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Commentary: A Behavioral Scientist Looks at the Science of Adolescent Brain Development

Laurence Steinberg
Temple University

In a remarkably short period of time – about ten years, by my estimation - the developmental neuroscience of adolescence has matured from a field in its infancy to one that is now approaching its own adolescence. The papers gathered in this special issue, from some of the world's leading scholars of adolescent brain development, showcase the richness, depth, and breadth of understanding that now characterizes this area of developmental science. As in any field that is barely a decade old, there remain many mysteries, contradictions, and unknowns. But given the short period of time during which systematic study has been ongoing, there is also a surprising degree of consistency, consensus, and replication.

In this brief commentary, I focus on several broad themes that cut across the articles in this collection, both respect to what we know (or at least have a reasonably good understanding of) and with respect to what we don't know. As a non-neuroscientist with no formal training in the study of brain development, it is beyond my expertise to delve deeply into the technical details of the research described by the contributors to this anthology. Nor is such an analysis necessary; the papers are uniformly clear and written at a level that will both interest other neuroscientists and edify behavioral scientists like myself, who increasingly find themselves needing to know about the neural underpinnings of the psychological and social phenomena they study. My focus in this commentary will be mainly on what these new studies reveal that will be of special interest to those who study adolescent behavioral development.

Let me begin with what will surely strike many readers as obvious, but which needs to be said strenuously and incontrovertibly. Taken together, the contributions to this issue demonstrate conclusively that the adolescent's brain is different from both the child's brain and the adult's brain. It is different with respect to both morphology and function, and at the levels of brain structures, regions, circuits, and systems. It is different with respect to grey matter (Gotgay & Thompson, this issue), white matter (Paus, this issue), structural connectivity (Schmithorst & Yuan, this issue; White et al., this issue), and neurotransmission (Doremus-Fitzwater et al., this issue; Wahlstrom et al., this issue). It is different in ways that are revealed in studies of sleep (Feinberg & Campbell, this issue), electrophysiology (Segalowitz et al., this issue), functional imaging (Luna et al., this issue; Somerville et al., this issue), pharmacological challenge (Wahlstrom et al., this issue), and stress reactivity (McCormick et al., this issue). It is different in ways that are consistent with studies of juvenile rodents (Doremus-Fitzwater et al., this issue) and non-human primates (Dahl & Forbes, this issue), and among males as well

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Address correspondence to: Laurence Steinberg Department of Psychology Temple University Philadelphia, PA 19122
laurence.steinberg@temple.edu.

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as females (Lenroot & Giedd, this issue). And it is different in ways that are consistent with the observations of behavioral scientists who study both normative development (Blakemore et al., this issue; Dahl & Forbes, this issue) and developmental psychopathology (Gotgay & Thompson, this issue).

I state the obvious here to preemptively respond the claims of some non-neuroscientists that the notion of “the adolescent brain” is some sort of myth (Epstein, 2007; Males, 2009). As this collection of papers makes eminently clear, the fact that there are significant changes in the brain during adolescence is no longer debatable - if indeed it ever was. Indeed, it appears that the brain changes characteristic of adolescence are among the most dramatic and important to occur during the human lifespan. Whether neurobiological differences between adolescents and adults should inform how society treats young people is open for debate, but whether such differences are real is not (Steinberg, 2009).

That said, it is important to note, as several of the contributors to this issue have stated, that “different” does not necessarily mean “deficient”; that while there are some universals in adolescent brain development, there are also important individual differences; and that the process of brain maturation in adolescence (or during any period, for that matter) unfolds within an environmental context that influences the course of neural development and moderates its expression in emotion, behavior, and cognition. It is also important to acknowledge that we currently have a better understanding of the ways in which adolescent brain development may contribute to psychopathology and problem behavior than we do of the ways in which it may contribute to normative development and positive functioning, and that researchers have paid more attention to the study of universals and processes of biological maturation than to individual differences and environmental influences. As the field matures, and as collaborations between neuroscientists and non-neuroscientists become more common, attention to these issues will most likely increase.

