Development and Psychopathology 21 (2009), 1–6 Copyright © 2009 Cambridge University Press Printed in the United States of America doi:10.1017/S0954579409000017

Special Section
Heightened Stress Responsiveness and Emotional
Reactivity During Pubertal Maturation: Implications
for Psychopathology

SPECIAL SECTION EDITORIAL

Heightened stress responsiveness and emotional reactivity during pubertal maturation: Implications for psychopathology

RONALD E. DAHL^a AND MEGAN R. GUNNAR^b

^aUniversity of Pittsburgh; and ^bUniversity of Minnesota

Abstract

The onset of adolescence, and more specifically the advent of pubertal maturation, represents a key developmental window for understanding the emergence of psychopathology in youth. The papers in this special section examine normative differences in the neurobiology of stress and emotional functioning over the peripubertal period. The work in this special section helps to fill in gaps in our understanding of key mechanisms that may contribute to increased vulnerabilities in behavioral and psychiatric morbidity during this developmental period.

The onset of adolescence, and more specifically the advent of pubertal maturation, represents a key developmental window for understanding the emergence of psychopathology in youth. The beginning of adolescence is a time of dramatic physical, social, cognitive, and emotional changes. These include rapid physical growth; the onset of sexual maturation; increases in risk taking and sensation seeking; burgeoning romantic and sexual interests; increased self-

This work was supported by the Canadian Institute of Advanced Research Program on Experienced-Based Brain Development fellowship (to M.R.G.) and R24 MH067346 and R01 DA018910 (to R.E.D.).

Address correspondence and reprint requests to: Megan R. Gunnar, Institute of Child Development, University of Minnesota, 51 East River Road, Minneapolis, MN 55455; E-mail: gunnar@umn.edu.

consciousness; early experimentation with alcohol and other substance use; and increasing rates of accidents, suicide, reckless behavior, depression, anxiety, and bipolar disorders. Not only do morbidity and mortality rates rise sharply across this developmental interval, but also the roots of many adult-onset health problems can be traced to patterns of behavior that can be identified in adolescence. Clearly, the period of transition into adolescence reveals complex issues that have enormous implications for public health.

The scope of these problem behaviors that emerge at this time spans the boundaries of normal and pathological development and interweaves elements of physical health, behavioral health, and developmental psychopathology. For example, a tendency toward increased risk taking and sensation seeking may, on the one hand, represent a set of normative developmental changes in adolescence. On the other hand, these maturational shifts in motivation also contribute to early pathways for a wide array of risky behaviors with significant health consequences such as smoking, substance use, reckless behavior, and increased vulnerabilities to some psychiatric disorders. Another dimension to the complexity of these issues is the increasing recognition of the need to understand the interplay between biological and social factors that can tip the balance at key points along trajectories toward pathological outcomes such as adolescent depression, anxiety, bipolar disorder, substance use disorders, eating disorders, suicide, aggression, and the high rates of fatal accidents that occur during this period of development.

Progress in understanding the developmental roots of these problems, and ultimately, in identifying optimal leverage points for early intervention, will require slicing through these levels of complexity with well-focused scientific investigations. Some of the most compelling questions aim at mechanistic understanding. For example, there is increasing recognition of the importance of Gene × Environment × *Development* interactions and their relevance to adolescent vulnerabilities (e.g., Caspi et al., 2005).

However, as illustrated in this special section, there are equally compelling issues (with respect to long-term clinical and social policy relevance) focusing on questions about specific developmental windows that may render particular neurobehavioral systems relatively more vulnerable. More specifically, as highlighted by the papers in this special section, there appears to be emerging evidence for increased emotional and stress reactivity in early adolescence, which may have roots in neurobehavioral changes at puberty. Although only two of the papers tested the role of pubertal maturation directly, all four papers focus on the developmental interval when pubertal maturation is accelerating (and in one case the data show that girls demonstrate the increased reactivity on an earlier time course than boys, consistent with the hypothesis that puberty-specific changes may underpin at least some aspects of these alterations in emotional response).

Why, one might ask, is this particularly relevant to understanding the development of psychopathology during adolescence? As noted by several theorists, the onset of puberty appears to present a crucial transition point that heralds the onset of increased risks in adolescence. Although psychosocial factors associated with adolescence continue to receive attention as components of proposed adolescent vulnerability, clinical investigators are increasingly focusing attention on puberty-associated neurobiological maturational changes in systems underlying stress and emotional functioning. This journal has been active in publishing theoretical and empirical articles encouraging this focus (e.g., Alloy, Abramson, Walshaw, Keyser, & Gerstein, 2006; Keshavan & Hogarty, 1999; Walker, Sabuwalla, & Huot, 2004).

