Developmental Cognitive Neuroscience 17 (2016) 57–67

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

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

Review

Dopaminergic reward sensitivity can promote adolescent health: A new perspective on the mechanism of ventral striatum activation

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ARTICLE INFO

Article history:
Received 20 June 2015
Received in revised form
26 September 2015
Accepted 26 October 2015
Available online 24 November 2015

Keywords:
Adolescence
Rewards
Risk taking
Health
Brain development

ABSTRACT

The prevailing view in the field of adolescent brain development is that heightened activity in the mesolimbic dopaminergic reward system serves as a liability, orienting adolescents toward risky behaviors, increasing their sensitivity to social evaluation and loss, and resulting in compromised well-being. Several findings inconsistent with this deficit view challenge the perspective that adolescent reward sensitivity largely serves as a liability and highlights the potential adaptive function that heightened striatal reactivity can serve. The goal of this review is to refine our understanding of dopaminergic reward sensitivity in adolescence. I review several studies showing that ventral striatum activation serves an adaptive function for adolescents' health and well being relating to declines in both risk taking and depression and increases in cognitive persistence and achievement.

Contents

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http://dx.doi.org/10.1016/j.dcn.2015.10.010

Adolescence is a sensitive and vulnerable developmental period marked by steep increases in risk-taking behavior, emotional lability, and poor behavioral regulation (Steinberg, 2005). Such changes relate to increased rates of depression and anxiety (i.e., internalizing problems) and conduct disorder and rule breaking behaviors (i.e., externalizing problems) that incur significant public health concern, driven by their high prevalence, chronicity, and adverse effects on functioning. For example, the onset of many psychiatric disorders emerges during adolescence, with depression rising 500% from childhood to adolescence and an additional 400% by the young adult years (see Thapar et al., 2012). Morbidity and mortality rates increase 300% from childhood to adolescence (CDC, 2014) with over 70% of adolescent deaths each year due to preventable causes including motor vehicle crashes, unintentional injuries, homicide, and suicide (CDC, 2013). Recent evidence from animal models and developmental neuroscience studies in human youth has shown that disruptions in reward processing may underlie increases in internalizing and externalizing symptoms during adolescence (Spear, 2011).

1. Adolescent peaks in reward sensitivity

Across many species, including rodents, nonhuman primates, and humans, adolescents show peaks in reward-related behaviors, providing strong evidence for the conservation of reward processing across evolution (Spear, 2011). Adolescent rats are more sensitive than their adult counterparts to the rewarding properties of a variety of positively rewarding stimuli, including novelty-seeking (Douglas et al., 2003), the rewarding effects of social interactions (Douglas et al., 2004), consummatory behavior (Friemel et al., 2010; Spear, 2011), and palatable testants (Vaidya et al., 2004; Wilmouth and Spear, 2009; Friemel et al., 2010). In human primates, inverted U-shaped developmental patterns have been observed in reward seeking behaviors. For example, human adolescents show peaks in self-reported reward-seeking and sensation seeking (Steinberg et al., 2009; Romer et al., 2010), greater sensitivity to positive feedback during a behavioral gambling task (Cauffman et al., 2010), and heightened preferences and reactivity to sweet substances (Galván and McGlennen, 2013; Post and Kemper, 1993). These behavioral shifts in reward seeking behaviors and preferences are driven, in part, by underlying neural changes in frontostriatal circuitry.

2. Dopaminergic changes in the adolescent brain

Frontostriatal circuits subserving reward processes are modulated by the neurotransmitter dopamine (DA) The primary reward circuit includes dopaminergic projections from the ventral tegmental area (VTA) to the nucleus accumbens, which release dopamine in response to reward-related stimuli (Russo and Nestler, 2013). The ventral striatum, and the nucleus accumbens in particular, has been recognized as a core node for incentive, reward driven behaviors (see Padmanabhan and Luna, 2014; Galván, 2014). DA signaling supports reinforcement learning, and DA modulation of striatal and prefrontal function influences affective and motivated behaviors that are altered in adolescence (Padmanabhan and Luna, 2014). Major components of the reward system that undergo particularly dramatic change during adolescence include projections from DA neurons deep in the base of the brain (e.g., VTA; substantia nigra) to subcortical regions including the striatum, as well as the prefrontal cortex (PFC) and other cortical regions including the amygdala and hippocampus. There are also GABAergic projections from the nucleus accumbens to the VTA. These include projections through the direct pathway, which is mediated by D1-type medium spiny neurons, which directly innervate the VTA, and projections through the indirect pathway, which is mediated by D2-type medium spiny neurons, which innervate the VTA through GABAergic neurons in the ventral pallidum (Russo and Nestler, 2013). All of these reward-regions are interconnected in complex ways (see Fig. 1).

The DA system undergoes significant reorganization over adolescence, which is implicated in the pathophysiology of various disorders that appear during adolescence (see Nelson et al., 2005; Spear, 2000; Wahlstrom et al., 2010a). Across rodents, non-human primates, and humans, increases in dopamine signaling peak during adolescence (see Wahlstrom et al., 2010b). Adolescent-specific peaks have been observed in the density of dopamine receptors D1 and D2 in the ventral striatum in rodents (Andersen et al., 1997; Tarazi et al., 1999; Teicher et al., 1995; Badanich et al., 2006; Philpot et al., 2009), although a few studies have not found functional differences in the nucleus accumbens of adolescent rodents compared to adults (Matthews et al., 2013; Sturman and Moghaddam, 2012). Moreover, DA concentrations and the density of DA fibers projecting to the PFC increase into adolescence (Benes et al., 2000), as well as the number of PFC projections to the nucleus accumbens (Brenhouse et al., 2008). In non-human primates, region-wide DA innervation peaks in adolescence (see Wahlstrom et al., 2010b).

Studies with humans have reported similar peaks in DA expression during adolescence. For example, in human post-mortem samples, DA levels in the striatum increase until adolescence and then decrease or remain stable (Haycock et al., 2003), both in terms of length of axons as well as total number of projecting axons (Lambe et al., 2000; Rosenberg and Lewis, 1994). There is also a peak in glutamatergic connectivity from the PFC to the nucleus accumebens, specifically in D1-expressing neurons (Brenhouse et al., 2008). Finally, fMRI studies, which allow for assessment of changes in neural systems innervated by DA, have shown that the ventral striatum is significantly more active among adolescents than children or adults when receiving secondary rewards (e.g., money; Ernst et al., 2005; Galván et al., 2006; Van Leijenhorst et al., 2010), primary rewards (e.g., sweet liquid; Galván and McGlennen, 2013), or social rewards (Chein et al., 2010; Guyer et al., 2009) as well as in the presence of appetitive social cues (Somerville et al., 2011). Such peaks in ventral striatum activation are associated with compromised cognitive control (Somerville et al., 2011) and increased self-reported risk taking (Galván et al., 2007). Some studies have also found that adolescents show blunted ventral striatum activation relative to children or adults when anticipating rewards (Bjork et al., 2004, 2010), and such blunted striatal activation is associated with greater risk taking behaviors (Schneider et al., 2012). Hypoactivation of the ventral striatum is argued to suggest that adolescents may attain less positive feelings from rewarding stimuli, which drives them to seek out greater reward-inducing experiences that increase activity in dopamine-related circuitry (Spear, 2000).

It is hypothesized that the DA system is at a functional ceiling during adolescence (Chambers et al., 2014), as evidenced by peaks in DA cell firing, overall higher tonic DA levels, greater DA innervation, and increased DA receptor densities (see Padmanabhan and Luna, 2014). Therefore, the mesolimbic DA system is thought to be in a state of overdrive during adolescence, which has