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Detrimental psychological outcomes associated with early pubertal timing in adolescent girls

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Abstract

Though often discussed as a discrete event, puberty comprises one segment of a larger developmental continuum and is notable for rapid transformation across a multitude of domains. Research suggests that an earlier rate of pubertal maturation in girls correlates with a number of detrimental outcomes compared with on-time or later maturation. The present review synthesizes the research on negative psychological sequelae of early pubertal timing in adolescent girls. Emphasis is on three theoretical perspectives by which precocious development is believed to affect the emergence of adverse outcomes: biological, psychosocial, and selection effects.

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As a developmental milestone, puberty is notable for its rapid and near-simultaneous transformation across biological, social, and psychological domains. As physical appearance matures, individuals must navigate changing social norms and expectations. This often necessitates a confrontation and, perhaps, reorganization of identity and self-perception. Aberrance in maturational timing seems to increase risk for emotional and behavioral problems during adolescence and early adulthood (Petersen & Taylor, 1980). In particular, girls who mature earlier than their peers seem to find pubertal adjustment especially challenging and are more likely to experience detrimental sequelae (e.g., Caspi & Moffitt, 1991; Ge, Conger, & Elder, 1996).

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Just as puberty itself is multiply determined, the negative outcomes associated with early pubertal timing span multiple domains. These include psychological, medical, sexual, social, and academic correlates (e.g., Caspi & Moffitt, 1991; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004; Stice, Presnell, & Bearman, 2001). The present review synthesizes the vast research conducted on negative psychological effects of early pubertal timing in adolescent girls. We have chosen to focus only on the female pubertal transition, as there is evidence that timing of puberty affects boys and girls in different ways (e.g., Graber et al., 1997, 2004) and the surfeit of research on both genders precludes inclusion in a single review.

It is worth noting that Ellis (2004) similarly integrated the female pubertal timing literature in a seminal review paper on precursors of early maturation. The current paper seeks to complement and to extend this earlier work by reviewing negative sequelae of early maturation. Taken together, the two papers converge to provide a comprehensive examination of the process of early physical development in adolescent girls. We refer readers seeking greater background on the precursors of pubertal timing to Ellis's work.

There are several potential explanations for the mechanism by which early physical development influences the emergence of adverse outcomes. These fall broadly into the categories of psychosocial, biological, or selection effects. Advocates of a *psychosocial process* note the influence of environmental context in coming of age. Early maturation precipitates a flurry of social changes for which girls may not be “developmentally ready” (Rierdan & Koff, 1993). Because maturation inadvertently aborts the resolution of developmental tasks from the preadolescent period, girls are forced to grapple with these social changes with fewer resources than later maturing peers.

Psychosocial theories are buoyed by empirical evidence that social context is a critical determinant of resilience versus vulnerability for early developers. Although all girls are sensitive to societal responses to their development, social contexts vary in their reactions to and treatment of early maturation. For example, Caspi, Lynam, Moffitt, and Silva (1993) observed that enrollment in a single-sex school may serve as a protective factor for early maturing girls. Similarly, Brooks-Gunn and colleagues noted that girls enrolled at competitive ballet schools, where maturation typically occurs later than population norms, were influenced by the nuances of this environment. Girls who matured on time according to population standards nevertheless defined themselves as early developers and were more likely to display negative consequences (Brooks-Gunn & Warren, 1985a, 1985b; Brooks-Gunn, Attie, Burrow, Rosso, & Warren, 1989). In fact, cultural anthropology research indicates that the pathological outcomes associated with early puberty are largely limited to the Western world; in some cultures, such as the Kipsigis of Kenya, early menarche is considered socially advantageous (Borgerhoff Mulder, 1989).

Biological theories of maturational dysfunction hinge on the physical and hormonal changes hastened by early puberty. The dramatic hormonal shifts associated with puberty may result in arousal, excitability, or excessive emotionality (Angold & Rutter, 1992; Brooks-Gunn & Warren, 1985a; Dahl, 2004). Negative affect may spike as the endocrine system enters gonadarche, or is “turned on” at the initiation of puberty (Angold, Costello, Erkanli, & Worthman, 1999; Angold, Costello, & Worthman, 1998; Brooks-Gunn & Warren, 1989). This may be complicated by neurological changes that spark an increased affinity for risk-taking and sensation-seeking early in adolescence, whereas the development of judgment and self-regulatory skills to master these impulses only evolves towards the end of maturation (Dahl, 2004).

Angold, Worthman, and Worthman (2003) have suggested that increased levels of gonadal sex steroids increase risk of negative affect regardless of timing of pubertal development or other depressive correlates, such as stressful life events. Indeed, it is intuitive that biological consequences should apply globally, as all females will undergo puberty eventually. Nevertheless, early maturers seem to differ from later-developing peers in several key biological ways. Gonadarche triggers sharp increase in pulses of gonadotropin-releasing hormone (or GnRH), which facilitates the pituitary gland's increased secretion of luteinizing hormone (LH), and follicle-stimulating hormone (FSH) (Ellis, 2004; Fechner, 2003; Paretch & Sippell, 2001). Early maturing girls not only begin secreting FSH at an earlier age, but also secrete more of the hormone than later maturing girls. They also have higher levels of serum estradiol. Such hormonal differences persist well into adulthood, until girls reach their late 20s and early 30s (Apter, Reinila, & Vihko, 1989; Apter & Vihko, 1983; Vihko & Apter, 1984).

