Personal-Accentuation and Contextual-Amplification Models of Pubertal Timing: Predicting Youth Depression

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Abstract

This research examined personal-accentuation and contextual-amplification models of pubertal timing, wherein personal and contextual risks magnify the effects of earlier pubertal maturation on youth depression. A sample of 167 youth (*M* age = 12.41 years, *SD* = 1.19) and their maternal caregivers completed semi-structured interviews and questionnaires at two waves. Consistent with a personal-accentuation model, earlier pubertal maturation more strongly predicted subsequent depression in youth with prior depression, certain personality traits, and maladaptive stress responses than in youth without these personal risks. Several of these effects were specific to earlier-maturing girls. Consistent with a contextual-amplification model, earlier pubertal maturation more strongly predicted subsequent depression in youth exposed to recent maternal depression and family stress than in youth without these contextual risks. These findings identify key characteristics of youth and their family context that help to explain individual variation in depressive reactions to earlier pubertal maturation. More broadly, this research contributes to integrative models of depression that consider the interplay among personal vulnerability, contextual risk, and developmental transitions.

Keywords

pubertal timing; depression; moderators

Developmental theory and research characterize the pubertal transition as a challenging stage, characterized by a confluence of biological, psychological, and social disruptions (Alsaker, 1995; Angold, Worthman, & Costello, 2003; Paikoff & Brooks-Gunn, 1991; Petersen & Taylor, 1980; Susman & Rogol, 2004). Moreover, this transition serves as a developmental context of risk for the onset of depression, especially in girls (Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Costello et al., 2003; Hankin et al., 1998; for a review, see Rudolph, Hammen, & Daley, 2006). Progressing through puberty early relative to one's peers poses a particularly strong risk for depression, especially in girls (for reviews, see Alsaker, 1995; Graber, 2003). Yet, many adolescents negotiate this stage with few ill-effects (Graber, 2003). Despite this variation, surprisingly little is known about what determines individual differences in depressive reactions to puberty. The goal of this research was to examine two models of pubertal timing—a personal-accentuation model and a contextual-amplification model—that explain these individual differences. According to these models, characteristics of youth and their contexts either magnify or temper the adverse emotional consequences of earlier pubertal maturation.[1]

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Pubertal Development and Depression

Biopsychosocial perspectives on puberty focus on the interacting biological, physical, psychological, and social changes that mark this transition (DeRose, Wright, & Brooks-Gunn, 2006; Graber, 2003; Lerner, 1998; Magnusson, 1988; Petersen & Taylor, 1980; Susman & Rogol, 2004). These changes have the potential to confer both advantages and risks. Whereas adolescence may be a time of increasing competence and resilience, as reflected in heightened cognitive and emotional capacities, it also may be a time of increasing risk for vulnerable youth (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009). With regard to risk, a growing body of research suggests that both biological (e.g., hormonal variation) and physical (e.g., morphological and other somatic changes) aspects of puberty contribute to depression and associated psychological distress (for reviews, see DeRose et al, 2006; Susman & Rogol, 2004). Several models have been proposed to explain these effects (for reviews, see Brooks-Gunn, Graber, & Paikoff, 1994; DeRose et al., 2006; Paikoff & Brooks-Gunn, 1991; Petersen & Taylor, 1980), including direct pathways (hormonal changes directly influence negative emotions and depression; Angold, Costello, Erkanli, & Worthman, 1999; Angold et al., 2003; Susman, Dorn, & Chrousos, 1991), indirect pathways (biological and physical changes stimulate psychological disruption and social reorganization, which then heighten risk for depression; Graber, Brooks-Gunn, & Warren, 2006), and interactive pathways (biological and physical changes interact with psychological and social challenges to predict depression; Conley & Rudolph, 2009; Graber, 2003; Magnusson, 1988).

Beyond the independent or interactive effects of various components of puberty, biopsychosocial perspectives increasingly implicate youths' *timing* of puberty (i.e., pubertal status relative to age; Graber et al., 2006; Susman & Rogol, 2004; Weichold, Silbereisen, & Schmitt-Rodermund, 2003) as a key determinant of risk for depression and associated distress (for reviews, see Alsaker, 1995; Weichold et al., 2003). According to the stage-termination hypothesis, earlier maturation presents a particular risk for maladjustment. Earlier-maturing youth face the adolescent transition before they have successfully completed prior developmental tasks and developed the skills necessary to cope with the challenges of adolescence (Peskin, 1973; Petersen & Taylor, 1980). Thus, earlier-maturing youth are faced with a great deal of novelty and ambiguity that they are required to negotiate prior to many of their peers (Caspi & Moffitt, 1991). Moreover, earlier-maturing youth tend to be exposed to riskier social contexts (e.g., affiliation with older and norm-breaking peers; Weichold et al., 2003) and thus may face even greater challenges than their on-time and later-maturing peers.

In support of this hypothesis, earlier-maturing girls show heightened risk for depression and associated emotional difficulties relative to their on-time and later-maturing peers (Brooks-Gunn & Warren, 1985; Ge, Conger, & Elder, 1996; Ge, Conger, & Elder, 2001a; Hayward et al., 1997; Kaltiala-Heino, Kosunen, & Rimpela, 2003; Siegel, Aneshensel, Taub, Cantwell, & Driscoll, 1998; Stattin & Magnusson, 1990) although some research does not support this effect (Angold, Costello, & Worthman, 1998). Findings among boys are mixed (for a review, see Huddleston & Ge, 2003). Whereas some research links elevated depression and internalizing symptoms with earlier maturation (Alsaker, 1992; Ge, Brody, Conger, & Simons, 2006; Ge, Conger, & Elder, 2001b; Kaltiala-Heino et al., 2003; Susman et al., 1991; Susman et al., 1985), other research links these symptoms with later maturation

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[1]To distinguish between the moderating role of personal versus contextual risks, the term *accentuation* is used to describe a model that focuses on personal vulnerabilities (see Caspi & Moffitt, 1991) whereas the term *amplification* is used to describe a model that focuses on contextual risks (see Ge, Brody, Conger, Simons, & Murry, 2002). However, these models propose the same type of moderating effect, namely that the adverse effects of earlier maturation are magnified under certain conditions.

(Benjet & Hernández-Guzmán, 2002; Crockett & Petersen, 1987; Dorn, Susman, & Ponirakis, 2003; Huddleston & Ge, 2003; Weichold et al., 2003). These inconsistencies are likely accounted for, in part, by methodological discrepancies across studies but also may stem from individual differences in youths' emotional reactions to earlier maturation, suggesting the need for more complex models of pubertal timing effects.

Integrative Models of Pubertal Timing Effects

Developmental psychopathology perspectives highlight the complex interactions among personal characteristics of youth, their contexts, and developmental factors in shaping trajectories of psychological health or ill-being (Cicchetti & Cohen, 2006; Sameroff, 1987). In this vein, developmental scientists emphasize the need to view puberty in context (Caspi & Moffitt, 1991; Conley & Rudolph, 2009; Ge et al., 1996; Graber, 2003; Paikoff & Brooks-Gunn, 1991). According to Caspi and Moffitt's (1991) accentuation model, earlier pubertal timing accentuates pre-existing individual differences in vulnerability. More specifically, this model proposes that dispositional characteristics of youth are most likely to be expressed during periods of social change, when youth are faced with novelty, ambiguity, and challenge. During these times, youth with strong internal resources will cope effectively, thereby buffering them from distress, whereas youth with vulnerabilities will show ineffective coping, thereby accentuating their vulnerabilities. Consistent with this model, earlier menarche magnifies aggression and delinquent behavior in girls who show childhood behavioral problems (Caspi & Moffitt, 1991) and who engage in maladaptive responses to stress (Sontag, Graber, Brooks-Gunn, & Warren, 2008). Similarly, earlier maturation magnifies psychological distress (i.e., internalizing symptoms and depression) in girls with prior elevated distress (Ge et al., 1996).[2]

Researchers also have proposed that the adverse effects of earlier maturation are amplified by contextual factors (Caspi, Lynam, Moffitt, & Silva, 1993; Ge et al., 1996; Graber, 2003; Magnusson, 1988; Rudolph, 2009; Simmons & Blyth, 1987). Indeed, findings across a few studies suggest that earlier maturation interacts with various aspects of youths' contexts, including the sex (Ge et al., 1996) and age (Stattin & Magnusson, 1990) composition of their peer groups, the quality of their peer relationships (Conley & Rudolph, 2009), their engagement in romantic relationships (Natsuaki, Biehl, & Ge, 2009), and their exposure to life stress (Ge et al., 2001a; Silberg et al., 1999) to predict depression and associated distress. Interestingly, one study revealed that pubertal status (based on hormonal assessment) did not interact with life stress to predict depression (Angold et al., 2003). Given that pubertal timing, particularly when assessed in terms of visible somatic changes, has significant psychological and social implications, its effects may be more susceptible than hormonal status to moderation by social context.

The present research was designed to extend these personal-accentuation and contextual-amplification models to identify other characteristics of youth and their contexts that magnify or temper the adverse influence of earlier pubertal maturation on depression. In particular, this study focused on (a) three types of personal risks—prior depression (i.e., lifetime history and recent symptoms), personality attributes of youth (i.e., depressive personality traits and negative self-focus), and maladaptive responses to stress, and (b) two

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[2]Note that the Caspi & Moffitt (1991) model construes earlier puberty as a moderator (and accentuator) of the effects of prior dispositional characteristics on adjustment. In contrast, the present research construes prior dispositional characteristics as a moderator (and accentuator) of the effects of earlier puberty on adjustment. In both models, earlier pubertal timing is presumed to confer risk in part due to the novelty and challenges presented by this experience. Moreover, both models predict statistical interactions between earlier puberty and personal risks. The models diverge in the statistical interpretation of these interactions: Whereas the former model frames pubertal timing as the moderator, the latter model frames personal risks as the moderator.

types of contextual risk—exposure to lifetime and recent maternal depression, and exposure to lifetime and recent family adversity.