Many of the papers in this collection reaffirm some established principles about adolescent brain development, but with important added caveats and nuances. For example, evidence continues to accumulate that brain maturation continues throughout adolescence, with some of the most significant changes taking place in prefrontal areas. It is now well established that there is a decline in grey matter and an increase in white matter in these regions during adolescence, but as several authors note, there is some evidence that these changes are not due solely to synaptic pruning and myelination, as had been widely assumed. With regard to the first, it now appears that some of the change in the grey matter density that had been attributed to synaptic pruning may instead be due to other cellular processes as well as increases in white matter, which would affect estimates of grey matter density (Gotgay & Thompson, this issue). Moreover, as Paus (this issue) points out, the extent to which increases in white matter are due to changes in myelin versus changes in axonal diameter is not yet known; although both would improve the flow of information across neural pathways, changes in axonal caliber may have implications for particular neurotransmission processes. However, regardless of whether the absolute extent of synaptic pruning to take place in the prefrontal cortex during adolescence is somewhat smaller than had been believed, the basic notion that the density of prefrontal grey matter follows a $\cap$ -shaped trajectory over development, with a peak somewhere around age 11 (earlier for girls, later for boys) still finds considerable empirical support and therefore continues to direct our attention to the study of behavioral phenomena in adolescence that are linked in one way or another to prefrontal functioning. That said, there is also growing evidence that important changes occur during adolescence in other brain regions, including the parietal and temporal cortices, as well as the cerebellum. The implications of these structural changes for adolescent behavioral development are far less understood than are those involving the prefrontal cortex.

As Gotgay and Thompson (this issue) note, the evidence also points to early adolescence as a time of considerable brain plasticity, which has several important implications. To the extent that patterns of synaptic proliferation and elimination are contextually-dependent (something that is not yet known but crucial to examine), we should expect to see considerable individual differences in brain structure and function that can be linked to differences in experience. In this sense, brain plasticity in adolescence makes this period a time of considerable opportunity for intervention. At the same time, however, heightened brain plasticity in adolescence may contribute to increased vulnerability to certain forms of psychopathology, many of which begin or intensify during adolescence. This suggestion is further bolstered by studies demonstrating differential responsivity to stress during adolescence as compared to adulthood, as discussed by McCormick and colleagues in this issue (see also Walker et al., 2004).

Although researchers continue to study patterns of grey matter development in adolescence, increased attention is now being given to changes in white matter during this period, in part stimulated by the growing use of DTI to study changes in structural connectivity. Indeed, whereas synaptic pruning in the frontal lobe was the main focus of attention in previous research on structural aspects of brain development in adolescence, white matter development has clearly stolen some of the scientific limelight. It is now clear that adolescence is a time of dramatic changes in fiber tracts that link different brain regions and structures (Schmithorst & Yuan, this issue). This increase in structural connectivity is, not surprisingly, paralleled by increases in functional connectivity, which has significant implications for our understanding of changes in adolescent behavior, especially with regard to cognitive control (Luna et al., this issue). An important lesson for behavioral scientists about brain development in adolescence therefore is that it is not, as the popular media would suggest, all about synaptic pruning in the frontal lobe.

As research begins to link changes in white matter (within prefrontal regions but, perhaps more important, in pathways linking prefrontal areas to other brain regions) to changes in adolescent behavior (a topic of study that has just now started receiving interest), we will gain further insight into the neural underpinnings of behavioral development in adolescence. For instance, work by Paus and his colleagues (Grosbras et al., 2007; Paus et al., 2008) has demonstrated that individual differences in individuals' vulnerability to peer pressure are correlated with differences in structural and functional connectivity in ways that link the development of resistance to peer influence to improvements in the coordination of emotion and cognition. This finding is also consistent with research showing that individual differences in structural connectivity during early adolescence are correlated with delay discounting performance, such that individuals with more highly organized white matter are less likely to be drawn to immediate rewards (Olson et al., 2008). It is worth noting, however, that these findings are hard to reconcile with a recent report from Berns et al. (2009) indicating that structural maturity of white matter is correlated with *more*, not less, risk-taking. It is too early in the development of this area of work to render an explanation for this apparent inconsistency, but my guess is that research on the behavioral correlates of inter-regional connectivity ultimately may prove more informative than that on the correlates of grey or white matter density, especially in the study of self-regulation.