Additionally, it is possible that early development may interact with normative hormonal changes in such a way as to be particularly detrimental for early maturers (a psychobiological model). Increases in hormones, particularly estradiol, during puberty may heighten a girl's sensitivity to environmental conditions, resulting in disproportionate increases in negative affect following discouraging events, such as exchanges with peers or parents (Brooks-Gunn & Warren, 1989). Because early maturers must endure the consequences of hormonal changes at a time when the majority of their later-developing peers are more stable, this may amplify feelings of being isolated or misunderstood by others. Correspondingly, hormonally triggered erratic behavior may be perceived by others in such a way as to result in conflict, social problems, or the cessation of longstanding childhood friendships.

Early maturers are further compromised by rate of physical development. It appears to take less time for early maturers to progress from Tanner stage 2, characterized by breast budding and pubic hair development, to menarche than it does for later maturers (Apter & Vihko, 1985). While this is hypothesized to be the result of increased estradiol levels, it has unmistakable psychological implications. Early maturing girls must already cope with significant body and lifestyle changes at an earlier age than peers, and before they may be emotionally ready for biological maturity. It seems likely that the restricted period of time allowed to assimilate physical changes might exacerbate feelings of confusion and fear in early developers.

The final theoretical explanation of pubertal timing effects has received less research attention. However, it is possible that the negative sequelae observed with early pubertal timing may evolve from *selection effects*. Girls who experience early menarche are more likely to bear children at an early age, obtain low-paying jobs, experience a startling array of psychological difficulties, and rear children in environments characterized by chronic stress (e.g., Belsky, Steinberg, & Draper, 1991; Comings, Muhlemann, Johnson, & MacMurray, 2002; Graber et al., 1997, 2004; Udry, 1979; Udry & Cliquet, 1982). Because the timing of pubertal maturation is largely heritable (Doughty & Rodgers, 2000; Meyer, Eaves, Heath, & Martin, 1991; Mustanski, Viken, Kaprio, Pulkkinen, & Rose, 2004; Rowe, 2002; Treloar & Martin, 1990), they are also more likely to transmit an earlier age of physical development to their daughters. It may therefore be difficult to discriminate the distinct consequences of early pubertal development from the expected outcomes caused by such genetic or environmental predispositions for problem behavior. An inadvertent failure to account for these family-level confounds would result in an amplification of the effects of pubertal timing on developmental outcomes.

A second potential role of selection in outcomes associated with early development would be through genotype–environment correlation (rGE). That is, while the timing of puberty is largely heritable, early maturation ignites a series of negative environmental responses that influence the course of future development. For example, precocious maturation may cause peers to behave differently towards early maturing girls, which results in social difficulties and feelings of isolation (evocative rGE). Early menarche might also represent active rGE, which occurs when individuals select or construct environmental experiences that are correlated with their genetic propensities. In the case of early maturation, early developing girls may seek out friends who are similarly mature or find themselves attracted to older boys, both of which might result in weakening peer relationships. To date, much of the work on selection effects has concentrated on predictors of pubertal timing, such as father absence (e.g., Comings et al., 2002; Mendle et al., 2006) or socioeconomic status (Obeidallah, Brennan, Brooks-Gunn, Kindlon, & Earls, 2000). Nevertheless, investigation of how selection theory might apply to outcomes (e.g., Burt, McGue, DeMarte, Krueger, & Iacono, 2006) represents a needed area of scientific inquiry and interest.

Consequences of early pubertal timing

In the following section, we present findings on psychological sequelae associated with early pubertal timing. For each correlate, we also evaluate theoretical perspectives and how well each explains the finding. The majority of the studies presented in this review utilize age at menarche as an indicator of pubertal timing. Although early menarche and early pubertal timing are typically considered psychologically synonymous, it is worth noting Ge, Conger, and Elder's (2001) perspective that menarche is a particularly dramatic and shocking signal of physical development. Given that menstruation is both a more sudden and more vivid transition than other developments, its advent may be especially resonant for girls.

Depression

Consistent with the significant inequality in depression rates among adult women and men (Nolen-Hoeksema, 1991), girls are more likely to manifest symptoms of depression during adolescence than boys (Angold & Rutter, 1992; Nolen-Hoeksema & Girgus, 1994). While rates of depression by gender are comparable during childhood, multiple studies suggest that gender differences in symptomatology emerge somewhere between the ages of 11 and 16 (Angold et al., 1998; Ge et al., 2001; Ge, Lorenz, Conger, Elder, & Simons, 1994; Patton et al., 1996; Petersen, Sarigiani, & Kennedy, 1991). Researchers have speculated that the female pubertal transition, in and of itself, is a stressful life event that places all individuals at an increased risk for depressive affect (Angold et al., 1998; Petersen et al., 1991). It is additionally likely the hormones associated with puberty may influence the manifestation of depressive symptoms (Angold & Rutter, 1992; Angold et al., 1998, 1999; Brooks-Gunn & Warren, 1985a, 1989).

Evidence is mixed as to whether puberty alone is a sufficient instigating process for depression (e.g., Angold et al., 2003; Ge et al., 2001). Given that not all girls develop internalizing psychopathology during adolescence, the role of individual factors cannot be neglected (Hayward & Sanborn, 2002). Although negative affect increases in accordance with a period of rapid estrogen rise such as puberty, social factors and the interaction of

hormones with negative life events explain more of the variance in negative affect than hormones alone (Brooks-Gunn & Warren, 1989).

Consistently, the best replicated explanation for the emergence of depressive symptomatology in adolescent females implicates pubertal timing rather than pubertal status (Ge et al., 1996, 2001; Rierdan & Koff, 1991). Girls who experience an earlier age of menarche manifest higher levels of depressive affect than either boys or later maturing female peers (Patton et al., 1996). They are more likely to attempt suicide (Graber et al., 1997) and meet DSM-IV diagnostic criteria for an episode of major depressive disorder (Stice et al., 2001).