Personal Risk

Prior depression

According to a personal-accentuation model (Caspi & Moffitt, 1991), depressed youth would be particularly ill-equipped to cope with the challenges of earlier maturation. Depressive symptoms may directly interfere with youths' negotiation of this developmental challenge. For example, symptoms such as anhedonia and fatigue may undermine motivation and energy that are needed to deal with the many changes that youth encounter during this transition; symptoms such as low self-worth may intensify the potential negative self-evaluative consequences of earlier maturation. Recent depressive symptoms also may indirectly intensify subsequent depressive reactions to earlier maturation. Research shows that earlier-maturing depressed youth generate more stress in their relationships (Rudolph, 2008) and show greater affiliation with deviant peers (Ge et al., 1996) than on-time and later-maturing depressed youth, suggesting that these youth actually face more challenging contexts that could exacerbate their risk for subsequent depression. Beyond recent depressive symptoms, an earlier history of depression may magnify the depressive consequences of earlier maturation. Depression may lead to long-term deficits in a variety of competencies (Rohde, Lewinsohn, & Seeley, 1990) and may undermine the development of supportive social networks (Stice, Ragan, & Randall, 2004), thereby compromising youths' ability to successfully negotiate the pubertal transition. Thus, the adverse effects of earlier maturation on subsequent depression may reflect, in part, continuity or exacerbation of prior disorder and associated impairment (Caspi & Moffitt, 1991).

Personality attributes

Earlier maturation also may present difficulties for youth with certain personality attributes. The adolescent transition is marked by a heightened focus on self-evaluation, social comparison, and self-consciousness (Harter, 1990; Tobin-Richard, Boxer, & Petersen, 1983), stemming in part from the "social-stimulus value" (Brooks-Gunn & Warren, 1989, p. 41) linked to the somatic manifestations of puberty. Because earlier-maturing youth and others in their social contexts are likely to be acutely aware of their differing physical and social status, this self- and social scrutiny may be intensified. Although earlier-maturing youth with a positive sense of self and an optimistic outlook on the world may emerge relatively unscathed from this scrutiny, those with a negative view of the self and a pessimistic outlook on the world may be emotionally vulnerable, resulting in heightened depression. The present study examined the hypothesis that personality traits characterized by a negative preoccupation with the self and the world, namely depressive personality traits and negative self-focus, would heighten earlier-maturing youths' susceptibility to depression.

Depressive personality traits and negative self-focus are conceptualized as relatively stable individual differences in personality. Negative self-focus reflects a tendency to engage in negative self-evaluation, social comparison, and excessive concern about social evaluation. Depressive personality reflects a tendency toward self-criticism, pessimism, brooding, passivity, and negative preoccupations (Klein, 1990; Rudolph & Klein, 2009). Although depressed individuals often express such tendencies, these personality attributes are viewed as stable characteristics that exist even in the absence of symptoms. Consistent with this view, research indicates that such characteristics are distinct from depressive disorders (Klein, 1990; Klein & Shih, 1998). Moreover, they predict depression over time after adjusting for earlier depression (Rudolph & Klein, 2009), supporting the idea that these

personality attributes serve as a risk factor rather than merely an indicator of depression. It was expected that these personality traits would accentuate the depressogenic effects of earlier maturation.

Coping and responses to stress

Earlier-maturing youth face significant reorganization in their peer relationships (e.g., affiliation with older peers, entrance into deviant peer groups, early onset of romantic and sexual relationships) and heightened interpersonal stress (Ge et al., 1996; Rudolph, 2008; Stattin & Magnusson, 1990; for reviews, see Silbereisen & Kracke, 1993; Weichold et al., 2003). Internal resources for coping with these relationship challenges likely play a large role in determining individual differences in depressive reactions to earlier maturation. Specifically, youth who engage in goal-directed efforts to cope with interpersonal stress (e.g., problem solving, emotion regulation) are likely to be successful at negotiating these complex social changes, and may therefore be buffered from the depressogenic effects of earlier maturation. In contrast, youth who show involuntary, dysregulated responses to interpersonal stress (e.g., rumination, physiological arousal, denial, inaction) are likely to be more susceptible to the depressogenic effects of earlier maturation.

Contextual Risk

Maternal depression

Youth with depressed mothers may be at a disadvantage when negotiating the challenges of earlier maturation. Depressive symptoms deplete maternal resources for engaging in warm and nurturing parenting (for a review, see Goodman & Gotlib, 1999), thereby interfering with efforts to provide scaffolding and support during this stressful time. Even youth whose mothers have a prior history of depression may be ill-prepared to deal with earlier maturation. Maternal depression is linked to a range of difficulties in youth, including interpersonal deficits and heightened stress (Hammen, Shih, & Brennan, 2004), which may compromise youths' efforts to cope with earlier maturation. Of course, maternal depression also may represent a genetic marker of risk; in this case, onset of depression during the pubertal transition may reflect the developmental expression of a genetic liability rather than a functional association between maternal and youth depression. In either case, earlier maturation would be more strongly linked to depression in youth of currently or formerly depressed mothers than in youth of nondepressed mothers.

Family adversity

Exposure to family adversity also may moderate the depressogenic effects of earlier maturation. Earlier-maturing youth who are exposed to stressful family contexts face simultaneous challenges that may overwhelm their psychological resources (Simmons & Blyth, 1987). Moreover, puberty in general, and earlier maturation in particular, stimulates changes and challenges in family relationships (Paikoff & Brooks-Gunn, 1991; Steinberg, 1987). Whereas healthy families can likely mobilize the necessary resources to deal effectively with these challenges, unhealthy families (e.g., those with high levels of disruption, conflict, or stress) typically lack these resources (Paikoff & Brooks-Gunn, 1991). Finally, youth exposed to severe adversity may suffer from a host of difficulties (e.g., insecure sense of self, interpersonal deficits, emotion regulation deficits; for reviews, see Geiger & Crick, 2001; Rudolph, 2009) that interfere with their ability to deal effectively with earlier maturation. Thus, earlier-maturing youth exposed to a history of family adversity and recent family stress may be more susceptible to depression than those in healthy family contexts.

Sex Differences in the Personal-Accentuation and Contextual-Amplification Effects of Earlier Maturation

As discussed earlier, research provides a mixed picture of sex differences in the contribution of earlier maturation to depression. Whereas some research suggests that these effects are specific to girls, other research notes a similar pattern in boys (for reviews, see DeRose et al., 2006; Huddleston & Ge, 2003). In general, there are sex-differentiated implications of puberty that often favor boys (e.g., a gain in status; increases in desirable somatic characteristics such as muscularity and strength that confer social value; Alsaker, 1995; Weisfeld, 1999). Moreover, earlier-maturing girls and boys may differ in their susceptibility to personal-accentuation and contextual-amplification effects; thus, examining sex differences in these effects may provide insight into the contribution of earlier maturation to depression in girls versus boys.

Both personal and contextual risks may magnify the depressogenic effects of earlier maturation to a greater extent in girls than in boys. First, some research suggests that depression generates more problems in girls' than boys' relationships (Prinstein, Borelli, Cheah, Simon, & Aikins, 2005; Rudolph, Ladd, & Dinella, 2007). Depressed girls may therefore face more difficulty than depressed boys navigating the social challenges of earlier maturation. Second, because many of the puberty-induced somatic changes are socially undesirable in girls but socially desirable in boys (Alsaker, 1995; Petersen, Silbereisen, & Sorensen, 1996; Tobin-Richards et al., 1983), a tendency toward negative preoccupation and brooding, as reflected in depressive personality traits and negative self-focus, is more likely to magnify the depressogenic effects of earlier maturation in girls than in boys. Third, compared to boys, girls place more emphasis on maintaining intimate relationships and resolving interpersonal problems, and show more dependency (Rose & Rudolph, 2006). Thus, girls with maladaptive styles of responding to stress may have more trouble than boys negotiating the challenging interpersonal dynamics associated with earlier maturation. Fourth, because maternal depression (Sheeber, Davis, & Hops, 2002) and interpersonal stress (Hankin, Mermelstein, & Roesch, 2007) predict depression more strongly in girls than in boys, earlier-maturing girls may be more vulnerable than boys to the amplifying effects of maternal depression and family adversity on depression.

Overview of the Present Study

In sum, this study examined the proposal that pre-existing personal and contextual risks would magnify the contribution of earlier maturation to subsequent depression. Moreover, girls were expected to be especially sensitive to these effects. These hypotheses were examined in the context of a longitudinal study investigating the emergence of depression over a one-year period. Youth and their maternal caregivers completed semi-structured interviews to assess youth and maternal depression, depressive personality traits, and family adversity (i.e., lifetime history and recent stress). Participants also completed questionnaire-based measures of puberty, negative self-focus, and responses to interpersonal stress. Because the present study focused on the psychological and social implications and context of puberty, morphological development and other somatic manifestations of puberty were the target of assessment.

Method

Participants

Participants were 167 youth (86 girls, 81 boys; *M* age = 12.41 years, *SD* = 1.19; 77.8% White, 22.2% minority) and their maternal caregivers (88.6% biological mothers; 1.8% stepmothers; 4.2% adoptive mothers; 5.4% other) recruited from several Midwestern towns.