Evidence also grows concerning important changes in subcortical processes during adolescence. Especially important are increases in dopaminergic activity during early adolescence in pathways linking limbic, striatal, and prefrontal areas (Wahlstrom et al., this issue). These changes, documented in both human and animal studies, have been linked to changes in reward-directed activity, as described by Doremus-Fitzwater et al. (this issue), but as Dahl and Forbes (this issue) point out, the pubertal period is a time of changes in appetitive behavior more generally, and not simply in reward-driven behavior. Dopamine, of course, plays a role in reward anticipation and reward-seeking, but it also has been implicated in motivated

action more broadly (Angier, 2009). It makes perfect evolutionary sense that individuals are more motivated by appetitive inclinations, more oriented toward sensation-seeking, and more willing to take risks during adolescence, when they must leave the natal environment and seek out mates (Casey et al., 2008; Spear, 2010; Steinberg, 2008). From an evolutionary perspective, adolescent risk-taking is a *good* thing, not a bad one.

These three changes – the change in the ratio of grey to white matter in prefrontal areas, the increase in connectivity between prefrontal and other regions, and the increase in dopaminergic activity in prefrontal-striatal-limbic pathways – provide the basis for a theory that links brain maturation in adolescence to increased vulnerability to risky behavior. The basic framework, articulated in slightly different versions by many writers, including Casey (Casey et al., 2008), Dahl (2004), Ernst (Ernst et al., 2006), Spear (2009), and myself (Steinberg, in press), posits that middle adolescence is a time of heightened vulnerability to risky and reckless behavior because of the temporal disjunction between the rapid rise in dopaminergic activity around the time of puberty, which leads to an increase in reward-seeking, and the slower and more gradual maturation of the prefrontal cortex and its connections to other brain regions, which leads to improvements in cognitive control and in the coordination of affect and cognition. As dopaminergic activity declines from its early adolescent peak, and as self-regulatory systems become increasingly mature, risk-taking begins to decline.

From this perspective, middle adolescence (roughly 14 to 17) should be a period of especially heightened vulnerability to risky behavior, because sensation-seeking is high and self-regulation is still immature. And in fact, many risk behaviors follow this pattern, including unprotected sex, criminal behavior, attempted suicide, and reckless driving (Steinberg, 2008). How do we explain risky behavior that follows a different developmental trajectory? I would argue that the reason that certain other risky behaviors, such as binge drinking, peak a little later in development is because there are more constraints on opportunities to engage on them during middle adolescence (e.g., the prohibition of alcohol sales to individuals under 21 in the United States), and not because the underlying psychological processes are different.

This general model of adolescent brain development has been extended beyond the study of risk-taking in several ways. First, as Blakemore and colleagues (this issue) discuss, adolescence is also a time of important changes in the processing of social and emotional information, much of which is subserved by the same regions and systems that undergird the motivational and self-regulatory changes described by writers who have focused on risk taking. Whether and how these various sets of changes may be linked is an important question (Albert & Steinberg, in press). For instance, there is evidence that adolescents are highly responsive to the social rewards afforded by positive peer evaluation and that such rewards activate the same brain regions as non-social rewards (Guyer et al., 2009).

We are currently engaged in a program of research in our lab designed to examine the impact of peers on the neural underpinnings of risk-taking. Building on experimental work showing age differences in the degree to which peer presence evokes risky behavior (Gardner & Steinberg, 2005), with peers affecting risk-taking among adolescents but not adults, we have attempted to bring peer context into the scanner (Chein et al., 2009). Using an event-related fMRI design, we are examining age differences in neural activation at the moment of decision-making in different risk-taking tasks. To manipulate peer context, we measure task-related neural activation for each participant during two separate sessions. In one session, the participant completes the tasks while their peers are observing their performance from the scanner control room; in the other session, the participant completes the task with no observation. In each case, the participant is made aware of the condition. Consistent with our predictions, preliminary data indicates that adolescents activate socio-emotional reward regions (e.g., medial PFC, ventral striatum) more strongly when making risky decisions while

being observed by their peers than when they do so alone. In contrast, our early data suggest that adults show few differences in activation of socio-emotional reward centers in the peer condition as compared to when they are alone. In this program of work, we have also replicated the earlier finding, but with completely different tasks, showing that the presence of peers increases adolescents' risk taking but has no such effect on adults. We also have evidence that the presence of peers leads adolescents to more steeply discount delayed rewards, leading to increased preference for immediate, although smaller, ones (O'Brien & Steinberg, 2010). This is a potentially important finding, since it provides further evidence that the impact of peers on risky decision-making may be mediated specifically by their effects on reward processing.