The most common explanations for the effect of pubertal timing on depressive symptoms imply a psychosocial relationship precipitated by the physical changes associated with puberty. Girls who exhibit obvious signs of physical maturation may find it difficult to maintain friendships with same-sex peers who have not developed at a similar rate (Peterson et al., 1991). Because menarche is a more dramatic and sudden transition than other pubertal developments, experiencing menarche prior to one's peers may additionally foster a sense of emotional distance (Ge et al., 2001). Given their precocious physical appearance, older individuals may ascribe unwarranted perceptions of maturity onto these girls, who may consequently be forced to confront environmental challenges before they are emotionally or cognitively prepared to do so (Ge et al., 1996). This might provoke negative affect, fear, and/or confusion.

It is important to note that, in the case of depressive symptoms, a psychosocial model is not necessarily inconsistent with a biological one. As previously discussed, it is possible that hormonal changes or levels may intensify negative emotional responses to environmental circumstances. Alternatively, hormonal shifts may affect girls' behavior in a way that directly influences interactions with peers, parents, boys, or other. This, in turn, results in perplexing or distressing social consequences that impact psychological well-being.

To date, there has been no research investigating the correlation between early maturation and depression from a selection perspective. Nevertheless, as both early development and adolescent depression are genetically influenced (e.g., Glowinski, Madden, Bucholz, Lynskey, & Heath, 2003; Mustanski et al., 2004), there are several methods by which selection may impact this relationship. One possibility is that the genes which influencing depression are the same as those influencing timing of menarche—a situation known as *pleiotropy*, in which a single gene affects multiple traits. A second hypothesis is that early menarche provokes adverse environmental circumstances, by placing girls in situations for which they are unprepared both cognitively and emotionally. This would be consistent with theories that risk for adolescent depression may be explained by genetic predisposition to experiencing stressful life events (Silberg, Pickles, & Rutter, 1999) and is, again, not incongruous with psychosocial hypotheses.

Other internalizing symptoms

In addition to specific diagnostic associations, early puberty has been associated with more generalized internalizing symptoms (Hayward et al., 1997; Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003). Early maturers are also more likely to report psychosomatic symptoms—including abdominal pain, sleep disturbances, headache, upset stomach, breathlessness, tremors, and heart palpitations—than peers during early adolescence (Aro & Taipale, 1987). Anxiety has been less frequently studied in conjunction with timing of pubertal maturation than other clinical domains. However, given the comorbidity of

anxiety and depression, it is not surprising that there also seem to be links between early menarche and anxious symptoms (Patton et al., 1996). In particular, panic attacks have been shown to occur more frequently among sixth- and seventh-grade girls who display advanced signs of physical development (Hayward et al., 1992). Anxiety also seems to be more common in young adults who experienced early pubertal maturation (Graber et al., 2004). The same social mechanisms that account for depressive affect in girls might also produce these internalizing symptoms: anxiety might be a response to ambiguous or upsetting social interactions. Psychosomatic symptoms might also be viewed as an overattentive bodily response, perhaps triggered by the onslaught of physical changes accompanying puberty.

Eating disorders

Onset of menstruation requires a certain body mass index and may be precipitated by a gain in weight and body fat. Additionally, menarche may serve as a demarcation for the advent of disordered eating cognitions. Comparisons of pre- and post-menarcheal adolescents indicate that, prior to menarche, girls tend to be less cognizant of the relation between diet and exercise as a means of weight loss (Abraham & O'Dea, 2000). Regardless of the respective timing of maturation, adolescent girls are also more likely to consider themselves fat and restrict eating following menarche (O'Dea & Abraham, 1999). In fact, a staggering 19% of adolescent females are estimated to engage in some form of disordered eating behavior or to display distorted body perception (Foreyt, Poston, Winebarger, & McGavin, 1998).

Early maturation, however, may magnify the relatively normative concerns with body shape and weight initiated by menarche. Early maturing girls are more likely to report body dissatisfaction and poor self-esteem during adolescence and to engage in excessive dieting and disordered eating (Blyth, Simmons, & Zakin, 1985; Graber et al., 1997; Keel, Fulkerson, & Leon, 1997; McCabe & Ricciardelli, 2004). Poor body image seems to persist among early maturers even after same-age peers have achieved puberty (Graber, Brooks-Gunn, Paikoff, & Warren, 1994).

While the above studies confirm the relationship between early maturation and chronically disturbed eating patterns, it is less clear whether age of pubertal onset affects eating disorder symptoms at a diagnostically severe level. Fairburn, Welch, and Doll (1997) reported that adult inpatient bulimics were more likely to have experienced early menarche than community controls or other inpatients with other disorders. Similarly, bulimia and subclinical bulimia nervosa symptoms have been associated with early menarche in samples of Finnish teenagers (Kaltiala-Heino, Rimpela, Rissanen, & Rantanen, 2001; Ruuska, Kaltiala-Heino, Koivisto, & Rantanen, 2003). However, Stice et al. (2001) found that earlier menarche was associated with dieting and body dissatisfaction, but not diagnoses of anorexia, bulimia, or binge-eating disorder.

