



# Understanding and treating social anxiety: lessons learnt from developmental cognitive neuroscience



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Social anxiety disorder (SAD) is common and disabling, and generates significant personal and economic costs. Individuals with SAD experience a paralyzing fear of negative evaluation from others and, as a result, social situations are endured with extreme distress or are avoided. The majority of adult SAD begins in youth [1], marking late childhood and early adolescence as a developmentally sensitive juncture for the emergence of persistent social fears and worries. While models of child and adolescent social anxiety have emphasized various risk factors to explain individual differences, less is understood about how typical developmental changes may influence the expression of risks during the transition to adolescence.

Here, I present a brief overview of current models of SAD in children and adolescents. Next, I argue how a myriad of social and biological changes across adolescence may enable some of the risk factors associated with SAD to be expressed in this age range. Finally, I consider whether the cascade of typical neurodevelopmental changes operating across adolescence also provides a more

optimal platform upon which targeted interventions can yield stronger and longer-lasting benefits to anxiety resilience across the lifespan.

### Why do some children & young people develop social fears & worries?

Extensive work has identified biases at several stages of information processing in children and adolescents with social anxiety. Such biases may give rise to a preferential processing of threatening over benign information, which may maintain social anxiety by shaping socially avoidant behaviors. Biases have been reported at early stages of processing, such as in the allocation of attention to threatening stimuli [2], although studies vary in the direction of these biases, with some studies finding faster reaction times to probes that replace threatening stimuli (reflecting hypervigilance) and others reporting slower reaction times (reflecting attention avoidance). Our group have also studied biases during learning, for example, in acquiring and failing to extinguish fearful associations between neutral

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(social) cues and negative outcomes [3]. We have also studied biases at later stages of processing, such as in the interpretation of social situations [HALLER SPW *ET AL.* A NOVEL PICTURE-BASED TOOL FOR MEASURING INTERPRETATION BIASES IN ADOLESCENTS (2013), MANUSCRIPT IN PREPARATION]: when viewing themselves in various social scenarios, adolescents with greater levels of social anxiety are more likely to endorse negative explanations, and less likely to endorse positive explanations for their peers’ social behavior, regardless of whether the picture contains connotations of positive, negative or ambiguous outcomes. Interestingly, these biased interpretations only appear to characterize adolescent social anxiety; in children, findings are more mixed [4].

Neuroimaging studies have begun to investigate the extent to which these cognitive biases in adolescent SAD might reside in perturbations within various brain circuits. Earlier studies using simple social stimuli, such as faces with negative emotional expressions to tap SAD-related neural perturbations, have gradually paved the way for more ecologically valid paradigms that aim specifically to capture aspects of age-typical peer interaction, such as anticipating peer feedback, receiving rejecting and accepting feedback from peers, and responding flexibly in uncertain social situations [5–7]. These studies show that relative to healthy adolescents, SAD adolescents manifest heightened amygdala sensitivity and perturbations in the striatum in response to socially provocative stimuli/exchanges. However, differences also extend into regions that are connected with these structures including the medial and ventral lateral prefrontal cortex (PFC), insula, and the anterior cingulate cortex [7]. Thus, similar to adult SAD, adolescent SAD is also characterized by functional perturbations in frontal–amygdalae–striatal circuits.

While it is unclear where these neurocognitive profiles originate, there is evidence to suggest that social anxiety symptoms may partly be inherited [8] and partly acquired through social learning mechanisms [9]. It is likely that nature and nurture correlate, and interact to produce neurocognitive biases in SAD youth, but this awaits empirical demonstration.

**Does the transition into adolescence bring out risks associated with social anxiety?**

While we are beginning to gain a basic understanding of why some youth develop social anxiety and others do not, an outstanding question is why the peak age of onset of SAD occurs in

the transition to adolescence. The transition to adolescence is associated with a myriad of age-typical social and biological changes. Widespread changes occur in the environment: in educational curricula and associated classroom routines, time spent with peers and a corresponding increase in the salience of peer relationships, and finally, more subtle, culturally influenced changes in societal expectations about independence and responsibility. However, recent research has also revealed parallel changes in biology, not just in the body, through circulating pubertal hormones, but also in regional brain anatomy and functional circuits [10]. Indeed, studies have reported linear increases in white matter volume (attributed to myelination) and age-associated decreases in gray matter volume, which may reflect a process of experience-dependent pruning. These age-associated declines vary across regions, with subcortical regions maturing sooner and regions of the PFC maturing last [11].