Notably, although these models are based on the idea that *normative* neurobiological changes impact systems that underlie stress and emotion functioning, the evidence that such changes are observed *normatively* is relatively sparse.

Models of normal adolescent brain development increasingly emphasize interactions between neural systems underpinning cognitive control functions and those that are more reactive to social and affectively salient stimuli. This provides a general framework for considering the dramatic changes in social and emotional domains in early adolescence, which place new demands on the still-maturing neural systems that are critical for mediating arousal, orientation, attention, and affect, as well as for regulating and integrating these affects in line with long-term, goal-directed behaviors (the maturation of cognitive control; see Dahl, 2004; Ernst, Pine, & Hardin, 2006; Nelson, Leibenluft, McClure, & Pine, 2005; Steinberg et al., 2006).

Yet, one of the core hypotheses, pubertyspecific changes in affective systems, has received surprisingly little empirical investigation. As highlighted by the papers in this special section, this is beginning to change.

The four studies in this special section provide data that begin to fill key gaps in the literature. Each examines peripubertal changes in neurobiological functioning. The focus of these studies is on changes in emotion and stress systems.

The first paper by Silk et al. (2009) examines pupillary and behavioral response to emotional

words in a sample of youth in two stages of puberty: pre- to early and mid- to late. The pupil becomes more dilated in response to stimuli that require greater cognitive load or that have greater emotional intensity reflecting activity of corticolimbic circuits involving the anterior cingulate cortex, amygdala, and midbrain reticular formation. Pupil dilation thus provides a peripheral index of brain activation in response to a specific stimulus. Silk et al. (2009) demonstrate that, controlling for participants' age, mid-/late pubertal children evidence a greater peak pupillary reactivity to these words than do pre-/early pubertal children. The mid-/late pubertal subjects also report greater emotionality during the laboratory visit and remember more emotion words during free recall.

The second paper by Quevedo, Benning, Gunnar, and Dahl (2009) examines affective influences on two reflexes: eye-blink startle and postauricular (PA) reflex as measures of the defensive and appetitive motivational systems, respectively. The design of the study (focusing on youth that were the same age, but varied on pubertal maturation) is particularly important as a way to disentangle puberty-specific maturational changes. They examined emotion modulation (pleasant, neutral, and aversive pictures) of these reflexes in 12- and 13-year-old adolescents who were selected to be pre-/early or mid-/late pubertal. Compared to pre-/early pubertal children of the same age, mid-/late pubertal adolescents exhibited higher amplitude eye-blink startles and emotion modulation of the PA reflex. Furthermore, only among the mid-/late pubertal subjects were individual differences in emotion modulation of the appetitive reflex associated with self-reports of behavioral and personality characteristics.

The third and fourth papers by Stroud et al. (2009) and Gunnar, Wewerka, Frenn, Long, and Griggs (2009), respectively, focus on whether the reactivity of stress sensitive neurobiological systems increase over the transition to adolescence. This question is critical because it has been argued that heightened risk for psychopathology in adolescence not only involves heightened emotionality, but also heightened stress responses to emotional provocation. Although previous work in this area has primarily focused on basal activity of stress-sensitive systems, both

Stroud and colleagues (2009) and Gunnar and colleagues (2009) expose participants to stressevocative tasks to examine normative changes in stress reactivity at the transition to adolescence. Stroud et al. (2009) demonstrate increases in activity of the hypothalamic-pituitary-adrenal (HPA) axis to a performance stressor and increases in sympathetic reactivity to a social rejection stressor among older (13- to 17-yearolds) versus younger (7- to 12-year-olds) participants. Similarly, Gunnar and colleagues (2009), using a performance stressor (Trier Social Stress Test), found significant changes in HPA reactivity around the transition to adolescence, and further showed that pubertal stage correlated with increased HPA activity. In contrast to Stroud and colleagues (2009), however, they found that sympathetic activity, measured using the preejection period, was increasingly modulated with age.

3

On the one hand, there are several limitations to these studies and clearly a need for further well-controlled investigations in these areas. More rigorous measures of puberty, measures of reproductive hormones, designs that specifically disentangle puberty and age (such as in Quevedo et al., 2009), and importantly, longitudinal studies, will be needed to advance understanding in these important areas. Studies of high-risk samples followed through this maturational interval as well as low-risk normal controls would also increase the ability to answer a deeper set of clinically relevant questions. On the other hand, we believe that taken together, this body of work has broad significance, pointing toward developmental changes in stress responsiveness and emotional reactivity that may have puberty-specific neurobehavioral underpinnings. Because these changes were observed in healthy, low-risk children they provide a basis for models positing that puberty-specific changes in these systems may heighten the risk for psychopathology in vulnerable youth.

