening (Barrett, Rapee, Dadds, & Ryan, 1996; Bögels & Zigterman, 2000; Dineen & Hadwin, 2004; Miers et al., 2013).

A series of studies (Barrett et al., 1996; Chorpita, Albano, & Barlow, 1996; Dadds & Barrett, 1996) further demonstrated how a discussion with parents led anxious youth to endorse more anxious responding and choose more avoidant solutions to hypothetical threat scenarios than they had prior to the parental influence. As previously mentioned, children with anxiety disorders often have parents who themselves have anxiety disorder diagnoses and there is a growing research base to show that adults with mental health issues demonstrate qualitatively different parenting styles compared to adults without mental health concerns (Reder et al., 2000). For example, Turner et al. (2003) discovered anxious parents to be more physically withdrawn from their children and experience more subjective anxiety while their children engaged in non-threatening play. Others have observed anxious parents to demonstrate less emotional warmth, more catastrophic interpretation, and more open criticism of their children (Hirshfeld et al., 1997; Moore, Whaley, & Sigman, 2004; Whaley, Pinto, & Sigman, 1999), which likely does little to foster a sense of social competence or dispel a fear of negative evaluation in children.

Research on the intergenerational continuity of social fears suggests an early link between parent and child social anxiety as well as provides some evidence for social referencing and negative evaluative concerns as potential transmission mechanisms. Aktar and colleagues (2014) assessed toddler reactions to a social referencing paradigm. Relative to toddlers of non-anxious parents, parental lifetime social anxiety disorder predicted greater fear and avoidance during a social referencing paradigm. Other comorbid parental anxiety disorders did *not* produce significant effects. Parental expressed anxiety during the social referencing situations predicted greater toddler fear and avoidance in the task at 12 months, but not at 30 months, indicating the possibility for developmental differences in risk susceptibility. These findings were robust across both fathers and mothers, highlighting the need for further study of fathers' roles in social anxiety development. Fear of negative child evaluation is another proposed mechanism of social anxiety transmission from parent to child. Schreier and Heinrichs (2010) assessed the degree to which parents worry that their child (9–16 years of age) will face negative evaluation in social situations, and found that for both fathers and mothers, this particular facet of parental anxiety was positively associated with child social anxiety. Moreover, maternal (but not paternal) fear of negative child evaluation mediated the relationships between maternal social anxiety and child social anxiety symptoms, as well as maternal social anxiety and child emotional problems. The authors speculate that mothers with these particular fears may express greater anxiety not only in their own social situations, but also in situations their child faces, passing on threatening interpretations and avoidance behaviors specifically related to social situations. Collectively, these results highlight the contribution that specific elements of parental attitudes and behavior make to child social anxiety development.

In addition to its role in the development of social anxiety, evidence links elements and quality of the parent-child interaction to long-term impairment, as

well as treatment response. Specifically, Festen and colleagues (2013) observed that child perceptions of greater maternal negative affect, and lower emotional warmth, were related to less favorable response to cognitive behavioral treatment for social anxiety, although paternal temperament and parenting style was not related to changes in social anxiety severity from pre- to posttreatment. Exposure to maltreatment from caregivers carries lasting consequences, as adults with social anxiety disorder who experienced childhood emotional abuse or neglect not only reported greater symptom severity and lower quality of life (Bruce, Heimberg, Goldin, & Gross, 2013; Bruce, Heimberg, Blanco, Schneier, & Liebowitz, 2012), but also showed higher attrition rates for both cognitive-behavioral therapy (Bruce et al., 2013) and pharmacotherapy (Bruce et al. 2012).

Given the increasingly apparent role of parent-related factors in the development and/or maintenance of child anxiety, it is not surprising that supported treatments for child anxiety problems (particularly early child anxiety problems) are increasingly incorporating parent-based methods focusing on parent-child interactions and parental patterns of anxiety and avoidance accommodation (e.g., Comer, Puliafico, Aschenbrand et al., 2012; Comer, Furr, Cooper-Vince et al., in press).

Peer Influence

Even for non-anxious youth, one's peer standing in childhood can predict social functioning and emotional adjustment in adulthood (Gettinger, 2003; Kupersmidt, Coie, & Dodge, 1990; Kupersmidt & Dodge, 2004; Parker & Asher, 1987). For youth with social phobia, however, avoidance behavior is a key component of both the development and maintenance of this condition (see Vasey & Dadds, 2001). Beidel and Morris (1995) reported the majority of children with social phobia described unstructured peer encounters in the school setting as their most feared of social situations, which Albano (1995) notes overlaps considerably with the social situations they most try to avoid. A pattern of social avoidance early in childhood reduces the likelihood of encountering positive social contingencies, gaining adequate social skills or developing interpersonal relationships (Boivin, Hymel, & Burkowski, 1995; Hymel, Bowker, & Woody, 1993; Messer & Beidel, 1994), which negatively impacts social functioning over the long term (Rubin, LeMare, & Lollis, 1990; Strauss, Lahey, Frick, Frame, & Hynd, 1988). Children diagnosed with social phobia are less communicative, more behaviorally withdrawn and otherwise socially passive in their interactions with peers (Alfano, Beidel, & Turner, 2006; Spence, Donovan, & Brechman-Toussaint, 1999). They lack basic social skills (Beidel & Turner, 1998; Miers et al., 2013) and their social standing and peer relationships often suffer as a result. Socially anxious children are disliked by peers and are both passively neglected and actively rejected in social spheres (Boivin et al., 1995; Chen, DeSouza, Chen, & Wang 2006; Deater-Deckard 2001; Gazelle & Ladd 2003; Ladd, 2006; Nelson, Rubin, & Fox, 2005; Newcomb, Bukowski,

