

Putting pubertal timing in developmental context: Implications for prevention

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Abstract:

This article examines selected findings regarding the consequences of difference in timing of pubertal onset in order to build an explanatory model of puberty in context. We also seek to shed light on possible prevention efforts targeting adolescent risk. To date, there is substantial evidence supporting early onset effects on both internalizing and externalizing problems during the adolescent decade and possibly beyond. However, such effects do not directly speak to preventive intervention. The biological, familial, and broader relationship contexts of puberty are considered along with unique contexts for early maturing girls versus boys. Finally, we identify potential strategies for intervention based on these explanatory models.

Keywords: adolescent | puberty | psychopathology | stress | vulnerability

Article:

INTRODUCTION

Puberty has often been identified as an important marker, if not a causal factor, of changes in adjustment and behavior from childhood to adolescence. Puberty, as it is has been described, “. . . should not be considered a de novo event but rather as a phase in the continuum of the development of gonadal function and the ontogeny of the hypothalamic-pituitary-gonadal system in the fetus, through puberty, to the attainment of full sexual maturation and fertility. . .” (Grumbach & Styne, 2003, p. 1115). Notably in their introductory comments about puberty, Grumbach and Styne assert that pubertal development will have “. . . profound psychological effects” (p. 1115). At the same time, most individuals traverse not only puberty but also adolescence without serious emotional or behavioral problems calling into question the assertion of profound effects. To some extent, this assertion is true in that the attainment of reproductive capacity occurs via transformation of nearly every system in the body— neural, cardio-pulmonary, skeletal-musculature—occurring over a significant period of years; for example, maturation of secondary sexual characteristics such as pubic hair growth or breast development

typically spans 4–5 years. The experience of these changes for the individual is likely profound, but at the same time, completely ordinary.

Of significance is that puberty does not occur in a social vacuum but is experienced in a diverse and changing contextual environment. Girls and boys receive feedback about their developing bodies, and the behavioral expectations commensurate with physical maturity from peers, parents, teachers, culture, and the media. Most individuals integrate their own perceptions of their development with contextual feedback and navigate the psychosocial dimensions of puberty quite well (Graber & Brooks-Gunn, 1996). To understand why puberty confers risk for some individuals requires examination of the interaction of individual developmental processes within the broader social context.

Both the biology and psychosocial experience of puberty demonstrate substantial individual differences. Prior studies have found biological, or more specifically hormonal, effects on emotional and behavioral changes at puberty (Susman & Dorn, 2009). However, effects have not been pervasive or large with the most consistent findings linking puberty to changes in sexual arousal, interest, and behavior as well as aggression (e.g., Graber, Brooks-Gunn, & Archibald, 2005; Halpern, 2006).

Notably, the timing of the onset and likely also the tempo of progression of pubertal development vary widely within the range of normative development. Tanner (1962) originally estimated a 5-year span in variation in age of onset of growth spurt within a particular age cohort and gender. Off-time development, that is being earlier or later than one's same gender peers, has been identified as a non-normative aspect of puberty that confers risk for serious problems including internalizing and externalizing problems and disorders. It is beyond the scope of the present discussion to examine all possible pubertal effects—hormonal, timing, or other—rather we highlight selected findings regarding pubertal timing in order to build an explanatory model of puberty in context and to shed light on possible prevention efforts targeting adolescent risk.

Notably, timing effects on psychopathology are often observed during the adolescent period with a few studies demonstrating sustained effects into adulthood (Foster, Hagan, & Brooks-Gunn, 2008; Graber, Seeley, Lewinsohn, & Brooks-Gunn, 2004). Interestingly, effects of early timing for girls have demonstrated substantial consistency despite a wide range of methods used to assess pubertal timing and psychopathology (Graber, 2008). Moreover, while much of this work was initially conducted with Caucasian samples, subsequent research has often replicated and extended findings across ethnic groups. In a study of African American young adolescents, Ge, Brody, Conger, and Simons (2006) found negative effects of early maturation for both girls and boys on internalizing and externalizing symptoms. We have also reported on comparable effects of early timing on externalizing behaviors among African American and Latino youth (Lynne, Graber, Nichols, Brooks-Gunn, & Botvin, 2007). While variations in findings by subgroups exist, to date, the trend has been toward more similarity than differences in early timing effects.

