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Emerging Adulthood Brain Development

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[-] Abstract and Keywords

Emerging adulthood (EA) is marked by a prolonged developmental transition to adulthood, dynamic personal and environmental circumstances, and unique patterns of vulnerability to psychological dysfunction. Neurodevelopment in childhood and adolescence has been studied extensively, but EA has not yet received its due attention from developmental cognitive neuroscience. The existing evidence shows that neurodevelopment continues throughout EA in support of emerging adult roles. The data suggest a frontolimbic fine-tuning model of brain development in EA that holds that adult functions are promoted through the strengthening of prefrontal regulation of limbic function and a newly emerging balance between prefrontal subregions involved in modulating approach and avoidance. Considering the overlap between these neurodevelopmental processes and the peak incidence of numerous psychological disorders in EA, it seems that individual differences in the dynamics of emerging adulthood neurodevelopment may not only underlie differences in functioning, but also risk for psychological disorder.

Keywords: emerging adulthood, brain development, neurodevelopment, developmental cognitive neuroscience

Introduction

As individuals in industrialized societies move past adolescence and into their third decade of life, they—and their brains—are consolidating new adult functions and roles. In the emerging-adult brain are the neural systems that support identity and role exploration as the individual navigates new feelings of instability and freedom in life, grapples with feeling in-between adolescence and adulthood, and transitions from the perception of future life possibilities to managing their emergence (Arnett, 1998). Brain structure and function during this period are the biological substrates that influence continually unfolding developmental trajectories that may or may not set a course toward well-being in adulthood. Although most developmental neuroscience work has focused on early childhood through adolescence, here, the focus is shifted to neurodevelopment in emerging adulthood (EA).

The life stage of EA is a developmental period marked by a “prolonged transition to adulthood” that has arisen due to social and cultural pressures in industrialized and postindustrialized societies, such as more years spent on education, delays in marriage, gender equality, and greater social freedom (Arnett, 1998). In this environment, the transition to adulthood is more gradual, with a sense of becoming fully adult delayed until the late 20s (Arnett, 2005). Thus, brain development during EA supports increasing independence and responsibility in the context of still present parental and societal support.

The evidence demonstrates that brain development does not stop in adolescence; changes in brain structure and function are ongoing in EA (Benes, 1989; Giedd et al., 1999; Yakovlev & Lecours, 1967). Specifically, neurodevelopment in EA involves prominent changes in association cortices and the frontolimbic systems involved

in executive, attention, reward, and social processes. In addition, alterations in neurodevelopmental trajectories in EA may underlie differences in functioning and new vulnerabilities to psychopathology evident in this developmental window.

This chapter synthesizes extant findings related to EA neurodevelopment and outlines a frontolimbic fine-tuning model of EA neurodevelopment. Specifically, the model argues that the transition from adolescence to EA involves both a shift toward greater balance between bottom-up and top-down functioning as executive and self-regulatory mechanisms of the prefrontal cortex (PFC) develop to interact with reactive processes (Casey, Jones, & Hare, 2008*b*; Steinberg, 2008), as well as the fine-tuning of top-down PFC regulation that enhances sensitivity to negative consequences and promotes future-oriented decision making.

This chapter also describes how neural systems underlying the transition to adulthood may create a “permissive environment” (Benes, 1988, 2000) that makes individuals vulnerable to the emergence of psychiatric disorder in EA. Although life satisfaction tends to increase as individuals navigate EA (Schulenberg & Zarrett, 2006), and discussions of developmental vulnerability to psychopathology typically focus on childhood and adolescence (Paus, Keshavan, & Giedd, 2008), there are significant increases in psychopathology in EA (Kessler & Wang, 2008).

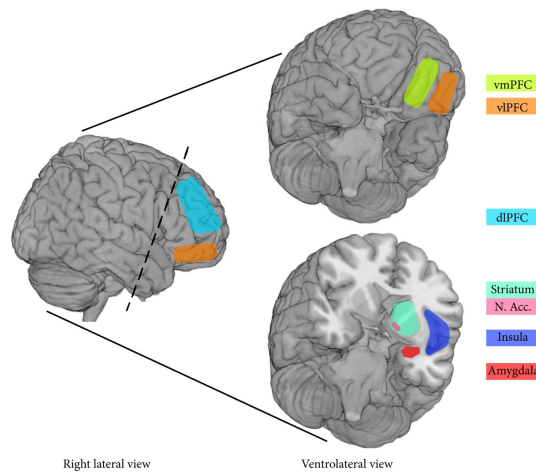
EA is associated with great instability across environmental and psychological domains, which may lead to affective disorder and substance use (Arnett, 2005). Median ages of onset for some anxiety (phobia, separation) and impulse-control disorders are around puberty, but EA is also a highly vulnerable period (Kessler et al., 2005): median ages of onset for anxiety (panic, generalized, and post-traumatic stress; 19–31 years), substance use (20 years), and mood disorders (depression, bipolar; 30 years) all fall during EA in the United States (Kessler & Wang, 2008). The peak in the percentage of individuals using alcohol is ages 21–25 (25%), and peak illicit drug use is in ages 18–20 (24%), followed closely by 21–25 (20%) (Substance Abuse and Mental Health Services Administration, 2012). Mental disorders diagnosed in childhood and adolescence are typically primary or first-onset disorders, whereas those diagnosed in EA are more often comorbidities (disorders occurring in addition a prior diagnosis; Kessler et al., 2005), which may have a heavier toll on mental health (Kessler & Wang, 2008).

Current developmental science focuses on the seeds of typical and atypical development sown during or prior to the flux of adolescence (Paus et al., 2008; Sowell et al., 2003). Neuropsychiatrically, EA may be no less of a dynamic sensitive period (Masten, 2004): (a) EA (like adolescence) is indeed a period of substantial psychological (and as described later, neural) development, and (b) onsets of psychiatric disorders—especially devastating comorbidities—reach their peak during EA. As is the case with adolescence, understanding EA brain development can lead to insight into the mechanisms underlying typical development and psychiatric vulnerability.

The following sections outline the basic principles of brain organization and development, followed by evidence from postmortem and structural neuroimaging studies of brain structure development in EA. Next, we review research on the developmental changes in brain function during EA, with a focus on recent studies utilizing functional neuroimaging methods. Finally, the roles of plasticity and culture in EA brain development are discussed.

Development of Brain Structure in EA

Principles of Brain Organization and Development



Right lateral view
Click to view larger

Ventrolateral view

Figure 1. Brain regions developing in emerging adulthood. Regions undergoing extensive neurodevelopment in EA are primarily in frontolimbic and central executive systems, including limbic structures (striatum, nucleus accumbens, insula, and amygdala) and ventral prefrontal cortex (ventrolateral and ventromedial) and in the dorsolateral prefrontal cortex.

The brain exhibits specialization and integration of functions that allow for the complexity of human behavior (see Figure 1). The brain is oriented with input (sensory) functions in the back (occipital, parietal, and temporal lobes) and output (motor) functions in the front (frontal lobe). Brain functions are organized hierarchically, with more complex, multimodal, associative functions built on more basic sensory functions (Fuster, 2001). This hierarchical pattern is also observed in the temporal unfolding of neurodevelopment—sensory systems tend to develop earlier, and more complex association systems (which integrate and associate information from multiple sensory modalities) develop later (Gogtay et al., 2004). Gogtay and colleagues produced time lapse movies of cortical maturation from age 4 to 21 based on longitudinally acquired brain scans, showing that maturation begins in primary somatosensory areas of the dorsal (top) parietal lobe spreading forward and down across the cortex, with the dorsal PFC maturing last (2004). Importantly, as described in detail later, the brain systems undergoing protracted maturation and development into EA support the psychological demands of this period as individuals work to increase future-oriented behavior and decrease risky decision making.

Postmortem and Comparative Studies

Early studies of brain development relied on postmortem dissection of brain tissue and comparative neuroanatomical studies of nonhuman versus human brains. These studies revealed that brain structure is changing in EA through myelination (Benes, 1989), pruning of synapses (Huttenlocher & Dabholkar, 1997), and the formation of new connections (Benes, Vincent, Molloy, & Khan, 1996).

Initial postmortem studies provided the first evidence that brain maturation continues well into the fourth decade of life, particularly with respect to increases in white matter volume (Yakovlev & Lecours, 1967). The brain is composed primarily of gray matter (neuronal cell bodies) and white matter (myelinated neuronal axons and glial cells); the integrity of white matter fibers is crucial for the transmission of information between neurons and for maintaining the processing speed, timing, efficiency, and synchrony that generate functional brain networks (Fields & Stevens-Graham, 2002).

