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Gaining insight into adolescent vulnerability for social anxiety from developmental cognitive neuroscience

Justin D. Caouette, Amanda E. Guyer*

Department of Human Ecology, Center for Mind and Brain, University of California, Davis, CA, United States

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ABSTRACT

Social anxiety disorder (SAD) markedly impairs daily functioning. For adolescents, SAD can constrain typical development precisely when social experiences broaden, peers' opinions are highly salient, and social approval is actively sought. Individuals with extreme, impairing social anxiety fear evaluation from others, avoid social interactions, and interpret ambiguous social cues as threatening. Yet some degree of social anxiety can be normative and non-impairing. Furthermore, a temperament of behavioral inhibition increases risk for SAD for some, but not all adolescents with this temperament. One fruitful approach taken to understand the mechanisms of social anxiety has been to use neuroimaging to link affect and cognition with neural networks implicated in the neurodevelopmental social reorientation of adolescence. Although initial neuroimaging studies of adolescent SAD and risk for SAD underscored the role of fear-processing circuits (e.g., the amygdala and ventral prefrontal cortex), recent work has expanded these circuits to include reward-processing structures in the basal ganglia. A growing focus on reward-related neural circuitry holds promise for innovative translational research needed to differentiate impairing from normative social anxiety and for novel ways to treat adolescent SAD that focus on both social avoidance and social approach.

1. Introduction

Nearly everyone experiences normative, non-impairing levels of anxiety. Anxiety can be a motivator to overcome challenges, such as meeting an important deadline. It can also be adaptive in certain contexts, such as protecting one's social status until becoming familiar enough with new people to let down one's guard. For some individuals, however, anxiety becomes debilitating to the extent that daily functioning is markedly impaired. Furthermore, impairing levels of anxiety that onset early in life can interrupt the developmental progression of typical life experiences, such as socializing with peers at school.

One such anxiety disorder that commonly has an onset early in life is social anxiety disorder (SAD). SAD is indicated uniquely by an extreme, irrational and impairing fear of social situations, such as being criticized or negatively evaluated by other people (American Psychiatric Association, 2000). Although SAD can be diagnosed in early and middle childhood, a disproportionately high prevalence of cases emerges in late childhood and early adolescence (Beesdo et al., 2010; Stein et al., 2001). The age-of-onset distribution for SAD is unique from any other anxiety disorder. SAD onset rates increase considerably at age ten and plateau in the early twenties, whereas onset rates for generalized anxiety disorder (GAD), panic disorder, and specific phobias increase more steadily during this same period (Beesdo et al., 2010). About 50% of SAD cases onset by age 13, with 90% reaching onset by age 23 (Stein, 2006).

* Corresponding author at: 267 Cousteau Place, Center for Mind and Brain, University of California, Davis, CA 95618, United State. Tel.: +1 530 297 4445; fax: +1 530 297 4400.
E-mail address: aeguyer@ucdavis.edu (A.E. Guyer).

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Given its age-of-onset patterns and characterization, SAD can constrain normative development in a life stage typically marked by expanding social experiences, a strong need for social approval and high social status, and an increased investment in friendships and romantic relationships. A multitude of expected, stage-dependent changes that facilitate social and emotional development renders adolescence as a period when fears of social evaluation or humiliation that characterize SAD can become especially pronounced. These changes in adolescence include but are not limited to brain maturation and puberty (Blakemore and Choudhury, 2006; Forbes and Dahl, 2010; Giedd, 2008; Nelson et al., 2005; Sisk and Zehr, 2005), broadened social opportunities and more exposure to unfamiliar peers (Graber and Brooks-Gunn, 1996; Smetana et al., 2006), and new motivations for peer and romantic relationships (Steinberg, 2008). Not only can these shifts contribute to vulnerability for SAD, they can exacerbate its consequences, as in the case of adolescents who withdraw from peers and social situations at a time when establishing healthy peer relationships is important for well-being.