Families represented a range of socioeconomic classes as reflected in income level [16.7% below 30,000; 48.7% $30-59,999; 21.6% $60,000-89,999; 13.0% over $90,000]. Youth were selected for the longitudinal study based on school-wide screenings with the Children's Depression Inventory (Kovacs, 1992). Youth with a range of CDI scores were recruited, over-sampling slightly for youth with severe symptoms (15.8% of the screening sample, 20.3% of targeted youth, and 24.1% of recruited youth had scores > 18). Participants were recruited based on CDI scores, having a maternal caregiver in the home, and proximity (within one hour) to the university. Exclusion criteria included having a non-English speaking maternal caregiver or having a severe developmental disability that interfered with the assessment.

Youth whose families did and did not consent to participate in the study did not differ in sex, $\chi^2(N = 468, df = 1) = .39$, *ns*, ethnicity (white versus minority), $\chi^2(N = 468, df = 1) = .02$, *ns*, or CDI scores, $t(280) = 1.11$, *ns*. Participants ($M = 12.41$, $SD = 1.19$) were slightly, but not meaningfully, younger than nonparticipants ($M = 12.65$, $SD = .89$), $t(275) = 2.28$, $p < .05$. Relevant data (i.e., depression scores) were available for 151 (96%) of the original sample at Wave 2 (W_2). Youth without data at W_2 did not differ from those with complete data in sex, $\chi^2(N = 158, df = 1) = .08$, *ns*, age, $t(156) = .20$, *ns*, ethnicity (white versus minority), $\chi^2(N = 158, df = 1) = 1.54$, *ns*, or any of the W_1 variables, $ts(134-156) \leq 1.50$, *ns*, with the exception of depressive personality, $t(137) = 3.63$, $p < .001$. Specifically, youth who dropped out of the study had significantly higher scores on depressive personality ($M = 1.98$, $SD = .62$) than did youth who remained in the study (M = 1.41, SD = .26). Multiple imputation procedures were used to deal with missing data (see Results).

Procedures

Families were recruited through phone calls to the primary maternal caregivers. Interested families completed an in-person, three- to four-hour initial assessment. Caregivers provided written informed consent, and youth provided written assent. Youth and their caregivers were then interviewed separately. A follow-up interview was completed one year later. To compensate families for their time, caregivers were given a monetary reimbursement and youth were given a gift certificate at each assessment.

To avoid contamination of the diagnostic and life stress information, two different staff members conducted these interviews. Diagnostic interviews were conducted by a faculty member in clinical psychology, a post-doctoral fellow in clinical psychology, psychology graduate students, or a post BA-level research assistant. All diagnoses were made through consultation with a clinical psychology faculty member or a post-doctoral student in clinical psychology. Life stress interviews were conducted by a post-doctoral student in clinical psychology, psychology graduate students, a post BA-level research assistant, or advanced undergraduate students. Interviewers underwent extensive preliminary training with the first author (or, for the life stress interview, a highly trained experienced interviewer) that involved: (a) reviewing the interview protocols and diagnostic criteria, (b) listening to prior audiotaped interviews and making independent diagnoses, and (c) role-playing and feedback. They also received extensive feedback based on audiotapes of their interviews, as well as during consensual coding sessions.

Measures

Table 1 presents descriptive data on the measures for girls and boys.

Assessment of Pubertal Timing

Pubertal maturation—Two measures of pubertal status were administered. First, youth and caregivers completed the Pubertal Development Scale (PDS; Petersen, Crockett,

Richards, & Boxer, 1988). This measure includes five questions that assess stage of growth spurt, body hair, skin changes, voice changes and facial hair (boys), and breast development and menarcheal status (girls) using a 4-point scale (1 = *No development* to 4 = *Development is complete*). Onset of menarche is rated using a dichotomous response (1 = *No*, 4 = *Yes*). Reliability and validity of the PDS are well-established (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987). Data were available for 132 youth and 140 caregivers. Because strong correlations were found between youth and caregiver reports ($r = .88$, $p < .001$, for girls, and $r = .72$, $p < .001$ for boys), composites were formed for each item by averaging across informants ($\alpha = .86$ for girls and boys). When information was available from only one informant, this information was used.

Second, youth and caregivers rated youths' stage of development based on line drawings of the five Tanner stages (Morris & Udry, 1980). Girls were rated on breast development and pubic hair growth; boys were rated on genital development and pubic hair growth. Participants checked which of the drawings most closely matched the youth's current stage of development. Data were available for 118 youth and 140 caregivers. Validity of this measure has been established through significant associations with physician ratings based on physical exams (Morris & Udry, 1980; Schlossberger, Turner, & Irwin, 1992). In the present sample, strong correlations were found between girls' and caregivers' reports ($r = .83$, $p < .001$, for breast development; $r = .69$, $p < .001$, for pubic hair development). Moderate correlations were found between boys' and caregivers' reports ($r = .47$, $p < .01$, for genital development; $r = .65$, $p < .001$, for pubic hair development). Composites were formed for each of the two items by averaging across informants. When information was available from only one informant, this information was used.

Confirmatory factor analyses in this data set yielded well-fitting measurement models using the seven items (five PDS and two Tanner) as indicators for a latent variable (*authors omitted for blind review*). Thus, a composite score was created by standardizing the scores for each measure within sex and averaging the scores across the two measures. Higher scores reflected *more advanced* pubertal status. To create an index of pubertal timing, residualized scores were computed separately for girls and boys by regressing pubertal status onto chronological age. Higher scores reflected *earlier* maturation relative to one's agemates. This conceptualization and operationalization of pubertal timing (i.e., level of maturation relative to age) is consistent with a large body of theory and research on pubertal timing (e.g., Dorn et al., 2003; Ge et al., 2002; Ge et al., 2001b; Susman & Rogol, 2004; Steinberg, 1987; Weichold et al., 2003).

Assessment and Coding of Depression

Interviewers individually administered the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version-5 (K-SADS-E; Orvaschel, 1995) to youth and their caregivers to assess youth depression. Consensual diagnoses were assigned using a best-estimate approach (Klein, Ouimette, Kelly, Ferro, & Riso, 1994) to integrate information across youth and caregiver report.

For each period and type (e.g., major depression, dysthymia) of depression (both diagnosable episodes and subclinical symptoms), interviewers used Diagnostic and Statistical Manual of Mental Disorders criteria (DSM-IV-TR; American Psychiatric Association, 2000) to assign ratings of depressive symptoms on a 5-point scale: 0 = No symptoms, 1 = Mild symptoms, 2 = Moderate symptoms, 3 = Diagnosis with mild to moderate impairment, and 4 = Diagnosis with severe impairment. Based on DSM-IV criteria, these ratings considered the number, severity, frequency, duration, and resulting impairment of the reported symptoms. Thus, subclinical symptoms (i.e., mild or moderate) reflected the presence of symptoms that failed to meet one or more of the DSM-IV criteria

(e.g., the youth had fewer than the required number of symptoms or had the required number of symptoms for less than the required duration). These ratings were then summed to create separate continuous depression scores for (1) youths' lifetime history of depression (up until one year prior to the W_1 interview) and (2) youths' level of depression at the time of each assessment (i.e., during the past month). Higher ratings reflect more severe symptoms within a single diagnostic category, the presence of symptoms from multiple categories, and/or multiple periods of depression (for similar rating approaches, see Davila, Hammen, Burge, Paley, & Daley, 1995; Hammen, Shih, Altman, & Brennan, 2003; Hammen, Shih, & Brennan, 2004; Rudolph et al., 2000). Thus, these scores represent composite indexes of several different markers of depression severity. Validity of these scores was established through significant correlations with self-report measures of depressive symptoms (rs =.53 to .58, ps < .001). Moreover, this continuous index of depression is consistent with contemporary conceptualizations, derived in part from taxometric analyses, that view depression as best represented by a dimensional continuum rather than a discrete category (Fergusson, Horwood, Ridder, & Beautrais, 2005; Hankin, Fraley, Lahey, & Waldman, 2005; Shih et al., 2006). Based on independent coding of audiotapes of 42 interviews, strong inter-rater reliability was found for the depression ratings (one-way random-effects intraclass correlation coefficient [ICCs] = .95 - .97).

Of the 167 youth, 12.6% had a lifetime history of clinical depression (a rating of 3 or 4 for at least one episode) prior to the year before the W_1 interview; an additional 18.0% had a lifetime history of subclinical symptoms (i.e., a rating of 1 or 2 for at least one episode). At W_1, 11.4% met diagnostic criteria for clinical depression; an additional 13.2% experienced subclinical symptoms. At W_2, 8.8% met diagnostic criteria for clinical depression; an additional 18.2% experienced subclinical symptoms.

Assessment of Personality Attributes

Depressive personality traits—Interviewers individually administered a 16-item assessment of depressive personality traits to youth and caregivers (Klein, 1990). Based on conceptualizations of depressive personality described by Klein and colleagues (Klein, 1990; Klein & Shih, 1998), this measure was intended to capture stable personality traits of youth rather than acute symptoms of depression. Interviewers first provided a general probe that indicated an interest in youths' traits throughout their lives. To avoid confounding acute symptoms of depression with depressive personality traits, this probe specified that youth and caregivers should consider traits expressed during nonsymptomatic periods. Interviewers then asked participants to indicate the extent to which they would describe youth as someone who generally displayed a variety of traits (e.g., "Critical of yourself. Hard on yourself. Feels like you could or should do better than you do." "Puts yourself down a lot." "Thinks about things all the time that make you feel bad, unhappy, tense, or upset." "Thinks a lot about bad things that have happened, things that you haven't done well, or feel like you're not as good as other kids." "Gloomy, as if everything goes wrong, and things won't work out very well in your life." "Complains a lot."). Interviewers read each question aloud and asked follow-up questions with specific probes (e.g., requests for examples, clarifications). After gathering information from both youth and their caregivers, interviewers provided consensual ratings of how typical each trait was of youth on a scale of 1 (*Not at all*) to 3 (*Definite*).

