Developmental Influences on Interpersonal Stress Generation in Depressed Youth

Karen D. Rudolph
University of Illinois at Urbana–Champaign

Abstract
This research tested the hypothesis that the transition through adolescence, particularly undergoing puberty early relative to one's peers, would amplify the effect of depression on the subsequent generation of interpersonal stress. This hypothesis was examined in 158 youth (M age = 12.39 years, SD = 1.21) using semistructured interviews of depression and life stress. Three indexes of development—chronological age, pubertal status, and pubertal timing—were examined as possible moderators of the stress-generation effect. As anticipated, depression predicted interpersonal stress generation in early-maturing but not late-maturing youth. These findings provide an important developmental context for theory and research on stress generation.

Keywords
depression; stress generation; puberty

Consistent with interpersonal theories of depression, the stress-generation perspective holds that depressed individuals create stress in their relationships, either as a direct consequence of symptoms or as a result of depressotypic attributes and behaviors (Hammen, 2006). Indeed, research links depression to interpersonal stress generation in adults (Hammen, 1991), adolescents (Daley et al., 1997; Hammen, Shih, & Brennan, 2004), and a mixed-age sample of youth (Rudolph et al., 2000). Although depression predicts interpersonal stress generation across a broad age range, this process may be intensified during developmental stages that involve significant reorganization of relationships. In fact, no study to date has specifically examined the interpersonal stress generation effect of depression prior to puberty. The present study investigated the hypothesis that depressed youth generate particularly high levels of interpersonal stress when they negotiate the complex, demanding, and emotionally charged relationships that evolve during the transition to adolescence.

Developmental theories view adolescence, and particularly the pubertal transition, as a challenging stage of development during which youth face many normative disruptions and stressors within their relationships (Paikoff & Brooks-Gunn, 1991; Simmons, Blyth, Van Cleave, & Bush, 1979; Slaff, 1990). These interpersonal challenges are believed to be most salient when youth mature early relative to their peers. According to the stage-termination hypothesis (Petersen & Taylor, 1980), early pubertal timing interrupts normative developmental trajectories, such that early-maturing youth are ill prepared to deal with the adolescent transition, and they lack the support structures enjoyed by on-time youth. Moreover, early-maturing youth tend to enter into more complex and potential risky relationships than do
their peers (Weichold, Silbereisen, & Schmitt-Rodermund, 2003). Expanding on this hypothesis, Caspi and Moffitt (1991) proposed that early pubertal timing serves as a transitional stressor that accentuates preexisting individual differences in vulnerability. Thus, depressed youth may generate particularly high levels of stress in their relationships when faced with the social challenges of adolescence and the pubertal transition; moreover, this amplification effect may be magnified in early-maturing depressed youth. One recent study (Cole, Nolen-Hoeksema, Girgus, & Paul, 2006) revealed that the stress-generation effect of depression increased from early childhood through adolescence but did not elucidate whether this trend was due to chronological age or the progression through puberty.

The present study investigated whether three specific indexes of development—chronological age, pubertal status (absolute level of maturation), and pubertal timing (level of maturation relative to one’s peers)—moderated the interpersonal stress-generation process in depressed youth. It was hypothesized that pubertal timing would most likely amplify this process, such that depression would contribute more strongly to interpersonal stress generation in early-developing than in on-time or late-developing youth. Moreover, this study examined whether this amplification effect differed across sex. Research examining sex differences in the influence of puberty is mixed. Some research suggests that pubertal development confers social and emotional costs for girls but benefits for boys (Crockett & Petersen, 1987; Simmons et al., 1979). However, other research suggests that this transition contributes to difficulties in girls and boys (Ge et al., 2003; Huddleston & Ge, 2003). Particularly relevant to the present study, both early-maturing girls and early-maturing boys experience more risky and challenging interpersonal contexts (for a review, see Weichold et al., 2003). Thus, early maturation may intensify the adverse interpersonal effects of depression in both groups.

