Adolescent Brains and Risk-Taking

In a concise and insightful Letter to the Editor, David Moshman [2011] lays out his arguments against five core assumptions of those who attribute the risk-taking behaviors of adolescents to their immature brains. My reading of both the popular press and literature on adolescent development suggests that Moshman is one of the few scholars who turn a critical eye toward the neuroscientific and behavioral evidence on this topic. He should be commended for reminding us all that we should treat this evidence the same way that we treat any other form of evidence.

As we move forward in this area of research, I think it is important to keep in mind the following additional points. First, it is scientifically unwise to attribute causal status to a factor (e.g., brain development) before we clearly understand how this factor could produce its putative effects. So far, reviews of neuroscientific research [e.g., Byrnes, 2011] have revealed that brain regions and neural architectures seem to subserve three aspects of cognition: content (e.g., representation of a face or a concept), processes (e.g., retrieval or inhibition), and working memory capacity. When researchers hypothesize that brain development is responsible for changes in behavior between two age periods, they need to (a) specify which aspect of cognition is likely to be affected (e.g., content or working memory capacity?), (b) specify which mechanism of brain development led to this change (e.g., myelination or dendritic arborization?), and (c) consider whether the account makes sense. Otherwise the account is just hand-waving, and the proponent may be embarrassed when our understanding is more fully articulated 20 years from now (à la phrenology or Lemarkianism). For example, one theory of adolescent risk-taking is that the nucleus accumbens (NAcc) in the subcortical basal ganglia reaches maturity before the prefrontal cortex (PFC) [Casey, Getz, & Galvan, 2008]. Activation of the NAcc is often higher when people process rewards and the PFC is involved in inhibiting responses. In this model, higher adolescent risk-taking is explained by arguing that adolescents and adults would be more attracted to rewarding outcomes than children, but adolescents would not be able to control themselves as well as adults due to an immature PFC.
or immature connections between the PFC and NAcc. Given this account, we can ask: In what way is the adolescent NAcc more mature than a child’s NAcc (more or fewer neurons)? Why does this structural change lead to greater activation? What aspect of reward processing does this affect? And why would anatomical changes in the PFC and NAcc lead to greater inhibition? Not all answers to these questions would be sensible or plausible. In addition, the account could have a mechanistic explanation where people at all ages are slaves to neural activation that precedes their thoughts and actions.

Second, the definition of ‘the adolescent period’ in many psychological and neuroscientific studies is insufficiently imprecise. Researchers who study the development of deductive reasoning and decision-making (including me) have sometimes found that 16-year-olds and adults perform significantly better than 13-year-olds, but there are often small, if any, differences between 16-year-olds and adults. Moreover, some of the neuroscientific studies comparing adolescents and adults [e.g., Casey et al., 1997; Ernst et al., 2005] used age ranges such as 7–13 and 9–17 for adolescents. With sample sizes less than 20, children below the age of 12 in these samples could easily affect the results. In the design of future studies, researchers would be wise to expect that behavioral differences might emerge between 9-, 13-, 16-, and 24-year-olds; lumping any of these age groups together would be unwarranted and lead to misleading conclusions.

Third, researchers need to avoid the tendency of generalizing age differences in the performance on one task to all tasks in the domain of risk-taking. In my reviews of the literature on risk-taking [e.g., Byrnes, Miller, & Schafer, 1999; Byrnes, 2003], I have repeatedly discovered studies showing considerable within-person variability in people’s tendency to take risks. This is the norm. To be sure, there are some studies that show how small subsets of young adolescents will engage in three stereotypical forms of risk-taking (cigarette smoking, alcohol consumption, and unprotected sex). However, this finding does not mean all of us who take one kind of risk usually take any other kind of risk. It is for this reason that the choice of content matters in any study. For example, there are a few studies showing the neural basis of adolescent risk-taking in which adolescents showed greater activation of the NAcc than adults when they won a small monetary prize (e.g., four dollars). I don’t know about you, but four dollars would not excite my NAcc, but it would excite my 17-year-old son’s! We should also be cautious about generalizing from the adolescent period of rats (lasting 2 weeks) in which they engage in more peer play to the behavior or cognitions of human adolescents. Note that it is possible that other content besides money could produce the same effects, but in the absence of such data, we need to be cautious in overgeneralizing.