It is also important to consider these papers within a broader context of related findings indicating peripubertal changes in social/affective systems. For example, there are relevant advances investigating adolescent increases in sensation seeking, which appears to be linked to pubertal maturation. As Steinberg et al. (2008) have described, a deeper understanding

of adolescent risk taking may require separating two overlapping and interacting domains: sensation seeking versus impulsivity, which are sometimes confounded (conceptually methodologically). A key dimension of sensation seeking centers on an affective or appetitive domain; it is a bit like a hunger or desire to experience novel, exciting, arousing experiences, even if these entail some risks. In contrast, impulsivity has a primarily cognitive dimension, related to the ability to inhibit and delay action through cognitive control. In many risk-taking situations (and measures) these two overlap. Yet, some sensation seeking is not impulsive (e.g., an adolescent who saves up money to take hang-gliding lessons or goes on a rock-climbing trip is being quite planful in her pursuit of thrilling sensations). Similarly, a great deal of impulsive behavior is not aimed at achieving excitement (e.g., impulsively turning off the alarm clock in the morning and falling back asleep). Most relevant to the discussion here, these two follow different developmental trajectories. It appears that sensation seeking increases during early adolescence (with some evidence that this occurs in direct association with onset of puberty (Martin et al., 2002; Steinberg et al., 2008) with a peak in midadolescence, whereas impulsivity follows a relatively linear decline from ages 10 through 30 (Steinberg et al., 2008). This provides another example where delineating a deeper understanding of the puberty-specific changes (and the underlying neural mechanisms of the normal developmental changes in relevant affective and motivational systems) can inform prevention and early intervention strategies for high-stakes issues such as adolescent risk taking and reckless behavior, which contributes to high rates of mortality.

A second specific domain within the framework of developmental social/affective neuroscience where there is emerging evidence of puberty-specific maturational changes in face processing (Blakemore, 2008; McGivern, Andersen, Byrd, Mutter, & Reilly, 2002). Children's recognition memory for unfamiliar faces steadily improves from about ages 5 to 10, exhibits a temporary decline in accuracy from about 11 to 12, and recovers to adult levels by about 16 years of age (Carey, Diamond, & Woods, 1980). These

results have been interpreted as evidence for a transition in the prominent information-processing strategy used to encode and remember faces that may be tied to changes in lateralization in the brain during puberty. More recently, brain imaging studies investigating the functional organization of face-related activation in children and adolescents have been converging on the finding that such organization only begins to look adultlike in middle adolescence. For example, most functional magnetic resonance imaging (fMRI) studies of face processing indicate that the fusiform face area is still undergoing significant maturational changes in early adolescence (Aylward et al., 2005; Gathers, Bhatt, Corbly, Farley, & Joseph, 2004; Passarotti et al., 2003; Scherf, Behrmann, Humphreys, & Luna, 2007). There also are several studies indicating developmental changes during adolescence that involve the broader face-processing networks that include anterior temporal, prefrontal, and limbic regions such as the amygdala (Baird et al., 1999; McClure et al., 2004; Nelson et al., 2003; Thomas et al., 2001).

Another example of social/affective changes linked to puberty is the strong evidence from both animal and human studies that supports a link between increasing levels of reproductive hormones and sensitivity to social status (Archer, in press; Josephs, Sellers, Newman, & Mehta, 2006; Mazur & Booth, 1998). These data also fit with some anthropological perspectives on risk taking in adolescence, which can be viewed as an adaptive willingness to demonstrate bravery to acquire more social status. Consider, for example, an adolescent growing up in a hunter-gatherer society who must make his first successful kill of a dangerous animal with a spear to attain adult status in his society, in this case, a willingness to act boldly despite fear and relative lack of skills and knowledge can be a risky, but necessary, step to succeed. Indeed, in many situations, it seems that adolescents do *not* become more fearless after puberty (e.g., in the Quevedo et al., 2009, paper fear/startle responses *increased* at puberty), but rather it may be that adolescents become more highly motivated to act boldly despite their feelings of fear. This may assist youth along the path of learning to master their fears (and at times, to transform these fears into "thrills"), particularly when they perceive that acting in a brave or even reckless way might bring them increased recognition by peers or more social status. These findings support the idea that adolescents may be more prone to peer and status-sensitive influences on risky decision making and that peer influences and other social context variables may play an important role in explaining risky behavior during adolescence as described by Steinberg (2004).

