

The Teenage Brain: Surging Hormones—Brain-Behavior Interactions During Puberty

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Abstract

In this paper, we discuss the surging hormones of puberty and their influences on adolescent behavior. We describe why these issues represent an interesting and important area of investigation, emphasizing their contributions to a specific set of developmental processes at the heart of the transition from childhood to adolescence. We briefly review the neuroendocrine underpinnings of human puberty. Our review focuses on evidence for behavioral (and neurobehavioral) effects of gonadal hormones and emphasizes the social and affective dimensions of these hormonal effects. More broadly, we consider how these hormonal events contribute to brain-behavior interactions that can bias early adolescent trajectories in both positive and negative directions, and in ways that may begin as small influences but can spiral into large-scale effects over time. These influences also appear to play an important role in functional and structural brain development during adolescence. Finally, we offer some thoughts on directions for future research in these areas.

Keywords

behavior, puberty, social-affective development, testosterone

The Initial Surge of Hormones Occurs Early

Although the title of this special issue highlights the *teenage* brain, it is important to recognize that the foundational events at the onset of human adolescence—the surge of hormones that starts the cascade of physical changes known as puberty—typically begin *before* the teenage years. In the United States, the average age of menarche (the onset of menstrual periods) in girls is 12.5 years of age. Moreover, menarche occurs near the final stages of puberty and is usually preceded by 2 to 3 years of hormonal and physical changes (including breast development, the development of pubic and axillary hair, rapid physical growth, and sexually dimorphic changes in facial structure). This surge of hormones, which marks the end of childhood and the onset of adolescence, typically begins by 9 to 10 years of age in girls and by 10 to 12 years of age in boys. Thus, the effects these hormones have on developing bodies and brains (and behavior) usually begin well before the teens.

It is important to acknowledge that some of the consequences of these rising levels of hormones can continue to grow in amplitude throughout the teenage years and may influence brain-behavior interactions for several years. However, in order to understand the proximal effects of this

surge of pubertal hormones on neurobehavioral systems, we should focus not on the teenage years but, rather, on this (usually) earlier window of development when there is a dramatic change in hormonal levels, from very low prepubertal levels to relatively high midpubertal levels.

Why study the effects of surging pubertal hormones?

Until recently, relatively few studies of adolescent brain development have focused on puberty and pubertal hormones. In fact, there has been little consistency in the field as to how “adolescence” is operationalized. This lack of consistency is reflected in the large variability in the age ranges used to define “adolescents” (from 10 to 25 years of age) and in the huge range of and variability in developmental processes that occur across this interval. A strong case can be made for the scientific advantages of posing well-specified questions about particular aspects of adolescent development.

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Focusing on pubertal maturation—and, more specifically, on the hormonal components of puberty—is one example of this approach. Moreover, there are at least four reasons to focus on this particular developmental process. First, puberty is *the* fundamental basis of the transition from childhood to adolescence. Second, the biological components of pubertal maturation provide targets for the development and testing of mechanistic hypotheses (e.g., about the effects of particular hormones on specific neural systems at specific points in development). Third, the focus on puberty creates unique opportunities for translational research with animal studies (e.g., it is difficult to specify what ages in mice, rats, or monkeys correspond to “teenage” years in humans, but sharp pubertal increases in testosterone and estradiol create direct parallels across species). Finally, there is growing evidence for shifts in social and affective processing during puberty that may play a crucial role in biasing developmental pathways in ways that can have enormous long-term effects on health, education, and well-being.

Why focus on social and affective influences of pubertal hormones?

As reviewed recently in Crone and Dahl (2012), growing evidence from studies in both humans and animals has suggested that, amid the myriad of social, emotional, cognitive, and biological changes during adolescence, pubertal hormones may influence (bias) some neural and neurobehavioral tendencies of social and affective processing. Examples of such neural and neurobehavioral correlates of social and affective processing include (but are not limited to) activity within the striatum during reward processing, activity within the amygdala and striatum during the processing of emotional stimuli, and activity within the anterior medial prefrontal cortex and temporal-parietal junction during social-cognitive reasoning (Crone & Dahl, 2012).

Changes in these brain areas and circuits appear to sensitize youth to their social world. A tendency to direct increased attention and motivation to social domains may have adaptive advantages in this developmental window. The fundamental task of adolescence—to achieve adult levels of social competence—requires a great deal of learning about the complexities of human social interactions. Puberty appears to create a neurobehavioral nudge toward exploring and engaging these social complexities. These tendencies to explore and engage can promote adaptive social and affective learning across adolescence; however, these same tendencies appear to create some vulnerabilities to negative developmental trajectories.