& Pattee, 1993; Oh et al., 2008; Ollendick, Greene, Weist, Oswald, 1990; Rubin & Krasnor 1986; Stewart & Rubin 1995).

Aversive conditioning experiences have been conceptualized as avenues to the development of social anxiety (Beidel & Turner, 1998; Hofmann & Barlow, 2002), and this has gained some support in information-processing models of social anxiety (Huijding, Wiers, & Field, 2010; de Hullu, de Jong, Sportel, & Nauta, 2011). As opposed to random acts of violence, bullying can have unique social consequences that make it a potent impetus for social evaluation concerns. Bullying behavior is common in school settings (Batsche & Knoff, 1994), where it often occurs in the presence of peers and maintains humiliation of the victim as the primary goal (Smith et al., 1999; Smith & Brain, 2000). It should come as no surprise that bullying victims are at increased risk for developing anxiety problems (see Hawker & Boulton, 2000). Victims of bullying have been shown to demonstrate increased anxiety and shyness, are more withdrawn, have lower self-esteem, display poorer social skills and experience more interpersonal difficulties than other youth (Graham, Bellmore, & Mize, 2006; Haynie et al., 2001; Hazler, 1996). Unfortunately, this relationship may be bi-directional as there is also evidence that introversion, social skills deficits, and lower self-esteem can serve as risk factors for victimization through bullying (Egan & Perry, 1998; Schwartz, Dodge & Cole, 1993). La Greca and Harrison (2005) studied victimization among adolescents and found that even non-violent, indirect harassment could result in high social anxiety if accompanied by a poor quality friendship with a best friend, certainly a possibility for youth with social phobia who are prone to both neglect and rejection by peers. Conversely, they found the positive aspects of a close friendship to be a protective factor against social anxiety but not depression. Further demonstrating the importance of peer interactions, it appears that victimization by peers is related to objective, rather than perceived, hyper-arousal and dysregulation in socially anxious children (Erath, Tu, & El-Sheikh, 2012). Peer rejection may also play a role in the maintenance of social fears, as it interferes with children's use of distraction as a coping strategy for social anxiety (Wright, Banerjee, Hoek, Rieffe, & Novin, 2010).

Unfortunately, there is often a correlation between youth who are victims of bullying by peers and their status as victims of physical, sexual, and/or emotional abuse in the home (Baldry, 2003), suggesting these youth represent victims on a wider social scale (Gladstone, Parker, & Malhi, 2006). Chronic traumatic childhood experiences that come at the hands of influential friends or family can teach children the social environment is a potentially dangerous place best avoided. Gladstone and colleagues (2006) examined a sample of adults presenting to a depression clinic and found that significantly more of those who were bullied also met criteria for a diagnosis of lifetime social phobia as well as agoraphobia. The humiliating aspect of bullying can lead youth to conceptualize their history of abuse as the result of personal shortcomings rather than the result of the unsolicited harassment of others, fueling social anxiety concerns. For example, Singh and Bussey (2009) examined children's self-efficacy for coping with peer aggression

and found increased coping self-efficacy to be associated with less social anxiety. In contrast, lower levels of general self-efficacy are associated with higher levels of social anxiety symptoms, mediating the relationship between negative self-statements and symptom severity (Rudy, Davis III, & Matthews, 2012).

Adolescents begin the process of forging their independence, which involves gradually separating from the social sphere of the immediate family, assuming more autonomy over decision-making, and developing close peer and romantic relationships to increasingly rely upon for support (Choudhury, Blakemore, & Charman, 2006; Eccles et al., 1993; Steinberg & Morris, 2001). By adolescence, teens spend nearly double the time with peers as they do with parents or other adults (Csikszentmihalyi & Larson, 1984) and the presence of close friendships facilitates children's transition to adolescence (Hartup, 1996). This developmental transition is a challenging one and adolescents face more stressors on average than younger children (Colton & Gore, 1991; Compas, Hinden & Gerhardt, 1995). As the opinions and approval of peers become more relevant to the adolescent's development of a sense of self and consolidation of their social standing outside of the family unit, self-consciousness and a fear of negative evaluation become more salient stressors (Fordham & Stevenson-Hinde, 1999; Hymel et al., 1990; Parker & Asher, 1987; Steinberg, 2005). This period is replete with the types of social demands and developmental tasks that one would normally expect to provoke a certain degree of social anxiety (Albano & Detweiler, 2001). Some studies have noted an increase in social fears experienced during adolescence (Weems & Costa, 2005; Westenberg, Drewes, Goedhart, Siebelink, & Treffers, 2004) leading others to posit it is an increase in avoidance observed during adolescence that results in the sudden jump in the onset of social anxiety in adolescents (Rapee & Spence, 2004; Sumter et al., 2009; see also Chartier, Hazen, & Stein, 1998; Muris, 2006; Rao et al., 2007).

