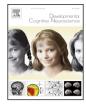
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# Neurocognitive bases of emotion regulation development in adolescence



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#### ABSTRACT

Emotion regulation is the ability to recruit processes to influence emotion generation. In recent years there has been mounting interest in how emotions are regulated at behavioural and neural levels, as well as in the relevance of emotional dysregulation to psychopathology. During adolescence, brain regions involved in affect generation and regulation, including the limbic system and prefrontal cortex, undergo protracted structural and functional development. Adolescence is also a time of increasing vulnerability to internalising and externalising psychopathologies associated with poor emotion regulation, including depression, anxiety and antisocial behaviour. It is therefore of particular interest to understand how emotion regulation develops over this time, and how this relates to ongoing brain development. However, to date relatively little research has addressed these questions directly. This review will discuss existing research in these areas in both typical adolescence and in adolescent psychopathology, and will highlight opportunities for future research. In particular, it is important to consider the social context in which adolescent emotion regulation, scaffolding the development of emotion regulation during this time may be a fruitful preventative target for psychopathology.

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#### 1. Introduction

Emotion regulation has been broadly defined as the monitoring, evaluation and modifying of emotional reactions in order to accomplish goals (Thompson, 1994). This can include both implicit emotion regulation, i.e. processes which occur automatically and largely outside conscious awareness and occur at very early stages of the emotion regulation process, and explicit emotion regulation, which involves using conscious strategies to modify emotional responses (Gyurak et al., 2011). Fully functional emotion regulation requires the ability to recognise the emotional significance of perceived stimuli, to appreciate the need for regulation, and then to select and implement an appropriate strategy (Sheppes et al., 2015). As such, it requires the co-ordination of multiple high-level processes including executive functions (Kesek et al.,

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2009) and in some cases social cognitive skills such as perspective taking.

Adolescence (approximately spanning the ages 10–19; Sawyer et al., 2012) is of considerable interest from an emotion regulation perspective for several reasons. Developmentally, this period is associated with significant biological and physical changes, a growing need for independence, academic and employment pressures and fluctuating social relationships (Casey et al., 2010). These challenges are often accompanied by increased emotional reactivity and stress. As will be discussed in more detail below, it has been hypothesised that ongoing brain development renders adolescents less able to successfully regulate their emotions, putting them at greater risk for anxiety and stress related disorders (Powers and Casey, 2015). Indeed the period of adolescence has been associated with an increasing incidence of internalising and externalising symptoms (Lee et al., 2014; Paus et al., 2008; Spear, 2000). This suggests that adolescents may be particularly vulnerable to emotional dysregulation, although it is worth noting that, as with adults, it can be difficult to distinguish whether these behaviours result from poor regulation, increased affective responses, or both.

Cognitively, high-level executive and social processes needed for emotion regulation, including working memory, inhibitory control, abstract thought, decision making and perspective taking, all undergo development during adolescence (e.g. Blakemore and Robbins, 2012; Dumontheil, 2014; Sebastian et al., 2010a; Somerville and Casey, 2010). Development of these cognitive processes appears to be underpinned by structural and functional development at the neural level, particularly in the protracted development of parts of prefrontal cortex and the remodelling of connections between prefrontal and limbic regions (see below). Simultaneously, adolescents are learning to negotiate increasingly complex social contexts (Sebastian et al., 2010a; Vartanian, 2000). It is possible that the interactions between these neurocognitive processes and social pressures could contribute to the observation that aspects of adolescent emotional processing and regulation development appear to follow a non-linear trajectory. In turn, this may at least partially explain increased emotional volatility and risk taking at this stage of life relative to both adulthood and earlier childhood (Casey and Caudle, 2013). Adolescence may therefore be a critical phase for the development of adaptive emotion regulation, with long-term consequences for future regulatory success and mental health. It has been suggested that adolescence is a period of heightened learning and flexibility (Casey et al., 2008; Steinberg, 2005). It could therefore be a critical phase for the development of adaptive emotion regulation strategies and in turn the implementation of interventions. Targeting this window of opportunity could have positive long-term consequences for mental health (Wekerle et al., 2007).

