



Review

Beyond stereotypes of adolescent risk taking: Placing the adolescent brain in developmental context[☆]



Daniel Romer^{a,*}, Valerie F. Reyna^b, Theodore D. Satterthwaite^c

^a Annenberg Public Policy Center, University of Pennsylvania, United States

^b Human Neuroscience Institute, Cornell University, United States

^c Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, United States

ARTICLE INFO

Keywords:

Brain development
Dopamine
Decision-making
Cognitive control
Experience

ABSTRACT

Recent neuroscience models of adolescent brain development attribute the morbidity and mortality of this period to structural and functional imbalances between more fully developed limbic regions that subserve reward and emotion as opposed to those that enable cognitive control. We challenge this interpretation of adolescent development by distinguishing risk-taking that peaks during adolescence (sensation seeking and impulsive action) from risk taking that declines monotonically from childhood to adulthood (impulsive choice and other decisions under known risk). Sensation seeking is primarily motivated by exploration of the environment under ambiguous risk contexts, while impulsive action, which is likely to be maladaptive, is more characteristic of a subset of youth with weak control over limbic motivation. Risk taking that declines monotonically from childhood to adulthood occurs primarily under conditions of known risks and reflects increases in executive function as well as aversion to risk based on increases in gist-based reasoning. We propose an alternative Life-span Wisdom Model that highlights the importance of experience gained through exploration during adolescence. We propose, therefore, that brain models that recognize the adaptive roles that cognition and experience play during adolescence provide a more complete and helpful picture of this period of development.

1. Introduction

Recent theorizing and research regarding the neurodevelopment of the adolescent brain has generated considerable attention in both the popular media and the scientific literature. The most striking generalization stemming from this work is that the adolescent brain does not fully mature until at least age 25, with the implication that adolescent decision-making and judgment is similarly limited up to this age (Casey et al., 2008; Giedd, 2004; Steinberg, 2008). This conclusion rests on research indicating that the myelination and pruning of the prefrontal cortex (PFC) continues into adulthood, well after ventral limbic regions that control motivation and reward have achieved these milestones. As a result, it is proposed that adolescents suffer from a structural as well as functional deficit in the ability of the PFC to exert top-down control over drives that are spurred by the limbic motivational system, leading to less than “rational” behavior during adolescence. The basic dynamics

of these neurobiological imbalance models are illustrated in Fig. 1 (Casey et al., 2008), showing that limbic structures are activated in excess of prefrontal cognitive control regions during the adolescent period.

A key feature of such imbalance models is the suggestion that a developmental deficit in PFC cognitive control limits adaptive decision making by adolescents.¹ However, when Giedd et al. (1999) first presented evidence of declining PFC gray matter volume in adolescents, they attributed the phenomenon to the role that experience plays in sculpting the brain during this developmental period. As they put it, the decline in PFC gray matter “may herald a critical stage of development when the environment or activities of the teenager may guide selective elimination during adolescence.” (p. 863). In other words, gray matter decline in the PFC could reflect pruning that results from the experience that adolescents gain during this period rather than a direct marker of increasing behavioral control. As Spear (2010) also noted, pruning may

[☆] DR was supported by National Institute on Drug Abuse (R01DA033996); VFR was supported by National Cancer Institute (R21CA149796); National Institute on Nursing Research (R01NR014368-01) and National Institute of Food and Agriculture (NYC-321423 and NYC-321436); TDS was supported by National Institute on Mental Health (R01MH107703 and K23MH098130). We thank James Bjork, Joseph Kable, Kathryn Mills, and Flaura Winston for helpful comments on earlier versions of this paper. However, the conclusions reached in this paper do not necessarily reflect the views of the funding agencies or prior readers.

* Corresponding author.

E-mail address: dan.romer@appc.upenn.edu (D. Romer).

¹ In a recent review of imbalance research, Casey (2015) prefers not to describe imbalance as a “deficit” but rather a “brain that is sculpted by evolutionarily based biological constraints...” Nevertheless, these constraints are seen as contributing to a “200% increase in preventable deaths (accidents, suicide, homicide)...” during adolescence (p. 296–297).

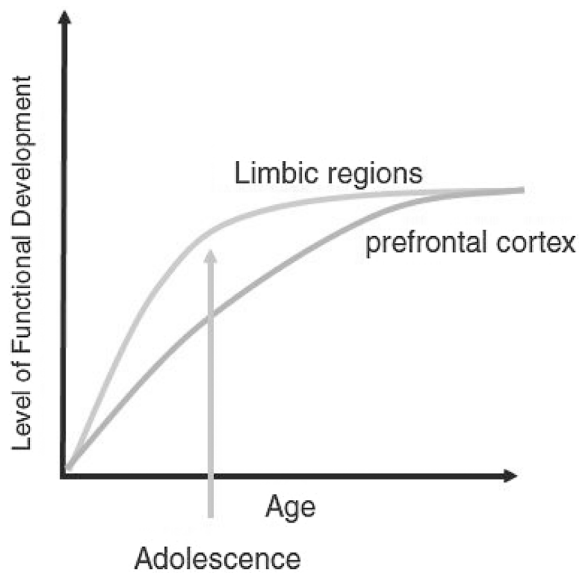


Fig. 1. Casey et al. (2008) model of imbalance between prefrontal versus limbic control over behavior in adolescence.