It should be noted that late maturation also has negative effects on girls during mid adolescence, but these tend to disappear over time. In general, late maturation has been associated with positive adult outcomes for young women (e.g., Graber et al., 2004). For boys, late maturation is associated with subclinical problems during adolescence and substance and disruptive behavior problems during the entry into adulthood.

For the purposes of this discussion, our focus is on early maturation, why these effects emerge, and the trajectories they may be associated with. In particular, whereas early maturation may be linked to internalizing and externalizing problems and disorders, prevention or intervention efforts are not greatly advanced by this basic information. Some parents and pediatricians do in fact treat early maturation directly in order to offset potential negative experiences of early timing. Specifically in girls, medications developed for treating clinically precocious puberty are used to stop or slow the progression of puberty in girls who are early maturers, yet still within the normal range of maturation (for a discussion, see Graber 2008). In contrast to considering direct intervention on puberty, we seek to examine explanatory models that shed light on factors that may be more amenable to prevention or health promotion efforts.

Puberty and Timing in Developmental Context

Brain Development. As Grumbach and Styne (2003) noted, puberty is not a *de novo* event. It begins during fetal development with the organization of the hypothalamus–pituitary–gonadal (HPG) system but is essentially dormant postnatally. Activation of the system is indicated by increases in gonadotropin-releasing hormone (GnRH) in childhood. Despite years of research, the specific trigger that results in GnRH release is unknown (Susman & Dorn, 2009). Timing of onset for any single individual is likely the result of a complex set of processes including genes and gene expression, changes in neurotransmitter activities (i.e., GABA and glutamate), and hormones, such as leptin, that are associated with growth and adiposity (Banerjee & Clayton, 2007; Styne & Grumbach, 2007).

These neuroendocrine changes do not happen in isolation but are influenced by environment and behavior; for example, nutrition, health, and physical activity have long been known to influence the timing of onset and rate of progression of puberty (e.g., Frisch, 1983). Moreover, it is often forgotten that HPG activation is interconnected with other biological and physiological changes associated with growth and maturation. As described by Casey, Jones, and Hare (2008), recent studies of adolescent brain development have demonstrated substantial changes in maturation of the dopaminergic system, particularly during early adolescence. Dopaminergic systems are associated with processing of social information; for example, human and animal studies suggest that these neural developments are linked to increased sensation seeking or reward sensitivity during early to mid adolescence (e.g., Steinberg, 2008, this volume). In contrast, neural changes associated with improved cognitive control processes (e.g., executive function, emotion regulation) progress across the adolescent decade. The differences in the timing of these two dimensions of neural development potentially result in a period of heightened responses to socio-

emotional cues with lagging cognitive controls. Much of pubertal development occurs between the age of 9–13 for girls and 10–14 for boys, and dopaminergic systems are influenced by gonadal hormones. Thus, puberty may have an activating role on changes in dopaminergic systems and commensurate changes in processing social information observed during early adolescence. Delineation of both dopaminergic and cognitive control processes speak to developmental changes in reward sensitivity, risk taking, and more broadly decision-making for all adolescents.

In contrast, beyond what is experienced within the population, subgroups of adolescents are on pathways for serious psychopathology during adolescence and adulthood. Some evidence suggests that individuals with preexisting heightened emotionality may have elevated risk for more serious problems in early adolescence, or peak pubertal development (Casey et al., 2008), via the interaction of prior individual differences in brain development and puberty. However, less is known about individual differences in brain development making it difficult to ascertain the extent to which individual differences in neural processes are explanatory for effects of observed links between pubertal timing and adolescent risk behaviors.

Family Context. The assertion that puberty is not a “de novo event” applies to puberty as part of not only biological maturation but also as part of psychosocial development. For most children, the most consistent and persistent context of psychosocial development is the family. Numerous studies and reviews have considered parent–child relationship changes concurrent with puberty and across the adolescent decade (e.g., Laursen & Collins, 2009; Paikoff & Brooks-Gunn, 1991). Regardless of timing of maturation, children who enter adolescence with poorer quality family relationships are likely to continue to have low-warmth and high-conflict relationships, and higher risk for psychopathology during adolescence (Steinberg, 2001). And, early maturers, particularly girls, often experience poorer quality relationships than other youth during adolescence and even young adulthood (e.g., Graber et al., 1997, 2004). Impaired relationships may be one reason why early maturers have higher rates of symptoms and disorder. Or the reverse processes may be at work; that is, the stress of child psychopathology may have adverse effects on relationships. We have found evidence for both processes (for a review, see Graber & Sontag, 2009). However, the interconnections of parent–child relationships and psychopathology do not begin with puberty. While puberty may impact the course of psychopathology or its link with family relationships, family relationships influence the timing of the onset of puberty, itself.