Other studies suggest that the relationship between menarche and eating pathology may be contingent on the spectrum of symptomatology. For example, although earlier menarche has been associated with bulimia nervosa (Fairburn et al., 1997; Kaltiala-Heino et al., 2001), it does not seem related to anorexia nervosa (Fairburn, Cooper, Doll, & Welch, 1999; Ruuska et al., 2003). This difference may be partially explained by the divergent attitudes towards dating that typify these disorders. Engagement in disordered eating among early maturing girls is moderated by dating behavior (Cauffman & Steinberg, 1996; Smolak, Levine, & Gralen, 1993). Anorexia is characterized by a fearful, disgusted response to

sexuality, whereas bulimia is associated with early sexual activity (Kaltiala-Heino et al., 2001). It is therefore possible the effects of pubertal maturation on eating pathology may be confined to disorders whose symptoms are not incongruous with romantic activity. Since anorexics are less likely to engage in dating or sexually intimate behavior than other populations of eating disorders, they may therefore fail to experience the moderating effect of heterosocial behavior on the relationship between early menarche and eating pathology.

While the relationship between early puberty and eating pathology is theoretically intriguing, it is worth noting that pubertal timing by itself accounts for relatively minimal proportions of variance in eating disorders (typically less than 2%; Stice, 2003). Interestingly, concurrent life stressors seem to intensify the effects of early puberty on body dissatisfaction (Blyth et al., 1985). If we consider initiation of dating a stressful life transition, these results are congruent with the moderating effect of romantic relationships on the association between menarche and disordered eating obtained by Cauffman and Steinberg (1996) and Smolak et al. (1993).

The research on stressful life events implies that effect of pubertal timing may be amplified or exacerbated by contextual stressors. This supports a psychosocial explanation. Because the menarche-incited weight gain physically distances early maturing girls from both the current idealized thin body type and the bodies of their less-developed peers (Graber et al., 1994; McCarthy, 1990; Stice, Hayward, & Cameron, 2000) a comparatively earlier age of menarche confers increased risk for eating pathology by triggering body dissatisfaction and insecurity (Graber et al., 1994). Girls may internalize changing physical appearance as a way that they are “different” from peers. This may be manifest as feelings of self-consciousness or behavioral attempts to “reduce” their bodies via diet and exercise.

By invoking weight gain, curviness, and other morphological changes associated with maturation, psychosocial explanations for the menarche–eating pathology association implicitly rely on biological differences. While a psychobiological theoretical synthesis seems sensible, a strictly biological explanation for this association is less compelling. Both anorexics and bulimics display elevated levels of the androgen hormone dehydroepiandrosterone (DHEA) and its sulfate, DHEAS (Monteleone, Luisi, & Colurcio, 2001). Conversely, lower levels of luteinizing hormone, follicle-stimulating hormone, and estradiol have been observed in women with both anorexia and restricting subtypes of bulimia nervosa (Devlin et al., 1989; Pirke et al., 1987). Although early maturing girls begin to produce DHEAS prior to peers, they do not seem to differ in overall levels of DHEAS production (Vihko & Apter, 1984). In fact, the symptoms of eating disorders—particularly bingeing and restricting dieting—can actually result in dysfunctional hormone levels (Resch, Szendei, & Haasz, 2004). This renders conclusions about the role of biology in eating pathology and early menarche particularly nebulous.

Academic achievement

Early maturing girls are more likely to exhibit poor academic performance in high school than on-time or later maturing peers. This may be an artifact of the link between early maturation and problem behavior. For example, early maturers are more likely to report getting in trouble at school, absenteeism and truancy (Graber et al., 1997; Simmons & Blyth, 1987; Stattin & Magnusson, 1990). They report less interest in academic subjects and are less likely to pursue college educations (Stattin & Magnusson, 1990). Although Simmons and Blyth (1987) found a correlation between early pubertal timing and lower

grades, this has not been replicated in other studies (Dubas, Graber, & Petersen, 1991; Graber et al., 1997; Stattin & Magnusson, 1990).

Conversely, later maturation has been associated with higher grades (Dubas et al., 1991). Attempts to explain this phenomenon through specific cognitive capabilities indicated that later maturing girls perform better on tests of spatial reasoning (Hassler, 1991). However, verbal fluency performance—a better determinant of academic achievement—does not seem related to pubertal timing. Moreover, there is no discernable link between Tanner stage of pubertal development and advancement of cognitive capabilities (Orr, Brack, & Ingersoll, 1988).

These adolescent trends in achievement generate differences in career and income level that persist through adulthood. Early maturing girls tend to bear children before their peers and to report that subsequent difficulties with childcare affect pursuit of higher education. Consequently, they tend to be employed in lower-paying, less prestigious jobs (Stattin & Magnusson, 1990). This effect is magnified in girls with extremely precocious maturation. Among a sample of girls with menarche prior to 11 years of age, only 2% pursued higher education (Magnusson, Stattin, & Allen, 1985). Additional analyses demonstrated that this relationship was not confounded either by intelligence or parental education levels; however, it was mediated by contact with an older social network.

Rather than a qualitative cognitive difference, a psychosocial mechanism may better explain the association between later maturation and grade performance. Because later maturing girls tend to be less popular and less attractive to boys, they may devote more time to schoolwork. In contrast, early maturing girls are more likely to engage in dating and precocious romantic relationships, which may distract them from academic work (Simmons & Blyth, 1987). However, because academic achievement predicts future socioeconomic potential, it is additionally important to consider selection effects. Girls who experience early maturation are more likely to obtain lower SES employment and transmit earlier ages of development to their daughters. Although research in this area is sparse, the inclusion of socioeconomic demographics into analyses seems to eliminate associations between early menarche and educational attainment (Koivusilta & Rimpela, 2004).