Method

Participants

Participants were 158 youth (82 girls, 76 boys; \( M \) age = 12.41 years, \( SD = 1.19 \), range = 9.6 to 14.8; 76.6% White, 23.4% minority) and their female caregivers. Families were diverse in income (16.4% below $30,000; 48.4% $30,000–$59,999; 22.2% $60,000–$89,999; 13.1% over $90,000). On the basis of the availability of puberty data, these youth were selected from 167 families participating in a longitudinal study of depression. Youth were selected for the longitudinal study on the basis of school-wide screenings with the Children’s Depression Inventory (CDI; Kovacs, 1980). The screening sample (\( N = 1,985 \)) represented 80% of targeted participants. From this sample, we selected potential participants (\( n = 468 \)) along the range of the CDI, oversampling 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 on the basis of CDI scores, having a maternal caregiver in the home, and proximity to the university, until the target sample was attained. Exclusion criteria included having a non-English-speaking maternal care-giver or a severe developmental disability. Participants and non-participants in the longitudinal study did not differ in sex, \( \chi^2(1, N = 468) = .39, \text{ } ns; \) ethnicity, \( \chi^2(1, N = 468) = .02, \text{ } ns; \) or CDI score, \( t(280) = 1.11, \text{ } ns. \) Participants (\( M \) age = 12.41 years, \( SD = 1.19 \)) were slightly younger than nonparticipants (\( M \) age = 12.65 years, \( SD = 0.89 \)), \( t(275) = 2.28, \text{ } p < .05. \) Wave 2 (\( W_2 \)) stress data were available for 148 (93.7%) of the 158 participants. Youth with \( W_2 \) data versus youth without \( W_2 \) data did not differ in sex, \( \chi^2(1, N = 158) = .61, \text{ } ns; \) age, \( t(156) = .71, \text{ } ns; \) ethnicity, \( \chi^2(1, N = 158) = .26, \text{ } ns; \) or Wave 1 (\( W_1 \)) depression, pubertal status, or dependent interpersonal stress, \( ts(156) \leq 1.77, \text{ } ns. \)

Procedures

Caregivers and youth were interviewed separately during a 3- to 4-hr assessment. Separate interviewers conducted diagnostic and life-stress interviews. Families received a financial...
reimbursement and youth received a gift certificate. Follow-up assessments were completed approximately 1 year later (\(M\) time interval = 11.7 months, \(SD = 0.96\)).

**Measures**

Table 1 presents descriptive data. On the basis of the Pubertal Development Scale, girls were significantly more developed than boys, \(t(143) = 4.65, p < .001\). On the basis of self- and caregiver-reported Tanner stages, girls were somewhat more developed than boys, \(t(149) = 1.73, p < .10\). 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. There were no sex differences in depression or dependent interpersonal stress, \(ts(146–156) < 1, ns\), likely because of the fact that these sex differences emerge during middle adolescence (Costello & Angold, 2006; Ge, Lorenz, Conger, Elder, & Simons, 1994; Rudolph & Hammen, 1999), and more than half of this sample was younger than 13 years.

**Depression**—Trained interviewers administered the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Epidemiologic Version 5; Orvaschel, 1995) to youth and caregivers to assess youths’ depression; information was combined using a best-estimate approach (Klein, Ouimette, Kelly, Ferro, & Riso, 1994). On the basis of Diagnostic and Statistical Manual of Mental Disorders (4th ed.; American Psychiatric Association, 1994) criteria regarding the number, severity, and frequency of symptoms and impairment, interviewers rated depressive psychopathology on a 5-point scale (0 = no symptoms, 1 = mild symptoms, 2 = moderate symptoms, 3 = diagnosis with mild to moderate impairment, 4 = diagnosis with severe impairment). We assigned separate ratings for each category of depression (e.g., major depression, dysthymia), and we summed them to create continuous depression scores for the past month. Higher ratings reflected more severe symptoms within a single diagnostic category and/or the presence of symptoms from multiple categories. Independent coding of 25% of the interviews yielded strong reliability (one-way random-effects intraclass correlation coefficient [ICC] = .97). Of the 158 youth, 11% met diagnostic criteria for major depression or dysthymia at the initial interview (a rating of 3 or 4 for at least one episode). An additional 13% experienced some symptoms of depression within the past month (a rating of 1 or 2 for at least one episode). Table 2 presents descriptive information about levels of depression (i.e., mean scores on the continuous scale; percentages of youth with moderate symptoms and clinical diagnoses) within developmental groups. We formed three groups for each index (chronological age, pubertal status, and pubertal timing) by dividing youth into those who fell in the bottom, middle, and top thirds of the sample.