This review will present mounting behavioural and neural evidence on the development of implicit and explicit emotion regulation in adolescence, and will highlight potential research directions. We will first briefly discuss the structural development of brain regions involved in the detection, expression and regulation of emotion across adolescence (see Blakemore, 2012; Giedd, 2008; Giedd and Rapoport, 2010; Lenroot and Giedd, 2006; Paus, 2005 for more comprehensive reviews of adolescent structural brain development). We will then review human behavioural and neuroimaging data investigating the development of different aspects of emotion regulation, ranging from automatic implicit emotional control (e.g. the ability to filter out emotional information via attentional control mechanisms) through to explicit and effortful strategy use. This section of the review will broadly follow the Process model of emotion regulation (e.g. Gross, 1998), and the recent Extended Process model (Sheppes et al., 2015). fMRI evidence suggests that, while conceptually quite different, there may be continuities in the way these implicit and explicit processes are

instantiated at the neural level (e.g. Drabant et al., 2009). It is worth noting that due to the existence of other comprehensive reviews in the field (Blakemore and Robbins, 2012; Casey and Caudle, 2013; Steinberg, 2008) we will not cover risky decision-making, although emotion regulation abilities doubtless play a role here.

### 2. Adolescent brain development: relevance to emotion regulation

Adolescence is characterised by a period of heightened emotional reactivity, instability and risk-taking. Several studies using self-report questionnaires have found hypersensitivity to peer rejection and peer influence in adolescents relative to adults and children (Kloep, 1999; Larson and Richards, 1994; O'Brien and Bierman, 1988). Moreover, in a longitudinal study it was found that average emotional states over a week became more negative across early adolescence but this decline in emotions ceased by late adolescence (18 years of age) (Larson et al., 2002). Stability of daily emotional states also increased with age. Increased emotional reactivity has also been demonstrated in behavioural studies where experimental ostracism has been manipulated. In one study, overall mood was found to be significantly lower after ostracism in the adolescent group and state anxiety was higher in the young adolescent group (12-14 years of age) but there were no differences between conditions on either measure for adults (Sebastian et al., 2010a). Adolescents also place a higher emphasis on rewards, particularly social rewards, compared to adults which may make the perceived benefits outweigh the perceived risk (Reyna and Farley, 2006; Steinberg, 2008).

Over the past few decades, neuroimaging studies have begun to suggest that ongoing structural and functional brain development during adolescence may contribute to adolescent-specific behaviours. Evidence suggests that structural brain development in brain regions subserving emotion regulation continues into adulthood (Paus et al., 2008). For example, the prefrontal cortex (PFC), is central in the generation and maintenance of emotion regulation strategies (Ochsner and Gross, 2008; and see below). Subdivisions of the PFC most implicated in emotion processing and regulation include the dorsolateral (dIPFC), ventrolateral (vIPFC) and ventromedial regions (vmPFC) (Kalisch, 2009; Ochsner and Gross, 2008; see Box 1). Development of the PFC is particularly protracted, with reductions in cortical grey matter volume, density and thickness continuing into adolescence and even into the third decade of life (Gogtay et al., 2004; Shaw et al., 2008).

These reductions are thought to index a maturational process. One theory is that they correspond to synaptic pruning, i.e. the elimination of redundant synapses (Blakemore, 2008). Post-mortem studies have shown that synaptic density gradually increases during childhood, peaks in early adolescence, and then reduces by roughly 40% during adolescence and early adulthood before stabilising, following an inverted-U shaped pattern (Huttenlocher and de Courten, 1987). This synaptic pruning in adolescence fine-tunes the remaining connections into specialised functional networks, which might result in more efficient cognitive processing (Blakemore, 2008). However, others have suggested that a reduction in the number of synapses during adolescence is unlikely to have such a large effect on cortical volume as measured by MRI, as cortical grey matter contains numerous cellular elements including neural cell bodies, axons, dendrites, glial cells and blood vessels. Instead, grey matter decline may reflect an artefact of increased myelination of intra-cortical axons (Bourgeois and Rakic, 1993; Paus, 2005; Paus et al., 2008). Unfortunately, methodological limitations make it difficult to directly link developmental change in the living brain as measured with structural MRI with changes in the underlying cellular anatomy.

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