A second extension is the application of this model beyond the study of risk-taking and into the realm of psychopathology more generally (Paus et al., 2008; Steinberg et al., 2006). As I noted earlier, and as several contributors to this issue describe, many forms of psychopathology onset or intensify during adolescence. Some, but not all, of these forms of psychopathology in one way or another involve appetitive or affective dysregulation (e.g., depression, substance abuse, eating disorders). This leads to the very reasonable hypothesis that at least some forms of adolescent psychopathology are related to abnormalities in the remodeling of the dopaminergic system at puberty (which would affect appetitive and affective functioning) or in the morphological changes of the prefrontal cortex or its connections to other brain regions over the course of adolescence (which would affect self-regulation).

A couple of points about this “dual systems” framework are noteworthy. First, from where I sit as a generalist in adolescent development, this neuroscientific model of adolescence is the first new “grand theory” of adolescence to be proposed in the last 50 years. I noted about a decade ago (Steinberg & Morris, 2001) that the field has more or less abandoned the major grand theories of adolescence that previously had dominated research (psychoanalytic theory, Erikson's theory of identity development, and Piaget's theory of cognitive development), but nothing as ambitious or sweeping has replaced these viewpoints. The emergent neuroscientific perspective described in this commentary and in several of the contributions to this issue (I use the term “emergent” because the framework is still developing) has the potential to structure a new, overarching model of normative and atypical adolescent development. Notably, the basic notion that early adolescence is characterized by a dramatic increase in appetitive drive that remains relatively unchecked until self-regulatory systems mature is eerily similar to the basic Freudian model of adolescence (change “appetitive drive” to “libido” and “self-regulatory systems” to “ego development” and see for yourself) that modern-day empiricists have derided. As they say, “*plus ça change*.”

Second, it is ironic that the remarkable increase in research on adolescent brain development during the past decade has outpaced research on the very psychological phenomena that this period of brain maturation presumably causes. One limitation of much extant psychological research is that few studies typically include an age range spanning preadolescence, adolescence, and early adulthood, likely because psychologists have been more interested in development during early and middle adolescence than during late adolescence or young adulthood. Consequently, there are many behavioral and self-report studies that compare children and adolescents, fewer that compare adolescents and adults, and almost none that compare children, adolescent, and adults all at once and allow for the detection of trends that may not be linear. This is especially problematic where reward-seeking and risk-taking are concerned, because, in light of research indicating the curvilinear nature of the developmental trajectory of dopaminergic receptor remodeling, there is reason to think that these behaviors increase until middle adolescence and then decline. In contrast, given what we know about maturation of the prefrontal cortex and its connections with other brain regions, the developmental course of cognitive control would be expected to increase linearly and into the decade of the 20s.

Ironically, some of the best *behavioral* data on these matters now come from functional brain imaging studies of reward processing (e.g., Adelman et al., 2002; Bjork et al., 2004; Ernst et al., 2005; Fareri et al., 2008; Galvan et al., 2006; May et al., 2004; Van Leijenhorst et al., 2009) and self-regulation (e.g., Durston et al., 2002; Luna et al., 2001; Rubia, et al., 2006; Stevens et al., 2007; Tamm et al., 2002; Velanova et al., 2008). It is true that there are limits on how closely one can mimic real world situations in laboratory paradigms, but these seem no more troublesome to me than the constraints on any experimental research. Indeed, one reason these studies are so important is that in the lab it is possible to construct tasks that have equivalent meaning to people of different ages and, if necessary, to manipulate performance so that brain functioning can be compared both with and without equivalent performance (see Blakemore et al., this issue; Luna et al., this issue).

To the extent that a goal of research on brain development is to better understand adolescent behavior, it is important to complement the neurobiological studies with both experimental and field research designed to examine whether the basic principles of development identified in the lab have parallels in the real world. In order to do this, my colleagues and I conducted a large cross-sectional study of 935 individuals between the ages of 10 and 30 that tests these very hypotheses. Our findings indicate that reward sensitivity, as indexed on the Iowa Gambling Task (Cauffman, et al., in press); preference for immediate rewards, as indexed by performance on a delay discounting task (Steinberg, et al., 2009); and sensation-seeking, as indexed by self report and performance in a video driving game (Steinberg, et al., 2008), follow a $\cap$ -shaped function, increasing between preadolescence and mid-adolescence, peaking between 14 and 16, and then declining. In contrast, impulse control, as indexed by self report and unhurried decision-making during the Tower of London task (Steinberg et al., 2008); anticipation of future consequences, as indicated by self report (Steinberg et al., 2009), strategic planning, as indexed by self report (Steinberg et al., 2009) and Tower of London performance (Albert, et al., 2009); and resistance to peer influence as indexed by self-report (Steinberg & Monahan, 2007) and experimentally manipulated exposure to peers (Gardner & Steinberg, 2005), all increase linearly from preadolescence through late adolescence and, in some respects, early adulthood. Moreover, and as predicted within this model, individuals' preference for risky activity – the extent to which they believe that the benefits of risk-taking outweigh the costs – is higher during mid-adolescence than before or after and predicted independently and jointly by measures of reward-seeking and self-regulation (Steinberg, 2009).