The goal of this paper is to focus on the proximal roots of these changes—the specific neurobehavioral effects that occur during the initial surge of pubertal hormones—and to consider how these effects contribute to a complex set of social and affective changes that can impact long-term developmental trajectories.

Neuroendocrine and Hormonal Aspects of Puberty

Puberty is an endocrinological event leading to sexual maturation. In the process of achieving reproductive maturity, this surge in hormones plays a central role within a larger set of biological changes, which include rapid physical growth; sexually dimorphic alterations in facial structure, voice, and body characteristics; metabolic changes; the activation of new drives and motivations; changes in sleep and circadian regulation; and a wide array of social, behavioral, and emotional changes.

The start of puberty is characterized by the reactivation of the hypothalamic-pituitary-gonadal (HPG) axis. The first step is when the hypothalamus begins to release large amounts of gonadotropin-releasing hormone (GnRH) in a pulsatile manner during sleep (Delemarre-van de Waal, 2002). The HPG axis is active during prenatal and early postnatal life and then becomes quiescent throughout childhood. Puberty actually represents a reactivation. The exact mechanisms that trigger this reactivation of pulsatile GnRH release at puberty remain unclear, but there has been rapid progress in understanding several key aspects (Navarro & Tena-Sempere, 2012).

The first outward signs of puberty are breast development in girls and genital development in boys. The development of these secondary sexual characteristics occurs, on average, at age 10 in girls and age 11.5 in boys (for a review, see Euling et al., 2008). In addition to the gonadal hormones testosterone and estradiol, the adrenal androgen dehydroepiandrosterone (DHEA) also plays a role in the development of secondary sexual characteristics, including pubic hair (girls and boys) and facial hair (boys; Delemarre-van de Waal, 2002). Changes in levels of growth hormone also contribute to pubertal growth and metabolic changes. In addition, other neuroendocrine systems also appear to contribute to neurobehavioral changes during puberty. For example, oxytocin and vasopressin, involved in social bonding, appear to undergo changes in adolescence in some species. However, there is a dearth of data on humans that directly addresses these influences, so most of this review will focus on gonadal hormones.

The development of reproductive capabilities involves not only changes in the body, but also changes in neural systems. The brain is a major target for sex-steroid hormones. Sex steroids act on the brain in two different ways: first, through *organizational effects* that permanently change the structure of the brain (e.g., neuronal number, myelination, dendritic branching), and second, through *activational effects* that temporarily change the activity of neural systems (e.g., the hormonal activation of neural systems that underpin mating behavior in animals after puberty; McCarthy & Arnold, 2011).

Until recently, it was believed that organizational effects of sex hormones took place only during prenatal and early postnatal life, and that puberty reflected only activational effects

of sex hormones. However, recent animal work has shown clear evidence of further organizational effects during puberty, such as the addition of new neurons to parts of the hypothalamus and amygdala (Ahmed et al., 2008). There is also evidence for brain-organizational effects of pubertal hormones in humans—for instance, decreases in global and focal gray matter and increases in white matter (Ladouceur, Peper, Crone, & Dahl, 2012; Peper, Hulshoff Pol, Crone, & van Honk, 2011). However, human studies have a limited capacity for disentangling the direct effects of hormones from indirect effects (e.g. what might appear to be “organizational” effects could be indirect structural changes resulting from new patterns of behavior correlated with puberty). Accordingly, we will focus on activational effects of pubertal hormones, reviewing briefly the evidence for how specific hormones appear to influence behavior.

Pubertal Hormones and (Neuro) Behavioral Changes During Adolescence

Testosterone

Increased levels of testosterone during adolescence have been associated with increased approach-related behaviors, such as proactive aggression (van Bokhoven et al., 2006) and risk taking (Vermeersch, T’Sjoen, Kaufman, & Vincke, 2008b) among boys and sensation seeking and sensitivity to rewards (Forbes et al., 2010) among both boys and girls.

With respect to brain activity, increased levels of testosterone in boys predict enhanced activation in the ventral striatum after high-risk gambles (Op de Macks et al., 2011). The ventral striatum is a subcortical brain region that is active when a person receives or expects a reward (Haber & Knutson, 2010). Therefore, one interpretation of these findings might be that heightened gonadal hormones “sensitize” the brain’s reward system, making adolescents more reactive to rewards in general. However, there is also evidence that social rewards are particularly important during this period of development, given that pubertal testosterone is a strong predictor of status-relevant motivation and behavior. In boys, higher levels of testosterone predict increased social aggression (controlling for age; Rowe, Maughan, Worthman, Costello, & Angold, 2004). The social environment plays an important role: The effect of testosterone on aggression was found only when individuals’ status was threatened (Josephs, Mehta, & Carre, 2011; Josephs, Sellers, Newman, & Mehta, 2006). Moreover, bullied girls were found to produce less testosterone and bullied boys to produce more testosterone than their nonbullied counterparts (Vaillancourt, deCatanzaro, Duku, & Muir, 2009). These findings demonstrate that the social environment not only mediates the effects of testosterone on behavior but also influences actual levels of testosterone itself.