Importantly, maturation does not occur at the same rate and time across the brain. Fibers in sensory and motor regions, as well as long integrative fibers, mature early in life. In contrast, shorter integrative fibers—intercortical fibers within regions of association cortex—remain under development through the fourth decade of life (Yakovlev & Lecours, 1967). These association cortices are essential for higher cognitive functions involved in typical adult human behavior, such as integrating multiple streams of information in support of reasoning, planning, and decision-making processes.

Postmortem evidence also suggests ongoing EA neurodevelopment in the frontolimbic system, which connects the brain's affective (limbic) and executive (frontal) networks and is critical for integrating emotional information into cognitive processing (Benes, 1998) in support of higher cognitive functioning (Damasio, 1994). In early EA, there

are large increases in the myelination of corticolimbic fibers in this system (Benes, 1989, 1998; Benes, Turtle, Khan, & Farol, 1994).

Comparative data from the rat have also shown that frontolimbic connections continue to increase into adulthood (Cunningham, Bhattacharyya, & Benes, 2002). Specifically, there is a marked increase in the density of fibers from the amygdala to rat analogues of the human frontal cortex (anterior cingulate and ventromedial prefrontal cortices). The amygdala is involved in processing salient and emotional information (Adolphs, 2010; Davis & Whalen, 2001), providing signals to higher order regions of medial PFC to shape multimodal sensory processing (Bechara, Damasio, Damasio, & Lee, 1999; LeDoux, 1993) and exerting an affective influence on complex and social behavior (Adolphs, 2010; MacLean, 1985). Feedback is then sent to basic sensory cortices to facilitate processing of and responding to goal-relevant information (Sah, Faber, Lopez de Armentia, & Power, 2003). Functioning of this system is thus critical for adaptive behavior, and altered frontolimbic functioning is implicated in psychological disorders such as schizophrenia (Gur et al., 2007; Schneider et al., 1998), depression (Munafò, Brown, & Hariri, 2008), and anxiety (Davis, 1992; Hahn et al., 2011).

For example, temperamentally defensive cats exhibit greater amygdala response to threatening sensory information, illustrating the role of the frontolimbic system in processing highly salient information to generate fear and withdrawal behaviors (Adamec, 1991; LeDoux, 1998). The functioning of this system has also been implicated in temperamental differences in human developmental trajectories (Schwartz, Wright, Shin, Kagan, & Rauch, 2003). Young adults who were temperamentally fearful as infants and at risk for anxiety show heightened amygdala response to novel as compared to familiar faces (Schwartz et al., 2003), and differences in frontolimbic functioning may mediate the relations between childhood temperament and adult psychopathology (Hardee et al., 2013). Hardee and colleagues (2013) found that more negative functional connectivity between amygdala and insula is associated with higher levels of internalizing symptoms among young adults with (but not those without) a history of fearful temperament, suggesting the possibility that altered frontolimbic function may underscore the relation between early temperamental risk and later psychopathology.

In sum, evidence from postmortem and nonhuman animal studies shows that the neural systems supporting core psychological functions in the transitional period of EA are under ongoing development throughout this developmental window. The association cortices and frontolimbic system continue to mature during EA in support of executive and emotional processes important for the transition to adulthood, particularly social-emotional behavior. Increased white matter in the frontolimbic system may “[enable] an individual to modulate anxiety and fear and become more socially adept” (Cunningham et al., 2002), whereas individual differences in development of this system may be related to differences in social functioning and risk for psychological disorder (Giedd et al., 2008).

Human Magnetic Resonance Imaging

Postmortem studies provide high spatial resolution at the cellular level, but they lack the power of in vivo and longitudinal approaches. Recent advances in noninvasive techniques for observing brain structure, in particular magnetic resonance imaging (MRI), provide evidence that converges with and expands on early postmortem findings. MRI scans can capture millimeter resolution images of the living brain. The brain's structural anatomy can be revealed in exquisite detail by acquiring a single, several-minute-long MRI image (structural MRI), or the brain's functioning in time can be revealed by acquiring a time series of second-long images (functional MRI; fMRI; see Thompson et al., 2005). These MRI techniques have been particularly exciting for developmental researchers because participants' brains can be studied (i) across multiple time points, thus providing a longitudinal picture of brain development, and (ii) while engaging in specific tasks of interest, thus providing a “living picture” of brain functioning in context.

In EA, structural MRI has revealed general increases in white matter and decreases in gray matter across the brain (Bartzokis et al., 2001; Giedd et al., 1999; Sowell et al., 2003). Gray matter volume increases in preadolescence and decreases rapidly postadolescence to age 40, whereas white matter volume increases to age 43, followed by rapid decline (Giedd et al., 1999; Sowell et al., 2003; Sowell, Trauner, Gamst, & Jernigan, 2002).

Importantly, there is regional variability in these trends consistent with postmortem evidence, showing development in the association cortex and the frontolimbic system during EA. In one of the first studies, Elizabeth Sowell and

colleagues demonstrated how brain structure in EA is distinct from adolescence (Sowell, Thompson, Holmes, Jernigan, & Toga, 1999). Specifically, when comparing a group of emerging adults (23–30 years old) to a group of adolescents (12–16 years old), the emerging adults displayed reduced gray matter volume in frontolimbic regions of dorsal, medial, and orbitofrontal cortex, as well as in the striatum.

Additional cross-sectional studies have shown regional variability in observed nonlinear changes in gray and white matter volume from childhood through adulthood. First, gray matter density declines are most prominent and rapid through EA in dorsal frontal, ventromedial prefrontal, and dorsal parietal cortices (Sowell et al., 2003). This is consistent with the earlier group study (Sowell et al., 1999) and with postmortem findings showing that the process of myelination extends into adulthood in these regions of association cortex (Yakovlev & Lecours, 1967). Second, anterior cingulate, limbic, and primary sensory cortices showed more linear gray matter decline. These are basic sensory and affective regions where myelination is completed earlier, prior to the youngest participants in the Sowell study (Benes et al., 1994; Yakovlev & Lecours, 1967), and, structurally, these areas seem to be more stable during EA.

Individual differences in these typical neurodevelopmental patterns may underlie differences in psychological functioning and risk for psychological disorder in EA (Giedd et al., 2008). For example, in schizophrenia—which has peak onset in EA—there is evidence for a “lower and later peak” in gray matter density (Douaud et al., 2009). In particular, schizophrenia is associated with reduced volume in PFC (Kuperberg et al., 2003; Narr et al., 2005a; Narr et al., 2005b) and medial temporal lobe, critical regions of the frontolimbic system (Shenton, Dickey, Frumin, & McCarley, 2001; Wright et al., 2000). During EA, rapid, nonlinear gray matter decline in frontolimbic regions implicated in schizophrenia, anxiety, and depression may heighten the sensitivity of those regions to external pressures and make the individual vulnerable to disorder (Benes, 2000; Rapoport, Giedd, & Gogtay, 2012). As Benes pointed out, typical neurodevelopmental processes may “establish a ‘permissive’ environment” for the onset of psychological disorders (Benes, 2000, p. 262). When there is temporal overlap between peak onset of a psychological disorder and ongoing, active neurodevelopment in neural systems associated with that disorder, it is important to ask whether perturbations to the neurodevelopmental process may constitute a “trigger” for the onset of disorder.

Summary and Future Directions

In summary, postmortem, comparative, and neuroimaging studies have converged to show neurodevelopment in EA that is continuous with adolescence in some respects and divergent in others. The general trend of gray matter decrease and white matter increase is a continuation of processes that begin around the onset of adolescence. However, this process does not unfold simultaneously across the brain; as adolescence progresses and EA begins, the brain regions undergoing gray matter loss and white matter increase shift to higher order association, frontal executive, and frontolimbic cortices. These structural changes may support the reduction in risk taking and facilitate the increasingly well-regulated, future-oriented, and planful behavior seen in EA.