Importantly, the relationship between peer rejection and social anxiety is reciprocal. Verduin and Kendall (2008) had children rate the perceived anxiety and likeability of anxious and non-anxious children they had never met after viewing recordings of them. Children's ratings of the perceived level of anxiety being experienced by the child in the recording was positively correlated with those children's own self-reported anxiety, and in fact peers' ratings of the perceived level of anxiety was significantly higher among children with social anxiety disorder. Further, peer ratings of children's likeability were inversely related to peer perceptions of the anxiety experienced by the child, and was particularly low for recorded children with social anxiety disorders. Accordingly, there appears to be a transactional interplay between peer influences and social anxiety.

THE MAINTENANCE OF CHILDHOOD SOCIAL ANXIETY: A COGNITIVE-BEHAVIORAL MODEL

When left untreated, childhood social anxiety can be unremitting and persist as a chronic condition associated with psychological and physical comorbidities, as well as reduced quality of life—including decrements in social functioning,

role functioning, educational attainment, and financial independence (Comer, Blanco et al., 2011; Mendlowicz & Stein, 2000). In fact, only 20–40% of social anxiety disorder cases remit within 20 years of onset, and only 40–60% remit within 40 years (Comer & Olfson, 2010). Having reviewed a number of etiologic factors associated with the development of social anxiety in youth, we now turn our attention to factors that serve to maintain social anxiety. Specifically, we draw heavily on the cognitive-behavioral model of social anxiety offered by Rapee and Heimberg (1997), and highlight key developmental factors to be considered when applying this maintenance model of social anxiety to youth.

What occurs when a socially anxious child confronts a situation that he or she perceives to hold the potential for negative evaluation? Evidence suggests a transactional relationship between cognitive processes and social behaviors, in which negative beliefs about social situations and others' perceptions lead to behavioral avoidance and social withdrawal, which in turn serve to further reinforce negative beliefs and perpetuate avoidance and withdrawal. Chronic avoidance of social situations denies the child important opportunities for corrective experiences that might contradict his or her negative assumptions, opportunities to master his or her anxiety in uncomfortable situations, and opportunities to learn to successfully navigate developmentally appropriate social interactions.

Rapee and Heimberg's (1997) model begins with the notion that individuals with social phobia attach a fundamental importance to being positively appraised by others, and such individuals assume other people are inherently critical (i.e., likely to evaluate them negatively). The model further proposes that when encountering a social situation—whether real, anticipated, or considered in retrospect—the individual forms a mental representation of his or her appearance and behavior as perceived by those around (i.e., the perceived "audience"), which is informed by long-term memory (e.g., prior experiences, recollections of physical appearance), internal cues (e.g., physical symptoms), and external cues (e.g., "audience" feedback). Attentional resources are allocated simultaneously to this internal mental representation and to any perceived threat in the social environment (e.g., someone laughing). The individual also forms a mental representation of the standard against which he or she believes the audience will evaluate his or her performance. According to Rapee and Heimberg (1997), the potential for negative evaluation is assessed by evaluating the discrepancy between the mental representation of one's performance and the mental representation of the standard against which the performance is believed to be evaluated. In a socially anxious individual, a high perceived likelihood for negative evaluation, in which there is a discrepancy between the two mental representations, results in physiological (e.g., increased heart rate), cognitive (e.g., thinking "I'm making a fool of myself"), and behavioral (e.g., blushing) consequences, which in turn further color one's mental representations of oneself and the situation, and the cycle is renewed.

While Rapee and Heimberg's (1997) model provides an overall picture of how youth social anxiety is developed and maintained, it is important to acknowledge the individual contribution of elements that fall under the model's

"cognitive" consequences of perceived dissonance between performance and standards. For example, the concept of negative post-event processing—a tendency to repeatedly review aspects of a social situation or performance in great detail—has been extended to young adults and older adolescents (e.g., Cody & Teachman, 2011), as well as pre-adolescents (e.g., Schmitz, Krämer, & Tuschen-Caffier, 2011). Dysfunctional post-event processing occurs more frequently in 10–12-year-old adolescents with high versus low social anxiety (Schmitz et al., 2011), and is linked to both anxiety levels experienced during social evaluative situations (Schmitz et al., 2011), and to increasingly negative appraisals of personal performance in these situations over the course of several days (Cody & Teachman, 2011; Schmitz et al., 2011). Post-event processing is often conceptualized as part of a larger repetitive cognitive tendency exercised by socially anxious youth: rumination (Wong & Moulds, 2009). Over the course of six months, adolescent social anxiety symptoms directly predict rumination and, via their link with rumination, indirectly predict co-rumination (Jose, Wilkins, & Spendelow, 2012). In Jose and colleagues' (2012) 13–16-year-old sample, girls engaged in rumination and co-rumination more frequently than boys, and associations between social anxiety symptoms and rumination were more robust for females than males, indicating the potential for differential treatment targets and a need for further study of differences across genders.

