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In Search of Explanations for Early Pubertal Timing Effects on Developmental Psychopathology

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ABSTRACT—Early pubertal maturation has been identified as a potential risk factor for internalizing and externalizing problems during adolescence. However, questions about the mechanisms that link early pubertal timing and psychopathology remain. In this article, we describe four hypotheses that explain the effects of early pubertal maturation. The hormonal influence hypothesis predicts that an increase in hormones at puberty leads to increased psychopathology. The maturation disparity hypothesis focuses on the gap between physical, social, and psychological maturation in early maturers that exacts the toll on individuals' adjustment. The contextual amplification hypothesis proposes that experiencing early pubertal transition in a disadvantaged context increases the risk for psychopathology. Finally, the accentuation hypothesis maintains that preadolescent vulnerabilities and challenges during early pubertal transition together increase problems. This article concludes with a consideration of how these hypotheses individually and collectively generate new lines of research linking early pubertal maturation and psychopathology.

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Researchers have accumulated substantial evidence that early pubertal maturation constitutes a significant risk factor for psychopathology: Adolescents who undergo pubertal maturation earlier than their same-age, same-sex peers are more likely to have a number of detrimental outcomes, including problem behaviors, substance use, and emotional distress in adolescence (Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997) and adulthood (Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004). For instance, Figure 1 illustrates that over the course of early to mid adolescence, girls whose menarche occurred before age 12.5 (i.e., earlier maturers) tended to have increasingly higher levels of psychological distress than did girls who had their first periods between ages 12.5 and 13.5 (i.e., on-time maturers) or those whose first periods came after age 13.5 (i.e., later maturers; Ge, Conger, & Elder, 1996). Although scholars have converged in recognizing the significant roles played by pubertal timing in the developmental risks for psychopathology, they diverge on how to best explain this pubertal timing effect. In what follows, we describe four emerging lines of thinking for explaining why early puberty exerts its influences on externalizing and internalizing psychopathologies. These models include the hormonal influence, maturation disparity, contextual amplification, and accentuation hypotheses. The focus of this report is the effect of *pubertal timing* (i.e., timing at which an individual undergoes puberty), rather than pubertal transition (i.e., physical maturation during puberty) per se.

THE HORMONAL INFLUENCE HYPOTHESIS

The rising trajectories of externalizing and internalizing psychopathologies at puberty make a hormonal explanation intuitively appealing. Several researchers maintain that the rise in the adrenal and gonadal hormones at puberty increases risks for developing psychopathologies (e.g., Angold, Costello, Erkanli, & Worthman, 1999).

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Two components of puberty, *adrenarche* and *gonadarche*, are important in the study of hormonal influence on risk for psychopathology. Adrenarche, which typically occurs between ages 6 and 9, refers to the maturation of the hypothalamic-pituitary-adrenal (HPA) axis; in this period, adrenal androgens (e.g., dehydroepiandrosterone [DHEA] and its sulfate [DHEAS]) begin to rise. There is some evidence to suggest that adrenal androgens are related to dominance, depression, and antisocial conduct (Angold et al., 1999). Gonadarche, which begins at approximately ages 9 to 11, involves the maturation of the hypothalamic-pituitary-gonadal (HPG) axis. Hormones of the HPG axis, gonadotropins (i.e., follicle stimulating hormone [FSH] and luteinizing hormone [LH]) and sex steroids (i.e., estradiol, progesterone, and testosterone), increase rapidly during the pubertal transition (Nottelmann et al., 1987). Although the results are somewhat mixed, individual differences in concentration in testosterone and estradiol are related to negative affect, behavior problems, and aggressive tendencies (Angold et al., 1999).