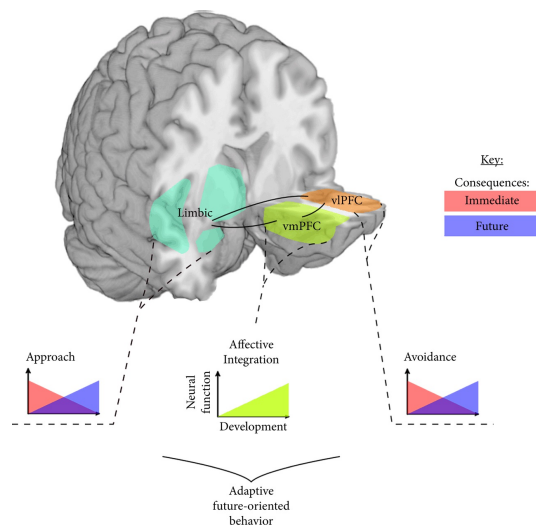
The study of structural brain development in EA is in its infancy, and several pressing issues need to be addressed. First, the neurobiological changes underlying changes in gross brain structure (gray and white matter density) in EA are not yet clear. For example, increases in white matter may be due to increased myelination, but could also be related to increases in axon diameter in the absence of changes in myelin (see Paus et al., 2008, for a critical discussion of the possible underlying neurobiological bases of post-childhood changes in brain structure). More nuanced methods will be needed to transition from gross measures of brain volume to distinguishing specific neurobiological processes and more subtle aspects of brain structure development. This is particularly important given developmental transitions from the sweeping structural and functional changes seen early in life to the relatively subtle shifts in brain–behavior relations (often dependent on context) that emerge with age. Also, along with advancing methods should come a greater focus on analyses of systems and networks, rather than discrete regions or volumes, in order to understand structural changes in a more functionally relevant way. For example, Wu and colleagues (2013) used diffusion tensor imaging (an MRI-based approach for imaging white matter tracts) to show that the coherence (“straightness”; Beaulieu, 2002) of superficial white matter in the frontolimbic system increases from ages 10 to 18, although the amount of myelin does not. Thus, superficial white matter fibers, which are among the slowest maturing white matter, may be developing in more subtle ways than simple volumetric measures can capture, thus illustrating advancing methodological and analytic techniques that will be needed in

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research on EA neurodevelopment.

Second, what are the driving forces that trigger EA neurodevelopmental processes, and how do they differ from the drivers of neurodevelopment in childhood and adolescence? For example, the onset of pubertal hormones is an important factor in early-adolescent increases in limbic function. However, the biological and environmental forces that elicit changes in association cortex and frontolimbic structure in EA are not clear. There is evidence that white matter development is influenced more by genetic factors in adolescence than adulthood (Chiang et al., 2011) and that later synapse formation is influenced more by environmental demands (Kleim, Lussnig, Schwarz, Comery, & Greenough, 1996), suggesting the possibility that environmental, social, and cultural factors may be central drivers of neurodevelopment in EA.

Development of Brain Function in EA



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Figure 2. Fine-tuning model of frontolimbic system development from adolescence through emerging adulthood in support of adaptive future-oriented behavior. Plots depict the fine-tuning of neural processing of approach and avoidance—specifically, the shifting balance from immediate or easy consequences (red) to future or more effortful consequences (blue). Approach: EA neurodevelopmental changes in limbic processing are in the context of the approach to immediate versus future rewards. Although amygdala and insula have previously been associated with processes of negative emotion, recent research suggests they have a more general role in saliency (Adolphs, 2010; Menon & Uddin, 2010). In the fine-tuning model, the primary development in limbic function is in enhancing the saliency of and emotional response to receiving future rewards (i.e., rewards received at a significant cost or that delay the behavior that generated them), relative to immediate ones. Avoidance: EA neurodevelopmental changes in vlPFC processing are in the context of the avoidance of immediate versus future punishments. The primary function of vlPFC development is in enhancing the saliency and emotional anticipation of receiving future punishments relative to immediate ones. Affective integration (green): vmPFC development in EA supports an increasing integration of affective information from approach/avoidance networks to generate future-oriented behavior. Solid black lines indicate network connections. Importantly, the functions depicted (approach, avoidance, affective integration) are not the exclusive purview of associated regions; rather, the regions are considered the central node in network development for their associated functions. At bottom, integrated frontolimbic network processing is feed-forward into the organization of adaptive future-oriented behavior.

Functional neuroimaging examines brain activity while individuals rest or perform experimental tasks, providing insight into brain functioning as it happens. This technique has allowed researchers to study the development of brain function in EA, revealing development of the frontolimbic brain system underlying reward processing, executive functions, and social behavior (see Figure 2). Specifically, from adolescence to adulthood, there is a shifting functional balance as the limbic system becomes increasingly regulated by PFC to facilitate future-oriented behavior. The neurodevelopmental literature previously considered EA as an endpoint, during which prefrontal systems have simply “come online.” However, recent data question the notion of EA as a period of stability following adolescent flux. Instead, the evidence reviewed here suggests that, in addition to the emerging frontolimbic balance, EA may be another period of instability (and thus vulnerability) in which plasticity facilitates fine-tuning of the frontolimbic system.

From Adolescent to Emerging Adult Models

Neurodevelopmental models broadly agree that adolescence is marked by the development of brain systems involved in social-emotional functioning (Burnett, Sebastian, Cohen Kadosh, & Blakemore, 2011). The “social brain” (Frith, 2007) is organized hierarchically, with more complex functions (executive, regulatory, planning, and social judgment) built on intermediate functions (social emotions and theory of mind) and more basic functions (reward, basic emotion, and face processing; Adolphs, 2003; Fuster, 2001; Taber-Thomas & Tranel, 2012). From adolescence to EA, there is a shift in the balance of limbic (affective, motivational) and frontal (executive, regulatory) systems, with limbic functioning developing earlier and frontal functioning coming online over the course of adolescence and into EA (Casey et al., 2008b; Ernst, Pine, & Hardin, 2006; Nelson, Leibenluft, McClure, & Pine, 2005; Steinberg, 2008).

According to the social information processing network model described earlier, limbic development is triggered by pubertal hormones and drives a “social reorientation”—or a dominance of basic social reward processing—in adolescence (Nelson et al., 2005; Spear, 2000). As the frontal system develops, it allows for the integration of social and reward information from multiple streams (Kringelbach & Rolls, 2004; Rolls, 2000) to more adaptively guide goal-directed behavior (Miller & Cohen, 2001; Watanabe, 1996), and early-life disruption to PFC functioning results in a stunting of social-emotional development (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Taber-Thomas et al., 2014).

From a dynamic systems perspective, neurodevelopment in puberty is a “phase transition” or period of flux (Granic & Patterson, 2006). During the flux of adolescence, reorganization is facilitated and behavior is less stable, perhaps making the individual more vulnerable to risk-taking behavior (Steinberg, 2008). As adolescence progresses, the system settles into a new, more stable functional arrangement (Granic & Patterson, 2006; Thelen & Smith, 2006; Van der Maas & Molenaar, 1992), in which a balance is struck between frontal and limbic system function. However, epidemiological and psychosocial evidence does not support the notion that EA is a stable endpoint of postadolescent functioning (Masten, 2004).

EA is, in fact, a period of vulnerability for psychological disorder (Kessler et al., 2005; Schulenberg & Zarrett, 2006) and is marked by significant changes in environment and personal development (Arnett, 2000) that may usher in a new period of developmental flux. Here, we review research that supports the proposition of a newly emerging frontolimbic balance in EA, as well as accompanying periods of flux in behavior and functioning. We argue that EA functional neurodevelopment is marked by both increased top-down prefrontal function, as well as by an emerging balance between ventromedial (approach) and ventrolateral (avoidance) prefrontal regulatory function.

Incentive Processing

At a foundational level, human behavior is motivated by the pursuit of incentives and avoidance of punishment (Fareri, Martin, & Delgado, 2008; McClure, York, & Montague, 2004). Thus, flexible, well-regulated incentive processing is essential to adaptive behavior. For example, seeking social rewards can be adaptive (friendship, love), but if uncontrolled can result in risky behavior (promiscuity or drug use due to peer influence). Consistent with the increasingly self-regulated behavior observed in EA, there are functional changes in the neural systems underlying incentive processing, including the PFC (executive, regulatory), limbic system (affective, motivational), and striatum (action-outcome associations; Balleine, Delgado, & Hikosaka, 2007; McClure et al., 2004; Rolls, 2000).

In an early fMRI study of incentive processing development, Bjork and colleagues (2004) scanned adolescents (12–17 years old) and emerging adults (22–28 years old) while they performed a monetary incentive delay task. Participants saw a cue that indicated whether quickly responding to an upcoming stimulus would yield monetary gain or avoid monetary loss. Task difficulty was rigged so participants could only respond quickly enough on roughly 66% of trials, making participants “earn” their reward. In both groups, preparing to respond to the gain cue activated the striatum and insula, with ventral striatum activation positively correlating with potential gain and also with age; ventral striatum and extended amygdala activation were greater for emerging adults than adolescents. This increased preparatory activation suggests that emerging adults may have a lower threshold for engaging incentive-processing circuitry to motivate behavior. In the context of emerging adult behavior in the real world, this lower threshold may allow for delayed or less intense rewards (stopping at the yellow light for safety) to compete with more immediate or salient rewards (showing off for your passengers by speeding ahead).