Negative social schemas represent another maladaptive cognitive feature observed in socially anxious adolescents that plays a role in learned reactions to perceived social challenges. Calvete, Orue, and Hankin (2013) found bidirectional relationships between adolescents' social schemas and automatic thoughts over time, with negative underlying schemas regarding social situations (e.g., preoccupation with others' approval; Young, 1999) both giving rise to and perpetuated by automatic thoughts (e.g., "If I talk to them, they will just think I am stupid"). In Calvete and colleagues' (2013) sample, specific automatic thought categories (i.e., negative self-appraisal) mediated the relationship between other-directed social schemas and social anxiety symptoms over six months. The relationship between broader schemas, individual thoughts, and the development and maintenance of social anxiety is further enriched by the influence of threat-related automatic associations. Cognitive biases active in social situations, such as those encompassed by negative social schemas, provoke fear and worry. In turn, anxious feelings strengthen these schemas both directly, by building associations between discomfort and social situations, and indirectly, by interfering with appropriate behavior and coping in social situations, fulfilling negative expectations (Huijding, Wiers, & Field, 2010). Comparing 12–15-year-old adolescents with subclinical versus low social anxiety, de Hullu, de Jong, Sportel, and Nauta (2011) found that youth with elevated social anxiety viewed social cues and situations as threatening, with both automatic and explicit threat-related associations linked to social anxiety reports. These results have both theoretical and clinical implications, suggesting that socially anxious adolescents' assessments of social situations as threatening may have

origins in both controlled and uncontrolled thought processes. Collectively, these cognitive styles and tendencies likely contribute to the perceived discrepancy between mental representations of personal performance and evaluative standards, thereby maintaining social anxiety.

Given the specific cognitive processes associated with the maintenance of social anxiety, it is not surprising that social anxiety disorder typically onsets during adolescence. Adolescence is a developmental stage characterized by substantial advancement in perspective-taking, meta-cognition, self-awareness, and self-reflection (Kuhn, 2009), during which time peer group approval increases in importance and independent social functioning is expected. Let's consider the example of a socially anxious teenager giving an oral report at the front of his classroom. As a socially anxious teen, he places a tremendous importance on being positively regarded, while at the same time he perceives his classmates to be fundamentally critical. During his oral report, he forms a mental image of how his performance is being perceived, and this image is informed by prior experiences giving oral reports (e.g., he remembers that he lost his place the last time he read out loud), internal cues (e.g., his stomach is turning and his heart is racing, which means he is not in full control of himself), and external cues (e.g., a girl in the first row just yawned). The teen also holds an image of what he believes the standard for an oral report should be (e.g., classmates hanging onto every word he speaks), and the discrepancy between this perceived standard and his image of how he is currently being perceived suggests to him that there is a high perceived likelihood for negative evaluation. This discrepancy causes the teen to have further physiological symptoms (e.g., sweating, blushing) and negative beliefs about his performance. The teen's attempt to simultaneously monitor the environment for evidence of negative evaluation *and* engage in the task of giving the oral report divides the teen's attention and actually disrupts the child's performance, which in turn elicits *actual* negative feedback from classmates (e.g., his classmates look puzzled or shift in their seats as he slows down, he starts to mumble and begins to sweat). This, in turn, prompts further negative mental images of his performance and increased anxiety symptoms, which further affects his performance, and the cycle is renewed.

EVIDENCE-BASED TREATMENT OF SOCIAL ANXIETY

The cognitive-behavioral model of social anxiety suggests several points of intervention in the psychosocial treatment of social anxiety disorder. These include: (1) negative beliefs about oneself, (2) negative beliefs about social situations and other people, (3) negative predictions about the outcomes of situations in which one could be evaluated, (4) patterns of avoidance associated with these negative predictions, (5) attentional focus on social threat cues while in social situations, and (6) negative performance evaluation after engagement in social situations. A comprehensive cognitive-behavioral treatment plan incorporates *skill-building* and *exposure tasks*, and can be implemented in

either group or individual formats (Albano & DiBartolo, 2007; 2007; Kendall & Hedtke, 2006; Beidel, Turner, & Young, 2006). We now discuss these two treatment phases in turn.

The *skill-building* phase of cognitive-behavioral treatment for youth social phobia focuses on the acquisition of various skills that reduce anxiety and facilitate social interactions. Early skill-building sessions emphasize the cognitive components of social anxiety by first introducing the concepts of automatic thoughts and rational responses. Self-monitoring is emphasized as children are taught to identify their own anxious thoughts and thinking errors. Some treatments (e.g., Kendall & Hedtke, 2006) also introduce relaxation training during these early sessions. After children get these concepts down, therapists engage children in cognitive restructuring exercises, teaching them how to challenge their dysfunctional beliefs (e.g., a child afraid of tripping in public would be taught to ask herself "Have I ever tripped in front of others before?" "What are the realistic chances that I would trip in front of others today?" "Even if I did actually trip in front of others, would that be *so* bad?"). After cognitive restructuring is successfully addressed, treatment moves to problem-solving strategies targeted to expand the repertoire of coping behaviors available in handling problematic social situations. Typically avoidance and social withdrawal are the only problem-solving strategies that socially anxious children have found effective in reducing their anxiety. Of course, this strategy only works in the short term, and over the long term chronic avoidance leaves the child underequipped to master anxious experiences. The final stage of the skill-building treatment phase typically focuses on identifying and strengthening appropriate social and assertiveness skills.