Substance use

Early pubertal development is associated both with an earlier initiation of smoking and drinking (Wiesner & Ittel, 2002; Wilson et al., 1994) and an increased likelihood of a lifetime diagnosis of substance abuse (Graber et al., 1997; Stice et al., 2001). Conversely, late pubertal maturation predicts abstinence well beyond the end of puberty (Stattin & Magnusson, 1990). Although some experimentation with cigarettes and alcohol may be considered “normal” adolescent behavior (Moffitt, 1993), early maturing girls tend to smoke and consume alcohol in greater quantities and at greater frequencies than less developed peers (Lanza & Collins, 2002; Wichstrom, 2001; Wiesner & Ittel, 2002). They are also more likely to smoke marijuana regularly and to experiment with other illegal drugs (Magnusson et al., 1985; Prokopcakova, 1998).

The effects of pubertal timing on substance use do not seem to be mediated by either emotional distress or depressive symptoms (Tschann et al., 1994; Wiesner & Ittel, 2002). Notably, there is some evidence that effects of pubertal timing on substance use may be significant only for those individuals whose development remains consistently advanced compared to same-age peers (Dick, Rose, Pulkkinen, & Kaprio, 2001). Girls who exhibit an

initial earlier maturation but whose developmental rate then slows until maturation is comparable to that of peers may not engage in detrimental substance use. In a within-family twin comparison, Dick, Rose, Viken, and Kaprio (2000) found that, among twins discordant for age of menarche, the earlier maturing twin was more likely to engage in smoking and drinking at a younger age and to report a greater frequency of smoking and drinking at age 16 than the later-developing one.

Dick et al.'s work supports a causal, psychosocial role of early maturation, as twins are concordant for home environment and genetic variables that also influence substance use. Additional support for psychosocial factors comes from examination of dating behavior. Prematurely advanced signs of physical development in girls tend to promote affiliations with older boys or adult boyfriends (Caspi et al., 1993). This contact with an older social group increases accessibility to alcohol and illicit drugs (Stattin & Magnusson, 1990; Tschann et al., 1994). Presence of an older boyfriend is not only associated with substance use, but mediates the relationship between chronological age and risky sexual behavior (Mezzich et al., 1997). It is possible that early maturing girls might attempt to bridge an obvious chronological gap by engaging in smoking and drinking, which may be perceived as adult behavior (Wichstrom, 2001). Alternatively, since substance use has been shown to moderate the relationship between adult sexual partners and parent–daughter conflict (Mezzich et al., 1999), girls with poor parental relationships may utilize or seek adult partners as parental surrogates or supplements.

Sexual activity

Girls who experience an earlier age of menarche begin to date before their peers (Kim & Smith, 1998). In concordance with earlier engagement in dating, early maturers tend to have an earlier initiation of sexual intercourse and sexually intimate behaviors such as kissing and petting (Flannery, Rowe, & Gulley, 1993; Lam, Shi, Ho, Stewart, & Fan, 2002; Wyatt, Durvasula, Guthrie, LeFranc, & Forge, 1999). By the age of 18, girls who have experienced an early onset of menarche are more than twice as likely to have given birth or terminated a pregnancy than peers (Udry, 1979).

Psychosocial, biological, and selection hypotheses all account for associations between pubertal timing and early sexual experience. Psychosocially, one consequence of a sexually mature appearance is that girls become more attractive to older males and are consequently more likely to become involved with older boyfriends (Marin, Coyle, Gomez, Carvajal, & Kirby, 2000; Mezzich et al., 1999). Involvement with older males is, in turn, associated with sexual activity in adolescent girls (Gowen, Feldman, & Diaz, 2004; Marin et al., 2000). Biologically, rises in DHEA, testosterone, and estradiol are thought to trigger feelings of sexual attraction (McClintock & Herdt, 1996). As age of first sex and age of menarche share common genetic pathways (Rowe, 2002), selection theories suggest that this association is mediated through genetic transmission.

Is earlier engagement in sexual activity pathological? Ellis (2004) notes that there is no conclusive empirical support that earlier timing of menarche is associated with risky sexual behavior, higher number of sexual partners, or unstable pairbonds. Rather, timing of first intercourse and first pregnancy are linked with timing of menarche for the simple reason that girls who experience earlier puberty experience sexual attraction and transition to reproductive status at an earlier age.

We would argue, nevertheless, that early sexual activity is one example of a correlate of early maturation that triggers problematic sequelae. Medically, girls who experience early menarche prior to the age of 12 are more likely to test positive for the human papillomavirus (HPV), a sexually transmitted virus associated with increased risk for cervical cancer (Fisher, Rosenfeld, & Burk, 1991). Early maturers are also at a greater risk for teen pregnancy: the time from menarche until 50% of menstrual cycles are ovulatory is approximately 1 year if menarche occurs before age 12 but 4.5 years if menarche occurs at 13 or older (Apter & Vihko, 1983).

Emotionally, many of the negative outcomes associated with early physical maturation are either mediated or exacerbated by engagement in romantic relationships. We have previously cited the examples of substance use (Mezzich et al., 1997), eating pathology (Cauffman & Steinberg, 1996; Smolak et al., 1993), and academic achievement (Stattin & Magnusson, 1990). Kaltiala-Heino, Kosunen, and Rimpela (2003) have noted an association between early menarche, early sexual activity, and depression during adolescence. They suggest that depressive affect might result from sexual activity occurring before adolescents were prepared. Certainly, as discussed, presence of an older boyfriend increases likelihood of sexual activity (Marin et al., 2000). More distressing, however, is the increased frequency of both date rape (Vicary, Klingaman, & Harkness, 1995) and unwanted sexual advances (Marin et al., 2000) among early maturing girls. Because of the implicit power differential that occurs between an adolescent girl and an older sexual partner, early maturing girls may be placed in hazardous situations with fewer emotional and cognitive resources than grown women.