Interestingly, Bjork and colleagues found that adolescent and emerging-adult brain responses did not differ in response to actually receiving the earned rewards. Other studies have found that adolescents do show greater striatal response than emerging adults when rewards are “unearned” (i.e., minimal effort was needed to secure them); for example, when rewards are given for reporting which side of the screen a stimulus appeared on (Galvan et al., 2006), guessing which color will be randomly selected (Ernst et al., 2005), or playing a slot machine (Leijenhorst et al., 2010). Thus, it seems that earned incentive processing is relatively stable from adolescence to EA (Bjork et al., 2004), whereas unearned incentives become less enticing over that period (Ernst et al., 2005; Galvan et al., 2006). That is, rewards that are easy to acquire have become disincentivized for emerging adults, which may allow rewards that require more effort to win the day. This could be an important underpinning for the development of self-regulatory and future-oriented functioning in EA by reducing motivation for less effortful gratification (e.g., drugs) and shifting the incentive-processing balance toward gratification requiring more effort (e.g., education, work).

An increased motivation to engage in more effortful behavior may require increased top-down regulatory control of incentive processing. Indeed, frontolimbic connectivity increases from adolescence to EA (Bos, Cohen, Kahnt, & Crone, 2012). In response to the failure to win a reward, emerging adults exhibit greater ventrolateral PFC (vlPFC) activation than adolescents (Leijenhorst et al., 2010) and less amygdala activation (Ernst et al., 2005), suggesting that top-down inputs may dampen the salience of loss in EA. This is not to say that negative feedback is ignored in EA, but rather that emerging adults may respond more adaptively to negative feedback (Wilmouth & Spear, 2004).

One study found that the extent of vlPFC response to unearned rewards was more focal in emerging adults than adolescents, which was interpreted as more developed top-down regulation in EA. However, given (1) the involvement of vlPFC in processing negative feedback (Kringelbach & Rolls, 2004; O’Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001) and (2) increased vlPFC engagement in EA in response to negative feedback (Leijenhorst et al., 2010), less extensive vlPFC activation in EA in response to reward may indicate that the sensitivity of vlPFC has shifted from reward to negative feedback: that is, rather than the more “focal” vlPFC activity in EA indicating greater efficiency or development of vlPFC functioning in general, the vlPFC may actually be less responsive to positive feedback (but more response to negative feedback) in EA as compared to adolescence. The ventral PFC is involved in updating expectations of incentives in order to guide decision making (O’Doherty, 2007; Wallis, 2007), and a shift in ventral PFC function toward negative feedback may facilitate the adjustment of decision making following negative feedback in emerging adults (Leijenhorst et al., 2010).

In sum, it seems that from adolescence to adulthood, the sensitivity of incentive processing to unearned rewards decreases while the development of adaptive prefrontal regulation of limbic function increases (Casey, Getz, & Galvan, 2008a; Ernst et al., 2005; Fareri et al., 2008; Galvan et al., 2006; Leijenhorst et al., 2010). These changes in incentive-related neural processing may underlie the shift in goal-directed behavior toward outcomes requiring more effortful, self-regulated, and future-oriented processing.

Future-Oriented Decision Making

The focus in developmental cognitive neuroscience is typically on adolescent impulsivity and risk taking (Fareri et al., 2008); here, the attention is shifted to neural processes underlying increased future-oriented (less risky) decision making in EA. Risk taking, approach, and novelty seeking may be important for the development of independence, self-esteem, and social affiliation (Nelson et al., 2005; Spear, 2000). However, once the foundation of social development is laid, future-oriented behavior is crucial to facilitating the adoption of adult roles and responsibilities by minimizing behavior with negative consequences (e.g., injury, drug addiction, hangover; Reyna & Farley, 2006) in favor of long-term advantageous behavior.

Such adaptive decision making in the real world requires the ability to predict future consequences based on past experience, a process supported by the medial aspect of the ventral PFC (vmPFC), which integrates limbic and PFC information processing (Rolls, 2000). Patients with vmPFC injury demonstrate a myopia for the future that results in disruptions in real-world and social behavior (Dimitrov, Phipps, Zahn, & Grafman, 1999; Eslinger & Damasio, 1985), as in the well-known case of Phineas Gage (Harlow, 1868). Gage, a railroad foreman, was tamping explosives when his tamping rod struck a spark and was unfortunately fired upward, through his skull and vmPFC. Although he miraculously survived with his physical health and most cognitive functions intact (e.g., sensory processing, language, and memory), Gage’s personality changed dramatically—he went from being a well-liked and respected

leader to being rude, impatient, and unable to hold down a job. His physician Dr. John Harlow noted at the time that the “balance.... his intellectual faculties and animal propensities, seems to have been destroyed” (Harlow, 1868).

In the laboratory, the impairments associated with vmPFC injury have been captured by the Iowa Gambling Task (IGT), a neuropsychological test that involves learning which of four options are advantageous versus disadvantageous in the long-term based on the cumulative consequences of prior choices (Bechara, Damasio, Damasio, & Anderson, 1994). As typical adults play the IGT, they develop an anticipatory emotional arousal to disadvantageous options, so-called *somatic markers* that bias choice away from the disadvantageous options and drive an increasing preference for advantageous options (Bechara, Damasio, Tranel, & Damasio, 1997; Damasio, 1994). Injury to the vmPFC disrupts the generation of somatic markers, causing an impairment in the ability to use the emotional consequences of past experience to guide behavior and, accordingly, a failure to learn which options are advantageous on the IGT (Bechara et al., 1997; Bechara, Tranel, Damasio, & Damasio, 1996).

Developmentally, performance on the IGT improves with age from late childhood to EA (Overman, 2004). Emerging adults and adolescents do not differ at the start of the game, but as the game goes on, emerging adults make more advantageous choices than do adolescents (Cauffman et al., 2010). This neuropsychological evidence is consistent with a developmental increase in vmPFC functioning facilitating the generation of somatic markers that encode the affective predictions of future consequences. This, in turn, biases decision making toward long-term advantageous options. Indeed, compared to adolescents, emerging adults exhibit greater vmPFC and dorsal anterior cingulate (dACC) cortex activation when making risky monetary decisions, such as choosing between a high-risk/high-reward option (10% chance of winning \$4) and a low-risk/low-reward option (90% chance of winning \$0.50), and greater activation in these regions correlates with less risk taking (Bjork, Smith, Danube, & Hommer, 2007; Eshel, Nelson, Blair, Pine, & Ernst, 2007).

The dACC is involved in conflict monitoring and the detection of risky contexts (Botvinick, Cohen, & Carter, 2004; Bush, Luu, & Posner, 2000). In risky contexts, both adults and adolescents exhibit dACC activation when potentially negative consequences are large, but only adults activate dACC when potentially negative consequences are small (Bjork et al., 2007). One possibility is that emerging adults have a lower threshold for detecting risky circumstances, which may increase the likelihood of engaging prefrontal (dACC) regulatory circuitry to drive future-oriented choices. This would be consistent with evidence that adults intuitively detect and avoid risk without conscious deliberation (Reyna & Farley, 2006). Although adolescents are able to understand risks (Reyna & Farley, 2006), emerging adults have a greater ability to bring intuitive information about risk online in the moment of choice to guide their decision making (Casey et al., 2008a; Geier & Luna, 2009; Reyna & Farley, 2006).

It seems that developing PFC functioning in EA may both (1) modulate approach behavior and (2) engage avoidant behavior. These dual PFC functions (Kringelbach & Rolls, 2004) may generate future-oriented behavior to facilitate the emergence of adult roles. For example, when selections of advantageous and disadvantageous options on the IGT are examined separately (Cauffman et al., 2010), the selection of advantageous options peaks in adolescence and declines in EA. This is consistent with enhanced reward sensitivity and approach in adolescence, with PFC regulation of approach increasing in EA (Galvan et al., 2006). Sensation seeking and risky behavior show a decrease after the age of 16 (Zuckerman, Eysenck, & Eysenck, 1978), again suggesting that the approach system becomes more regulated from adolescence to EA to facilitate future-oriented behavior. In contrast, the avoidance of disadvantageous options increases from adolescence to EA (Cauffman et al., 2010), perhaps due to PFC-driven increases in avoidance in response to negative feedback.