A reliability analysis revealed that three items (quiet-introverted, extremely conscientious, and self-disciplining) showed low item-total correlations (average = .09; average for other items = .49). Moreover, deletion of these three items from the scale increased the alpha. Thus, these three items were omitted. Scores were computed as the average of the 13 remaining items, with higher scores reflecting more depressive personality traits. The

measure had adequate internal consistency (αs = .85 and .81 at Waves 1 and 2, respectively) and was moderately stable over time ($r = .56$, $p < .001$). Research has established strong concurrent and discriminant validity of the construct of depressive personality (Klein, 1990; Klein & Shih, 1998).

Negative self-focus—Youth completed an 8-item questionnaire assessing negative judgments about their abilities or worth, a focus on social comparison, and concerns about social evaluation (e.g., "I was disappointed in myself." "I felt like other kids were better than me." "I worried about what other people thought of me."). They rated how much each item described them on a 5-point scale (1 = *Not at all* to 5 = *Very Much*). Scores were computed as the average of the 8 items, with higher scores reflecting more negative self-focus. The measure had high internal consistency (αs = .90 and .91 at Waves 1 and 2, respectively) and was relatively stable over time ($r = .57$, $p < .001$). Providing evidence of validity, the negative self-focus measure correlates significantly with a variety of associated constructs tapping negative self-appraisals, including heightened social-evaluative concerns, negative relational self-views, lower levels of perceived control, and a negative attributional style (|r|s = .39 - .57, ps < .001).

Assessment of Responses to Stress

The Responses to Stress Questionnaire (RSQ; Connor-Smith et al., 2000) was used to assess youths' effortful coping versus involuntary, dysregulated responses to stress. This measure distinguishes engagement with, and disengagement from, stressors. It includes four subscales, which demonstrated high internal consistency in the present sample: effortful engagement coping (efforts to resolve stressors or one's response to stressors, such as problem solving and emotion regulation, or to adapt to stressors, such as cognitive restructuring; 21 items; $\alpha = .86$), effortful disengagement coping (e.g., denial, avoidance, wishful thinking; 12 items; $\alpha = .78$), involuntary engagement (e.g., rumination, emotional and physiological arousal; 15 items; $\alpha = .93$), and involuntary disengagement (e.g., inaction, emotional numbing; 12 items; $\alpha = .89$).

Youth reported how much they engaged in each type of response to peer stressors (e.g., fighting with other kids, having problems with a friend) on a 4-point scale (1 = *Not at All* to 4 = *A Lot*). Consistent with previous research (Connor-Smith et al., 2000; Flynn & Rudolph, 2007), to correct for base-rate differences in the endorsement of responses to stress (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001), proportion scores were calculated as the total score for each subscale divided by the total score on the RSQ. Higher scores reflect higher levels of each type of response to stress. The four subscales were relatively stable over time (average $r = .54$, ps < .001).

Confirmatory factor analyses across multiple samples during the original measure development supported the proposed distinctions between effortful engagement and disengagement, and between involuntary engagement and disengagement. Moreover, despite a large correlation between the involuntary engagement and disengagement factors, a two-factor model was found to fit significantly better than a one-factor model (Connor-Smith et al., 2000). Correlations with another well-validated measure of coping established strong convergent and discriminant validity of the individual subscales, supporting the use of separate subscales (Connor-Smith et al., 2000).

Assessment of Family Context

Maternal depression—Interviewers individually administered the nonpatient version of the Structured Clinical Interview for the DSM (SCID IV-NP; First, Spitzer, Gibbon, & Williams, 1996) to caregivers to assess maternal depression. Each period and type of

depression was coded using the same procedures as used for the K-SADS. Thus, two separate depression ratings were calculated for (1) mothers' lifetime history of depression (up until one year prior to the W_1 interview) and (2) mothers' level of depression at the time of each assessment (i.e., during the past month). Strong inter-rater reliability was found for the depression ratings (one-way random-effects ICCs = .94 - .98).

Of the 167 maternal caregivers, 35.3% had a lifetime history of clinical depression (a rating of 3 or 4 for at least one episode) prior to the year before the W_1 interview; an additional 10.8% had a lifetime history of subclinical symptoms (i.e., a rating of 1 or 2 for at least one episode). At W_1, 14.4% met diagnostic criteria for clinical depression; an additional 13.2% experienced subclinical symptoms. At W_2, 17.3% met diagnostic criteria for clinical depression; an additional 14.8% experienced subclinical symptoms.

Recent family stress—To assess recent family stress, interviewers administered the Youth Life Stress Interview (Rudolph & Flynn, 2007) separately to youth and their caregivers. This semi-structured interview uses the contextual threat method to determine the nature and intensity of episodic life stress experienced by youth during the preceding year (Brown & Harris, 1978; Rudolph & Hammen, 1999). The present focus was on episodic stress experienced within the family (e.g., parent-child, marital, and sibling relationships) during the interval between the W_1 and W_2 assessments. Interviewers first asked a general open-ended question about the occurrence of stressful events within the family, and then provided prompts about specific types of events (e.g., argument with a parent, argument between parents, parental remarriage, change in household composition). Follow-up questions were asked to elicit detailed information about each event, the timing and duration of the event, and objective consequences. Based on this information, interviewers presented a narrative summary of each event to a team of coders with no knowledge of the youth's diagnosis or subjective response to the event.

Integrating information from youth and caregivers, coders rated the stressfulness or negative impact of each event, from 1 (*No Negative Stress*) to 5 (*Severe Negative Stress*), reflecting how stressful the event would be for a typical child in those circumstances. If only the youth or the caregiver reported an event, that information was used for coding. Episodic family stress scores were calculated as the total of the objective stress ratings for each family event with a stress rating above 1. To assess reliability, 160 life events were coded by two independent teams. High reliability was found for the objective episodic stress rating (one-way random-effects intra-class correlation coefficient [ICC] = .90) as well as for the categorization of events as family-related (Cohen's κ = .91).

Lifetime family adversity—The Lifetime Adversity section of the Youth Life Stress Interview (Rudolph & Flynn, 2007) was administered to youth and their caregivers. This interview assesses youths' exposure to severe negative family life events and circumstances across their lifetime (up until one year prior to the W_1 interview) using the same type of contextual threat method as the assessment of recent episodic stress. First, a general probe was used to assess exposure to particularly stressful events and circumstances, emphasizing that the focus was on very serious rather than everyday problems. Second, specific probes were used to assess the occurrence of the following types of adversity: death of a close family member, extended separation from parents, parental separation or divorce, exposure to severe marital conflict, and severe chronic illness of a close family member. Follow-up questions were asked as needed to assess the context of the event. An independent rating team provided a consensual rating on a 10-point scale that reflected the overall level of adversity experienced by youth, considering the likely impact of the events for a typical child in the same circumstances. For reliability purposes, two independent teams of raters

coded a subset of 40 interviews. High reliability was found for ratings of adversity (ICC = .99).

Results

Data Imputation

To account for missing data, multiple imputation (MI) was used (Graham & Hofer, 2000; Rubjn, 1987; Schafer, 1997). MI yields more valid parameter estimates and standard errors than traditional methods of handling missing data (e.g., listwise deletion; Jeličić, Phelps, & Lerner, 2009). Shafer's (1999) NORM program was used to compute five imputed data sets. All analyses (e.g., computation of descriptive statistics, *t*-tests, bivariate correlations, regression equations, simple slopes) were conducted separately for the five imputed data sets, and results were combined in NORM, which uses Rubin's (1987) formulas to calculate parameter estimates, their standard errors, and the appropriate degrees of freedom for statistical tests. It should be noted that the degrees of freedom varied substantially as a function of the between- and within-imputation variance of the parameter estimates.

Preliminary Analysis of Sex Differences

A series of *t*-tests was conducted to provide descriptive information about sex differences in the variables (see Table 1). Based on the Pubertal Development Scale, girls were significantly more developed than were boys, $t(2,761) = 5.15$, $p < .001$. Likewise, based on self- and caregiver-reported Tanner stages, girls were significantly more developed than were boys, $t(10,045) = 1.98$, $p < .05$. These differences, along with the absence of a sex difference in age, are consistent with the fact that pubertal maturation occurs earlier in girls than in boys. Girls reported somewhat higher levels of effortful engagement than did boys, $t(389,548) = 1.79$, $p < .10$. Boys reported significantly higher levels of effortful disengagement, $t(396,175) = 2.56$, $p < .05$, and involuntary disengagement, $t(91,922) = 2.08$, $p < .05$, than did girls. The absence of sex differences in depression and family stress is likely due to the fact that these sex differences tend to emerge during middle adolescence (about age 13; e.g., Costello et al., 2003; Ge, Lorenz, Conger, Elder, & Simons, 1994; Rudolph & Hammen, 1999), and more than half of the present sample was younger than 13 years old.