Delinquency

Given that sexual activity, poor school attendance, and alcohol use are hallmarks of externalizing behavior, it is not surprising that early menarche has also been associated with delinquent norm violations such as shoplifting, vandalism, fighting, weapon possession, and illegal drug use (Caspi et al., 1993; Caspi & Moffitt, 1991; Flannery et al., 1993; Haynie, 2003; Magnusson et al., 1985). Although Stattin and Magnusson (1990) did not find a relationship between age of maturation and adolescent delinquency, they did find that women aged 18–33 with criminal records were more likely to report menarche prior to age 12. Repeat criminal offenders were more likely to report menarche prior to age 11. Similarly, Haynie (2003) found early maturation significantly predicted engagement in violent delinquent behavior (such as burglary, fighting, gang membership, and shooting or stabbing another person).

Just as affiliation with an older friend group and older boyfriend facilitates access to alcohol, psychosocial theory suggests that friendships with older adolescents would enable delinquent behaviors for early maturing girls. Magnusson et al. (1985) posited that older teenagers may have more tolerant attitudes towards norm violations, which they convey to chronologically younger friends. This results in an increased frequency of situations in which rule-breaking may occur and a more positive attitude towards these situations. However, Haynie (2003) observed friends' engagement in delinquency influenced early maturers' likelihood of delinquent behavior—regardless of friend age. Interestingly, enrollment in an all-girls school seems to serve as a protective factor for the association between early puberty and delinquent behavior. Compared to counterparts in same-sex schools, early maturing girls in mixed-sex school environments had more familiarity with

delinquent peers and more opportunities to engage in delinquent behavior by the age of 13 (Caspi et al., 1993).

Despite the importance of social factors such as peers, a consideration of selection effects is also important in understanding antisocial behavior. Children do not select into peer groups at random, but rather become friends with children who are like them (Guo, 2005; Jaccard, Blanton, & Dodge, 2005). This means that aggressive children are more likely to become friends with other aggressive children. With regard to the delinquency and early maturation association, support for a selection effect comes from research demonstrating that early maturing girls with childhood behavior difficulties are more likely to exhibit delinquent adolescent behavior than peers who have matured at a comparably advanced rate, but who have no previous history of behavioral difficulties (Caspi & Moffitt, 1991). Moreover, antisocial behavior is accentuated both by familial predisposition (see Rhee & Waldman, 2002, for a review) and by social and family environmental factors, such as friend characteristics (e.g., Allen, Porter, McFarland, Marsh, & McElhaney, 2005; Scaramella, Conger, Spoth, & Simonds, 2002). A pertinent focus for future research would be to examine the extent early puberty influences delinquency independent of these predilections and confounding factors for antisocial behavior. One recent attempt (Burt et al., 2006) suggested that social factors may be more closely linked to conduct problems for girls who experience early menarche; for girls with on-time or late menarche, genetic factors appear to be more influential.

At this time, a biological explanation does not sufficiently explain the link between early physical development and delinquent activity. Although aggressive behavior is related to neurobiological factors, links between hormones and externalizing behavior in adolescents remain ambiguous, with much of the research and significant results obtained only for boys. For example, there appears to be a weak association between aggression and higher circulating testosterone (Book, Starzyk, & Quinsey, 2001), higher levels of LH, and lower levels of FSH levels in boys (Susman et al., 1987). Results have not been replicated in girls, and—in the case of FSH, which is higher in early maturers—contradict an expected association.

Racial/ethnic differences in timing

Given that minorities tend to experience pubertal changes such as breast growth, pubic hair development, and menarche prior to Caucasians (Herman-Giddens et al., 1997; Wu et al., 2002), there is a surprising paucity of research regarding racial and ethnic differences in the relation between pubertal timing and emergent adolescent problem behavior. The association between an earlier age of menarche compared to one's peers and increases in depressive symptomatology has been replicated within an entirely African-American sample (Ge, Kim, Brody, Conger, & Simons, 2003). Additionally, Striegel-Moore et al. (2001) found that early physical development was equally associated with poor body image and dieting in both black and white girls.

Results from other studies, however, provide contradictory evidence. Notably, one comparison found that post-menarcheal Caucasians were more depressed than same-aged pre-menarcheal peers. This was not the case for African-American or Hispanic girls (Hayward, Gotlib, & Schraedley, 1999). Similarly, Michael and Eccles (2003) reported that Caucasian girls who experienced early menarche experienced greater levels of depressive affect, anger, and eating problems. In contrast, there was no association between menarcheal timing and negative psychological outcomes among African-American girls.

Cavanagh (2004) documented the clearest evidence of differential effects of pubertal timing across races when investigating age of first sexual intercourse. Using data from the National Longitudinal Study of Adolescent Health, Cavanagh compared sexual debut, age of menarche, and friendship group characteristics across white, Latina, and African-American adolescent girls. Among whites, friendship group seemed to mediate the association between early menarche and age of sexual initiation. Girls who experienced an earlier puberty tended to have friends who demonstrated risky behavior and poor academic achievement, which consequently influenced sexual debut. In contrast, Latina girls also demonstrated an association between early menarche and early sexual activity, but this was not as strongly affected by peer friendships. Instead, frequency of interactions with older boys was a far more important predictor of sexual activity for Latinas than for whites or African-Americans. Lastly—and perhaps most interestingly—there were no meaningful associations obtained between early pubertal timing, age of first sexual activity, and friendship characteristics among African-American girls.

