Developmental Cognitive Neuroscience 13 (2015) 11–20

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: http://www.elsevier.com/locate/dcn

Review

Social anxiety disorder in adolescence: How developmental cognitive neuroscience findings may shape understanding and interventions for psychopathology

Simone P.W. Haller[a,*], Kathrin Cohen Kadosh[a], Gaia Scerif[a], Jennifer Y.F. Lau[a,b]

[a] Department of Experimental Psychology, University of Oxford, Oxford, UK
[b] Department of Psychology, Institute of Psychiatry, King's College London, London, UK

ARTICLE INFO

Article history:
Received 18 December 2013
Received in revised form 6 February 2015
Accepted 6 February 2015
Available online 28 February 2015

Keywords:
Social anxiety
SAD
Cognitive bias
Neurocognitive development
fMRI
Adolescence

ABSTRACT

Social anxiety disorder represents a debilitating condition that has large adverse effects on the quality of social connections, educational achievement and wellbeing. Age-of-onset data suggests that early adolescence is a developmentally sensitive juncture for the onset of social anxiety. In this review, we highlight the potential of using a developmental cognitive neuroscience approach to understand (i) why there are normative increases in social worries in adolescence and (ii) how adolescence-associated changes may 'bring out' neuro-cognitive risk factors for social anxiety in a subset of individuals during this developmental period. We also speculate on how changes that occur in learning and plasticity may allow for optimal acquisition of more adaptive neurocognitive strategies through external interventions. Hence, for the minority of individuals who require external interventions to target their social fears, this enhanced flexibility could result in more powerful and longer-lasting therapeutic effects. We will review two novel interventions that target information-processing biases and their neural substrates via cognitive training and visual feedback of neural activity measured through functional magnetic resonance imaging.

Contents

1. Introduction

Adolescence is a transitional period beginning with the onset of puberty and culminating in the assumption of a stable adult role (Lerner and Steinberg, 2004). There is considerable individual variability in both its onset and length. While the onset of adolescence is likely to be dependent on the release of pubertal hormones which set in motion a cascade of physical developments (e.g., dimorphic secondary sexual characteristics, neural changes), the offset is more contingent on sociocultural norms (Blakemore and Mills, 2014; Forbes and Dahl, 2010; Goddings et al., 2014; Herting et al., 2014; Peper and Dahl, 2013; Sisk and Foster, 2004). Adolescence is characterized by changes in psychological make-up too, particularly in social-affective (the experience and regulation of emotion in response to social cues) and social-cognitive (the reasoning about the social world and others' mental states) abilities. These changes are observed in and driven by maturing neural circuits (Nelson et al.,

* Corresponding author at: Department of Experimental Psychology, University of Oxford, Tinbergen Building, 9 South Parks Road, OX1 3UD Oxford, UK. Tel.: ++44 (0) 1865-271382.
E-mail address: simone.haller@psy.ox.ac.uk (S.P.W. Haller).

http://dx.doi.org/10.1016/j.dcn.2015.02.002

2005; Haller et al., 2014) and are likely to be adaptive as adolescents begin to engage with increasingly complex peer networks (Steinberg and Silverberg, 1986).

Adolescence is also a period of vulnerability for the emergence of many psychiatric conditions. One of these is social anxiety disorder (SAD), a debilitating condition characterized by a paralyzing fear of negative evaluation from others (Clark and Wells, 1995; Rapee and Spence, 2004). Since peer interactions carry important learning experiences for adolescents, avoidance of social exchanges, often used as a way to cope with social anxiety, is likely even more impairing and disruptive during this time (Miers et al., 2014). Social anxiety may be viewed as an 'adolescent disorder'. Indeed, age-of-onset data show that around 75% of more extreme and persistent forms of social anxiety have their onset by mid-adolescence with a median age of onset of 13 years (Gregory et al., 2007; Kessler et al., 2005a,b; Wittchen et al., 1999). Although several neurobehavioral hypotheses have been proposed to account for the general vulnerability to psychiatric conditions in adolescence (e.g., Sturman and Moghaddam, 2011), there is less understanding of how typical age-associated changes in adolescence may serve as vehicles for the expression of social anxiety risks in particular. In this article, we consider *whether* and *why* SAD risk factors emerge at the adolescent juncture, with a focus on the underlying neuro-developmental mechanisms that enable risk factors to find expression. We will first review changes in the functional architecture of social-affective and -cognitive brain circuits in adolescence. Next, after reviewing the cognitive characteristics and neural correlates of adolescent SAD, we will suggest how adolescent changes may 'bring out' aspects of SAD-risk.

More recently, adolescence has also been suggested as a period of heightened learning and flexibility (Crone and Dahl, 2012). This raises the question of whether adolescence may be an optimal period for targeting risks associated with SAD through translational interventions. A second set of goals of this paper is to (a) highlight how typical neuro-developmental changes can allow flexible and adaptive long-term learning about social-emotional events and, (b) highlight how developmental cognitive neuroscience research can inform the timing of psychological treatments for SAD. We ask whether there is greater social-affective plasticity in adolescence and how this might enable age-appropriate interventions to bring stronger, longer-term benefits.