In particular, Belsky, Steinberg, and Draper (1991) delineated an evolutionary model of socialization in which stressful family environments lead to behavioral problems and earlier onset of pubertal development as well as subsequent reproductive strategies. Stressful family environments have multiple dimensions; structure, relationship turmoil, and other factors may all be aspects of stress in the family environment. Several studies have now demonstrated that father absence during childhood as well as quality of family interactions actually predict earlier maturation in girls (for a listing of studies to date, see Susman&Dorn, 2009). Prospective studies,

including our own, have identified the quality of family relations as important predictors of earlier maturation in girls (e.g., Graber, Brooks-Gunn, & Warren, 1995). In our work, we demonstrated that low warmth and more conflict in family relations were associated with earlier maturation among a homogeneous sample of girls (i.e., with minimal father absence) accounting for maternal age at menarche and any initial signs of breast development. In subsequent work, Ellis, McFadyen-Ketchum, Dodge, Pettit, and Bates (1999) reported that affection in parent–child interactions when girls were in preschool (age 4–5) predicted pubertal timing differences in early adolescence. Most recently, Belsky et al. (2007) predicted earlier maturation in girls from parent–child interactions in early childhood and childhood in a large national longitudinal study. Across studies, poorer quality family relationships predict earlier maturation in girls with minimal or no effect in boys. In addition, separate from the quality of relationships or father absence, girls who experience sexual abuse prior to the onset of puberty also have earlier ages of menarche (Trickett & Putnam, 1993; Zabin, Emerson, & Rowland, 2005). As with poor quality family relationships, childhood sexual abuse has been identified as a predictor of several disorders, including depression (Trickett & Putnam, 1993).

Stress Processes. How psychosocial factors influence pubertal development has yet to be explained. Commonly, such experiences are thought of as stressors. Diathesis stress models have been central in explaining the development of psychopathology. In these models, neither stress nor vulnerability alone predicts subsequent disorder. Although significant stressful occurrences commonly precede depressive episodes, as well as other problems, and dysregulation of the physiological stress system occurs in the face of high levels of psychosocial stress, stressful events rarely fully explain changes in affect or onset of disorder. Sources of vulnerability or “diathesis” such as emotion regulation, physiological responses to stress, or temperament, often become stable during childhood and adolescence (Ingram & Luxton, 2005). Of course, the distinction of stressor versus vulnerability is rarely clear. In the case of pubertal timing, disentangling stressors and vulnerabilities is particularly challenging.

As described in other articles in this volume and elsewhere, activation of the limbic–hypothalamus–pituitary–adrenal (L-HPA) system occurs in response to novelty and stress—often social stressors. Animal studies and recent work in humans have demonstrated that early experiences may result in long-term alterations in the L-HPA system. Suomi (1999) in lab and natural settings has documented the effects of gene–environment interactions on indicators of adjustment in rhesus monkeys; specifically, developmental outcomes of genetic-based vulnerabilities in responses to stress were moderated by the quality of maternal behavior. In this work, reactive monkeys, those with particularly high or sustained physiological responses to stressors, had better or worse social outcomes depending upon the quality of maternal care in infancy (i.e., high versus low nurturance). The effects of parenting on the developing L-HPA system speak to how physiological vulnerabilities or perhaps invulnerabilities to stress may be established via early experience. Evidence in animal and human studies indicates that quality of care in infancy predicts subsequent L-HPA functioning—not merely interacting with

vulnerabilities but having an active role in establishing them. For example, Roisman et al. (2009) reported that blunted cortisol response upon wakening, as assessed in adolescence, was predicted by maternal sensitivity over the first 3 years of life and the time spent in child care. These effects were small but consistent with animal models and the emerging human literature on early deprivation.