Correlational Analyses

Table 2 presents intercorrelations among the variables for girls and boys. These correlations provide information about the general pattern of associations among the variables. In girls, earlier pubertal maturation was significantly associated with higher levels of depression (W_1, lifetime, and W_2). In boys, earlier pubertal maturation was nonsignificantly associated with lower levels of W_1 and lifetime depression, and with higher levels of W_2 depression. Comparison of the correlations using Fishers r-to-Z transformations revealed that the correlations of pubertal timing with W_1, lifetime, and W_2 depression were significantly different between girls and boys, $Zs \geq 2.05$, $ps < .05$. In girls, each of the risks was significantly associated in the expected direction with subsequent depression, with the exception of a nonsignificant association with W_1 maternal depression and a marginally significant association with maternal lifetime depression. In boys, W_1 depression, depressive personality traits, negative self-focus, effortful engagement, involuntary engagement, W_1 maternal depression, recent family stress, and lifetime family adversity were significantly associated in the expected direction with subsequent depression; lifetime depression and involuntary disengagement were marginally associated with subsequent depression. Comparison of the correlations using Fishers r-to-Z transformations revealed that the following variables were significantly more strongly correlated with W_2 depression in girls than in boys: W_1 depression, lifetime depression, negative self-focus, involuntary

disengagement, and recent family stress, $Zs \geq 2.02$, $ps < .05$. Effortful engagement was marginally more strongly correlated with W_2 depression in girls than in boys, $Zs = 1.88$, $p < .10$.

Overview of Hierarchical Multiple Regression Analyses

A series of hierarchical multiple regression analyses was conducted to examine whether personal and contextual risks magnified the contribution of earlier maturation to subsequent depression. For each analysis, the moderating effect of sex was first examined. The three main effects (pubertal timing, moderator, and sex) were entered at the first step, all two-way interactions were entered at the second step, and the three-way interaction was entered at the third step. Analyses predicted W_2 depression, adjusting for W_1 depression (also entered at the first step). Consistent with procedures outlined by Aiken and West (1991; see also Friedrich, 1982), all predictors were standardized prior to creating interaction terms and inclusion in the regression analyses. Standardized W_2 depression scores were regressed on predictors, and unstandardized regression coefficients were interpreted. When the three-way interaction with sex was significant, the Pubertal Timing × Moderator interaction was examined separately for girls and boys. When the three-way interaction with sex was nonsignificant, analyses were rerun without sex in the model. Significant interactions were interpreted by solving the unstandardized regression equation to predict depression from pubertal timing at low (-1 SD), moderate (mean), and high ($+1$ SD) levels of the moderating variable (Aiken & West, 1991).

Moderating Effects of Personal Risks

Table 3 displays the two-way interactions between pubertal timing and personal risks in predicting depression. Of the eight moderators that were examined, significant three-way interactions (i.e., moderation by sex) were found for two variables and a marginally significant three-way interaction was found for one variable. Results for these three-way interactions are elaborated in the text and figures.

Depression—Analyses revealed significant Pubertal Timing × Youth Depression interactions for both W_1 (i.e., current) levels of depression and lifetime history of depression (see Table 3). For W_1 depression, earlier maturation predicted heightened subsequent depression in youth experiencing high (b = .35, $t(60) = 4.38$, $p < .001$) and moderate (b = .21, $t(482) = 3.64$, $p < .001$) but not low (b = .07, $t(65) = .78$, ns) levels of depression (see Figure 1a). Likewise, for lifetime history of depression, earlier maturation predicted heightened subsequent depression in youth experiencing high (b = .46, $t(34) = 5.33$, $p < .001$) and moderate (b = .23, $t(207) = 4.01$, $p < .001$) but not low (b = .00, $t(2,717) = -.04$, ns) levels of lifetime depression (see Figure 1b).

Personality attributes—Analyses revealed significant Pubertal Timing × Personality interactions for both depressive personality traits and negative self-focus (see Table 3). For depressive personality, earlier maturation predicted heightened subsequent depression in youth with high (b = .36, $t(75) = 4.21$, $p < .001$) and moderate (b = .22, $t(339) = 3.81$, $p < .001$) but not low (b = .08, $t(180) = 1.03$, ns) levels of these traits (see Figure 2a). For negative self-focus, a significant moderating effect of sex was found (b = .15, $t(40) = 2.62$, $p < .05$). Analyses were therefore conducted separately for girls and boys. These analyses revealed a significant Pubertal Timing × Negative Self-Focus interaction for girls (b = .31, $t(93) = 3.97$, $p < .001$, $\Delta R^2 = .07$) but not for boys (b = .05, $t(45) = .06$, ns, $\Delta R^2 = .00$). Specifically, earlier maturation predicted heightened subsequent depression in girls with high (b = .56, $t(436) = 5.06$, $p < .001$) and moderate (b = .25, $t(8,566) = 3.47$, $p < .001$) but not low (b = -.09, $t(288) = -.88$, ns) levels of negative self-focus (see Figure 2b). In boys,

analyses revealed a significant main effect of earlier maturation (b = .17, $t(690) = 2.31$, $p <$ .05) and a nonsignificant main effect of negative self-focus (b =.05, $t(667) =.57$, *ns*).

Responses to stress—Analyses revealed significant Pubertal Timing × Responses to Stress interactions for all four subscales (see Table 3). As displayed in Figures 3a and 3b, earlier maturation predicted heightened subsequent depression in youth who reported high (b = .37, $t(2812) = 4.35$, $p < .001$) and moderate (b = .22, $t(471 = 3.87$, $p < .001$) but not low (b = .07, $t(1010) = .85$, *ns*) levels of effortful disengagement, and those who reported high (b = .35, $t(338) = 4.53$, $p < .001$) and moderate (b = .20, $t(593) = 3.52$, $p < .001$) but not low (b = .06, $t(958) = .66$, *ns*) levels of involuntary engagement.

Two of the two-way interactions were further moderated by sex, significantly for involuntary disengagement (b = .18, $t(2,772) = 3.70$, $p < .001$), and marginally for effortful engagement (b = −.09, $t(7,406) = −1.70$, $p < .10$). Analyses were therefore conducted separately for girls and boys. For effortful engagement, the two-way interaction was significant for girls (b = −.30, $t(8,654) = −4.41$, $p < .001$, $\Delta R^2 = .07$) but not for boys (β = −.13, $t(1,971) = −1.62$, *ns*, $\Delta R^2 = .02$). Specifically, earlier maturation predicted heightened subsequent depression in girls who reported low (b = .51, $t(3206) = 4.72$, $p < .001$) and moderate (b = .15, $t(502) = 1.89$, $p < .10$) but not high (b = −.14, $t(672) = −1.29$, *ns*) levels of effortful engagement (see Figure 3c). In boys, analyses revealed a significant main effect of earlier maturation (b = .17, $t(309) = 2.14$, $p < .05$) and a nonsignificant main effect of effortful engagement (β = −.06, $t(42,127) = −.70$, *ns*).

For involuntary disengagement, the two-way interaction also was significant for girls (b = .34, $t(5,008) = 5.42$, $p < .001$, $\Delta R^2 = .09$) but not for boys (b = −.01, $t(3,378) = −.09$, *ns*, $\Delta R^2 = .00$). Specifically, earlier maturation predicted heightened subsequent depression in girls who reported high (b = .64, $t(113,258) = 5.58$, $p < .001$) and moderate (b = .24, $t(6193) = 3.49$, $p < .001$) but not low (b = −.06, $t(918) = −.82$, *ns*) levels of involuntary disengagement (see Figure 3d). In boys, analyses revealed a significant main effect of earlier maturation (b = .16, $t(370) = 2.14$, $p < .05$) and a nonsignificant main effect of involuntary disengagement (b = .10, $t(13,493) = 1.31$, *ns*).

Summary—In sum, results supported the idea that prior depression, personality traits, and maladaptive responses to stress accentuated the depressogenic effect of earlier maturation. Effects were specific to girls for a tendency toward negative self-focus and certain types of responses to stress; effects generalized across sex for recent and past symptoms of depression, depressive personality traits, and other types of responses to stress.

Moderating Effects of Contextual Risks

Table 4 displays the two-way interactions between pubertal timing and contextual risk in predicting depression. Of the four moderators that were examined, no significant three-way interactions (i.e., moderation by sex) were found.

Maternal depression—Analyses revealed a significant Pubertal Timing × Maternal Depression interaction for W_1 (i.e., current) levels of maternal depression but not for lifetime history of depression. Specifically, earlier maturation predicted heightened subsequent depression in youth whose mothers were experiencing high (b = .36, $t(98) = 4.06$, $p < .001$) and moderate (b = .24, $t(529) = 4.12$, $p < .001$) but not low (b = .12, $t(315) = 1.38$, *ns*) levels of W_1 depression (see Figure 4a). Past history of maternal depression did not significantly predict youth depression over time.

Family adversity—Analyses revealed a significant Pubertal Timing × Family Adversity interaction for recent family stress but not for lifetime family adversity. Specifically, earlier maturation predicted heightened subsequent depression in youth exposed to high (b = .39, $t(343) = 4.67$, $p < .001$) and moderate (b = .21, $t(447) = 3.72$, $p < .001$) but not low (b = .04, $t(607) = .47$, *ns*) levels of recent family stress (see Figure 4b). Lifetime family adversity did not significantly predict youth depression over time.

Summary—In sum, results supported the idea that recent maternal depression and family adversity amplified the depressogenic effect of earlier maturation for both girls and boys.

Discussion

Puberty represents a challenging developmental transition that has been implicated in rising rates of depression during adolescence, yet many youth successfully traverse this stage with few adverse emotional consequences. Understanding how puberty contributes to trajectories of development and psychopathology therefore requires elucidating individual differences in youths' reactions (Graber, 2003). Guided by personal-accentuation and contextual-amplification models, the present study makes substantive contributions to both theory and empirical research by identifying specific personal and contextual risks that magnify or temper the contribution of earlier maturation to subsequent depression.

Evidence for Personal-Accentuation Models of Earlier Maturation

Several personal vulnerabilities of youth accentuated the depressogenic effects of earlier maturation. As anticipated, earlier-maturing youth who experienced recent depressive symptoms were more vulnerable to heightened levels of subsequent depression than those with few recent depressive symptoms. This pattern is consistent with the idea that earlier maturation presents a particular challenge to youth with a predisposition toward psychological difficulties prior to this transition (Caspi & Moffitt, 1991). Thus, the heightened depression associated with earlier maturation may reflect continuity of prior dysfunction rather than the onset of new disorder—that is, earlier maturation does not necessarily confer risk for depression in previously healthy youth.