The mechanisms by which hormonal secretions in puberty affect psychopathology are undoubtedly highly complex. Several potential pathways have been discussed. One possible pathway that has been well-studied in animal models is pubertal hormones, particularly gonadal hormones, organizing neural circuits in the developing adolescent brain and leading to behavioral consequences (Sisk & Zehr, 2005). Another related possibility is that pubertal hormones are linked to psychopathology via alterations in stress sensitivity. Heightened stress reactivity during puberty has been demonstrated in recent studies examining increases in cortisol secretion (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009) and pupillary dilation reflecting brain activation (Silk et al., 2009). Finally, another emerging idea is that social and environmental factors may mediate the effects of pubertal hormones on behavior (Schultz, Molenda-Figueira, & Sisk, 2009). Physical

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changes at puberty, which are the external manifestation of underlying endocrine developments, call forth actual or perceived social reactions, which in turn are associated with psychopathology.

Hormonal alterations occur regardless of when puberty occurs. What is less clear, however, is what happens to behaviors if the timing of hormonal increases is accelerated and secretions are initiated earlier. One theoretical possibility is that the developing brain's sensitivity to pubertal hormones decreases with time, marking early maturation as a potential correlate of behavior (Schultz et al., 2009). Interesting experimental evidence indicated that the effect of testosterone treatments in castrated male hamsters most effectively facilitated mating behaviors when the treatments were given early in pubertal transition (Schultz et al., 2009). Another possibility is that earlier maturers not only begin secreting puberty-related hormones earlier but also secrete more of the hormones even after completing pubertal transition. For instance, girls diagnosed with premature puberche (the appearance of pubic hair before age 8) had higher levels of adrenal androgens from the early stages of puberty to after puberty (Ibanez, Street, Potau, Carrascosa, & Zampolli, 1997). Further examination is required to answer the question of whether and how early increases in pubertal hormones detrimentally affect adolescent mental health.

Although it is intuitively appealing to directly ascribe the rise of psychopathology at puberty to a surge of hormonal activities, the empirical findings for such a link in humans are fragmented and equivocal. Verifying a direct link requires a rigorous demonstration that puberty-related hormonal changes precipitate the increase in externalizing and internalizing psychopathologies. No existing empirical data we are aware of have systematically documented an "interlocking" dynamic between changes in hormonal activity and increases in psychopathology over time. Studies that chart hormonal changes and maladaptive developmental courses, track their progression, and trace their associated changes over time are urgently needed.

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It also is important for researchers to attend to the confounding nature of hormonal changes, puberty, and age in examining their relations to psychopathology. Many forms of psychopathology are age-related; so are hormonal changes and puberty. For instance, older adolescents, who are typically more physically mature than younger adolescents, may have higher levels of behavioral problems, not because they have higher levels of testosterone, but because they are older. As “being older” connotes myriad other risk factors in addition to rising hormones, isolating the real effect of hormones requires testing the effects of individual variations in hormonal levels on psychopathology controlling for age.

THE MATURATION DISPARITY HYPOTHESIS

Perhaps the most widely accepted but, paradoxically, least tested explanation for the link between early maturation and psychopathology is the maturation disparity hypothesis. According to this hypothesis, it is the gap between physical and psychosocial maturities that places early (physical) maturers at risk for developing psychopathology. Developmental change is sequential; thus, chronologically ordered developmental tasks in childhood must be completed successfully before the transition to adolescence to ensure normative adjustment. Because early maturers experience a briefer prelude to pubertal change than do their peers, they might be less well prepared socially and cognitively for the biological and psychosocial challenges at puberty. In the words of Caspi and Moffitt (1993, p. 257), these early maturers “are often ill informed about puberty; they are ill prepared by parents; they have few age-appropriate models for how to behave; and their cognitive and emotional levels of development lag behind their biological growth.”

Despite its plausibility, this hypothesis has more often been implied rather than directly tested. One reason for its relative neglect, we speculate, involves the conceptual difficulty in defining psychological “(im)maturity.” Furthermore, there exist empirical difficulties in

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demonstrating such effects, for it requires researchers to show that cognitively and emotionally immature “early bloomers” are at the highest risk for internalizing and externalizing problems. Most studies of puberty typically involve a relatively small group of early bloomers who constitute a small proportion of the sample. Further selecting cognitively and emotionally immature adolescents from an already small sample creates a serious statistical power problem.