Thus, in a diathesis-stress model of pubertal timing effects on emotion and behavior, atypical patterns of physiological responses to stress may be vulnerabilities, whereas off-time development may be a stressor. In our own work, we have begun to test such a model to determine whether atypical stress responses and early pubertal timing interact to predict development of psychopathology (Graber, Sontag, & Brooks-Gunn, 2009). Such models may explain the emergence of more serious problems during adolescence, both during and after puberty.

An important consideration is how both factors, stress and early timing, are associated with family relationships during puberty. In our study, atypical stress response was indicated by the failure to decrease in cortisol (over a 2-hr period) during a home visit; this response pattern was observed in about 20–25% of children. Of note is that interactions between stress response and timing were demonstrated for outcomes associated with specific social contexts—attention and effort in school, and family conflict—rather than general symptoms of adjustment. Early maturers who also had an atypical stress response demonstrated lowered attention and effort in school and experienced the greatest decline in this indicator from childhood (age 8–9) to early adolescence (age 11–12). Similar interactions were found for frequency and intensity of conflict between mothers and children with early maturers with atypical stress responses having higher rates of conflict in comparison to other children. As can be seen in Figure 1, other groups, that is early maturers with typical stress responses and non-early maturers regardless of stress response were equivalent in family conflict and school-related behaviors. Although indicators of family conflict did not change from childhood to early adolescence, the interaction effect was found at both times.

These findings are consistent with the hypothesis that family factors are inter-related developmentally with stress responses and maturational timing. In addition, family factors may be explanatory for early maturation effects on some aspects of adjustment at least within this window of development. One caveat is that we found timing and stress response interactions for boys and girls, whereas the effects of family stress on timing are most often demonstrated in girls and not boys. As such, psychopathology risks for early maturation also occur separately from family relationships that set girls on the course for early maturation. Thus, multiple pathways need to be tested to explain early timing effects. In addition, the literature on physiological responses to stress has at times been contradictory and does not lead to clear implications for how to offset the establishment or continuation of atypical stress responses across childhood.

Connecting Timing Pathways Across Development

Until recently, research on pubertal timing has focused on factors that predict either timing of pubertal onset and progression, or adolescent outcomes via pubertal timing. A few select studies have sought to identify pathways that encompass both aspects of timing. For example, using data from the Oregon Youth Substance Use Project (OYSUP), Lynne-Landsman, Graber, and Andrews (in press) have examined levels of family risk, across 1st–6th grades, in connection with subsequent pubertal timing and subsequent substance use in 8th grade. Numerous family factors have been identified as predictors of substance initiation and use during adolescence; thus, as expected, high family risk during childhood predicted higher cigarette, alcohol, marijuana, and inhalant use in 8th grade. Although only 16% of the sample was classified as having the highest family risk, 56% of early maturers were in the high-risk group. Moreover, early maturation was associated with elevated substance use only among those in high family risk environments. Interestingly, as with our aforementioned study, there was no gender difference in these findings; early maturation was associated with higher substance use for both girls and boys with high levels of family risk.