Earlier maturation also was associated with subsequent depression in youth with a lifetime history of depression but not those without such a history. The fact that a history of depression accentuated the effects of earlier maturation even after adjusting for recent depression suggests that youth with a history of depression suffer some core, lasting deficits that contribute to their emotional vulnerability in the face of earlier maturation. Perhaps a history of depression interferes with youths' development of adaptive relationships with family and peers and thus deprives them of social support networks that would help them to negotiate this challenging transition. Alternatively, it is possible that a history of depression reflects a genetic predisposition toward depression that is expressed during the adolescent transition. Interestingly, youth with high lifetime depression had particularly low levels of depression when they experienced later maturation. Although this pattern was unexpected and must be interpreted with caution, it is consistent with the biological sensitivity to context theory (Boyce & Ellis, 2005). According to this theory, certain youth possess attributes that foster maladaptive outcomes (i.e., health risk) under conditions of adversity but positive outcomes (i.e., health benefits) under conditions of support. It is possible that youth with a lifetime history of depression are especially attuned to their social contexts, such that they are at particular risk when faced with challenges in the context of earlier maturation but receive particular benefits from support received in the context of later maturation.

Extending personal-accentuation models, this research also identified personality attributes that magnified the depressogenic effects of earlier maturation. Specifically, earlier

maturation predicted heightened levels of subsequent depression in youth with depressive personality traits and in girls with a negative self-focus but not in youth without these personality attributes. The heightened self- and social scrutiny that often accompanies the pubertal transition (Harter, 1990; Zimmerman, Copeland, Shope, & Dielman, 1997), particularly for earlier-maturing youth, may trigger a preoccupation with negative self-perceptions and downward social comparison in youth who are predisposed to a pessimistic outlook on the self and the world. Such youth may be acutely sensitive to their discrepant physical and social status and thus may react to the pubertal transition with negative emotions, low self-worth, and others symptoms of depression. A negative self-focus may accentuate these effects in earlier-maturing girls but not boys because earlier maturation tends to promote a negative body image in girls but a positive body image in boys (Tobin-Richards et al., 1983); this negative body image may in turn act as a source of more global self-preoccupation for girls. Moreover, because girls, on average, mature earlier than boys (Tanner, 1969), earlier-maturing girls are more deviant than boys in their timing relative to the entire peer group, potentially intensifying the adverse effects of self-evaluative and social-comparison processes. Of course, it is important to consider the fact that depressive personality traits and negative self-focus may themselves reflect early precursors or symptoms of depression rather than entirely discrete personality traits. Although research suggests that such traits are distinguishable from depressive disorders, are relatively stable over time, and do not fluctuate significantly with mood states (Klein, 1990; Klein & Shih, 1998), the present research cannot determine the precise role of these traits in the emergence of depression.

Youths' coping styles also moderated the contribution of earlier maturation to subsequent depression. Specifically, earlier maturation predicted heightened levels of depression in youth who showed low levels of effortful engagement coping and high levels of effortful disengagement coping and involuntary responses to stress but not in youth with the opposite response styles. Youth who engage in goal-directed efforts to resolve peer stressors and to manage the associated negative emotions appear to be buffered from the adverse effects of earlier maturation. These youth are likely more adept at navigating the complex social landscape that emerges during the pubertal transition. In contrast, youth who react to peer stress through either disengagement (e.g., denial, avoidance) or involuntary, dysregulated responses (e.g., rumination, arousal, inaction) are likely less effective at handling the social challenges of puberty. For two types of stress responses, effortful engagement and involuntary disengagement, these protective and exacerbating effects were specific to girls. Because of girls' greater emphasis on connection-oriented social goals and their greater emotional reactivity to relationship problems relative to boys (Rose & Rudolph, 2006), earlier-maturing girls who have difficulty managing peer stress may be more susceptible than boys to depression.

Unexpectedly, youth who showed high levels of effortful disengagement actually had particularly low levels of depression when they experienced later maturation. Later-maturing youth likely face milder interpersonal challenges than earlier-maturing youth. It may be that disengaging from such stressors is actually an adaptive strategy that prevents youth from overreacting to minor hassles (e.g., ruminating about a minor problem with a friend) and, consequently, protects them from depression. This idea is consistent with other research suggesting that disengagement may be adaptive in the context of mild stressors (Abaied & Rudolph, in press).

In sum, several types of personal risk accentuated the contribution of earlier maturation to subsequent levels of depression; indeed, earlier-maturing youth without these vulnerabilities were no more likely to become depressed than were on-time and later-maturing youth. Interestingly, sex did not moderate the overall effect of earlier maturation on subsequent

depression. This finding is consistent with a growing body of research showing that, contrary to original beliefs, earlier-maturing boys also are at risk for depression (Ge et al., 2001b; Kaltiala-Heino et al., 2003). In fact, several researchers recently have emphasized the need to reassess the role of earlier maturation in boys' development (Alsaker, 1995; Graber, 2003). However, several personal risks did accentuate the effect of earlier maturation on levels of depression in girls but not in boys, such that this link was particularly strong in subgroups of girls with heightened risk. Thus, although earlier maturation may, on average, increase the likelihood of depression in girls and boys, more girls may express these effects due to pre-disposing vulnerabilities. These findings provide a more nuanced picture of the emerging sex difference in depression during adolescence, suggesting that this difference may be accounted for by a subgroup of highly vulnerable girls.

Evidence for Contextual-Amplification Models of Earlier Maturation

Evidence also emerged for contextual amplification of pubertal timing effects. Specifically, earlier maturation predicted heightened levels of subsequent depression in youth exposed to recent maternal depression and family stress but not in youth without these contextual risks. Maternal depression and family stress likely erode the emotional support and scaffolding on which earlier-maturing youth typically rely to negotiate the challenges of this stage. Moreover, these family risks serve as additional stressors to earlier-maturing youth at a time when they need to direct their internal resources toward dealing with developmental changes. Pre-existing family risks also may intensify problems and conflict associated with the normative reorganization of family relationships during puberty, such that unhealthy families experience greater difficulty dealing with youths' changing status (Paikoff & Brooks-Gunn, 1991). Collectively, these consequences of family risk may serve to amplify the depressogenic effects of earlier maturation.

Amplification effects of maternal depression and family stress also could be accounted for by a genetic liability to depression; that is, family risk could reflect a family loading for depression that is expressed during adolescence. Contrary to this explanation, however, lifetime history of maternal depression and family adversity did not exert an amplification effect, suggesting that recent maternal symptoms and family stress exert functional effects on the nature of family relationships that amplify risk for depression in earlier-maturing youth. This absence of a moderation effect for a lifetime history of maternal depression and family adversity may be due to the fact that our analyses adjusted for W_1 levels of depression; it may be that the effects of these distal risks already had been expressed earlier in youths' lives and thus did not amplify risk for heightened depression during the pubertal transition.

Interestingly, no sex differences in contextual amplification were found. Because maternal depression (Sheeber et al., 2002) and interpersonal stress (Hankin et al., 1997) are linked more strongly to depression in adolescent girls than in boys, it was expected that earlier-maturing girls would be more vulnerable to the effects of family risk than would earlier-maturing boys. However, earlier-maturing boys do experience heightened tension in mother-child relationships (Steinberg, 1987); thus, those who enter the pubertal transition with compromised family relationships may have difficulty negotiating this normative increase in parent-child conflict.

Theoretical and Practical Contributions and Caveats

Consistent with developmental psychopathology perspectives that emphasize complex Person × Environment × Development interactions (Cicchetti & Cohen, 2006; Sameroff, 1987), as well as developmental models that emphasize the social context of puberty (Caspi

& Moffitt, 1991; Graber, 2003), the present research reveals important personal and contextual variation in the contribution of pubertal timing to depression. Whereas youth with prior personal and contextual risks were susceptible to the depression-inducing effects of earlier maturation, youth without these risks were buffered from these effects. Although the size of the interaction effects tended to be small, the consistency in the pattern of effects was quite notable.

Continuing to elaborate and refine integrative models of puberty and depression will require building on these findings in several ways. First, given the present focus on understanding the psychological and social context of puberty, this study conceptualized pubertal timing in terms of youths' expression of secondary sexual characteristics and other somatic markers of maturation. Hormonal and morphological changes represent related, yet distinct, indexes of pubertal development. Future research in this area needs to examine whether similar personal-accentuation and contextual-amplification effects hold with hormonal assessments of puberty.

Second, this study focused on the independent contributions of several risk factors, which represented a reasonable first step toward identifying characteristics of youth and their contexts that moderate the effects of earlier maturation. However, it is likely that these characteristics reflect, at least in part, an interrelated network of factors that jointly magnify or temper risk for depression in earlier-maturing youth. For example, maternal depression was presumed to amplify risk in part by eroding family relationships and heightening family stress. Exploratory analyses in the present study did indeed reveal that pubertal timing interacted with maternal depression to predict family stress, and the Pubertal Timing × Family Stress interaction accounted for the contribution of the Pubertal Timing × Maternal Depression interaction to subsequent depression. Similarly, the amplifying effects of family risk may operate in part by heightening personal risks, such as fostering depressive personality traits or undermining youths' coping resources. Because of limited power, the present study could not incorporate tests of these complex, multi-determined pathways, but further development of comprehensive models of pubertal timing and depression will require a more thorough understanding of distinct versus shared mechanisms underlying the personal-accentuation and contextual-amplification effects that emerged in this study.