**Life stress**—We administered the Youth Life Stress Interview (Rudolph & Flynn, 2007), an adaptation of the Child Episodic Life Stress Interview (Rudolph et al., 2000), to assess episodic life stress using the contextual threat method (Brown & Harris, 1978; Hammen, 1991). This semistructured interview includes an initial open-ended question, followed by detailed probes to elicit information about the nature, intensity, timing, and context of episodic stressors experienced by youth during the preceding year. Interviewers present a narrative summary of each event to a team of coders with no prior knowledge about the youths’ diagnostic status or subjective reaction.

Integrating information across youth and caregiver reports, the coders rated the following: (a) the stressfulness of each event, from 1 (no negative stress) to 5 (severe negative stress)—events with ratings of 1 were excluded—and (b) the dependence of each event, or the extent to which the child contributed to the event’s occurrence, from 1 (completely independent) to 5 (completely dependent). Events with dependence ratings of 3 or above were categorized as dependent (Daley et al., 1997). The coders also categorized each event as (a) interpersonal, namely events that involved a significant interaction between the youth and another person or
that directly affected the relationship between the youth and another person, or (b) noninterpersonal (all other events). Given the present focus on the generation of interpersonal stress, a composite score of dependent interpersonal stress was calculated by summing the stress ratings across all relevant events. High reliability was achieved for ratings of stress (ICC = .90) and dependence (ICC = .96) and for event categorization (Cohen’s κ = .92).

**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 menstruation 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 care-giver reports (r = .88, p < .001, for girls; r = .72, p < .001, for boys), composites were formed for each item by averaging youth and caregiver reports (α = .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 on the basis of 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 youth and caregiver reports. 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 (Conley & Rudolph, in press). Thus, a composite score was created by averaging the seven items, standardized within sex. Composite scores were calculated for 146 participants (87% of the 167 youth) with at least five of the seven indicators. Because prior research has used the Tanner stages alone as an index of pubertal development (Dorn, Susman, Nottelmann, Inoff-Germain, & Chrousos, 1990; Morris & Udry, 1980), scores were calculated for another 12 participants with Tanner data only or Tanner data plus one to two PDS items. Thus, pubertal status scores were available for 158 of the 167 youth, yielding the present sample. Higher scores reflected more advanced pubertal status. To create an index of pubertal timing, we computed residualized scores separately for girls and boys by regressing pubertal status onto chronological age (Dorn, Susman, & Ponirakis, 2003). Higher scores reflected earlier maturation relative to one's agemates.

**Results**

**Intercorrelations**

Table 3 displays intercorrelations among the variables. As expected, depression was associated concurrently (in girls only) and over time (in girls and boys) with the generation of interpersonal stress. In girls, more advanced pubertal status and earlier pubertal timing were associated
concurrently and over time with stress generation. In boys, more advanced pubertal status was not associated with stress generation, but earlier pubertal timing was associated with stress generation at T2. Despite the more consistent pattern of correlations in girls than in boys, the size of these correlations did not significantly differ across sex. The positive and significant correlations between puberty (status and timing) and depression in girls were significantly greater than the negative and nonsignificant correlations between puberty and depression in boys (Zs ≥ 2.63, ps < .01).

**Contributions of Depression and Development to Interpersonal Stress Generation**

We conducted hierarchical multiple regression analyses to assess the contributions of depression, development (age, pubertal status, and pubertal timing), and sex to the generation of interpersonal stress over time. For each analysis, the main effects of T1 depression, T1 development, and sex were entered at the first step, the two-way interactions were entered at the second step, and the three-way Depression × Development × Sex interaction was entered at the third step. Analyses predicted T2 dependent interpersonal stress, adjusting for T1 dependent interpersonal stress (also entered at the first step). The continuous predictors were mean centered prior to analysis. Sex did not significantly moderate any of the Depression × Development interactions (βs < .17, ns); thus, regressions were rerun without sex. We interpreted significant interactions by solving the unstandardized regression equation to predict dependent interpersonal stress from depression at low (−1 SD), moderate (mean), and high (+1 SD) levels of development (Aiken & West, 1991).