The *exposure* phase of treatment builds on the socially anxious child's newly expanded repertoire of coping skills, giving them opportunities to practice these skills in increasingly fear-inducing contexts. These tasks give children practice experiencing, tolerating, enduring, and mastering distress. During cognitive-behavioral therapy for social anxiety disorder, the therapist and child develop a fear hierarchy of avoided social situations based on the child's idiosyncratic fears (e.g., calling a friend to get together, reading aloud, being assertive), and this hierarchy serves as an exposures roadmap. A graduated series of exposures is recommended, from lowest to highest fear-inducing situations. The goal is to always push the child further than they would naturally push themselves, while never pushing the child further than they are able to physically tolerate. Typically exposures begin as in-session tasks, carried out in the context of the warm and therapeutic relationship, and the child is expected to increasingly engage in out-of-session exposure tasks. These out-of-session exposures are critical for generalization of gains.

Importantly, some supported treatments for social anxiety disorder in youth place heavy emphasis on cognitive components (e.g., Beidel & Roberson-Nay, 2005), whereas other successful programs place less emphasis on cognition (Albano & DiBartolo, 2007; Comer, Puliafico, Aschenbrand et al., 2012).

Future work is needed to better understand subpopulations of socially anxious youth for whom cognitive treatments offer improved outcomes, and those youth for whom a more focused behavioral strategy may be appropriate. Developmental considerations suggest that cognitive strategies are particularly misguided in the treatment of early-onset social anxiety (i.e., below age seven) (Comer, Puliafico, Aschenbrand et al., 2012).

A public conception may exist that exposure tasks are not appropriate (or even cruel) within anxiety treatment, as evidenced by an editorial in the *New York Times* entitled "The Cruelest Cure" that described exposure tasks within a manualized CBT for adult anxiety (Barlow, 2002) as "surprisingly simple … but while many clinicians praise its well-documented results, others take a dimmer view of what one clinician calls 'torture, plain and simple'" (Slater, 2003, p. 34). Such perceptions could expectedly deter practitioners from adopting exposure-based treatment for socially anxious youth, as exposure tasks might be thought to rupture the therapeutic alliance. Importantly, Kendall et al. (2009) used growth curve modeling to examine the impact of exposure-based tasks on therapeutic alliance in the treatment of childhood anxiety disorders. Their analysis found no indication that therapeutic alliance suffers with the introduction of exposures. In fact, therapeutic alliance continued to grow across treatment after the introduction of exposure-based tasks. Given the paramount importance attributed to therapeutic alliance by a sizable proportion of mental health practitioners (e.g., Boisvert & Faust, 2006), these findings should inform dissemination efforts that promote the use of exposure-based treatments for childhood social anxiety disorder. Indeed, practitioners who are hesitant to consider in-session exposure-based strategies to treat childhood social anxiety out of concern for the therapeutic relationship may more readily consider exposure tasks in the context of Kendall et al.'s findings. Moreover, public rhetoric characterizing exposure-based treatments as "torture" (e.g., Slater, 2003) appears to be quite misguided, given that alliance does not diminish following the onset of in-session exposure tasks. Regrettably, such rhetoric may deter parents of socially anxious children from enrolling their children in treatments that research evidence supports.

Cognitive behavioral therapy for childhood social phobia can be implemented in a group or individual format. Many argue that a group treatment format for social anxiety disorder is preferable (Albano & DiBartolo, 2007; Beidel et al., 2006). Many children with social anxiety disorder feel isolated, and have never discussed their social anxiety with a peer. A group format, in which peers share their anxious thoughts and feelings with one another, facilitates opportunities to normalize the experience of social anxiety. In addition, a group format affords in-session and natural exposures because group members can serve as role-play partners or audience members. Moreover, peer group members can provide feedback to one another, and this feedback may be more credible than feedback provided by the adult therapist. That said, individual treatment for social anxiety disorder in children and adolescents has received considerable empirical support, as well (e.g., Kendall et al., 2008).

Regardless of the group or individual cognitive-behavioral therapy format, a focus on maintaining skills learned during treatment may be particularly crucial to the long-term success of youth with social anxiety. Kerns, Read, Klugman, and Kendall (2013) found that among eight to 14-year-olds with diagnosed anxiety disorders, children with social anxiety responded positively to individual cognitive-behavioral therapy at a rate similar to anxious youth without pretreatment social anxiety symptomatology, at both posttreatment and at one-year follow-up. However, these youth showed less maintained improvement at a 7.4-year follow-up than anxious youth without baseline social anxiety symptoms. These findings suggest that maintenance of treatment-related gains, perhaps through periodic booster sessions, may be especially important for youth with social anxiety.