Third, this research could not distinguish between two possible explanations for the observed interactions. On the one hand, these interactions could reflect synergistic effects of two independent risk factors, such that experiencing personal or contextual risks compounded the adverse effects of earlier maturation. On the other hand, these interactions could reflect a meaningful change in the association between earlier maturation and depression in the context of the risk factors. For example, earlier maturation may heighten risk for depression in youth with depressive personality traits because these youth have dual risk factors or specifically because they perceive pubertal changes in a particularly negative light. Likewise, earlier maturation may heighten risk for depression in youth with high levels of family stress because these youth face simultaneous challenges or specifically because high-stress families show a negative response to pubertal changes. Disentangling these alternative explanations will require more focused measurement of personal and contextual risks specifically in the context of puberty.

Fourth, although this study benefited from the use of a longitudinal design to examine the contribution of earlier maturation to depression over time, the one-year follow-up period provided a relatively short time-frame for examining these effects. It will be fascinating to examine whether pre-existing personal and contextual risks foreshadow long-term differences in depression and associated difficulties in earlier-maturing youth. Moreover, this study focused on the measurement of puberty at a single point in time; because puberty

is a dynamic process, the rate of maturation may have an influence on adjustment beyond the timing at any one particular point in development (e.g., Ge et al., 2003). Thus, future research should examine how changes in puberty over time interact with risks to predict trajectories of depression.

Fifth, in light of evidence for ethnic differences in the timing, meaning, and effects of puberty (Hayward, Gotlib, Schraedley, & Litt, 1999; Michael & Eccles, 2003), it is important to determine the generalizability versus specificity of these effects across ethnic groups. Although the present sample included some ethnic diversity, the sample size was insufficient for examining such ethnic differences. Whether or not the same risks serve as exacerbating or protective factors across ethnic groups remains an open question. More broadly, the progression through puberty has significantly different implications across cultures, with many cultures having positive views of this transition (Weisfeld, 1999); thus, the perspective of puberty as a high-risk period may better apply to Western than non-Western cultures.

Beyond these theoretical contributions and questions, the present study suggests important directions for depression prevention efforts. Given limited resources and the impracticality of targeting all earlier-maturing youth for prevention programs, this research provides specific information that can be used to identify earlier-maturing youth who are at particularly high risk for depression. Moreover, these findings point to potential targets of intervention. Specifically, strategies aimed at bolstering high-risk youths' self-appraisals and coping resources and at reducing stress associated with family risks may promote healthy development in earlier-maturing youth. Finally, broad psychoeducational efforts can address misconceptions in youth, families, and educators about the inevitable turmoil emerging from the pubertal transition, and encourage more realistic assessments regarding whether or not particular youth are at risk during this developmental stage.

Conclusion

Early perspectives on adolescence and the pubertal transition as a universal period of "storm and stress" are gradually transforming into more nuanced theories that consider individual and contextual variation in youths' negotiation of this developmental stage. Despite this growing sophistication in contemporary theories, relatively few theoretical models or empirical studies have elucidated which earlier-maturing youth are more or less susceptible to depression. The present study identifies several personal and contextual characteristics that explain these individual differences, and provides a basis on which to develop prevention and intervention efforts for depressed youth.

Acknowledgments

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(a) W₁ Youth Depression
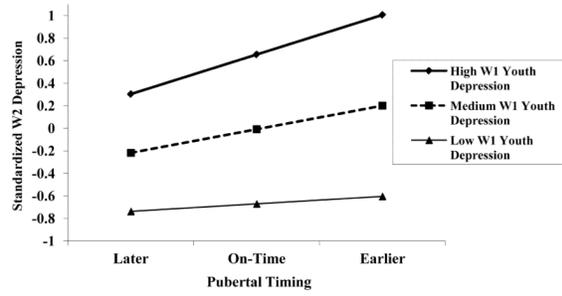
(b) Lifetime Youth Depression
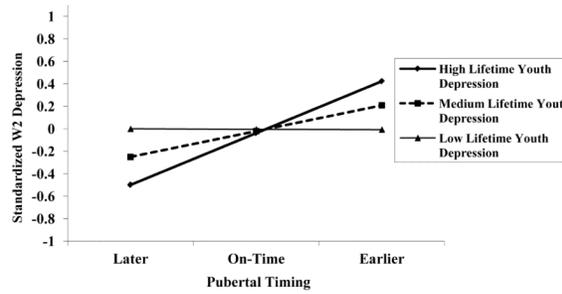
Figure 1.
Pubertal Timing × Depression interactions for (a) W_1 youth depression and (b) lifetime youth depression.

(a) Depressive Personality

(b) Negative Self-Focus – Girls
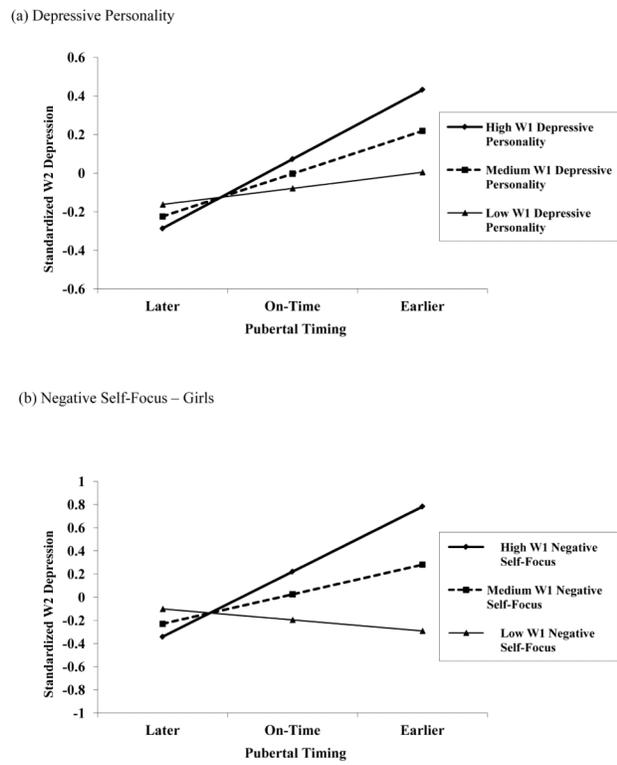
Figure 2.
Pubertal Timing × Personality interactions for (a) depressive personality traits and (b) negative self-focus (girls).

(a) Effortful Disengagement

(b) Involuntary Engagement

(c) Effortful Engagement – Girls

(d) Involuntary Disengagement – Girls

Figure 3.

Pubertal Timing × Responses to Stress interactions for (a) effortful disengagement, (b) involuntary engagement, (c) effortful engagement (girls), and (d) involuntary disengagement (girls).
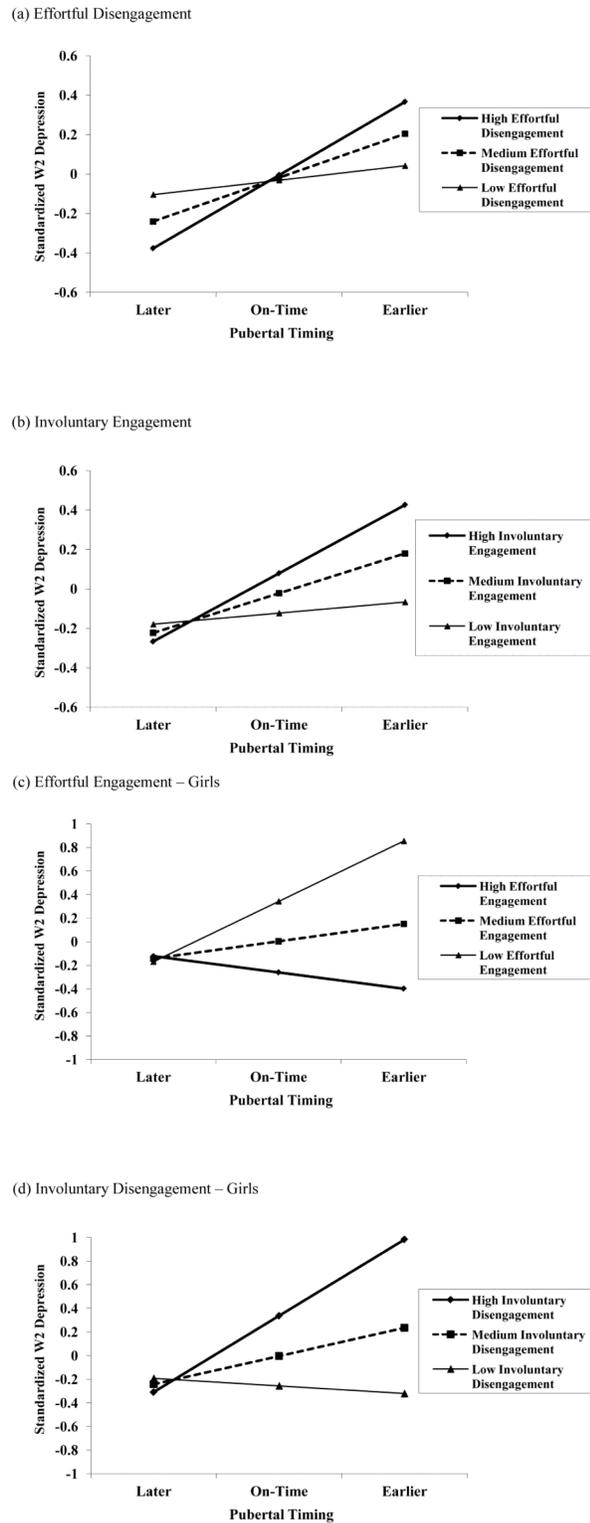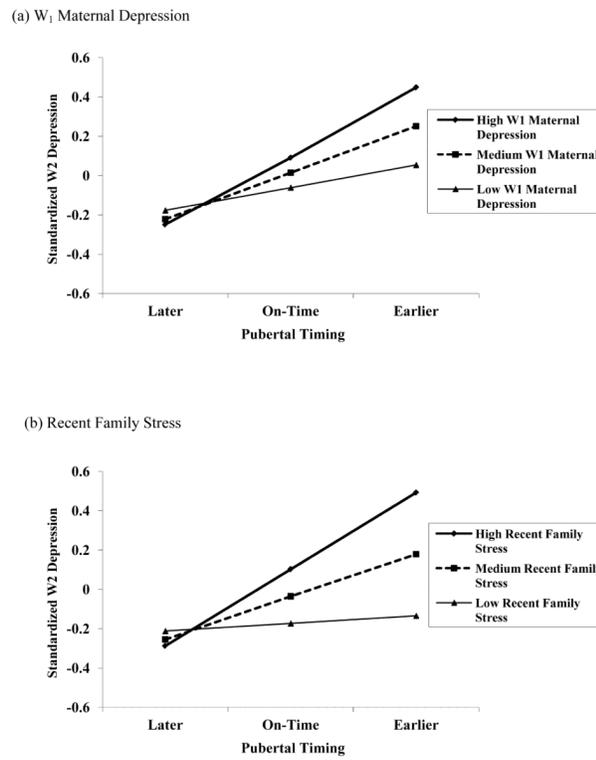
(a) W₁ Maternal Depression