Recent studies have also suggested that it is not simply high levels of testosterone that predispose adolescents to

these behaviors, but also interactions with other hormones or neurotransmitters (Montoya, Terburg, Bos, & van Honk, 2012). For example, relatively high levels of testosterone, together with relatively low levels of cortisol, have been implicated in delinquent behavior in adolescent boys (Popma et al., 2007; Yu & Shi, 2009).

Estradiol

The role of estradiol in motivated behavior in humans is less well studied than that of testosterone. In adolescent girls, a positive association was found between estradiol levels and (aggressive and nonaggressive) risk taking, controlling for the effects of age (Vermeersch, T’Sjoen, Kaufman, & Vincke, 2008a). The effects varied across the menstrual cycle: The association between estradiol and risk-taking was strongest during the mid-luteal phase, when estradiol levels are relatively high. Interestingly, during the proestrus phase of the menstrual cycle, when estradiol levels are also high, female rats exhibited attenuated inhibition compared with rats with low estradiol levels (Quinlan, Duncan, Loiselle, Graffe, & Brake, 2010). Moreover, this effect was not seen until puberty, which indicates that it is dependent on the surge of hormones at puberty (Quinlan et al., 2010). Taken together, these data suggest that increased pubertal estradiol in females is related to lessened behavioral inhibition and increased risk taking. Moreover, with respect to the relation between brain activity and risk-taking, it was found that higher estradiol levels in girls predict stronger activity within the ventral striatum after high-risk gambles, although this effect of estradiol was somewhat weaker than that of testosterone in boys (Op de Macks et al., 2011).

This emphasis on social and affective influences is not intended to imply that these are the only neurobehavioral effects of pubertal hormones. Clearly, some aspects of cognitive (and social-cognitive) function are impacted by pubertal hormones. For example, there is evidence that estrogen shapes dopamine-dependent cognitive processes (Jacobs & D’Esposito, 2011), as well as recent findings that pubertal hormones (testosterone, estradiol, and DHEA) directly influence brain activity within the anterior temporal lobe during social-emotional processing (Goddings, Heyes, Bird, Viner, & Blakemore, 2012). This is quite interesting given that the anterior temporal lobe has been implicated in the processing of social emotions such as guilt and embarrassment (Zahn et al., 2007).

Pubertal hormones and models of brain-behavior interactions

Recently, it has been proposed that pubertal maturation impacts social and affective processing in ways that contribute to an adolescent flexibility in cognitive engagement according to the social and motivational salience of the context (Crone & Dahl,

2012). The model suggests that interactions between social-affective processing systems in the brain and cognitive-control systems can lead to healthy adaptation to the complex and rapidly changing social contexts of adolescence. However, these interactions can also lead to negative trajectories, such as substance abuse or depression. Such negative trajectories may begin as small changes but, over time, can lead to patterns of behavior that have cascading effects: brain-behavior interactions with spiraling impact across adolescence.

Pubertal changes in sleep as an illustrative example

As a more in-depth illustration of how a small pubertal change in one neurobehavioral system can spiral into large-scale complex behavioral consequences, let us consider the powerful example of sleep and circadian changes during adolescent development. As has been observed in several species (Hagenauer, Perryman, Lee, & Carskadon, 2009), pubertal maturation is associated with a small shift in the tendency to prefer sleeping later in the circadian cycle. Elegant work in mice has recently shown that gonadal hormones influence the way the suprachiasmatic nucleus (the biological master clock in the hypothalamus) responds to light (Karatsoreos, Butler, Lesauter, & Silver, 2011).

In humans, there is evidence for a similar circadian shift in sleep preferences at puberty. The direct effect of these pubertal changes appears to be relatively subtle—for example, a typical midpubertal 12-year-old may experience slight (biological) tendencies to prefer staying up later and to sleep in later on weekends. Moreover, for most of human history (until the advent of electric lights), this slight tendency to prefer staying up later at night was unlikely to result in large, sustained effects on behavior because the prevailing darkness at night presented adolescents with limited opportunities to activate their hypothalamus with the type of light signals that create large-scale biological shifts in the circadian clock. However, in contemporary society, not only do youths have access to bright lights at any hour of the night, but it also appears that blue spectrum light from TV, computer, and personal-device screens may have particularly strong effects on the human circadian system. Even more importantly, several other social and motivational factors increase the likelihood that many adolescents will use these arousing light-signaling devices later into the night (Carskadon, 2011).