Importantly, the demands of balancing these complementary regulatory PFC functions may underlie vulnerability to psychopathology in EA (Kessler et al., 2005). For example, substance abuse, which peaks in EA, is associated with disrupted PFC function (Feil et al., 2010) and impaired performance on the IGT (Bechara & Damasio, 2002; Bechara et al., 2001; Bechara et al., 2002). More specifically, substance-dependent individuals have difficulty learning to avoid options linked to negative feedback (Thompson et al., 2012) and exhibit diminished regulation of approach-related striatal functioning (Feil et al., 2010), illustrating the psychiatric impact of disrupted frontolimbic regulation of approach and avoidance.

In contrast, hyperavoidance of negative feedback is an underlying feature of anxiety (Borkovec, Alcaine, & Behar, 2004), and generalized anxiety disorder is associated with enhanced sensitivity to negative feedback on the IGT (Mueller, Nguyen, Ray, & Borkovec, 2010), suggesting that there may be anxiogenic effects of enhanced PFC

sensitivity to negative feedback in EA (Cauffman et al., 2010; Kim, Gee, Loucks, Davis, & Whalen, 2011). In sum, whereas development of the PFC is crucial for adaptive future-oriented behavior in EA, the fine-tuning frontolimbic regulation of approach and avoidance may underlie the vulnerability to psychological disorder sometimes seen in EA.

Summary and Future Directions

In sum, with age, prefrontal functioning is enhanced leading to increased future-oriented (and decreased risky) behavior (Fareri et al., 2008). As the neural processing of incentives and reward becomes better regulated and fine-tuned, so do the prefrontal neural processes underlying regulatory control and behavior selection. Prefrontal development seems to involve a shift in sensitivity from positive to negative feedback that drives the reduction of approach and increase in avoidance behavior. Although this process is central to development in EA, it may also explain why EA is a period of vulnerability for psychological dysfunction.

It is important to note that these conclusions are based on the synthesis of a relatively small literature base but will hopefully stimulate further study to both test these conclusions and answer a number of other critical questions. For example, the processes underlying developing prefrontal functioning in EA remain unclear. It has been argued that functioning becomes more robust, focal (less diffuse) (Fareri et al., 2008), and efficient (Luna & Sweeney, 2004) from adolescence to EA. In addition, the frontolimbic system may have a lower threshold for engagement in EA in the context of organizing behavior and receiving negative consequences.

Roles of Plasticity, Culture, Social Context in EA Brain Development

The research thus far reviewed on the development of brain structure and function in emerging adulthood focuses almost entirely on Western individuals across ages, without regard for culture. Like our colleagues in the behavioral sciences (Henrich, Heine, & Norenzayan, 2010), neuroscientists commonly publish results on Western industrialized societies and assume they apply cross-culturally (Chiao & Cheon, 2010). In the neuroimaging literature, 90% of studies are on Western samples (Chiao, 2009), and the cross-cultural neuroscience research that has been done (1) has almost exclusively compared Western to industrialized East Asian cultures and (2) has not taken a developmental approach (Chiao & Cheon, 2010). Thus, the extant literature has not directly addressed the role of culture in brain functioning and development in EA. Nonetheless, research on neuroplasticity and cultural neuroscience does provide some initial evidence to support the notion that EA may be marked by development and fine-tuning of PFC function that is informed by the specific cultural demands faced by individuals.

Plasticity

The brain is plastic and can be reorganized in response to environmental demands to facilitate context-appropriate learning and adaptive behavior (Maguire et al., 2000; Maguire, Frackowiak, & Frith, 1997). This is particularly true of the PFC and frontolimbic regions involved in social-emotional and adaptive functioning (Davidson & McEwen, 2012; Kleim & Jones, 2008). Given that in industrialized societies there is a protraction of the environmental pressures that drive the transition to adulthood (Arnett, 2000), neurodevelopmental processes may occur earlier and more rapidly in cultures where there is more pressure to transition to adulthood. The longer duration of education required in postindustrialized economies (Douglass, 2007) may mean that experiences that promote the development of the frontolimbic system in EA are delayed and that earlier frontolimbic development would be observed in preindustrialized cultures.

Culture

Early forays into cultural neuroscience have focused on how individuals view themselves in relation to others, in particular individualism (as independent individuals) versus collectivism (as one of many interdependent individuals). The pursuit of individualistic goals is an important aspect of emerging adulthood in the West (Arnett, Grusec, & Hasting, 2007). One study of US and Japanese participants found that individualists showed greater PFC activation in response to self-statements such as "I am humble" (Chiao et al., 2009), which is consistent with the co-evolution of individualism and increased PFC function in EA. However, this finding was context specific—PFC response to qualified self-statements ("When talking to my mother, I am humble") was greater for collectivists than

individualists. Similarly, there is also evidence that greater adoption of American culture is associated with greater frontoparietal attentional control function (Hedden, Ketay, Aron, Markus, & Gabrieli, 2008). Specifically, acculturation was associated with frontoparietal activation when participants were asked to make absolute judgments, a basic perceptual analogue for judgments about social independence, versus relative judgments associated with interdependence. Again, the finding was context-specific—frontoparietal response to relative judgments was greater for individuals with greater adoption of East Asian culture.

Finally, there is evidence that among individuals from Western societies, but not from China, PFC activation is tuned to distinguish between self and other, again consistent with culturally appropriate PFC function for individualist cultures (where it is important to distinguish one's self from others) versus collectivist cultures (Zhu, Zhang, Fan, & Han, 2007). Taken together, these early findings from cultural neuroscience suggest that culture has important influences on the contextual-sensitivity of PFC networks, which are tuned to one's cultural values—a process that may be a core feature of EA brain development.

Emerging Social Context

In general, EA is associated with frontolimbic neurodevelopment and increases in future-oriented behavior. However, emerging adults may not take a linear path of steady increases in adaptive decision making from adolescence to adulthood, as evidenced by the spike in risky behavior in the early 20s; for example, alcohol and illicit drug use both peak early in EA, around age 21 (Substance Abuse and Mental Health Services Administration, 2012). Although the role of neural mechanisms in early EA substance use is only beginning to be studied (Beltz et al., 2013), the existing literature suggests two (compatible) possibilities.

First, the dynamic process of frontolimbic development may involve a brief disorganization of functional networks early in EA. This may be necessary to allow a new, more mature system to emerge (Lewis, 2000) but might also result in a temporary spike in risky behavior. Second, there may be an interaction between emerging social context and neural development that results in a vulnerability to risky behavior. The rapid changes in social context early in EA—for example, greater freedom and diminished social control (Arnett, 2005)—might outpace neurodevelopment, such that the shift in social context toward increased freedom outpaces neurodevelopment of regulatory systems needed to manage risky behavior. There is also some evidence that the rapidly emerging social context during the transition to college is associated with increased volume of frontolimbic brain structures (Bennett & Baird, 2006), although it is not clear if this association is causal or how it fits into the more general trajectory of EA frontolimbic development. Future functional brain studies will need to consider the impact of emerging social context on nonlinearities in EA behavioral and neural development by taking a more fine-grained longitudinal approach.

Summary

In sum, early work in cultural neuroscience suggests that PFC fine-tuning during EA may be at least partly driven by the environmental and cultural pressures. An increasing emphasis on cross-cultural neuroscience will be necessary to propel our understanding of brain function and development.

Conclusion

Neurodevelopment continues throughout EA in support of the prolonged emergence of adult functions and roles (Arnett, 1998). Specifically, EA is marked by the development of association cortices and the frontolimbic systems involved in executive, attention, reward, and social processes. The frontolimbic fine-tuning model proposed here holds that from adolescence to adulthood, adaptive, future-oriented behavior is promoted through both (1) increased prefrontal function and connectivity with the limbic system and (2) an emerging balance between ventromedial (approach) and ventrolateral (avoidance) prefrontal regulatory function.

Importantly, individual differences in this neurodevelopmental trajectory may underlie differences in functioning and risk for psychological disorders. Vulnerability to psychological disorder in EA may be increased by a “permissive environment” created by the dynamics of frontolimbic development (Benes, 1988, 2000). Specifically, an imbalance in frontolimbic function may result in a bias toward approach behavior and risk for externalizing and

substance abuse disorders or a bias toward avoidance behavior and risk for internalizing and mood disorders. When such disorder is comorbid with preexisting psychological dysfunction, the impacts are even more severe (Kessler & Wang, 2008). Increasing our understanding of these neural mechanisms of risk will provide insight into ways to modify developmental trajectories during an important developmental window to promote the healthy emergence of adult functioning.