Although there has been limited research on the reasons for racial differences in pubertal timing, hypotheses include cultural differences in diet and nutrition and exposure to environmental estrogens (Wu, Mendola, & Buck, 2002). Notably, deviations in timing of pubertal onset across race seem to be widening. Between 1966 and 1970, mean ages of menarche in the United States were 12.8 years for Caucasian girls and 12.5 years for African-American girls (Harlan, Harlan, & Grillo, 1980). Herman-Giddens et al. (1997) observed a mean age of 12.88 years for Caucasians and 12.16 for African-Americans. Similarly, Wu et al. (2002) reported means of 12.7 for Caucasians, 12.1 for African-Americans, and 12.2 for Mexican-Americans.

Implicit in any discussion of racial differences in pubertal timing in American girls is the dangerous conflation of race with socioeconomic status. Socioeconomic status, and not race, affects known predictors of pubertal timing such as nutrition, environmental stress, and family composition. In fact, Obeidallah et al. (2000) established that documented differences in age of pubertal onset between Caucasian and Latina girls disappeared after controlling for socioeconomic status. This might be viewed as support for a selection hypothesis. It also calls into question whether pubertal timing might serve as a proxy variable for poverty: are the negative correlates of pubertal timing attributable to the adverse effects of being reared in impoverished circumstances rather than a causal role of early maturation?

While socioeconomic and racial interactions with age of menarche within American samples might suggest cautious interpretations, research from international samples indicates an overall consistency of results. The association of early pubertal timing and early sexual initiation has been replicated in countries as diverse as Brazil (Schor, 1993), Slovakia (Prokopcakova, 1998), Jamaica (Wyatt et al., 1999), and Hong Kong (Lam et al., 2002). Within separate Finnish samples, early menarche has been shown to be related to bulimia (Kaltiala-Heino et al., 2001), internalizing symptoms (Ruuska et al., 2003), and alcohol use (Dick et al., 2001). In Norway, Wichstrom (2000, 2001) has found higher rates of suicide attempts and alcohol consumption among girls who report timing of puberty earlier than peers. Depressive and anxious symptoms have been associated with early menarche in Australia (Patton et al., 1996); in New Zealand, externalizing problems have been associated with early menarche (Caspi & Moffitt, 1991).

Decreasing age of puberty

Researchers have speculated about the progressive lowering of pubertal onset in Westernized countries. Tanner (1962) estimates the acceleration in age at menarche at between 3 and 4 months per decade. A study conducted by Pediatric Research in Office Settings (PROS), a research branch of the American Academy of Pediatrics, had 225 pediatric clinicians evaluate physical development in approximately 17,000 girls. Findings indicated a mean shift in initiation of pubertal onset of approximately 1 year since Marshall and Tanner's (1969) findings (Herman-Giddens et al., 1997). More recent estimates suggest that this lowering trend in age at menarche seems to be flattening out (Parent et al., 2003).

Several environmental explanations for the decrease in age of puberty have been proposed. One hypothesis is that exposure to environmental toxins may have either stimulated or mimicked the effects of estrogen in the body, thereby initiating pubertal onset. High levels of estrogens in meat and poultry are believed to have triggered epidemics of premature maturation in Italy and Puerto Rico (Fara et al., 1979; Saenz de Rodriguez et al., 1985), but there have been no similar localized outbreaks of precocious breast budding in the United States (Kaplowitz, 2004). Moreover, there remains no empirical evidence that puberty can be stimulated by exposure to environmental estrogens and samples of the alleged contaminated food were unavailable for testing (Paretsch & Sippell, 2001). Other hypotheses propose that manufactured chemicals may alter performance of the endocrine system or that hair products containing hormones may be a risk factor for early puberty (Zimmerman, Francis, & Poth, 1995). However, data supporting such explanations are insufficient and requires further research.

While shifts in mean maturational trends are theoretically intriguing, they do not contradict pubertal timing effects. Early maturation is defined relevant to a population; although the mean age of menarche may shift across generations, the proportion of individuals classified as early maturers will remain constant. More complicated to assess would be whether effects of pubertal timing occurred in previous generations, when the duration of childhood was artificially foreshortened by arranged marriages. Regardless, however, of whether effects of pubertal timing are a uniquely modern occurrence, we cannot neglect their undeniable impact on individual experiences. Interestingly, using classical sources such as the Hippocratic corpus, Amundsen and Diers (1969) establish a likely modal age of menarche in ancient Greece and Roman as between 13 and 14 years, younger than might be suggested by Tanner's estimates.

Discussion

Puberty encompasses a rapid and substantial period of change, second only to infancy in its scope and alacrity. Although typically described as the emergence of secondary sexual characteristics, such a definition fails to convey the complexity of this progression. Rather, puberty is better understood as an integrated transition across multiple domains: biological, psychological, cognitive, and social. As such, puberty is best conceptualized as one component of a larger development continuum.

Research consistently demonstrates that earlier physical maturation presages adolescent problem behaviors. Since puberty spans multiple developmental domains, it is unsurprising that the negative outcomes associated with early physical maturation cover an equally broad gamut, including depression, substance abuse, eating pathology, body

dissatisfaction, externalizing behavior, risky sexual behavior, abortion, and some aspects of academic achievement (e.g., Graber et al., 1997, 2004; Stattin & Magnusson, 1990; Stice et al., 2001; Udry, 1979). It is important to note that the effect sizes of pubertal timing vary by outcome and tend to be modest. For example, Stice et al. (2001) observed that early maturing girls were at 1.9 times risk for depression and 1.8 times risk for substance use. Similarly, Lanza and Collins obtained relative risk ratios of 2.0 for alcohol use, 1.6 for cigarette use, 2.7 for getting drunk, and 2.5 for marijuana usage (2002). In linear analyses, *R*-squared estimates indicate that maturational timing accounts for less than 2% for eating problems (Stice, 2003), 4% for delinquency (Flannery et al., 1993), and 6% for sexual activity (Flannery et al., 1993). Therefore, while early physical development is unlikely to predict adolescent problems singly, it seems to be one facet of a larger network of risk.