Together, this combination of social and behavioral factors has contributed to a situation in the United States where the average school-night bedtime among high school seniors is after 11:30 p.m. (despite the fact that the average wake-up time on school days for these students is 6:15 a.m.). The resulting chronic sleep deprivation (and catch-up sleep on weekends resulting in very late schedules) further amplifies the spiral of negative effects. It is currently estimated that up to 30% of U.S. high school students are chronically sleep

deprived as a result of these influences. Insufficient sleep has a cascade of negative effects on other aspects of emotion, behavior, cognition, and learning, which can, in turn, further interfere with the regulation of sleep and arousal (Carskadon, 2011).

The main point here is to illustrate how a relatively small pubertal change in particular neural systems—in this case, an increased circadian sensitivity to the environment (light and social cues)—can become amplified by multiple social factors across adolescence. Thus, while the direct effects of the initial increase in pubertal hormones may appear relatively small, the longer-term effects on brain-behavior interactions can become large-scale effects over time. This framework helps us understand why specific details about hormonal effects on neurobehavioral tendencies represent important scientific questions.

If we extend this example to consider the impact of a small pubertal increase in the motivational salience of social status, we can imagine a similarly complex cascade of changes (as with sleep patterns) interacting with complex contemporary social contexts. Most importantly, it highlights the power of social and cultural influences (e.g., the particular kinds of behaviors that bring increased attention and admiration early in the process), as well as the impact of early social failures. Clearly, a great deal more research is needed to empirically test features of this model—and to advance understanding of the specific hormonal mechanisms (and specific neural systems) that underlie these changes.

Conclusions and Important Areas for Future Research

Our most important conclusion is that more studies of adolescent brain development need to focus on puberty-specific and hormone-specific developmental processes. Studying puberty-specific effects on developmental processes requires designing studies to prioritize these questions—by focusing on the early ages of onset, using longitudinal designs, and obtaining appropriate measures of pubertal development (Shirtcliff, Dahl, & Pollak, 2009). In addition, it requires further development and refinement of heuristic models (and testing of key features of these models) to advance our understanding of how specific hormones impact specific neural systems to influence specific behavioral tendencies. Another important consideration entails recognizing that the biological signals created by a given hormone can sometimes have little to do with the *level* of the hormone in the blood or saliva, as opposed to the timing or pattern of hormone secretion. For example, it is well established that the fundamental trigger for the onset of puberty is the pulse frequency of gonadotropin-releasing hormone (rather than the level of the hormone).

Other priorities for future research include considering (and investigating) a broad range of hormones that change during puberty. These include not only changes in estradiol,

testosterone, and the adrenal androgens DHEA and DHEA sulphate but also changes in other neuroendocrine systems that appear to undergo developmental changes at puberty, such as growth-hormone regulation and hypothalamic-pituitary-adrenal-axis regulation. One particularly interesting line of investigation focuses on the oxytocin-vasopressin system that has been shown to be central to social-bonding motivation and behaviors and to influence social cognitive processes such as trust (which it does differently in in-group and out-group contexts). Given the dynamic social changes in adolescence—establishment of close friendships, intense motivations, and emotions associated with early sexual and romantic relationships, as well as changes in family relationships—it would be surprising if pubertal changes in these oxytocin and vasopressin systems were not intertwined with some of these developmental changes. There are some animal data showing pubertal changes in oxytocin, but translational work with human subjects has been limited (for relevant reviews, see Carter, 2003; Gordon, Martin, Feldman, & Leckman, 2011).

To conclude, our review demonstrates that pubertal hormones contribute to brain-behavior interactions. At least in some cases, these effects appear to be manifested as action tendencies, or behavioral tendencies, which can appear in some ways to have relatively subtle effects. Yet even small changes in behavioral tendencies (e.g., the biological shift in the tendency to go to sleep later) can, in some contexts, over time, lead to high-impact changes in patterns of behavior. There is growing evidence that these hormone-driven tendencies can impact long-term developmental trajectories in both positive and negative ways. Achieving a better understanding of these important developmental processes represents an exciting and pioneering area for transdisciplinary research.

Recommended Reading

- Carskadon, M. A. (2011). (See References). A comprehensive overview of what is known about sleep during pubertal transition and adolescent development.
- Josephs, R. A., Mehta, P. H., & Carre, J. M. (2011). (See References). A review that discusses socially and environmentally modulated effects on (pubertal) testosterone.
- Shirtcliff, E. A., Dahl, R. E., & Pollak, S. D. (2009). (See References). A thorough methodological paper that discusses various ways to quantify pubertal development in humans, each with its pros and cons.

Declaration of Conflicting Interests

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