Third, despite the basic heuristic utility of this emergent framework, one element that has been missing from most discussions linking adolescent brain development and behavior is the context in which adolescents live. Here I am not referring so much to the impact of context on brain development (although, as noted earlier, the likely plasticity of the adolescent brain makes this a crucial issue for future study), but the role of context in moderating the way that neural influences are expressed. The heightened vulnerability of middle adolescence will have different consequences in different settings, both as a function of available opportunities to engage in reward-seeking, and as a function of the degree to which external agents regulate adolescents whose self-regulation is still maturing. With respect to reward-seeking, as Dahl (2004) has noted, there are many ways for adolescents to satisfy their inclinations toward sensation-seeking that are not harmful or antisocial. And with respect to self-regulation, the degree to which adolescents engage in risky behavior is in part a function of opportunities to access substances and circumstances that place them at risk. The reason that the incidence of alcohol-related driving fatalities declined when the legal drinking age was raised from 18 to 21 has nothing to do with changes in the brain development of young people that somehow magically transformed high school students *en masse* into mature decision-makers. By the same token, increasing parental monitoring, placing restrictions on teen driving, or providing adult supervision during after-school hours would likely reduce teen pregnancies, car crashes, and juvenile crime, but for reasons that have nothing to do with neurobiological development.

Brain development undoubtedly influences adolescents' behavior, but it does so within a context.

The papers collected in this issue also make it abundantly clear that many important questions about adolescent brain development remain to be asked and answered. One fundamental issue that desperately needs research attention concerns the role of puberty in adolescent brain development. It has been hypothesized that changes in reward-seeking are linked to the impact of gonadal hormones on brain function (see Dahl & Forbes, this issue), but the extent to which this is the case is uncertain, as is the extent to which these puberty-dependent processes differ between males and females, whose endocrinological environments are obviously very different. (My reading of the literature on sex differences in brain development, reinforced by the contribution to this issue by Lenroot and Giedd, is that the area is littered with inconsistencies and that a coherent story has yet to be told, perhaps because there isn't much of one to tell.) Animal studies suggest that some of the changes in dopaminergic activity are directly attributable to the hormonal changes of puberty, that some are indirectly attributable to them (in that the presence of gonadal hormones may catalyze other processes), and that some are merely coincident with them, perhaps programmed to unfold along a developmental schedule established long before adolescence (Sisk & Foster, 2004). Conversely, it has also been hypothesized that the maturation of brain systems implicated in self-regulation is independent of puberty and perhaps more contextually dependent, but, again, there is little research that has examined this systematically. If the development of cognitive control is in fact experience-dependent, it is important to study which experiences matter most. Parents and educators understandably want to know whether it is possible to facilitate the development of self-regulation.

There are also new areas of research, some represented in this collection, that are only just beginning to draw attention. Exciting work is underway on the neural underpinnings of adolescent psychopathology, social cognition, and stress responsivity, and we can look forward to a dramatic increase in our understanding of these phenomena over the next decade. Studies of genetic influences on brain development and on brain-behavior relations in adolescence are still in their infancy, as is research on individual differences in brain development that may have genetic underpinnings. And research on neurotransmission, which has heretofore focused mainly on the role of dopamine, is beginning to expand into other substances that may also have special significance in adolescence. For example, puberty-related increases in gonadal hormones have been linked to a proliferation of receptors for oxytocin within the limbic system, including such structures as the amygdala and nucleus accumbens (Spear, 2009). Oxytocin neurotransmission has been implicated in a variety of social behaviors, including facilitation of social bonding and recognition and memory for positive social stimuli (Insel & Fernald, 2004). Whether oxytocin-related changes undergird some of the changes in social cognition described by Blakemore, or the impact of peers on reward seeking that my colleagues and I have been studying, is a question that warrants more attention.