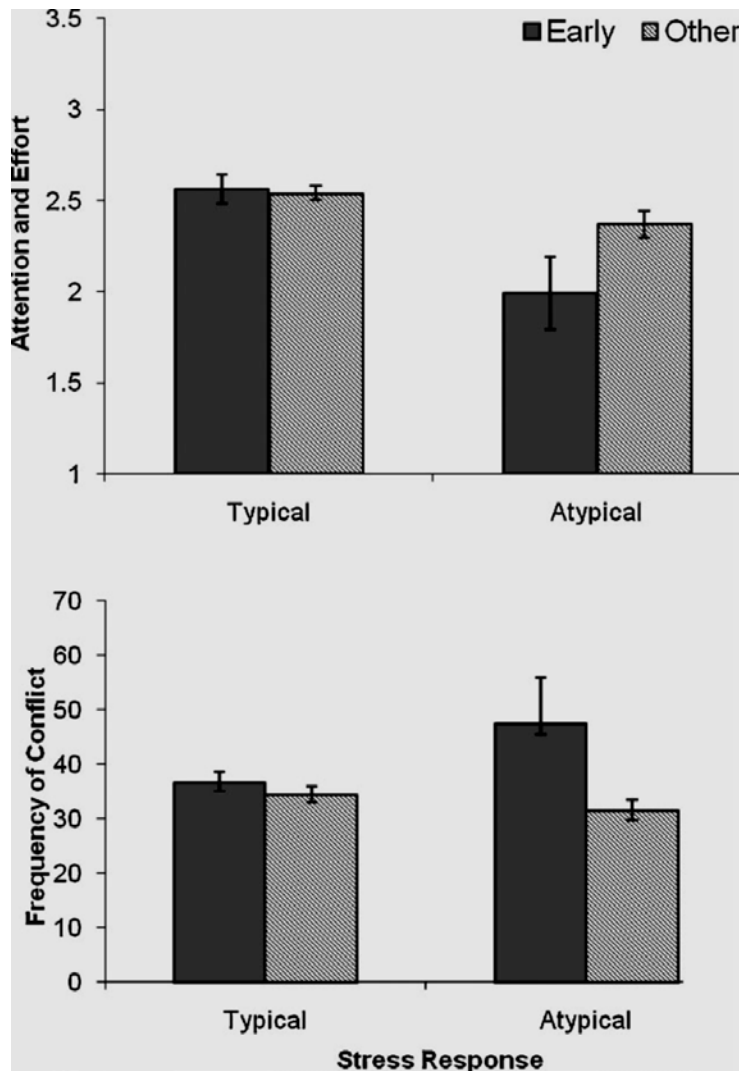


Figure 1. Interaction effects of atypical stress response (as indexed by pattern of change in cortisol) with early maturational timing. Effects are shown for boys and girls, 8–9 years of age. Upper graph shows the adjusted means for Attention and Effort in the classroom environment and the lower graph shows adjusted means for Frequency of Conflict in the past month. Error bars indicate significant differences.

Some adolescents, both early maturers and others, will likely initiate substance use via other developmental pathways; yet, these findings provide partial explanation of the early timing effects on an externalizing outcome via family risk. Given that there was no gender difference, family effects on timing in girls may be a by-product of the primary diathesis-stress model for substance use. That is, family stress interacts with early maturation; for girls, early maturation may have been causally linked with earlier family stress but that association need not be present for the development of early substance use as observed in both boys and girls.

Expanding Pathways to Emerging Relationships/Peer Processes

Understanding puberty in context extends beyond the family context especially for early timing effects on adolescent risk. Examination of emerging romantic relationships and peer processes may speak to pathways that are more salient for girls in contrast to boys and more salient for other outcomes such as internalizing symptoms and disorders. For example, affiliative stress, stress in one's relationships, or even in one's broader network of relationships is predictive of internalizing problems in girls and women (Graber & Sontag, 2009) likely because of the increased salience of interpersonal relationships to females in general (Kessler & McLeod, 1984; Rudolph, 2002). Initial studies of early maturation in girls demonstrated that externalizing problems such as earlier initiation of alcohol use were accounted for by affiliation with older peers (Stattin & Magnusson, 1990). In our own work, we found that affiliation with deviant peers was important for early timing effects on behavior in both boys and girls (Lynne et al., 2007). In this study, deviant peer affiliation accounted for early timing effects in aggressive and delinquent behavior across the middle school years (Lynne et al., 2007). Across several studies, affiliation with deviant peers as well as romantic partners who engaged in delinquent behaviors has been linked to increased psychopathology among early maturing girls (e.g., Halpern, Kaestle, & Hallfors, 2007). Thus, the transition into new relationships may be a critical factor in adjustment pathways, especially for adolescent girls, but likely for both boys and girls.

As noted, persistent deficits in interpersonal relationships such as lowered social support from family and friends occur at higher rates for early maturing girls and young women (Graber et al., 2004). Drawing upon AddHealth, Foster, Hagan, and Brooks-Gunn (2004) further delineated interpersonal relationships that led to entry onto high-risk health pathways for adolescent girls. In this study, they focused on the experience of partner violence, both verbal and physical, as it has known links to subsequent adjustment problems. Accounting for SES factors and prior delinquency, early maturation was associated with higher rates of physical and verbal partner

violence among girls. Notably, experience of abuse prior to adolescence was also an independent predictor of increased risk of experiencing intimate partner violence.