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References

- Adamec, R. E. (1991). Individual differences in temporal lobe sensory processing of threatening stimuli in the cat. *Physiology & Behavior*, *49*(3), 455–464.
- Adolphs, R. (2003). Cognitive neuroscience of human social behaviour. *Nature Reviews Neuroscience*, *4*(3), 165–178.
- Adolphs, R. (2010). What does the amygdala contribute to social cognition? *Annals of the New York Academy of Sciences*, *1191*(1), 42–61.
- Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature Neuroscience*, *2*(11), 1032–1037.
- Arnett, J. J. (1998). Learning to stand alone: The contemporary American transition to adulthood in cultural and historical context. *Human Development*, *41*(5–6), 295–315.
- Arnett, J. J. (2000). Emerging adulthood: A theory of development from the late teens through the twenties. *American Psychologist*, *55*(5), 469–480.
- Arnett, J. J. (2005). The developmental context of substance use in emerging adulthood. *Journal of Drug Issues*, *35*(2), 235–254.
- Arnett, J. J., Grusec, J. E., & Hastings, P. D. (2007). Socialization in emerging adulthood. In J. E. Grusec & P. D. Hastings, (Eds.), *Handbook of socialization: Theory and research* (pp. 208–239). New York: Guilford.
- Balleine, B. W., Delgado, M. R., & Hikosaka, O. (2007). The role of the dorsal striatum in reward and decision-making. *Journal of Neuroscience*, *27*(31), 8161–8165.
- Bartzokis, G., Beckson, M., Lu, P. H., Nuechterlein, K. H., Edwards, N., & Mintz, J. (2001). Age-related changes in frontal and temporal lobe volumes in men: A magnetic resonance imaging study. *Archives of General Psychiatry*, *58*(5), 461–465.
- Beaulieu, C. (2002). The basis of anisotropic water diffusion in the nervous system—a technical review. *NMR in Biomedicine*, *15*(7–8), 435–455.
- Bechara, A., & Damasio, H. (2002). Decision-making and addiction (part I): Impaired activation of somatic states in substance dependent individuals when pondering decisions with negative future consequences. *Neuropsychologia*, *40*(10), 1675–1689.
- Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, *50*(1–3), 7–15.
- Bechara, A., Damasio, H., Damasio, A. R., & Lee, G. P. (1999). Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *Journal of Neuroscience*, *19*(13), 5473–5481.
- Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science*, *275*(5304), 1293–1295.
- Bechara, A., Dolan, S., Denburg, N., Hinds, A., Anderson, S. W., & Nathan, P. E. (2001). Decision-making deficits,

linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. *Neuropsychologia*, 39(4), 376–389.

Bechara, A., Dolan, S., & Hinds, A. (2002). Decision-making and addiction (part II): Myopia for the future or hypersensitivity to reward? *Neuropsychologia*, 40(10), 1690–1705.

Bechara, A., Tranel, D., Damasio, H., & Damasio, A. R. (1996). Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. *Cerebral Cortex*, 6(2), 215–225.

Beltz, A. M., Gates, K. M., Engels, A. S., Molenaar, P. C., Pulido, C., Turrisi, R., Berenbaum, S. A., Gilmore, R. O., & Wilson, S. J. (2013). Changes in alcohol-related brain networks across the first year of college: A prospective pilot study using fMRI effective connectivity mapping. *Addictive Behaviors*, 38, 2052–2059.

Benes, F. M. (1988). Post-mortem structural analyses of schizophrenic brain: Study designs and the interpretation of data. *Psychiatric Developments*, 6(3), 213–226.

Benes, F. M. (1989). Myelination of cortical-hippocampal relays during late adolescence. *Schizophrenia Bulletin*, 15(4), 585–593.

Benes, F. M. (1998). Brain development, VII: Human brain growth spans decades. *American Journal of Psychiatry*, 155(11), 1489–1489.

Benes, F. M. (2000). Emerging principles of altered neural circuitry in schizophrenia. *Brain Research Reviews*, 31(2–3), 251–269.

Benes, F. M., Turtle, M., Khan, Y., & Farol, P. (1994). Myelination of a key relay zone in the hippocampal formation occurs in the human brain during childhood, adolescence, and adulthood. *Archives of General Psychiatry*, 51(6), 477–484.

Benes, F. M., Vincent, S. L., Molloy, R., & Khan, Y. (1996). Increased interaction of dopamine-immunoreactive varicosities with GABA neurons of rat medial prefrontal cortex occurs during the postweaning period. *Synapse*, 23(4), 237–245.

Bennett, C. M., & Baird, A. A. (2006). Anatomical changes in the emerging adult brain: A voxel-based morphometry study. *Human Brain Mapping*, 27, 766–777.

Bjork, J. M., Knutson, B., Fong, G. W., Caggiano, D. M., Bennett, S. M., & Hommer, D. W. (2004). Incentive-elicited brain activation in adolescents: Similarities and differences from young adults. *Journal of Neuroscience*, 24(8), 1793–1802.

Bjork, J. M., Smith, A. R., Danube, C. L., & Hommer, D. W. (2007). Developmental differences in posterior mesofrontal cortex recruitment by risky rewards. *Journal of Neuroscience*, 27(18), 4839–4849.

Borkovec, T. D., Alcaine, O. M., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. G. Heimberg (Ed.), *Generalized anxiety disorder: Advances in research and practice* (pp. 77–108). New York: Guilford.

Bos, W. van den, Cohen, M. X., Kahnt, T., & Crone, E. A. (2012). Striatum-medial prefrontal cortex connectivity predicts developmental changes in reinforcement learning. *Cerebral Cortex*, 22(6), 1247–1255.

Botvinick, M. M., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences*, 8(12), 539–546.

Burnett, S., Sebastian, C., Cohen Kadosh, K., & Blakemore, S. -J. (2011). The social brain in adolescence: Evidence from functional magnetic resonance imaging and behavioural studies. *Neuroscience & Biobehavioral Reviews*, 35(8), 1654–1664.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, 4(6), 215–222.

Emerging Adulthood Brain Development

- Casey, B. J., Getz, S., & Galvan, A. (2008a). The adolescent brain. *Developmental Review*, 28(1), 62–77.
- Casey, B. J., Jones, R. M., & Hare, T. A. (2008b). The Adolescent Brain. *Annals of the New York Academy of Sciences*, 1124(1), 111–126.
- Cauffman, E., Shulman, E. P., Steinberg, L., Claus, E., Banich, M. T., Graham, S., & Woolard, J. (2010). Age differences in affective decision making as indexed by performance on the Iowa Gambling Task. *Developmental Psychology*, 46(1), 193–207.
- Chiang, M. -C., McMahon, K. L., de Zubicaray, G. I., Martin, N. G., Hickie, I., Toga, A. W., Wright, M. J., & Thompson, P. M. (2011). Genetics of white matter development: A DTI study of 705 twins and their siblings aged 12 to 29. *NeuroImage*, 54(3), 2308–2317.
- Chiao, J. Y. (2009). Cultural neuroscience: A once and future discipline. In J. Y. Chiao (Ed.), *Progress in Brain Research* (Vol. 178, pp. 287–304). New York: Elsevier.
- Chiao, J. Y., & Cheon, B. K. (2010). The weirdest brains in the world. *Behavioral and Brain Sciences*, 33(2–3), 88–90.
- Chiao, J. Y., Harada, T., Komeda, H., Li, Z., Mano, Y., Saito, D., Parrish, T. B., Sadato, N., & Iidaka, T. (2009). Neural basis of individualistic and collectivistic views of self. *Human Brain Mapping*, 30(9), 2813–2820.
- Cunningham, M. G., Bhattacharyya, S., & Benes, F. M. (2002). Amygdalo-cortical sprouting continues into early adulthood: Implications for the development of normal and abnormal function during adolescence. *Journal of Comparative Neurology*, 453(2), 116–130.
- Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: Grosset/Putnam.
- Davidson, R. J., & McEwen, B. S. (2012). Social influences on neuroplasticity: Stress and interventions to promote well-being. *Nature Neuroscience*, 15(5), 689–695.
- Davis, M. (1992). The Role of the amygdala in fear and anxiety. *Annual Review of Neuroscience*, 15(1), 353–375.
- Davis, M., & Whalen, P. J. (2001). The amygdala: Vigilance and emotion. *Molecular Psychiatry*, 6(1), 13–34.
- Dimitrov, M., Phipps, M., Zahn, T. P., & Grafman, J. (1999). A thoroughly modern Gage. *Neurocase*, 5(4), 345–354.
- Douaud, G., Mackay, C., Andersson, J., James, S., Quested, D., Ray, M. K., Connell, J., Roberts, N., Crow, T. J., Matthews, P. M., Smith, S., & James, A. (2009). Schizophrenia delays and alters maturation of the brain in adolescence. *Brain*, 132(9), 2437–2448.
- Douglass, C. B. (2007). From duty to desire: Emerging adulthood in Europe and its consequences. *Child Development Perspectives*, 1(2), 101–108.
- Ernst, M., Nelson, E. E., Jazbec, S., McClure, E. B., Monk, C., Leibenluft, E., Blair, J., & Pine, D. S. (2005). Amygdala and nucleus accumbens in responses to receipt and omission of gains in adults and adolescents. *NeuroImage*, 25(4), 1279–1291.
- Ernst, M., Pine, D. S., & Hardin, M. (2006). Triadic model of the neurobiology of motivated behavior in adolescence. *Psychological Medicine*, 36(03), 299–312.
- Eshel, N., Nelson, E. E., Blair, R. J., Pine, D. S., & Ernst, M. (2007). Neural substrates of choice selection in adults and adolescents: Development of the ventrolateral prefrontal and anterior cingulate cortices. *Neuropsychologia*, 45(6), 1270–1279.
- Eslinger, P. J., & Damasio, A. R. (1985). Severe disturbance of higher cognition after bilateral frontal lobe ablation. *Neurology*, 35, 1731–1741.
- Fareri, D. S., Martin, L. N., & Delgado, M. R. (2008). Reward-related processing in the human brain: Developmental considerations. *Development and Psychopathology*, 20(Special Issue 04), 1191–1211.