As a scientist who has been working in the field of adolescent development long enough to remember when such a field didn't exist, I can say with great certainty that there has never been a more exciting time to be studying this phase of life. Much of this excitement comes from discoveries made within the laboratories of brain scientists, but the real excitement will come when this work is more fully integrated with psychological and contextual studies of this period of the life cycle. It is my hope that the next decade will be a time of great progress in the pursuit of this goal.

References

- Adelman N, Menon V, Blasey C, White C, Warsofsky I, Glover G, Reiss A. A developmental fMRI study of the Stroop Color-Word task. *Neuroimage* 2002;16:61–75. [PubMed: 11969318]
- Albert, D.; Steinberg, L. Peer influences on adolescent risk behavior. In: Bardo, MT.; Fishbein, DH.; Milich, R., editors. *Inhibitory control and drug abuse prevention: From research to translation*. Springer; New York: in press
- Albert D, Steinberg L, Banich M. Age differences in strategic planning as indexed by the Tower of London. 2009 Manuscript under review.
- Angier N. A Molecule of Motivation, Dopamine Excels at Its Task. *The New York Times*. October 26;2009 B1 and ff.
- Berns G, Moore S, Capra C. Adolescent engagement in dangerous behaviors is associated with increased white matter maturity of frontal cortex. *PLoS ONE* 2009;4:e6773. Published online, August 26, 2009. [PubMed: 19707512]
- Bjork J, Knutson B, Fong G, Caggiano D, Bennett S, Hommer D. Incentive-elicited brain activation in adolescents: Similarities and differences from young adults. *Journal of Neuroscience* 2004;24:1793–1802. [PubMed: 14985419]
- Casey BJ, Jones RM, Hare T. The adolescent brain. *Annals of the New York Academy of Sciences* 2008;1124:111–126. [PubMed: 18400927]
- Cauffman E, Shulman E, Steinberg L, Claus E, Banich M, Graham S, Woolard J. Age differences in affective decision making as indexed by performance on the Iowa Gambling Task. *Developmental Psychology*. in press.
- Chein, J.; DiSorbo, A.; Albert, D.; O'Brien, L.; Eagan, DE.; Steinberg, L. Neural markers of peer influence in adolescent risk taking; Paper presented at the biennial meeting of the Society for Research in Child Development; Denver. Apr. 2009
- Dahl R. Adolescent brain development: A period of vulnerabilities and opportunities. *Annals of the New York Academy of Sciences* 2004;1021:1–22. [PubMed: 15251869]
- Durston S, Thomas K, Yang Y, Ulug A, Zimmerman R, Casey BJ. A neural basis for development of inhibitory control. *Developmental Science* 2002;5:9–16.
- Epstein R. The myth of the teen brain. *Scientific American Mind*, April/May 2007:57–63.
- Ernst M, Nelson EE, Jazbec SP, McClure EB, Monk CS, Leibenluft, et al. Amygdala and nucleus accumbens in responses to receipt and omission of gains in adults and adolescents. *NeuroImage* 2005;25:1279–1291. [PubMed: 15850746]
- Ernst M, Pine DS, Hardin M. Triadic model of the neurobiology of motivated behavior in adolescence. *Psychological Medicine* 2006;36:299–312. [PubMed: 16472412]
- Fareri DS, Martin LN, Delgado MR. Reward-related processing in the human brain: Developmental considerations. *Developmental Psychopathology* 2008;20:1191–211.
- Galvan A, Hare TA, Parra CE, Penn J, Voss K, Glover G, et al. Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience* 2006;26(25):6885–6892. [PubMed: 16793895]
- Gardner M, Steinberg L. Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. *Developmental Psychology* 2005;41:625–635. [PubMed: 16060809]
- Grosbras M, Jansen M, Leonard G, McIntosh A, Osswald K, Poulsen C, Steinberg L, Toro R, Paus T. Neural mechanisms of resistance to peer influence in early adolescence. *Journal of Neuroscience* 2007;27:8040–8045. [PubMed: 17652594]
- Insel T, Fernald R. How the brain processes social information: Searching for the social brain. *Annual Review of Neuroscience* 2004;27:697–722.
- Luna B, Thulborn K, Munoz D, Merriam E, Garver K, Minshew N, Keshavan MS, Genovese CR, Eddy WF, Sweeney JA. Maturation of widely distributed brain function subserves cognitive development. *Neuroimage* 2001;13:786–793. [PubMed: 11304075]
- Males M. Does the adolescent brain make risk-taking inevitable?: A skeptical appraisal. *Journal of Adolescent Research* 2009;24:3–20.