Fortunately, recent development in social and cognitive neuroscience provides a fresh look at this hypothesis. Research has demonstrated that portions of the prefrontal cortex that subserve executive functions or self-regulatory control continue to develop well beyond puberty (Casey, Tottenham, Liston, & Durston, 2005; Nelson, Leibenluft, McClure, & Pine, 2005; Spear, 2000). From this view, the higher rates of psychopathology among early maturers are expected because their slow-developing neurocognitive systems are mismatched with the fast-approaching social and affective challenges at the onset of puberty (Nelson et al., 2005); early maturers’ consolidation of self-regulatory skills in both cognitive and emotional domains lags behind the social and emotional demands they face at the onset of puberty. Emerging data generated from this entirely different paradigm are beginning to shed light on the linkage between early pubertal maturation, brain maturation, and psychopathology.

THE CONTEXTUAL AMPLIFICATION HYPOTHESIS

The contextual amplification hypothesis focuses on the interaction effect between puberty processes and social contexts. Researchers subscribing to this hypothesis maintain that the rapid biological changes at puberty, coupled with adverse contexts (e.g., stressful environment, interpersonal challenges, family conflict, neighborhood disorder), further exacerbate these problems. It is reasoned that contextual circumstances can either facilitate or impede early puberty effects through the opportunities, norms and expectations, and implicit reward and punishment

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structures that the contexts provide. Adaptation is particularly difficult for children who negotiate an early pubertal transition in a stressful social environment because new challenges at the entry to puberty and a widening array of social stressors may overtax their relatively undeveloped coping resources.

A few studies of peer-group experiences at puberty provide the clearest case for the contextual amplification effect. Caspi, Lynam, Moffitt, and Silva (1993) found that early-maturing girls in mixed-sex schools engaged in more norm-violating behaviors than did their peers in girls-only schools. Ge et al. (1996) similarly reported that early-maturing girls in mixed-sex settings exhibited more psychological distress than those in same-sex contexts. These researchers inferred that risks of girls' early maturation could arise particularly in mixed-sex contexts because their sensitivity to peer norms and pressures from boys is heightened at puberty. Residing in a disadvantaged neighborhood, where opportunities for involvement in delinquent activities are abundant and collective supervision is lacking due to deteriorating informal social control, also places early maturers at risk for deviant peer association and externalizing behavior (Ge, Brody, Conger, Simons, & Murry, 2002).

Although the essence of the contextual amplification hypothesis lies in the moderating role of context, the picture becomes murkier when considering multiple pathways and influences together. Do early maturers select themselves into older peer networks (implying a mediating path), or does affiliating with older peers magnify the adverse effect of early maturation (implying a moderating effect)? Similarly, could it be that early maturation renders children more sensitive to normal variations in their social contexts, thus leading early-maturing youths to gravitate toward older and deviant peers and to engage in problem behaviors? Is disadvantaged neighborhood simply a macro proxy of greater opportunity to associate with deviant peers and to engage deviant acts, or do some

other properties of neighborhood disadvantages exert an independent effect? Stressful life experiences (Ge, Conger, & Elder, 2001), family adversities (Ge et al. 1996), deviant peers (Ge et al., 1996), lack of parental supervision and harsh parenting (Ge et al., 1996, 2002), and school and neighborhood conditions (Ge et al., 2002) are all intertwined, and each confounds the interpretation of the effects of the others. Better methodological design and statistics are required to tease apart their complex web of effects.

THE ACCENTUATION HYPOTHESIS

Finally, the accentuation hypothesis proposes that demanding life transitions characterized by high novelty, ambiguity, and uncertainty—early physical maturation being an example—tend to accentuate, rather than diminish, previous emotional and behavioral difficulties during those periods. This is because transitional events call forth an individually coherent and consistent way of approach and response that is likely to reveal each person's most salient disposition (Caspi & Moffitt, 1991). Thus, “[t]he reticent become withdrawn, the irritable become aggressive, and the capable take charge” (Caspi & Moffitt, 1993, p. 250). Here, early puberty is viewed as a precipitator that magnifies pre-existing individual differences.