Fourth, as Moshman notes, there are epigenetic causes of brain development including experience. Normally, when we argue for brain development causing behavior changes, we
assume some endogenous genetic factor causes a developmental mechanism (e.g., myelination) to alter the brain regardless of the adolescent’s circumstances. In other words, this change would occur even when the individual was asleep, it would happen at roughly the same age in most adolescents, and adolescents could not stop it from happening (just like puberty). If adolescents repeatedly take risks, experience the consequences, and these experiences alter their brains, we would expect differences between their brains and children’s brains, and between their brains and adult brains. But maturation, strictly speaking, is not the cause of this difference in neural architecture. By analogy, many adolescent males lift weights to give them more muscular biceps. Would we look at muscular adolescents and assume that this bodily change caused them to lift weights?

Finally, the alleged increases in adolescent risk-taking have to take into account contextual factors. If surveys showed that 12-year-olds engage in less unprotected sex than 17-year-olds, is this age difference due to brain changes leading the latter to engage in more sex or the fact that 17-year-olds spend more time alone with dates and away from parents than 12-year-olds? If this difference in opportunity were equated, would the age difference remain? Similarly, in my reviews of the literature, I found many studies where the rate of unplanned pregnancies was similar between adolescents and young married couples in their 20s and 30s. These unplanned pregnancies are not usually viewed or discussed in the same way, but only because of the difference in age and marital status.

If I were a betting man, I would not expect this commentary or Moshman’s to lead to more critical thinking in the area of adolescent risk-taking because a number of folk ideas about adolescents are very resistant to change. But my 40 years of postadolescent brain development would not let me bet; it would be too risky.

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References


Incorporating Behavior Risk Statistics in Teenage Brain Discussion

Based on developmental and cognitive research, David Moshman’s [2011] cogent disputation of current biologically and stage-based claims of teenage flaws postulates that ‘adolescents are qualitatively distinct from children but not from adults.’ Claims about the biological/developmental flaws of the ‘teenage brain’ bear a disturbing resemblance to past biodeterminist theories advanced to justify racial, gender, and nationalist hierarchies that statistics, tests, and physiological measures were interpreted to confirm.

For example, in 1920, ‘colored’ people, just 10% of America’s population, accounted for 25% of firearms deaths, 43% of murders, and half of all illegitimate births. Psychometric testing by Carnegie Foundation-backed psychologist H.H. Goddard labeled 80–90% of non-Western-Europeans ‘morons’ ruled by animalistic emotions who must be ‘carefully regulated’ by law and never allowed ‘to act upon their own initiative or their own judgment’ [Gould, 1981, p. 161]. Forensic measures duly found differences in brain structure seen as explaining the innate inferiority of ‘lower races.’

Biodeterminist theories benefited from two crucial errors in method: the ‘superior’ and ‘inferior’ populations were not compared under equivalent conditions, and any differences between the ‘superior’ and ‘inferior’ groups were interpreted to favor the former. Comparing new immigrants tested in English with English-fluent whites, or crime rates in poor chaotic districts with those in
established areas did not establish biological differences among populations even when multidisciplinary findings seemed to agree.

Today, scientists attribute even large statistical discrepancies between races, genders, and other adult populations to external factors rather than internal biologies. We do not pronounce Mississippians biologically inferior simply because for every race, both sexes, and all age levels, they suffer levels of traffic fatality rates five times higher, gun homicide rates four times higher, and other major risks substantially higher than residents of Massachusetts. Instead, we would note that Mississippians and Bay Staters display substantially different demographies, with the former suffering poverty levels nearly three times higher. Poverty is associated with risk of nearly every type [for elaboration on these points and sources, see Arnett, 2010; Males, 2010, 2011].

Given the history of biodeterminist claims, I suggest Moshman’s thesis of the adult-like nature of adolescent capabilities might be further tested by confronting an essential question: If adults and teens have equivalent competencies, how do we explain outcome statistics (for unintentional deaths, unplanned pregnancies, traffic crashes, criminal arrests, etc.) showing that teenagers as a class engage in fundamentally riskier behaviors than adults do?