Foster et al. (2008) have since developed the “subjective weathering” hypothesis, a model for girls’ development of depressive symptoms across adolescence that links family stress, early maturation, and romantic partner violence along with role transitions. This hypothesis draws upon the weathering hypothesis, in which Geronimus (1994) proposed that accelerated aging or physical weathering occurs among subgroups of women experiencing an accumulation of environmental, contextual stress exposure such as institutionalized racial/ethnic discrimination or socioeconomic disadvantage. Physical weathering is evidenced by disparities in health and mortality rates such as earlier onset of chronic disease and earlier mortality among disadvantaged groups.

Subjective weathering refers to the psychological experience of feeling that one is aging more rapidly or has an accelerated life course (Foster et al., 2008). Both physical and subjective weathering may be mechanisms that link childhood stress, maturational timing, and subsequent adult health-related behaviors and outcomes in females over the first decades of life. Using adolescent and adulthood data from AddHealth, Foster et al. (2008) found support for their model (see Fig. 2). Childhood trauma predicted earlier maturation in girls; earlier maturation was associated with increased risk for partner violence during adolescence, along with lowered rates of high school graduation; both partner violence and early maturation contributed to accelerated role transitions and subjective and physical weathering by young adulthood; these processes predicted depressive symptoms. Interestingly, as part of subjective weathering, early maturing young women reported feeling like they were older than other young adults—taking on more adult responsibilities, growing up faster. Such perceptions are, to some extent, accurate in that these young women had experienced more interpersonal traumas and stress resulting in earlier entry into adult roles. Further model testing may extend this model to other outcomes or this may be unique to depressive symptoms in girls and young women.

IMPLICATIONS FOR PREVENTION

In as much as puberty or pubertal timing are not targets of prevention but rather normative developmental transitions, accruing evidence for explanatory pathways sheds light on more meaningful targets of prevention. For example, findings on early maturation and atypical stress responses highlight the need to promote better coping strategies among pre-adolescents.

Currently many effective universal preventive interventions incorporate the teaching of emotion regulation, problem-solving, and anxiety reduction techniques. However, in studies conducted on program fidelity in the dissemination of evidenced-based interventions, sessions using interactive skill-building activities, such as emotion regulation and other coping strategies, are the most likely to be dropped due to time constraints and/or provider discomfort with the material (Ennett et al., 2003; Hansen & McNeal, 1999). Findings from the pubertal timing literature certainly

reinforce the need for the prevention field to strengthen implementation fidelity for these components.