2. How does the importance of peers change in adolescence?

A pronounced change in adolescence is the preoccupation with peers and romantic interests. Compared to adults, adolescents are far more concerned about peer feedback and respond more negatively to peer exclusion (Coleman, 1974; Kloep, 1999; O'Brien and Bierman, 1988; Reijntjes et al., 2006; Westenberg et al., 2004). Adolescents more frequently experience self-consciousness and use social comparison as a method of self-evaluation compared to pre-adolescent children (Butler, 1998; Elkind, 1967, 1985; Elkind and Bowen, 1979; Harter, 2006; Pfeifer et al., 2009). There is also an increased interest in understanding and tracking the mental states of peers, manifesting, for example, in increased awareness of their peers' likes and dislikes (fashion, music, gadgets or language neologisms). Studies using behavioral paradigms show a growing understanding of another's mental state in terms of visual perspective-taking, feelings and motivations ('mentalizing'; Dumontheil et al., 2010; Vetter et al., 2013) and more differentiated pro-social behavior (Güroğlu et al., 2014; Burnett Heyes et al., submitted for publication; Van den Bos et al., 2012).

These broad but pervasive adolescence-associated social-affective and cognitive changes may be mediated by prolonged maturation of functional (and structural) brain networks (Nelson et al., 2005). Indeed, over the last decade, there has been a surge in investigations of age- and/or puberty-related typical functional brain maturation of networks underpinning social-affective and cognitive processing (e.g., Blakemore, 2008; 2012). The networks of regions involve limbic and temporal areas and several functional sub-divisions of the pre-frontal cortex (PFC). Data suggest that these regions likely work in concert, as interactive networks enabling flexible responding to social-emotional cues.

Studies investigating developmental changes in the neural responses to social-affective stimuli can be divided into those investigating *automatic* regulatory responses to basic social and non-social threats and rewards, and those that probe more *controlled* regulatory responses. In the first category, complex, region-specific linear and quadratic trajectories across adolescence have been reported in the neural sensitivity of subcortical and cortical regions to social but also non-social threats and rewards. Broadly, these data suggest a peak in the neural responses of subcortical affect- and reward-processing regions such as the amygdalae and striatum to simple threatening or rewarding stimuli (e.g., monetary rewards and static, emotional faces) (e.g., Chein et al., 2012; Ernst et al., 2005; Hare et al., 2008; Pfeifer et al., 2011; Passarotti et al., 2009; Somerville et al., 2011; Van Leijenhorst et al., 2010). *When* peak sensitivities occur is not clear: while some researchers have documented curvilinear trends with a peak in mid-adolescence (Hare et al., 2008; Somerville et al., 2011), others have reported linear declines throughout adolescence with peaks in late childhood (Gee et al., 2013). Developmental trajectories of frontal areas in response to social-affective stimuli are equally, if not more, complex. Using basic inhibition-based paradigms (such as go/nogo tasks), which tap automatic regulatory responses to emotional face displays, studies have reported increased activity in functional portions of the PFC during response inhibition in adolescents compared to older age groups. These often occur in the presence of overall poorer behavioral performance (Dreyfuss et al., 2014; Somerville et al., 2011). Turning to paradigms that measure participants' automatic regulatory responses to more complex socially provocative stimuli (e.g., tasks that simulate online social exclusion), these have found more extensive differences occurring in the insula, anterior cingulate cortex (ACC) and medial and lateral functional subdivisions of the PFC between children, adolescents and adults (e.g., Moor et al., 2010, 2012; Guyer et al., 2009; Lau et al., 2011a,b; Masten et al., 2009; Sebastian et al., 2011). However, inconsistencies in the directionality of these differences across studies make drawing interpretations about developmental change difficult. The second category of studies investigating more effortful, controlled regulatory processing is more limited, but nonetheless show similarly inconsistent linear and quadratic trends in prefrontal activation (McRae et al., 2012; Monk et al., 2003; Pitskel et al., 2011). The inconsistency of findings across studies may stem from the use of different tasks tapping subtly different processes. Alternatively, variations may also arise from differences in the salience and relevance of the task context (Braams et al., 2014; Crone and Dahl, 2012).

As well as studying differences in individual regions, many studies have also explored adolescent changes in functional connectivity – the co-activation between different areas, either during 'resting state' or during a task. Resting state functional connectivity studies suggest that functional integration within networks increases across adolescence (Fair et al., 2007). A 'switch' from positive to negative connectivity in the amygdalae-medial prefrontal cortex (mPFC) network has also been reported across 4–22 year olds during the viewing of fearful emotional faces (Gee et al., 2013). Specifically, for children aged (4–9 years) increased amygdala activation was associated with increased mPFC activity, while from early adolescence (10–13 years) to adulthood higher