The role of family in the treatment of socially anxious youth merits comment and further consideration. Although family treatment modalities (i.e., treatments in which parents are present for most sessions) have shown efficacy in the treatment of children below the age of 12 (Kendall et al., 2008), when working with the adolescent social anxiety disorder peer group, treatment formats or individual treatment formats with minimal parent involvement (i.e., parents are present for only a handful of sessions) are typical (Albano et al., 1995). However, in considering the role of parental overprotection and overcontrol in the maintenance of anxiety (Chorpita & Barlow, 1998), we offer for consideration a model of intervention focused on increasing independence on the part of the adolescent, while addressing the parents' role in the maintenance of the disorder. This is especially important as adolescents will eventually need to transition to greater independent functioning by the end of high school, to meet various developmental tasks associated with emerging adulthood and role transitions (see Table 10.1; also, see Arnett, 2004).

TABLE 10.1 Developmental Tasks of Adolescents Transitioning to Adulthood

Establish emotional independence from parents.
Manage interpersonal issues and relationships independently
Take control of personal self-care (e.g., sleep, diet, exercise, self-soothing).
Develop self-identity ("This is who I am").
Manage own healthcare and develop appropriate healthcare-seeking behavior and doctor–patient relationships.
Accept sexual identity.
Complete basic educational requirements.
Enter college/workforce.
Manage own finances.
Develop and maintain lasting social relationships.
Develop and maintain lasting romantic relationship.
Separate successfully from parents/parental figures.
Formulate longer-term career goals.
Live independently from parents.

The functional impairments associated with social anxiety disorder may also impair an adolescent's ability to meet developmental milestones and effectively transition to more independent functioning. Confounded by these anxiety-associated impairments are the parents' concerns that certain opportunities will be missed and therefore, greater control involvement in the adolescent's functioning is reinforced for fear of their failing. Consider the situation where a junior in high school—an adolescent with social anxiety disorder—has difficulty with assertiveness and situations involving unfamiliar people. A college fair is being held at the school and all juniors are invited to attend to meet and ask questions of various college representatives. This particular student does not want to go and offers that "I won't know what to say." "I'm not even sure I want to go to their schools." "What if they ask me what major I want? I don't know!" While some anxiety is typical for this situation for any adolescent, social anxiety disorder makes the anxiety reaction much more intense and increases the pull towards avoidance. Then, instead of having the teen attend while stressed or not attend at all, a parent goes with the teen and asks all the questions, responds for the teen when a question is asked of him or her, and essentially, the parent does all the work of signing up for catalogues and information. While this may be fine in one or two circumstances, if the parent continues to overprotect and over involve him- or herself on behalf of the teenager, the natural process of learning how to manage these situations while anxious (as many other people are naturally anxious in these settings) will not occur. A dependence on others to assist in social problem-solving efforts, via a relationship between social anxiety and increasing social support-seeking strategies (Wright et al., 2010), may contribute further to lost opportunities for personal growth in adolescence.

As a result of our experience working with adolescents and emerging adults with social anxiety, we developed the Launching Emerging Adults Program (LEAP; Albano, 2014). LEAP integrates the core components of effective cognitive behavioral treatments for anxiety disorders in adolescents and adults, with specific components designed to address patient-caretaker dependency, role transitions, and attainment of behaviors necessary for independent adult functioning. Development of a transition-focused CBT protocol for this transition age is well justified on both theoretical and public health grounds. As summarized in this review, social phobia has its onset by adolescence and results in serious distress, disability, and functional impairments. Truncated educational attainment, failure to enter or advance in the work force, extended dependence on family or welfare systems, and poor marital outcomes occur with social anxiety disorder (see Detweiler, Comer & Albano, 2011; Hambrick, Comer & Albano, 2011). As noted in the CAMS follow-up study (Ginsburg et al., 2014), even the best of treatments do not confer long-term syndrome relief, and we argue that this may reflect at least partially a problem in not fully addressing the primary context of the child or adolescent—the family relationship. In addition, studies of developmental outcomes following treatment are lacking. Focusing on psychosocial treatment, given the chronicity of the disorder and its extended deleterious

impact on adult functioning, LEAP is established as a test of an integrated, developmentally-informed, psychosocial treatment protocol aimed at both syndrome relief and maximizing functional outcomes. LEAP is based on our experiences in treating transition-aged adolescents and young adults in our anxiety clinics (Albano & Detweiler, 2001; Albano & Hayward, 2004) and informed by critical reviews of the developmental psychology of emerging adulthood (e.g., Arnett, 2004).