Emerging Adulthood Brain Development

- Feil, J., Sheppard, D., Fitzgerald, P. B., Yücel, M., Lubman, D. I., & Bradshaw, J. L. (2010). Addiction, compulsive drug seeking, and the role of frontostriatal mechanisms in regulating inhibitory control. *Neuroscience & Biobehavioral Reviews*, 35(2), 248–275.
- Fields, R. D., & Stevens-Graham, B. (2002). New insights into neuron-glia communication. *Science*, 298(5593), 556–562.
- Frith, C. D. (2007). The social brain? *Philosophical Transactions of the Royal Society B: Biological Sciences*, 362(1480), 671–678.
- Fuster, J. M. (2001). The prefrontal cortex—An update: Time is of the essence. *Neuron*, 30(2), 319–333.
- Galvan, A., Hare, T. A., Parra, C. E., Penn, J., Voss, H., Glover, G., & Casey, B. J. (2006). Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience*, 26(25), 6885–6892.
- Geier, C., & Luna, B. (2009). The maturation of incentive processing and cognitive control. *Pharmacology Biochemistry and Behavior*, 93(3), 212–221.
- Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., Paus, T., Evans, A. C., & Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*, 2(10), 861–863.
- Giedd, J. N., Lenroot, R. K., Shaw, P., Lalonde, F., Celano, M., White, S., Tossell, J., Addington, A., & Gogtay, N. (2008). Trajectories of anatomic brain development as a phenotype. *Novartis Foundation Symposium*, 289, 101–195.
- Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., Nugent, T. F. III, Herman, D. H., Clasen, L. S., Toga, A. W., Rapoport, J. L., & Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences of the United States of America*, 101(21), 8174–8179.
- Granic, I., & Patterson, G. R. (2006). Toward a comprehensive model of antisocial development: A dynamic systems approach. *Psychological Review*, 113(1), 101–131.
- Gur, R. E., Loughhead, J., Kohler, C. G., Elliott, M. A., Lesko, K., Ruparel, K., Wolf, D. H., Bilker, W. B., & Gur, R. C. (2007). Limbic activation associated with misidentification of fearful faces and flat affect in schizophrenia. *Archives of General Psychiatry*, 64(12), 1356–1366.
- Hahn, A., Stein, P., Windischberger, C., Weissenbacher, A., Spindelegger, C., Moser, E., Kasper, S., & Lanzenberger, R. (2011). Reduced resting-state functional connectivity between amygdala and orbitofrontal cortex in social anxiety disorder. *NeuroImage*, 56(3), 881–889.
- Hardee, J. E., Benson, B. E., Bar-Haim, Y., Mogg, K., Bradley, B. P., Chen, G., Britton, J., Ernst, M., Fox, N. A., Pine, D. S., & Pérez-Edgar, K. (2013). Patterns of neural connectivity during an attention bias task moderate associations between early childhood temperament and internalizing symptoms in young adulthood. *Biological Psychiatry*, 74(4), 273–279.
- Harlow, J. M. (1868). Recovery from the passage of an iron rod through the head. *Publications of the Massachusetts Medical Society*, 2, 327–347.
- Hedden, T., Ketay, S., Aron, A., Markus, H. R., & Gabrieli, J. D. E. (2008). Cultural influences on neural substrates of attentional control. *Psychological Science*, 19(1), 12–17.
- Henrich, J., Heine, S. J., & Norenzayan, A. (2010). The weirdest people in the world. *Behavioral and Brain Sciences*, 33(2–3), 61–83.
- Huttenlocher, P. R., & Dabholkar, A. S. (1997). Developmental anatomy of prefrontal cortex. *Development of the Prefrontal Cortex: Evolution, Neurobiology, and Behavior*, 69–83.

- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 593–602.
- Kessler, R. C., & Wang, P. S. (2008). The descriptive epidemiology of commonly occurring mental disorders in the United States. *Annual Review of Public Health*, 29, 115–129.
- Kim, M. J., Gee, D. G., Loucks, R. A., Davis, F. C., & Whalen, P. J. (2011). Anxiety dissociates dorsal and ventral medial prefrontal cortex functional connectivity with the amygdala at rest. *Cerebral Cortex*, 21(7), 1667–1673.
- Kleim, J. A., & Jones, T. A. (2008). Principles of experience-dependent neural plasticity: Implications for rehabilitation after brain damage. *Journal of Speech, Language & Hearing Research*, 51(1), S225–S239.
- Kleim, J. A., Lussnig, E., Schwarz, E. R., Comery, T. A., & Greenough, W. T. (1996). Synaptogenesis and FOS expression in the motor cortex of the adult rat after motor skill learning. *Journal of Neuroscience*, 16(14), 4529–4535.
- Kringelbach, M. L., & Rolls, E. T. (2004). The functional neuroanatomy of the human orbitofrontal cortex: Evidence from neuroimaging and neuropsychology. *Progress in Neurobiology*, 72(5), 341–372.
- Kuperberg, G. R., Broome, M. R., McGuire, P. K., David, A. S., Eddy, M., Ozawa, F., Goff, D., West, W. C., Williams, S. C., van der Kouwe, A. J., Salat, J. H., Dale, A. M., & Fischl, M. (2003). Regionally localized thinning of the cerebral cortex in schizophrenia. *Archives of General Psychiatry*, 60(9), 878.
- LeDoux, J. (1993). Emotional memory systems in the brain. *Behavioural Brain Research*, 58(1–2), 69–79.
- LeDoux, J. (1998). Fear and the brain: Where have we been, and where are we going? *Biological Psychiatry*, 44(12), 1229–1238.
- Leijenhorst, L. V., Zanolie, K., Van Meel, C. S., Westenberg, P. M., Rombouts, S. A. R. B., & Crone, E. A. (2010). What motivates the adolescent? Brain regions mediating reward sensitivity across adolescence. *Cerebral Cortex*, 20(1), 61–69.
- Lewis, M. (2000). The promise of dynamic systems approaches for an integrated account of human development. *Child Development*, 71(1), 36–43.
- Luna, B., & Sweeney, J. A. (2004). The emergence of collaborative brain function: FMRI studies of the development of response inhibition. *Annals of the New York Academy of Sciences*, 1021(1), 296–309.
- MacLean, P. D. (1985). Brain evolution relating to family, play, and the separation call. *Archives of General Psychiatry*, 42(4), 405.
- Maguire, E. A., Frackowiak, R. S. J., & Frith, C. D. (1997). Recalling routes around London: Activation of the right hippocampus in taxi drivers. *Journal of Neuroscience*, 17(18), 7103–7110.
- Maguire, E. A., Gadian, D. G., Johnsrude, I. S., Good, C. D., Ashburner, J., Frackowiak, R. S. J., & Frith, C. D. (2000). Navigation-related structural change in the hippocampi of taxi drivers. *Proceedings of the National Academy of Sciences*, 97(8), 4398–4403.
- Masten, A. S. (2004). Regulatory processes, risk, and resilience in adolescent development. *Annals of the New York Academy of Sciences*, 1021(1), 310–319.
- McClure, S. M., York, M. K., & Montague, P. R. (2004). The neural substrates of reward processing in humans: The modern role of fMRI. *Neuroscientist*, 10(3), 260–268.
- Menon, V., & Uddin, Q. (2010). Saliency, switching, attention and control: A network model of insula function. *Brain Structure and Function*, 214(5–6), 655–667.
- Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, 24(1), 167–202.