- May JC, Delgado MR, Dahl RE, Stenger VA, Ryan ND, Fiez JA, Carter CS. Event-related functional magnetic resonance imaging of reward-related brain circuitry in children and adolescents. *Biological Psychiatry* 2004;55:359–366. [PubMed: 14960288]
- O'Brien, L.; Steinberg, L. Impact of peers on delay discounting; Poster to be presented at the biennial meeting of the Society for Research on Adolescence; Philadelphia. Mar. 2010
- Olson E, Collins P, Hooper C, Muetzel R, Lim K, Luciana M. White matter integrity predicts delay discounting behavior in 9- to 23-year-olds: A diffusion tensor imaging study. *Journal of Cognitive Neuroscience* 2008;21:1406–1421. [PubMed: 18767918]
- Paus T, Keshavan B, Giedd J. Why do so many psychiatric disorders emerge during adolescence? *Nature Reviews Neuroscience* 2008;9:947–957.
- Paus T, Toro R, Leonard G, Lerner J, Lerner R, Perron M, Pike G, Richer L, Steinberg L, Veillette S, Pausova Z. Morphological properties of the action-observation cortical network in adolescents with low and high resistance to peer influence. *Social Neuroscience* 2008;3:303–316. [PubMed: 18979383]
- Rubia K, Smith AB, Woolley J, Nosarti C, Heyman I, Taylor E, Brammer M. Progressive increase of frontostriatal brain activation from childhood to adulthood during event-related tasks of cognitive control. *Human Brain Mapping* 2006;27:973–993. [PubMed: 16683265]
- Sisk C, Foster D. The neural basis of puberty and adolescence. *Nature Neuroscience* 2004;7:1040–1047.
- Spear, L. The behavioral neuroscience of adolescence. Norton; New York: 2009.
- Steinberg L. A social neuroscience perspective on adolescent risk-taking. *Developmental Review* 2008;28:78–106. [PubMed: 18509515]
- Steinberg L. Should the science of adolescent brain development inform public policy? *American Psychologist* 2009;64
- Steinberg L. A dual systems model of adolescent risk-taking. *Developmental Psychobiology*. in press.
- Steinberg L, Monahan K. Age differences in resistance to peer influence. *Developmental Psychology* 2007;43:1531–1543. [PubMed: 18020830]
- Steinberg L, Morris A. Adolescent development. *Annual Review of Psychology* 2001;52:83–110.
- Steinberg L, Albert D, Cauffman E, Banich M, Graham S, Woolard J. Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. *Developmental Psychology* 2008;44:1764–1778. [PubMed: 18999337]
- Steinberg, L.; Dahl, R.; Keating, D.; Kupfer, D.; Masten, A.; Pine, D. Psychopathology in adolescence: Integrating affective neuroscience with the study of context. In: Cicchetti, D.; Cohen, D., editors. *Developmental psychopathology, Vol. 2: Developmental neuroscience*. Wiley; New York: 2006. p. 710–741.
- Steinberg L, Graham S, O'Brien L, Woolard J, Cauffman E, Banich M. Age differences in future orientation and delay discounting. *Child Development* 2009;80:28–44. [PubMed: 19236391]
- Stevens MC, Kiehl KA, Pearson GD, Calhoun VD. Functional neural networks underlying response inhibition in adolescents and adults. *Behavioural Brain Research* 2007;181:12–22.
- Tamm L, Menon V, Reiss A. Maturation of brain function associated with response inhibition. *Journal of the American Academy of Child and Adolescent Psychiatry* 2002;41:1231–1238. [PubMed: 12364845]
- Van Leijenhorst L, Zanolie K, Van Meel CS, Westenberg PM, Rombouts SARB, Crone EA. What motivates the adolescent? Brain regions mediating reward sensitivity across adolescence. *Cerebral Cortex*. 2009 Epub ahead of print: doi:10.1093.
- Velanova K, Wheeler ME, Luna B. Maturation changes in anterior cingulate and frontoparietal recruitment support the development of error processing and inhibitory control. *Cerebral Cortex* 2008;18:2505–2522. [PubMed: 18281300]
- Walker E, Sabuwalla Z, Huot R. Pubertal neuromaturation, stress sensitivity, and psychopathology. *Development and Psychopathology* 2004;16:807–824. [PubMed: 15704816]