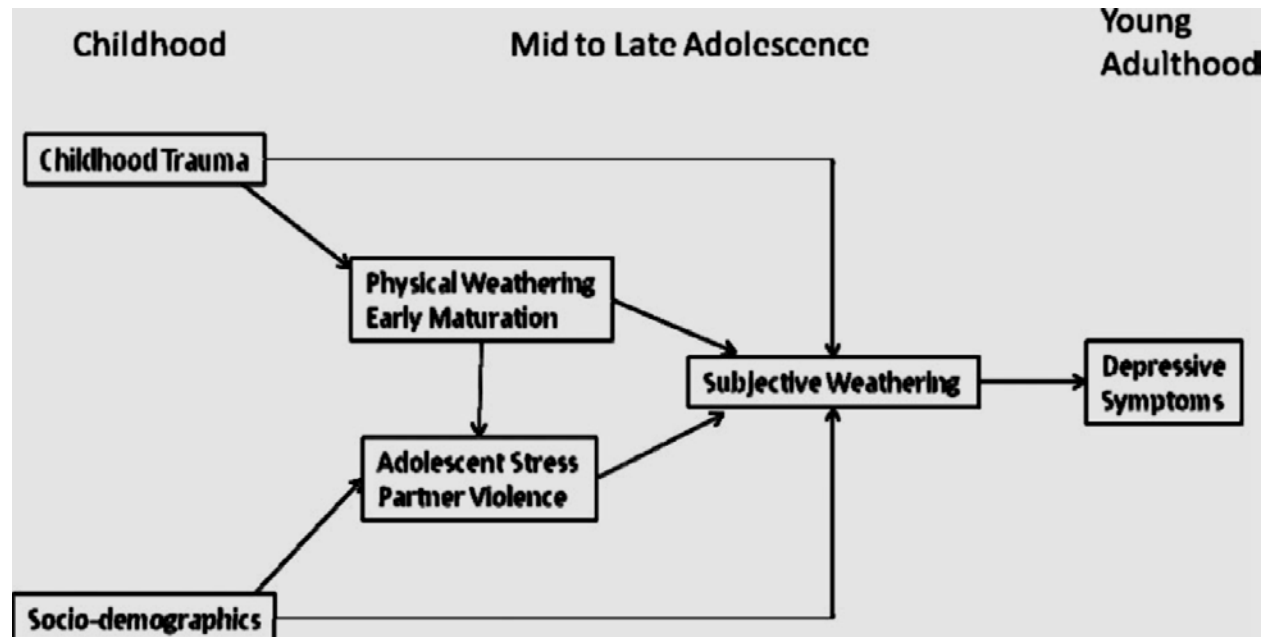


Figure 2. Subjective Weathering" model linking childhood experiences, maturational timing, and depressive symptoms. Redrawn from Foster et al., 2008.

As was consistently demonstrated, early maturation and stress are interconnected with family processes, making family-based initiatives an important approach for preventive interventions. This review demonstrates the need for family-based prevention initiatives to target relationship quality and to begin such initiatives in early childhood much like protocols proposed by Hussong (this volume). However, it may also be important for family based interventions targeting adolescents to address pubertal concerns directly. Mothers are perceived as being responsible for girls' education regarding menstruation and menarche (Costos, Ackerman, & Pardis, 2002; Houppert, 1999), yet most mothers report discomfort discussing the topic (Brumberg, 1997; Moore, 1995). Also, girls generally report dissatisfaction with the amount and quality of interactions with their mothers regarding menarche (Beausang & Razor, 2000; Costos et al., 2002). Likewise some girls have reported embarrassment and anger over their mother's insistence on discussing the event either privately or within the family (Martin, 1996; Uskul, 2004). Parental communication to daughters on pubertal changes in general can carry messages on the dangers of sexuality (Hawthorne, 2002; Martin, 1996; O'Sullivan, Meyer-Bahlburg, & Watkins, 2000; Teitelman, 2004), which may increase girls' discomfort with the subject (Beausang & Razor, 2000; Uskul, 2004). In addition, parents, who believe menstruation is a signal to males that a girl is sexually available, report using silence as a way to protect their daughters from sexual advances (Hawthorne, 2002). Therefore, parent-child communication components within preventive interventions should include how to address puberty in general,

menstruation in particular, and psychosocial effects of early maturation, rather than just any one of these topics.

Since families are not the only pathway to adolescent problem behavior, effective preventive interventions need to include peer-focused initiatives. Many social-skills based programs focus on the teaching of refusal skills. However, in light of the complexities of adolescent friendship networks and romantic relationships, it is unclear if refusal skill training alone is sufficient, especially among early maturing youth. Instead preventive interventions may need to focus on altering the formation of unhealthy peer networks and addressing the motivation of early maturing youth to develop unhealthy peer networks.

While altering the timing of pubertal development in order to decrease the number of early maturing youth is not possible, tailored interventions may assist in the appropriate delivery of preventive intervention messages to youth who are on different developmental trajectories. Tailored communication allows for the delivery of select messages and materials relevant to individuals within a target population based on each person's behavioral, attitudinal, or environmental characteristics (Kreuter, Farrell, Olevitch, & Brennan, 2000). This approach uses individual assessments to create messages and feedback written specifically for the individual. By assessing where an adolescent's development lies relative to her/his peers prior to the delivery of a preventive intervention, messages and materials can be designed to address the unique contextual issues that are associated with pubertal timing. Tailoring allows the intervention to incorporate contextual elements of individuals' daily lives that are directly linked to the outcomes of interest. Although tailoring is a relatively new methodology, evidence for its effectiveness is accruing, with both tailored print and computer-based health education messages showing greater effectiveness than regular health information (Kreuter et al., 2000).

Thus, while early maturation is only one of several potential risks for adolescent health, it is uniquely associated with substantial risk. At the same time, the context in which puberty occurs likely provides the most realistic opportunities for preventive interventions that would improve outcomes for early maturers and youth in general. Moreover, prevention research that incorporates information on timing with program evaluation is needed to determine whether different prevention strategies would, in fact, help to ameliorate risk for early maturing youth.

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