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- Mueller, E. M., Nguyen, J., Ray, W. J., & Borkovec, T. D. (2010). Future-oriented decision-making in Generalized Anxiety Disorder is evident across different versions of the Iowa Gambling Task. *Journal of Behavior Therapy and Experimental Psychiatry*, 41(2), 165–171.
- Munafò, M. R., Brown, S. M., & Hariri, A. R. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry*, 63(9), 852–857.
- Narr, K. L., Bilder, R. M., Toga, A. W., Woods, R. P., Rex, D. E., Szeszko, P. R., Robinson, D., Sevy, S., Gunduz-Bruce, H., Wang, Y. -P., DeLuca, H., & Thompson, P. M. (2005a). Mapping cortical thickness and gray matter concentration in first episode schizophrenia. *Cerebral Cortex*, 15(6), 708–719.
- Narr, K. L., Toga, A. W., Szeszko, P., Thompson, P. M., Woods, R. P., Robinson, D., Sevy, S., Wang, Y. -P., Schrock, K., & Bilder, R. M. (2005b). Cortical thinning in cingulate and occipital cortices in first episode schizophrenia. *Biological Psychiatry*, 58(1), 32–40.
- Nelson, E. E., Leibenluft, E., McClure, E. B., & Pine, D. S. (2005). The social re-orientation of adolescence: A neuroscience perspective on the process and its relation to psychopathology. *Psychological Medicine*, 35(02), 163–174.
- O'Doherty, J., Kringelbach, M. L., Rolls, E. T., Hornak, J., & Andrews, C. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience*, 4(1), 95–102.
- O'Doherty, J. P. (2007). Lights, camembert, action! The role of human orbitofrontal cortex in encoding stimuli, rewards, and choices. *Annals of the New York Academy of Sciences*, 1121(1), 254–272.
- Overman, W. H. (2004). Sex differences in early childhood, adolescence, and adulthood on cognitive tasks that rely on orbital prefrontal cortex. *Brain and Cognition*, 55(1), 134–147.
- Paus, T., Keshavan, M., & Giedd, J. N. (2008). Why do many psychiatric disorders emerge during adolescence? *Nature Reviews Neuroscience*, 9(12), 947–957.
- Rapoport, J. L., Giedd, J. N., & Gogtay, N. (2012). Neurodevelopmental model of schizophrenia: update 2012. *Molecular Psychiatry*, 17, 1228-1238.
- Reyna, V. F., & Farley, F. (2006). Risk and rationality in adolescent decision making implications for theory, practice, and public Policy. *Psychological Science in the Public Interest*, 7(1), 1–44.
- Rolls, E. T. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex*, 10(3), 284–294.
- Sah, P., Faber, E. S. L., Lopez de Armentia, M., & Power, J. (2003). The amygdaloid complex: Anatomy and physiology. *Physiological Reviews*, 83(3), 803–834.
- Schneider, F., Weiss, U., Kessler, C., Salloum, J. B., Posse, S., Grodd, W., & Müller-Gärtner, H. W. (1998). Differential amygdala activation in schizophrenia during sadness. *Schizophrenia Research*, 34(3), 133–142.
- Schulenberg, J. E., & Zarrett, N. R. (2006). Mental health during emerging adulthood: Continuity and discontinuity in courses, causes, and functions. In J. J. Arnett & J. L. Tanner (Eds.), *Emerging adults in America: Coming of age in the 21st century* (pp. 135–172). Washington, DC: American Psychological Association.
- Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants “grown up”: Adult amygdala response to novelty. *Science*, 300(5627), 1952–1953.
- Shenton, M. E., Dickey, C. C., Frumin, M., & McCarley, R. W. (2001). A review of MRI findings in schizophrenia. *Schizophrenia Research*, 49(1–2), 1–52.
- Sowell, E. R., Peterson, B. S., Thompson, P. M., Welcome, S. E., Henkenius, A. L., & Toga, A. W. (2003). Mapping cortical change across the human life span. *Nature Neuroscience*, 6(3), 309–315.
- Sowell, E. R., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*, 2(10), 859–861.

- Sowell, E. R., Thompson, P. M., Welcome, S. E., Henkenius, A. L., Toga, A. W., & Peterson, B. S. (2003). Cortical abnormalities in children and adolescents with attention-deficit hyperactivity disorder. *Lancet*, *362*(9397), 1699–1707.
- Sowell, E. R., Trauner, D. A., Gamst, A., & Jernigan, T. L. (2002). Development of cortical and subcortical brain structures in childhood and adolescence: A structural MRI study. *Developmental Medicine & Child Neurology*, *44*(1), 4–16.
- Spear, L. P. (2000). Neurobehavioral changes in adolescence. *Current Directions in Psychological Science*, *9*(4), 111–114. doi: 10.1111/1467–8721.00072
- Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, *28*(1), 78–106.
- Substance Abuse and Mental Health Services Administration. (2012). *Results from the 2011 National Survey on Drug Use and Health: Summary of national findings*. NSDUH Series H-44, HHS Publication No. (SMA) 12–4713. Rockville, MD: Substance Abuse and Mental Health Services Administration.
- Taber-Thomas, B. C., Asp, E. W., Koenigs, M., Sutterer, M., Anderson, S. W., & Tranel, D. (2014). Arrested development: Early prefrontal lesions impair the maturation of moral judgment. *Brain*, *137*, 1254–1261.
- Taber-Thomas, B. C., & Tranel, D. (2012). Social and moral functioning. In V. Anderson & M. H. Beauchamp (Eds.), *Developmental social neuroscience and childhood brain insult: Theory and practice* (pp. 65–90). New York: Guilford.
- Thelen, E., & Smith, L. B. (2006). Dynamic systems theories. In W. Damon & R. M. Lerner (Eds.), *Handbook of child psychology* (pp. 258–312). Hoboken, NJ: John Wiley & Sons.
- Thompson, L. L., Claus, E. D., Mikulich-Gilbertson, S. K., Banich, M. T., Crowley, T., Krmpotich, T., Miller, D., & Tanabe, J. (2012). Negative reinforcement learning is affected in substance dependence. *Drug and Alcohol Dependence*, *123*(1–3), 84–90.
- Thompson, P. M., Sowell, E. R., Gogtay, N., Giedd, J. N., Vidal, C. N., Hayashi, K. M., Leow, A., Nicolson, R., Rapoport, J. L., & Toga, A. W. (2005). Structural MRI and brain development. *International Review of Neurobiology*, *67*, 285–323.
- Van der Maas, H. L., & Molenaar, P. C. (1992). Stagewise cognitive development: An application of catastrophe theory. *Psychological Review*, *99*(3), 395–417.
- Wallis, J. D. (2007). Neuronal mechanisms in prefrontal cortex underlying adaptive choice behavior. *Annals of the New York Academy of Sciences*, *1121*(1), 447–460.
- Watanabe, M. (1996). Reward expectancy in primate prefrontal neurons. *Nature*, *382*(6592), 629–632.
- Wilmouth, C. E., & Spear, L. P. (2004). Adolescent and adult rats' aversion to flavors previously paired with nicotine. *Annals of the New York Academy of Sciences*, *1021*(1), 462–464.
- Wright, I. C., Rabe-Hesketh, S., Woodruff, P. W. R., David, A. S., Murray, R. M., & Bullmore, E. T. (2000). Meta-analysis of regional brain volumes in schizophrenia. *American Journal of Psychiatry*, *157*(1), 16–25.
- Wu, M., Lu, L. H., Lowes, A., Yang, S., Passarotti, A. M., Zhou, X. J., & Pavuluri, M. N. (2013). Development of superficial white matter and its structural interplay with cortical gray matter in children and adolescents. *Human Brain Mapping*, *35*(6), 2806–2816.
- Yakovlev, P. I., & Lecours, A. -R. (1967). The myelogenetic cycles of regional maturation of the brain. In A. Minkowski (Ed.), *Regional development of the brain in early life* (pp. 3–70). Oxford, UK: Blackwell Scientific.
- Zhu, Y., Zhang, L., Fan, J., & Han, S. (2007). Neural basis of cultural influence on self-representation. *NeuroImage*, *34*(3), 1310–1316.

Zuckerman, M., Eysenck, S. B., & Eysenck, H. J. (1978). Sensation seeking in England and America: Cross-cultural, age, and sex comparisons. *Journal of Consulting and Clinical Psychology*, 46(1), 139–149.

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