The 20-session LEAP intervention for adolescents and emerging adults (ages 15–28) with social phobia and related anxiety conditions, who are transitioning to adulthood, integrates components of cognitive-behavioral treatments for anxiety (e.g., self-monitoring, psychoeducation, cognitive-restructuring, exposure) with developmentally-informed interventions designed to promote healthy adult transitions. These components are meant to increase self-sufficiency by targeting patient skills deficits (e.g., realistic goal setting, problem-solving, assertiveness) and parental over-involvement (parent psychoeducation, goal setting, proactive cognitive coaching). It is hypothesized that anxiety and its comorbidities are maintained in part by parental behaviors that inadvertently allow the adolescent either active or passive avoidance of anxiety-challenging activities. For example, in our practice, we see many families where the parents continue to negotiate the adolescent's world well into the college years—going so far as to contact professors for course material and to make up missed exams. In response to this over-involvement, we propose an adaptation to traditional CBT approaches for social anxiety disorder whereby in addition to the fear and avoidance hierarchy of social situations, a developmental task hierarchy is also produced for the adolescent. This hierarchy involves tasks such as making and keeping one's own routine medical appointments, search for a summer job on your own, negotiating with teachers on your own, managing a bank account, setting up college visits and interviews on your own, and similar tasks. Whereby parental overprotection and overcontrol has been implicated in the maintenance of anxiety disorders (Parker, 1983), we suggest that these behaviors are synergistic with the anxiety and promote continued dependency on the parents and impede progress in mastering the developmental challenges of transitioning to adulthood. Parent inclusion in the LEAP protocol is meant to identify and critically evaluate parent-patient patterns of interaction to expose unhealthy relationships that may take the form of overprotection, overcontrol, or lack of confidence in the patient by the parent, and/or avoidance of taking on age-appropriate responsibility on the part of the patient (passive or active). Parents and teens receive several conjoint sessions to establish this developmental task hierarchy based on the items in Table 10.1 and then learn how to transfer responsibility for these tasks to the adolescent, while giving the parents some support for letting consequences fall where they may as the teen learns to manage on his or her own. Figure 10.1 presents a schematic of the LEAP model of therapy.

In addition to addressing developmentally relevant issues such as in LEAP, efforts have been made to improve intervention transportability. Lau, Pettit, and

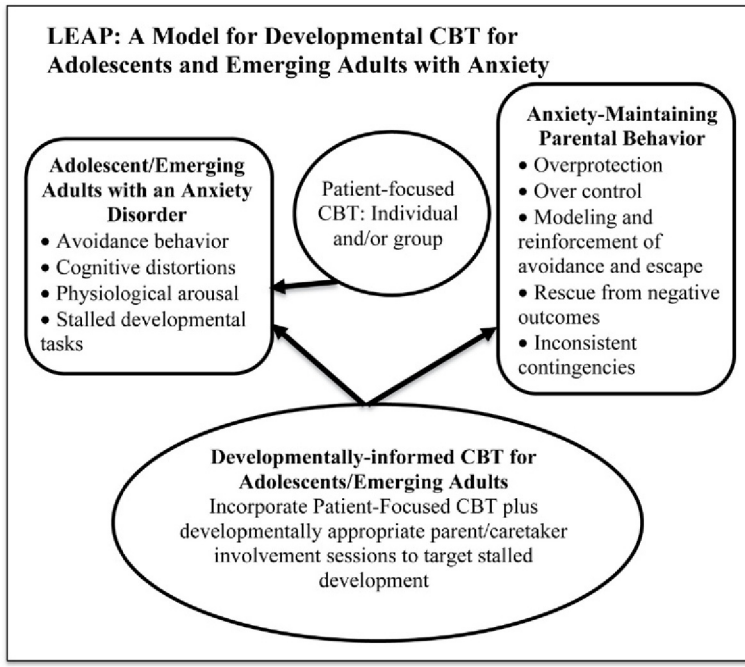

FIGURE 10.1 Adolescent Social Anxiety Treatment Model. *Adapted from A.M. Albano (unpublished).*

Creswell (2013) embedded training to alter negative interpretations of ambiguity, known as cognitive bias modification of interpretations (CBM-I), into 45 brief bedtime stories. Parents read these stories to their seven to 11-year-old children on three consecutive evenings. Compared to a monitored control group, children exposed to stories supplemented with CBM-I experienced a significant decrease in social anxiety symptoms post-brief intervention, and were more likely to interpret ambiguous situations as harmless. Though evidence is preliminary for several reasons, including a need for further research on the effectiveness of CBM-I as a standalone intervention component, parental administration of home cognitive bias modification training may be a useful preventive intervention for children with subclinical social anxiety symptoms. Moreover, the training modality shows promise in the areas of accessibility and cost-effectiveness, as the program may be implemented at home, using a parent-as-therapist approach. Further research is necessary to explore its potential as a prevention measure for children with subclinical social anxiety. Sarver, Beidel, and Spitalnick (2014), in the spirit of addressing dissemination obstacles, made an adjustment to an existing empirically supported social skills treatment for social anxiety. To reduce costs, time, and other resources associated with the homework and in-vivo peer generalization strategies utilized in Social Effectiveness

Therapy for Children (Beidel, Turner, & Young, 2006; Beidel et al., 2007), Sarver and colleagues assessed the feasibility and acceptability of using virtual environments housed on in-clinic and at-home personal computers. In this open trial, children and parents endorsed satisfaction with the virtual components, and parents indicated they would recommend the program to others. While further study is required to assess the efficacy of virtual components as substitutes for in-vivo exercises, these adjustments shows potential to improve dissemination efforts. Such findings specific to child social anxiety are parallel with broad trends across the field toward the increased use of new technologies to broaden the scope and accessibility of evidence-based care (see Comer & Barlow, 2014).