(b) Recent Family Stress

Figure 4.
Pubertal Timing × Family Risk interactions for (a) W₁ maternal depression and (b) recent family stress.

Table 1

Descriptive Statistics

	Girls		Boys	
	M	**SD**	**M**	**SD**
W_1 Pubertal Development Scale	2.55[a]	.77	1.99[a]	.60
W_1 Tanner Stages	3.01[a]	1.19	2.67[a]	1.00
W_1 Youth Depression	.67	1.41	.65	1.32
Lifetime Youth Depression	.98	1.57	.81	1.83
W_1 Depressive Personality	1.33	.34	1.29	.33
W_1 Negative Self-Focus	1.48	.68	1.59	.78
W_1 Effortful Engagement	.48[b]	.08	.46[b]	.07
W_1 Effortful Disengagement	.14[a]	.02	.15[a]	.03
W_1 Involuntary Engagement	.23	.05	.23	.04
W_1 Involuntary Disengagement	.16[a]	.04	.17[a]	.03
W_1 Maternal Depression	.88	1.46	.64	1.27
Lifetime Maternal Depression	2.58	3.98	2.35	3.62
Recent Family Stress	4.25	5.95	3.34	5.55
Lifetime Family Adversity	3.27	2.04	3.31	2.12
W_2 Youth Depression	.74	1.44	.48	.98

Note.

[a] Means differ at $p < .05$ or lower.

[b] Means differ at $p < .10$.

Table 2

Correlations Among Pubertal Timing, Personal Vulnerability, Contextual Risk, and Depression for Girls and Boys

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. W$_1$ Pubertal Timing	--	.30**	.39***	.24*	.23*	-.30**	.26*	.17	.29**	-.04	.06	.32**	.30**	.40***
2. W$_1$ Youth Depression	-.21	--	.66***	.65***	.67***	-.41***	.17	.32**	.41***	.19^	.27*	.43***	.40***	.76***
3. Lifetime Youth Depression	-.17	.63***	--	.42***	.41***	-.28*	.17	.18	.30**	.12	.27*	.22^	.38***	.60***
4. W$_1$ Depressive Personality	-.21	.67***	.40***	--	.52***	-.36***	.12	.29**	.34**	.16	.20	.36***	.47***	.55***
5. W$_1$ Negative Self-Focus	.09	.40***	.10	.35**	--	-.60***	.28**	.46***	.59***	.01	.01	.27*	.27*	.65***
6. W$_1$ Effortful Engagement	-.21^	-.23*	-.11	-.27*	-.48***	--	-.62***	-.79***	-.84***	.08	-.06	-.23*	-.16	-.51***
7. W$_1$ Effortful Disengagement	.15	.16	.26*	.07	.00	-.44***	--	.12	.57***	-.25*	-.04	.07	.11	.27*
8. W$_1$ Involuntary Engagement	.11	.23*	.12	.30*	.49***	-.83***	.02	--	.42***	.04	.03	.19	.11	.31**
9. W$_1$ Involuntary Disengagement	.19	.08	-.14	.14	.43***	-.78***	.07	.54***	--	-.06	.13	.23*	.14	.59***
10. W$_1$ Maternal Depression	-.25*	.42***	.31**	.19	.15	.13	-.06	-.05	-.15	--	.58***	.26*	.06	.14
11. Lifetime Maternal Depression	-.06	.46***	.56***	.21^	.20	.11	.10	-.08	-.23*	.46***	--	.10	.18^	.19^
12. Recent Family Stress	.08	.23*	.13	.08	.22^	-.23*	.11	.16	.20^	.18	.06	--	.25*	.57***
13. Lifetime Family Adversity	.11	.32**	.25*	.22^	.20	-.17	.14	.20^	.01	.08	.24*	.46***	--	.35**
14. W$_2$ Youth Depression	.10	.59***	.20^	.47***	.32**	-.26*	.04	.26*	.21^	.27*	.12	.27*	.30**	--

Note. Correlations above the diagonal are for girls; correlations below the diagonal are for boys.

^
$p < .10$.

*
$p < .05$.

**
$p < .01$.

***
$p < .001$.

Table 3

Predicting W_2 Depression From W_1 Depression, Pubertal Timing, Personal Vulnerability, and Pubertal Timing × Personal Vulnerability Interactions

	Predictors	b	t	ΔR^2
Step 1: Main Effects				.52
	Pubertal Timing	.23	4.08***	
	W_1 Depression	.67.	12.22***	
Step 2: Interaction				.02
	Pubertal Timing × W_1 Depression	.14	2.30*	
Step 1: Main Effects				.52
	W_1 Depression	.71	9.97***	
	Pubertal Timing	.23	4.14***	
	Lifetime Youth Depression	−.02	−.78	
Step 2: Interaction				.06
	Pubertal Timing × Lifetime Youth Depression	.23	3.99***	
Step 1: Main Effects				.53
	W_1 Depression	.76	8.06***	
	Pubertal Timing	.23	4.12***	
	Depressive Personality	.09	1.06	
Step 2: Interaction				.02
	Pubertal Timing × Depressive Personality	. 14	2.29*	
Step 1: Main Effects				.55
	W_1 Depression	.58	8.68***	
	Pubertal Timing	.21	3.76***	
	Negative Self-Focus	.18	2.56*	
Step 2: Interaction[a]				.05
	Pubertal Timing × Negative Self-Focus	.16	3.03**	
Step 1: Main Effects				.56
	W_1 Depression	.62	10.97***	
	Pubertal Timing	.1 9	3.34***	
	Effortful Engagement	−.16	−2.81**	
Step 2: Interaction[a]				.06
	Pubertal Timing × Effortful Engagement	−.24	−4.51***	
Step 1: Main Effects				.52

	Predictors	b	t	ΔR^2
	W_1 Depression	.67	12.07***	
	Pubertal Timing	.25	3.45**	
	Effortful Disengagement	−.01	−.19	
Step 2: Interaction				.02
	Pubertal Timing × Effortful Disengagement	.15	2.34*	
Step 1: Main Effects				.53
	W_1 Depression	.65	11.40***	
	Pubertal Timing	.22	3.88***	
	Involuntary Engagement	.08	1.41	
Step 2: Interaction				.02
	Pubertal Timing × Involuntary Engagement	.15	2.50*	
Step 1: Main Effects				.58
	W_1 Depression	.61	11.36***	
	Pubertal Timing	.18	3.22**	
	Involuntary	.24	4.35***	
Step 2: Interaction[a]				.05
	Pubertal Timing × Involuntary	.18	3.58***	

*
$p < .05$.

**
$p < .01$.

***
$p < .001$.

[a]Interaction was moderated by sex.

Note. bs, *t*s, and ΔR^2 represent statistics at each step of the regression equation.

Table 4

Predicting W_2 Depression From W_1 Depression, Pubertal Timing, Family Risk, and Pubertal Timing × Family Risk Interactions

	Predictors	b	t	ΔR^2
Step 1: Main Effects				.52
	W_1 Depression	.66	11.34***	
	Pubertal Timing	.24	4.11***	
	W_1 Maternal Depression	.04	.61	
Step 2: Interaction				.01
	Pubertal Timing × W_1 Maternal Depression	.12	1.99*	
Step 1: Main Effects				.53
	W_1 Depression	.87	10.84***	
	Pubertal Timing	.29	3.97***	
	Lifetime Maternal Depression	−.10	−1.44	
Step 2: Interaction				.00
	Pubertal Timing × Lifetime Maternal Depression	−.04	−.56	
Step 1: Main Effects				.56
	W_1 Depression	.60	10.68***	
	Pubertal Timing	.19	3.46***	
	Recent Family Stress	.22	3.78***	
Step 2: Interaction				.03
	Pubertal Timing × Recent Family Stress	.18	2.98**	
Step 1: Main Effects				.52
	W_1 Depression	.66	11.17***	
	Pubertal Timing	.22	3.90***	
	Lifetime Family Adversity	.03	.52	
Step 2: Interaction				.00
	Pubertal Timing × Lifetime Family Adversity	.05	.89	

*
$p < .05$.

**
$p < .01$.

***
$p < .001$.

Note. bs, *t*s, and ΔR^2 represent statistics at each step of the regression equation.