This issue has been muddied by the failure of many ‘teenage brain’ commentators to assess risk statistics accurately. National Geographic’s more positive but still stereotype-laden commentary on the ‘typical teen’ declares, ‘the period from roughly 15 to 25 brings peaks in all sorts of risky ventures and ugly outcomes. This age group dies of accidents of almost every sort (other than work accidents) at high rates… In the U.S., one in three teen deaths is from car crashes, many involving alcohol’ [Dobbs, 2011]. This mix-and-match assessment of adolescent risks is rigged in ways unfortunately typical of ‘teenage brain’ discussion. First, including the more dangerous ages 20–25 (who are not ‘adolescents’ or ‘teens’ and should by the article’s premise display less risky behaviors) actually boosts the risks the author then attributes to ‘teens.’ Second, that accidents comprise a high proportion of teenage deaths doesn’t suggest much; teens rarely die from cancer, heart disease, or other causes that claim older adults.

Third, elementary fact-checking would have revealed that teenagers are actually among the least endangered by accidents. According to Centers for Disease Control [2011] tabulations for unintentional (accidental) deaths per 100,000 population, younger teens (8.8 for ages 12–15 in 2007) and older teens (34.3 for ages 16–19) have rates well below those of every older adult age group. In fact, middle-agers suffer higher accidental death rates (46.3 for ages 45–54) even than young adults (44.7 for ages 20–24) – including higher risks for two of the three leading accidental causes (poisonings and falls) as well as substantially higher rates of fatality for other major categories, including suicide and firearms deaths. Researchers seem to have ignored the three-
decades’ explosion in middle-aged drug abuse mortality (as well as criminal arrest and imprisonment), which, despite their higher economic status, now renders 40- and 50-agers the riskiest group for external injury. The argument that adult risks begin in adolescence is both one-sided – teenage risk levels uncannily parallel those of adults around them – and suggests a serious cognitive/developmental flaw in grownups: the inability to change unhealthy behaviors [Males, 2010, 2011]. In any case, cherry picking those risks for which teens excel while ignoring those rampant in adulthood is prejudice, not science.

But even if we single out only the selected risks for which adolescents and young adults show higher rates than older adults – chiefly traffic crashes, criminal arrest, and firearms homicide – the unexamined question remains: are these just manifestations of higher levels of teenage poverty? The poverty levels of adolescents and young adults (around 20% live in households with incomes below federal guidelines) average nearly double those of middle-aged adults (around 11%), a divergence that persists within every race and locale. Teen populations also have very different racial breakdowns (44% are Hispanic or other nonwhite) compared to older adults (28% for ages 45–64).

One would expect, then, that developmental analysts and risk assessment scientists would employ standard adjustments to control for demographic and economic variances before comparing teenage and adult outcomes. Yet, my own review and those of others I queried (including journal editors) found plenty of controlled studies comparing teenaged groups to other teenaged groups and adult groups to adult groups, but no studies comparing adolescent to adult risk outcomes that controlled for differing sociodemographics [Males, 2010]. I argue this fatal omission invalidates previous conclusions regarding ‘adolescent risk taking’ that are based on risk statistics. Until teens can be shown to display a consistently worse constellation of risk outcomes compared to older adults under equivalent conditions, the statistical basis for ‘adolescent risk taking’ collapses.

I tested this question in several preliminary studies, all finding that low socioeconomic status, not young age, is the main factor in what we define as ‘adolescent risk taking.’ For example, compared straight across, fatal traffic crashes per 100 million miles driven for California drivers aged 16–19 (50 traffic crashes) is nearly four times the rate of drivers aged 45–54 (14 traffic crashes), the safest adult age. However, young age was not the only factor. Compared to older adults, teens tended to drive cars that were considerably older and smaller in more impoverished, crowded environments – conditions that sharply boosted crash rates for adults as well [Males, 2009]. When compared at equivalent poverty levels, the fatal crash rates for 16- to 19-year-olds closely resembled those of 45- to 54-year-olds. Similarly, compared straight across, the violent crime arrest rate per 100,000 population for Californians aged 15–19 was more than triple that of
those aged 45–54, but at an equivalent poverty level, the violent crime arrest rate for those aged 15–19 was somewhat below that of those aged 45–54 [Males, 2010].

There are numerous complications involved in comparing risk outcomes by age, and more analysis is needed. Still, leveling the economic playing field when comparing teenage and adult risk outcomes further suggests that Moshman’s thesis that teens are fundamentally like adults remains valid when risk statistics are incorporated.

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References


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