Finally, advancements in the development of efficacious psychosocial treatments for childhood social anxiety disorder have been paralleled by advances in the evaluation of psychopharmacologic interventions for childhood social anxiety disorder. Specifically, a number of selective serotonin reuptake-inhibitors (SSRIs) have shown efficacy in the treatment of several childhood anxiety disorders, including social anxiety disorder (Birmaher et al., 2003; RUPP Anxiety Study Group, 2001). The Child-Adolescent Anxiety Multimodal Study (CAMS) evaluated the relative efficacy of cognitive behavioral therapy, sertraline, and their combination in the treatment of childhood anxiety disorders, relative to pill placebo, in a randomized sample of 488 youth—ages seven to 17—treated across six treatment sites. Acute (Walkup, Albano, et al., 2008) outcomes of the CAMS trial found that the combination of cognitive behavioral therapy and sertraline offered a greater treatment response (~81% of treated youth deemed treatment responders by blind evaluators) than either of the two monotherapies alone. Roughly 60% of anxious youth treated with cognitive behavioral therapy alone and roughly 55% of anxious youth treated with sertraline alone were deemed treatment responders by blind evaluators. After six months of maintenance treatment, gains were maintained in CAMS treatment responders (Piacentini et al., 2016). However, in an uncontrolled follow-up study involving 288 participants of CAMS (ages 11–26; M = 17), nearly half had relapsed, whereas 46.5% of participants were still in remission. Treatment response was more likely to be maintained in males and with stable family functioning (Ginsburg et al., 2014). The CAMS findings document the availability of three effective short-term treatments for childhood anxiety disorders, with combination treatment offering a superior response rate to that offered by CBT or SSRI alone. However, relapse rates over the longer term clearly indicate that much work is necessary to evaluate ways to maintain treatment response for youth as they age into later adolescence and adulthood.

CONCLUSION/FUTURE DIRECTIONS

In summary, childhood social anxiety is highly prevalent, and often persists as a chronic condition associated with psychological and physical comorbidities, as well as reduced quality of life, educational attainment and financial independence,

as well as potential stalled development. Research has identified a host of biological, parenting, social, and cognitive factors associated with the development and maintenance of social anxiety in youth. Importantly, the majority of research on the development of social anxiety disorder has focused exclusively on risk factor main effects (i.e., focusing on only one domain of influence, such as structural abnormalities in the brain, susceptibility genes, or distorted cognitions). Despite theoretical accounts of how these factors may interact, a new generation of research is now needed to empirically evaluate the complex interplay among risk factors and to examine how these individual domains of influence transact with one another to eventuate in childhood social anxiety disorder.

Over the past decade, rapid advances in non-invasive neuroimaging technology now provide windows into the living brain in ways previously unimagined (Gerber & Peterson, 2008). These developments have greatly advanced our understanding of biological factors associated with the development of childhood social anxiety disorder, but it is nonetheless important to comment on the current quality of brain images. Despite the improved resolution of images of the live brain provided by current imaging technologies over prior technologies, the pictures offered are still crude when compared to the seemingly infinite complexity and detail of the human brain (see Peterson, 2003). In addition, with current technology, individuals must lay relatively still in a laboratory when undergoing magnetic resonance imaging, and thus our window into the functioning brain does not yet reveal brain functioning in naturalistic settings while performing complex, "real world" tasks. Moreover, research in molecular and population genetics has made it increasingly clear that childhood mental disorders do not exhibit simple, single-gene inheritance patterns. Research in childhood social anxiety disorder needs to now shift to the search for multiple susceptibility genes that may, under specific circumstances, increase vulnerability for the development of social anxiety disorder.

Despite the high prevalence and very heavy toll of childhood social anxiety disorder, systematic problems with the availability, accessibility, and acceptability of effective care prevent affected youth from receiving the services they need. Epidemiologic surveys document long delays in treatment-seeking and low rates of treatment among affected youth. Less than 5% of individuals with social anxiety disorder make contact with a service provider within the first year of disorder onset, and the median delay of treatment initiation after initial onset of social anxiety disorder is 16 years (Wang et al., 2005). Given the existence of highly effective treatments for childhood social anxiety disorder (e.g., Albano & DiBartolo, 2007; Beidel et al., 2006; 2007; Kendall & Hedtke, 2006), low rates and long delays in service use underscore the need for greater efforts to increase disorder awareness, enhance treatment access, and improve clinical recognition.

Finally, investigations of social anxiety in youth have been largely confined to industrialized regions of the world. Understanding the development and phenomenology of childhood social anxiety in regions beset by economic, educational, wartime, and health-related hardships is critical to understanding cultural

variations and the global burden of childhood social anxiety disorder and to planning mental health service delivery in these areas. Future efforts are needed to advance research methods in developing countries and refugee populations, and to advance effective care and mental health literacy in resource-poor regions of the world.

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