A growing empirical base has shown that adolescents who are affected by SAD or at risk for SAD exhibit heightened neural activation in appetitive-motivational systems (Bar-Haim et al., 2009; Guyer et al., 2014, 2012a, 2006; Haber and Knutson, 2010; Hardin et al., 2006; Helfinstein et al., 2011, 2012; Lahat et al., 2012; Perez-Edgar et al., 2013). This is an important consideration given that, historically, greater theoretical and research attention has been paid to the involvement of fear-avoidance neural systems, involving the amygdala for example, in SAD and risk for SAD (Davis, 1992; Kagan, 1996). The goal of this review paper is to highlight these recent findings from developmental cognitive neuroscience research that have deepened our understanding of the brain–behavior relationships in adolescent SAD and risk for SAD. This motivation stems from an understanding that the brain is a clear mediator of individual differences in behavior and that neurodevelopment in adolescence has distinct characteristics relative to other developmental periods. Although assessments based on other key physiological modalities (e.g., heart rate, heart rate variability) have provided critical insight into the development of and risk for SAD (Anderson and Hope, 2009; Beidel, 1988; Beidel et al., 1991; Porges, 2007) and relate to brain function (Thayer et al., 2012), the initial findings involving appetitive-motivational neural systems have come from studies using functional magnetic resonance imaging (fMRI). By mapping relationships among brain function, cognitive processes, and behavioral characteristics and phenotypes, we may eventually be able to use fMRI studies to test the effectiveness of treatments, such as cognitive behavioral therapy (CBT) on improving aberrant cognitive and affective processing in order to help alleviate anxiety (Pine et al., 2008).

Our review paper proceeds as follows. First, we provide a characterization of adolescent SAD including its symptoms, prevalence, treatment strategies, and association with other disorders. Second, we describe how a better understanding of adolescent SAD has also emerged from studies of early-life behavioral inhibition (BI). BI is a type of temperament characterized by heightened fear reactions to unfamiliar stimuli and extreme social reticence that confers a degree of risk for later SAD. Third, we briefly review established findings involving avoidance-based neural systems that have associated elevated amygdala activation with SAD and BI. Fourth, we discuss recent studies that have focused attention on the neural circuitry underlying reward motivation as a system to consider, in conjunction with avoidance systems, in the study of adolescent SAD and risk for SAD (readers are referred to Bishop, 2007; Britton et al., 2011; LeDoux, 2007), for more detailed reviews on anxiety and fear-avoidance circuitry). This recent work primarily includes differential patterns of social-information processing and performance-based reward processing in appetitive-motivational circuitry specific to adolescence, BI, and SAD. Fifth, we consider adolescence as a vulnerable period for SAD and suggest that a fuller characterization of when and why adolescents face heightened risk for SAD can be accomplished by examining: early-life BI, the distributed network of brain structures implicated in both fear-avoidance and appetitive-motivational processes, and adolescents' sensitivity to social evaluation and status (Fig. 1). We thus suggest a dimensional perspective of SAD that conceives of it as fueled by imbalances or conflicts between fear-avoidance and appetitive-motivational systems when responding to both social and non-social stimuli. We propose a conceptual model of adolescent vulnerability to SAD, central to which is the idea that socially anxious adolescents experience an approach-avoid conflict that arises from age-typical increased investment in peer evaluation coinciding with extreme fear of humiliation and embarrassment. Moreover, we stipulate that this conflict is mediated by aberrant functioning in a network of brain regions (i.e., amygdala, basal ganglia, and prefrontal cortex) that motivate cautious approach, and is moderated by individual differences in inhibited temperament that are rooted in childhood. Finally, we end with general conclusions based on the work reviewed.

2. Adolescent social anxiety disorder

SAD is defined by the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM) as a persistent and impairing fear of being in social or performance situations that involve potential evaluation or scrutiny by others. This fear often revolves around the potential for embarrassment and humiliation particularly within social contexts, such as public speaking, performing in class, starting conversations with peers, and social activities that involve interaction with others (e.g., dances, parties). The hypersensitivity to and fear of scrutiny from others that accompanies SAD heightens apprehension, arousal, and panic in these social and performance contexts (Heimberg et al., 2010; Kashdan and Herbert, 2001; Phan and Klumpp, 2010; Stein, 2006).

When exposed to the feared situation, the individual experiences anxiety despite the recognition of the excessive or unreasonable nature of this fear. The anxiety is often accompanied by physiological and behavioral reactions, such as elevated heart rate, sweating, shaking, crying, or refusal to speak. The individual will avoid the situations that put them at risk for public scrutiny, humiliation, or negative evaluation by others. Both adolescents and adults