This hypothesis has rarely been tested, with a few exceptions: Caspi and Moffitt (1991) showed that girls with prepubertal behavior problems display even more norm-violating behaviors in adolescence if they experience early menarche. More recently, Rudolph (in press) showed that early maturers with maladaptive stress responses manifested higher levels of subsequent depression than did youth without such personal vulnerabilities.

Empirical testing of this theoretically elegant hypothesis is not an easy task. Because its essence is the temporal order and timing of events, a rigorous examination of this hypothesis requires a longitudinal design based on a large representative sample, with detailed assessment of

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dispositional vulnerabilities before the onset of puberty and assessment of psychopathological outcomes after puberty onset. In addition, selecting “right” measures of existing dispositional vulnerabilities that are expected to be accentuated and that are expected to be manifested in “right” measures of psychopathology outcomes requires considerable conceptual understanding as well as methodological sophistication. Moreover, as the accentuation hypothesis implies a magnifying effect on existing vulnerabilities, choosing psychopathological outcomes with increasing variances over time presents a challenge, as most Likert-scale measures (i.e., scaled in a gradation, such as 1 = strongly agree, 2 = somewhat agree, 3 = neutral, 4 = somewhat disagree, 5 = strongly disagree) tend to have reduced variances over time due to statistical reasons such as ceiling effects (scores cannot take on a value higher than the limit imposed by the measurement scale) and regression toward the mean (scores that are extreme on the first assessment tend to be closer to the mean of the variable in the following assessment). These challenges may explain why there have been so few empirical tests of this hypothesis.

ADDITIONAL ISSUES: SEX AND RACIAL/ETHNIC DIFFERENCES

Although not a focus of this paper, sex and ethnic differences add considerable complexities when testing these hypotheses because (a) early maturation effects have been consistently observed for girls but the results are mixed for boys (Graber et al., 2004); (b) in adolescence, girls are more likely than boys to manifest internalizing psychopathology, while boys show more externalizing problems; (c) girls and boys undergo different hormonal changes at puberty (estradiol vs. testosterone, for example); (d) the two sexes differ in the sequence, timing, and manifestation of growth in primary and secondary sex characteristics, weight, and height, as well as in body composition; and (e) there are racial/ethnic differences in rates of physical maturation (Herman-Giddens et al., 1997). Although it is imperative to include both sexes and diverse samples to avoid

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gender and ethnic confounds, no clear consensus has been reached as to whether girls and boys and participants of different races/ethnicities should be analyzed separately when testing these hypotheses.

CONCLUSION

As psychopathology is the result of a complex web of biological, social, and psychological influences unfolding over the life course, to trace the causal pathways connecting early pubertal maturation to psychopathology is a challenging but exciting task. The four emerging explanations discussed in this article provide a conceptual basis for further studies of explanatory mechanisms. While these explanations offer many new challenges, they also hold the promise of exciting and innovative research characterized by integration across different fields, both within psychology and across disciplines. Although each of these explanations emphasizes a single dimension, they are by no means independent of each other, and they can help piece together the web of pathways from pubertal timing to developmental psychopathology. Not only will the examination of these hypotheses help explain developmental challenges uniquely faced by early maturers, they will also provide mental health practitioners with possible social and cognitive avenues to preventing early-maturing children from developing internalizing and externalizing psychopathology.

Recommended Reading

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Figure Caption

/fl/**Fig. 1.** Mean scores for girls' psychological distress (the Symptom Checklist-90-Revised) by pubertal timing (early, mid, or late) across four measurement occasions. From "Coming of Age Too Early: Pubertal Influences on Girls' Vulnerability to Psychological Distress," by X. Ge, R.D. Conger, & G.H. Elder, Jr., 1996, *Child Development, 67*, 3386–3400). Copyright 1996, Society for Research in Child Development. Adapted with permission.

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