With permission from Institute of Medicine (2011, p. 38).

be “an example of developmental plasticity whereby the brain is ontogenetically sculpted on the basis of experience to accommodate environmental needs.” Needs could vary dramatically across environments and cultures (Mata et al., 2016), potentially resulting in very different patterns of pruning and brain organization during adolescent brain development (Choudhury, 2010). For example, evidence has accumulated to suggest that differences in socioeconomic status, which are correlated with cultural influences, are associated with differences in brain structure (Brito and Noble, 2014; Noble et al., 2015). In particular, Noble et al. (2015) demonstrated that lower socioeconomic status was associated with diminished cortical surface area and reduced hippocampal volume even when controlling for maternal education. Such hippocampal volume reductions have been reported by other studies as well (Hanson et al., 2011; Hueston et al., 2017). Others have observed differences in language-related regions (Piccolo et al., 2016) and modular brain organization (Krishnadas et al., 2013). Future research should unpack influences of education, culture, and income (with concomitant effects on nutrition, access to healthcare, and other factors that may plausibly affect development) on specific aspects of brain development.

Rather than emphasizing the important role of culture and experience in shaping the development of the brain, researchers have instead focused on excess levels of maladaptive risk behavior, such as injury, drug use, pregnancy, and other unhealthy outcomes, as support for imbalance (Dahl, 2004; Steinberg, 2008; Casey, 2015). However, the stereotype of the impulsive, emotional, and distraught adolescent rests much more on the rise in adverse outcomes during this age period than on their overall prevalence (Institute of Medicine, 2011; Rivers et al., 2008). For the vast majority of adolescents, this period of development passes without substance dependence, sexually transmitted infection, pregnancy, homicide, depression, suicide, or death due to car crashes (Institute of Medicine, 2011; Willoughby et al., 2013). Indeed, the risks of these outcomes are often comorbid with each other (Biglan and Cody, 2003; Kreuger et al., 2002), leaving the average adolescent without great risk of life-altering consequences.

We do not question the reality that the adolescent period entails risk. What we challenge is the interpretation of the brain and behavioral underpinnings of this risk. Research suggests that the brain is structured to enhance development by encouraging movement toward independence and self-sufficiency, a process that supports exploration and learning (Luna and Wright, 2015; Murty et al., 2016; Spear, 2013).

Support for this view has been observed in both humans and other animals following the onset of puberty. Nevertheless, a focus on adverse outcomes leaves us with a biased picture that limits our ability to identify adaptive features of adolescent brain development within the context of the entire lifespan. Instead, we argue for a more nuanced interpretation of risk taking and its implications for healthy development. In particular, we outline the evidence regarding the role of sensation seeking, which although it peaks during adolescence does not reflect imbalance, as opposed to forms of impulsivity which either do not peak or only characterize a subset of youth. Our review of research regarding structural development indicates that the relation between brain structure and risk taking has failed to consider the implications of different forms of risk taking. Our analysis suggests that stereotypes of adolescents as particularly susceptible to unhealthy risk taking simplifies how adolescents think about risk and ignores the important role that experience plays in more adaptive forms of risk taking (Reyna et al., 2015a; Romer, 2010). In what follows, we consider what a broader perspective on adolescent brain development would suggest, how that helps to explain the way adolescents make decisions, and how these decisions can be improved.

1.1. The rise in sensation seeking

Consistent with stereotypes of young people, adolescents exhibit heightened attraction to novel and exciting experiences despite their evident risk (Chambers et al., 2003; Romer and Hennessy, 2007; Spear, 2010). This tendency, known as sensation seeking (Zuckerman, 2007), rises rapidly during adolescence. As seen in Fig. 2, a nationally representative U.S. survey of 1800 youth indicates that sensation seeking peaks around age 19 in males and 16 in females. A similar pattern has been observed across a wide range of countries (Duell et al., 2016). This rather striking pattern is regarded as a marker of rising dopaminergic activation during adolescence (Chambers et al., 2003; Wahlstrom et al., 2010) and may reflect activity in the midbrain dopamine pathway ascending from the ventral tegmental region (Ikemoto, 2007; Previc, 2009). This pathway traverses through the ventral striatum before branching into the orbital and ventromedial frontal cortex. These regions are heavily involved in recognition and anticipation of reward (Pagnoni et al., 2002; Schultz et al., 1997) and thus suggest a biological basis for increased attraction to novel and exciting experience during adolescence that declines as the brain transitions to adulthood (see Wahlstrom et al., 2010 for a review of evidence linking a peak in exploratory behavior during adolescence with changes in dopamine expression over the lifespan). A related personality cluster known as the behavioral activation system (BAS) is also believed to be related to

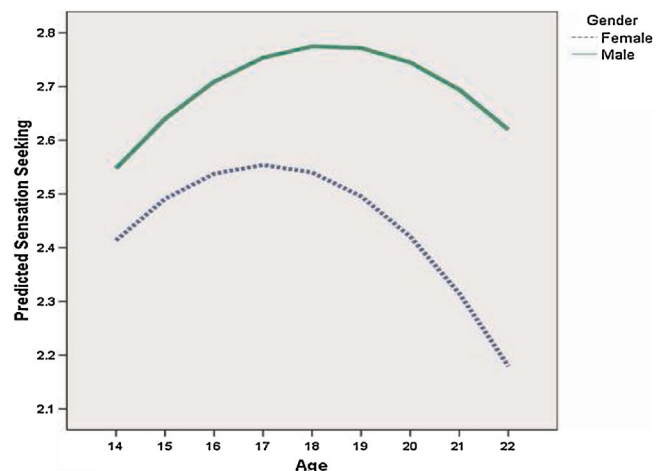


Fig. 2. Trends in sensation seeking by gender in a national U. S. sample. With permission from Romer (2010).

Download English Version:

<https://daneshyari.com/en/article/5735820>

Download Persian Version:

<https://daneshyari.com/article/5735820>

[Daneshyari.com](https://daneshyari.com)