Although it is not clear how these structural changes impact changes in brain function, there is nonetheless an emerging corpus of data also showing age-associated differences in brain activity between adolescents and adults, adolescents and children, and across phases of adolescence during various emotion and social processing tasks [12,13]. These data describe two patterns of change. First, there is increased engagement of subcortical structures (amygdalae and striatum) during the experience of emotionally provocative stimuli in adolescents (relative to adults and children), but a corresponding reduction in recruitment of regions of the PFC (relative to adults) – although the direction of these differences vary across studies and appear task dependent. These findings of the functional immaturity of PFC regions (which are often recruited during higher-order cognitive regulation of emotion) relative to early-maturing subcortical regions (which are implicated early in the response to negative and positive stimuli) have been used to explain why adolescents experience heightened emotional responses. The second set of age-associated findings describe the decreasing engagement of key social brain regions (medial PFC and anterior cingulate cortex) across adolescence through to adulthood, during tasks that measure responses to social stimuli, from the basic perception of animated objects to higher-order attribution of intent to another individual [13]. These changes have been interpreted to imply that as adolescents mature and gain more experience of complex social situations, their social



understanding increases and becomes increasingly automated, as reflected in parallel behavioral and neuronal changes.

However, how might this relate to SAD onset? Some have posited that for most adolescents, the prolonged changes in different brain circuits and the cognitive processes that they serve, can allow for more flexible responses to changing social environments to emerge [14]. However, we argue that for a minority of adolescents, these changes can reveal the risks associated with the acquisition of maladaptive and stable information-processing biases [HALLER SPW *ET AL.* A DEVELOPMENTAL ANGLE TO UNDERSTANDING THE MECHANISMS OF BIASED COGNITION IN SOCIAL ANXIETY (2013), SUBMITTED]. More particularly, we have suggested that in some adolescents, heightened responses to emotion can exert powerful effects on attention, highlighting possible social threats in the environment. Furthermore, as adolescents are increasingly able to understand the complexities of the social world that they inhabit, this could unleash, in some adolescents, biases associated with the interpretation of these social situations. This might explain why linkages between interpretation biases and social anxiety only emerge in adolescents, and not in children [4]. The extent to which these typical neurocognitive changes bring out social anxiety in some individuals may be made even more likely when actual changes in the environment are also experienced. For example, changing educational expectations and in the time spent with peers can, for some individuals, provoke heightened emotional responses and underscore the ambiguity of social situations, respectively.

### Do these age-typical neurocognitive changes associated with adolescence also mean greater plasticity to interventions aimed at targeting SAD?

While we have argued that age-typical neurocognitive changes can bring out vulnerability associated with SAD, it is also feasible that protracted maturational changes in the brain

can mean a greater responsiveness to positive environments. Indeed, if changes in gray matter structures do imply experience-dependent pruning – and moreover, some cognitive strategies do become increasingly stable and trait like in this period [15] – then it may be the case that acquiring adaptive strategies delivered through interventions in this age range may yield stronger and longer-lasting benefits.

There are a number of therapeutic strategies for targeting child and adolescent SAD. However, frontline psychological treatments for SAD have yielded poor outcomes [16] while there are concerns over sustained use of pharmacological treatments [17]. Our group has begun to study the effectiveness of interventions that target specific neurocognitive factors known to be associated with individual differences in social anxiety, and which are thought to be maturing in adolescence. One strategy has been to use cognitive bias modification training tasks to target emergent attention biases to social threat and interpretation biases of social situations [18]. Another strategy is to change activity in subcortical–prefrontal brain circuits, through the delivery of functional MRI-based neurofeedback on participants' own neural activity elicited during emotion processing [19]. The study of these interventions that are rooted in basic science is still in its infancy. However, a future step will be to examine whether the effects of such targeted interventions vary across age and whether sensitive periods for intervention occur.

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