Finally, one crucial line of investigation that is progressing rapidly is work focusing on maturational changes in reward-seeking during adolescence, including both animal studies as well as human studies including use of fMRI and event-related potential to examine neural systems of reward in adolescence (Ernst et al., 2005; Eshel, Nelson, Blair, Pine, & Ernst, 2007; May et al., 2004; van Leijenhorst, Crone, & Bunge, 2006). Currently, these studies converge on one point: all have found differences in activation of relevant circuitry between adolescents and adults. However, the studies diverge regarding the direction of these differences and in the

specific neural systems involved. Most relevant to the focus of this special issue, there are studies underway looking at puberty specific changes in neural systems of reward in youth who are matched for age and grade level but vary on pubertal maturation (see abstract by Forbes et al., 2008). It seems likely that this line of studies will contribute to understanding several aspects of these maturational changes.

5

In summary, the papers (and commentary by Spear) in this Special Section represent some key preliminary steps in a very important direction: providing initial evidence in support of the theory that early adolescence (and probably some puberty-specific changes in affective neural systems) contribute to increased reactivity and responsiveness to social and emotional stimuli. If these findings are replicated and extended, they may provide evidence that early adolescence is key period of vulnerability for the emergence of social and emotional changes leading to serious pathology; yet they may also point to opportunities for early intervention.

References

- Alloy, L. B., Abramson, L. Y., Walshaw, P. D., Keyser, J., & Gerstein, R. K. (2006). A cognitive vulnerability– stress perspective on bipolar spectrum disorders in a normative adolescent brain cognitive and emotional development context. *Development and Psychopathology*, 18, 1055–1103.
- Archer, J. (in press). Testosterone and human behavior: An evaluation of the challenge hypothesis. *Neuroscience and Biobehavioral Reviews*.
- Aylward, E. H., Park, J. E., Field, K. M., Parsons, A. C., Richards, T. L., Cramer, S. C., et al. (2005). Brain activation during face perception: Evidence of a developmental change. *Journal of Cognitive Neuro*science, 17, 308–319.
- Baird, A. A., Gruber, S. A. Fain, D. A., Maas, L. C., Steingard, R. J., Renshaw, P. F., et al. (1999). Functional magnetic resonance imaging of facial affect recognition in children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, 38, 195–199.
- Blakemore, S. J. (2008). The social brain in adolescence. *Nature Reviews Neuroscience*, *9*, 267–277.
- Carey, S., Diamond, R., & Woods, B (1980). The development of face recognition—A maturational component. Developmental Psychology, 16, 257–269.
- Caspi, A., Moffit, T. E., Cannon, M., McClay, J., Murray, R., Harrington, H., et al. (2005). Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-Omethyltransferase gene: Longitudinal evidence of a Gene × Environment interaction. Biological Psychiatry, 57, 1117–1127.

- Dahl, R. (2004). Adolescent brain development: A period of opportunities and vulnerabilities. Annals of the New York Academy of Science, 1021, 1–22.
- Ernst, M., Nelson, E. E., Jazbec, S., McClure, E.B., Monk, C.S., Leibenluft, E., et al. (2005). Amygdala and nucleus accumbens in responses to receipt and omission of gains in adults and adolescents. *NeuroImage*, 25, 1279–1291.
- Ernst, M., Pine, D. S., & Hardin, M. (2006). Triadic model of the neurobiology of motivated behavior in adolescence. *Psychological Medicine*, 36, 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. *Neuropsycholo*gia, 45, 1270–1279.
- Forbes, E. E., Hariri, A. R., Fiez, J. A., Ryan, N. D., Manuck, S. B., Brown, S. A., et al. (2008). Development of reward-related brain function from childhood to adulthood. Abstract presented at the Annual Meeting of the Society of Biological Psychiatry, Washington, DC.
- Gathers, A. D., Bhatt, R., Corbly, C. R., Farley, A. B., & Joseph, J. E. (2004). Developmental shifts in cortical loci for face and object recognition. *NeuroReport*, 19, 1549–1553.
- Gunnar, M. R., Wewerka, S., Frenn, K., Long, J. D., & Griggs, C. (2009). Developmental changes in hypothalamus–pituitary–adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology*, 21, 69–85.