Three broad theoretical categories—psychosocial, biological, and selection—have been proposed to account for the mechanism by which pubertal timing encumbers emotional adjustment. Psychosocial theories, both the most commonly used and empirically validated, revolve around interpersonal difficulties. Psychosocial theories are advantageous in that they effectively explain all outcomes associated with maturational timing, but problematic in that they fail to account for interindividual differences in propensity for psychopathology or early maturation. Biological theories concentrate on evidence that early maturers differ from peers both in initiation of hormonal increases and in overall hormone levels. This perspective explains outcomes such as depression and sexual activity quite well and meshes neatly with psychosocial theories for correlates such as eating disorders; it is less persuasive for academic achievement and externalizing outcomes. Selection research investigates how family environment and genetic predispositions might influence associations between early maturation and associated outcomes. Although there has been limited investigation in this domain, selection research seems to capture sexual activity and academic achievement well; understanding the role of selection in other correlates is more tenuous.

Future directions

All three theoretical perspectives require and would benefit from ongoing inquiry. However, ongoing research in two areas might be of particular utility: (1) genetically informed studies targeting selection effects and (2) studies investigating racial and ethnic differences in pubertal timing. With regard to selection effects, this perspective has received the least amount of research attention to date. Second, and more saliently, a key advantage of family studies is their ability to control for environmental differences. Therefore, psychosocial and biological theories can both be investigated and validated using genetically informed data. For example, studies such as those conducted by Dick et al. (2000) and Burt et al. (2006) are able to establish that associations between alcohol use and externalizing behavior, respectively, are not influenced by pre-existing genetic predispositions for such behavior. This supports a causal role of early maturation, as described by psychosocial theories.

It should be stressed that in advocating genetically informed studies we are not advocating mere heritability estimates (Turkheimer, 1998). Rather, research should attempt to elucidate the mechanisms by which early maturation affects the emergence of adolescent psychopathology. In particular, we believe that analyses should consider pubertal

timing an example of genotype–environment correlation. While the timing of puberty is genetically influenced, precocious maturation incites a chain of environmental reactions—such as attention from older males or feelings of isolation from peers—which shape the course of future development. This model is conceptually satisfying, because it captures the social triggers and repercussions associated with early maturation, while maintaining an integral role of biology and genes.

There are naturally drawbacks inherent in any research methodology. In this case, genetically informed data can be arduous to collect and require sophisticated analytic techniques and computer software. Moreover, although it is possible to detect presence of gene–environment correlation (rGE), it is not possible to ascertain whether such a correlation is active or evocative in nature. Genetic and environmental influences can also shift across populations and cohorts; for example, nutritional patterns may play a large environmental role in timing of menarche in impoverished populations but not for more affluent ones.

However, perhaps the greatest weakness of family studies lies in imprecise assessments of how racial differences in timing impinge on outcomes. The majority of genetically informed data utilize twins to estimate variance attributable to genetic, shared environmental, and nonshared environmental influences. These models are statistically driven by differences in concordance between monozygotic and dizygotic twin pairs. Variables such as race or ethnicity, however, present a conundrum for researchers for the simple reason that both monozygotic and dizygotic twins are always concordant. Although race is believed to be accounted for by shared environmental effects, Cholesky models can only generate a percentage of variance attributable to shared environment (termed C). It is impossible to tell if this effect of C is due to race or another shared environment variable—such as socioeconomic status or family structure—that would also be associated both with pubertal timing and various psychopathologies. Although analyses could circumvent this problem by using samples of stepsiblings or adopted siblings who are discordant for race, such cases are uncommon enough to make data collection onerous.

Certainly, the dearth of consistent results on how racial differences in pubertal timing affect outcomes is distressing. As psychosocial theories hinge on distress generated by early maturation, establishing cultural differences in treatment and perception of maturation might prove an especially apt starting point for this research. The majority of research conducted on pubertal timing assigns a label of “early maturation” to girls who reach certain physical milestones—generally menarche—in the first 20% of the sample. This is an objective measurement. However, given that many theories of pubertal timing involve a subjective sense of alienation, must early maturers be aware of their advanced development for psychosocial consequences to emerge? Can we expect the same sequelae if an early maturing girl does not feel isolated or different from peers?

Some preliminary research on differences between African-American and Caucasian girls suggests that African-American girls are less likely to find early maturation alarming or distressing (see Ge et al., 2003; Striegel-Moore et al., 2001 for exceptions). It has been suggested that this may illuminate a cultural difference, in that African-American girls may feel more prepared or excited for the challenges of adulthood (Michael & Eccles, 2003). Although one direct study of family attitudes toward menarche did not obtain racial differences (Teitelman, 2004), the sample size was quite small and perhaps not adequate for detection of group differences.

Conclusion

It is our belief that early physical development is best viewed as one risk factor for a variety of problems and psychopathology. The particular problems and symptoms manifest depend on individual qualities and predispositions. Continued research into issues associated with pubertal timing have the potential to yield direct clinical benefits. An increased awareness of the underlying mechanisms of early pubertal timing is vital for empathizing with the unique developmental challenges faced by early maturing girls. By understanding how a series of environmental reactions may be initiated through key biological events, it is possible to advance a multiple systems perspective for understanding the etiology of particular psychological problems.

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