- Josephs, R. A., Sellers, J. G., Newman, M. L., & Mehta, P. H. (2006). The mismatch effect: When testosterone and status are at odds. *Journal of Personality and Social Psychology*, 90, 99–1013.
- Keshavan, M. S., & Hogarty, G. E. (1999). Brain maturational processes and delayed onset in schizophrenia. Development and Psychopathology, 11, 525–543.
- Martin, C. A., Kelly, T. H. Rayens, M. K., Grogli, B. R., Brenzel, A., Smith, W. J., et al. (2002). Sensation seeking, puberty, and nicotine, alcohol, and marijuana use in adolescence. *Journal of the American Academy of Child* & Adolescent Psychiatry, 41, 1495–1502.
- May, J. C., Delgado, M. R., Dahl, R. E., Stenger, V. A., Ryan, N. D., Fiez, J. A., et al. (2004). Event-related functional magnetic resonance imaging of rewardrelated brain circuitry in children and adolescents. *Biological Psychiatry*, 55, 359–366.
- Mazur, A., & Booth, A. (1998). Testosterone and dominance in men. *Behavioral & Brain Sciences*, 21, 353–397.
- McClure, E. B., Monk, C. S., Nelson, E. E., Zarahn, E., Leibenluft, E., Bilder, R. M., et al. (2004). A developmental examination of gender differences in brain engagement during evaluation of threat. *Biological Psychiatry*, 55, 1047–1055.
- McGivern, R. F., Andersen, J., Byrd, D., Mutter, K. L., & Reilly, J. (2002). Cognitive efficiency on a match to sample task decreases at the onset of puberty in children. *Brain and Cognition*, 50, 73–89.
- 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, 163–174.
- Nelson, E. G., McClure, E. B., Monk, C. S., Zarahn, E., Leibenluft, E., Pine, D. S., et al. (2003). Developmental differences in neuronal engagement during implicit encoding of emotional faces: An event-related fMRI study. *Journal of Child Psychology and Psychiatry*, 44, 1015–1024.
- Passarotti, A. M., Paul, B. M., Bussiere, J. R., Buxton, R. B., Wong, E. C., & Stiles, J. (2003). The development of face and location processing: An fMRI study. *Developmental Science*, 6, 100–117.

- Quevedo, K. M., Benning, S. D., Gunnar, M. R., & Dahl, R. E. (2009). The onset of puberty: Effects on the psychophysiology of defensive and appetitive motivation. *Development and Psychopathology*, 21, 27–45.
- Scherf, K. S., Behrmann, M., Humphreys, K., & Luna, B. (2007). Visual category—Selectivity for faces, places, and objects emerges along different developmental trajectories. *Developmental Science*, 10, F15–F30.
- Silk, J. S., Siegle, G. J., Whalen, D. J., Ostapenko, L. J., Ladouceur, C. D., & Dahl, R. E. (2009). Pubertal changes in emotional information processing: Pupillary, behavioral, and subjective evidence during emotional word identification. *Development and Psychopa*thology, 21, 7–26.
- Steinberg, L. (2004). Risk taking in adolescence: What changes, and why? Annals of the New York Academy of Science, 1021, 51–58.
- Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. *Developmental Psychology*, 44, 1764–1778.
- Steinberg, L., Dahl, R., Keating, D., Kupfer, D., Masten, A., & Pine, D. (2006). Psychopathology in adolescence: Integrating affective neuroscience with the study of context. In D. Cicchetti & D. Cohen (Eds.), Developmental psychopathology: Vol. 2. Developmental neuroscience (pp. 710–741). New York: Wiley.
- Stroud, L. R., Foster, E., Papandonatos, G. D., Handwerger, K., Granger, D. A., Kivlighan, K. T., et al. (2009). Stress response and the adolescent transition: Performance versus peer rejection stress. *Development and Psychopathology*, 21, 47–68.
- Thomas, K. M., Drevets, W. C., Whalen, P. J., Eccard, C. H., Dahl, R. E., Ryan, N. D., et al. (2001). Amygdala response to facial expressions in children and adults. *Biology Psychiatry*, 49, 309–316.
- van Leijenhorst, L., Crone, E. A., & Bunge, S. A. (2006). Neural correlates of developmental differences in risk estimation and feedback processing. *Neuropsychologia*, 44, 2158–2170.
- Walker, E. F., Sabuwalla, Z., & Huot, R. (2004). Pubertal neuromaturation, stress sensitivity, and psychopathology. *Development and Psychopathology*, 16, 807–824.