Each of the analyses yielded a significant main effect of depression (see Table 4). For age and pubertal status, both the main effects of development and the Depression × Development interactions were nonsignificant. A significant main effect of pubertal timing indicated that early development predicted more interpersonal stress generation. However, the main effects of depression and pubertal timing were qualified by a significant Depression × Pubertal Timing interaction. As hypothesized, decomposition of this effect (see Figure 1) revealed that depression significantly predicted the generation of interpersonal stress in early-developing, β = .36, t(143) = 3.84, p < .001, but not late-developing, β = .00, t(143) = −0.04, ns, youth. A moderate effect was found for on-time youth, β = .18, t(143) = 2.48, p < .05.

**Supplemental Analyses**

To examine an assumption of stress-generation theory, namely that depression is particularly likely to lead to the generation of stress within relationships (Hammen, 2006), we conducted an additional set of hierarchical multiple regression analyses to investigate the specificity of these findings to interpersonal versus noninterpersonal stress. These analyses paralleled the first set with the following additions: (a) each analysis predicted one type of dependent stress, adjusting for the alternate type of dependent stress, and (b) because co-occurrence of depression and externalizing disorders are associated with heightened stress generation (Rudolph et al., 2000), each analysis adjusted for T1 externalizing psychopathology, also assessed with the Schedule for Affective Disorders and Schizophrenia for School-Age Children.

For the prediction of interpersonal stress, findings were highly similar to those reported in Table 4 with one exception: The main effect of depression became marginal for the age analysis and nonsignificant for the pubertal status and timing analyses. The main effect of pubertal

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1 This regression was rerun adjusting for T2 depression. The Depression × Pubertal Timing interaction remained a significant predictor of T2 dependent interpersonal stress, β = .13, t(141) = 1.80, p < .05, one-tailed. This conservative test of our hypothesis suggested that the interaction effect was not accounted for by concurrent (T2) depression.

2 These regressions also were conducted using a dummy-coded categorical variable representing youth with versus without depressive symptoms. These analyses yielded highly similar results, namely nonsignificant Depression × Age and Depression × Pubertal Status interactions and a significant Depression × Pubertal Timing interaction, β = .25, t(143) = 2.88, p < .01.
timing, $\beta = .18, t(141) = 2.48, p < .05$, and the Depression × Pubertal Timing interaction, $\beta = .18, t(141) = 2.53, p < .05$, remained significant. After we adjusted for noninterpersonal stress and externalizing psychopathology, depression significantly predicted interpersonal stress generation in early-developing, $\beta = .29, t(141) = 2.86, p < .01$, but not on-time, $\beta = .12, t(141) = 1.53, ns$, or late-developing, $\beta = −.05, t(141) = −0.44, ns$, youth. In all three regressions, externalizing psychopathology did not significantly predict interpersonal stress generation, $\beta$s $\leq .06, ts(141) \leq .56, ns$.

For the prediction of noninterpersonal stress, nonsignificant effects were found for the main effect of depression, $\beta$s $\leq .12, ts(141) \leq 1.46, ns$, the main effect of development, $\beta$s $\leq .06, ts(141) \leq 0.84, ns$, and the Depression × Development interactions, $\beta$s $\leq .09, ts(141) \leq 1.20$. In each regression, externalizing psychopathology significantly predicted noninterpersonal stress generation, $\beta$s $\geq .33, ts(141) \geq 3.97, ps < .001$.

**Discussion**

Consistent with stress-generation theory, this study revealed that depression predicted prospective increases in the generation of interpersonal stress, suggesting that depressed youth actively shape their social environments in ways that may contribute to the continuity of depression. Building on prior work, this study also provided insight into the developmental context of stress generation. Specifically, pubertal timing, but not chronological age or pubertal status, amplified the interpersonal stress-generation process in depressed youth. This effect was quite robust, remaining significant even after adjusting for co-occurring externalizing psychopathology. Analyses of specificity revealed that this amplification effect was specific to interpersonal but not noninterpersonal stress.

These findings support models of development and psychopathology that focus on the adverse psychosocial effects of early pubertal timing. In particular, they support Caspi and Moffitt’s (1991) interactive “transitional stressor” model of early maturation, which proposes that individual differences are magnified under conditions of uncertainty and novelty, when youth are required to negotiate new challenges. The transition through puberty is marked by a reorganization of youths’ social contexts and heightened demands on their coping resources. Undergoing the transition prior to one’s peers contributes to a host of particularly challenging social experiences, such as heightened family tension, affiliation with older and norm-breaking peer groups, and early entrance into romantic relationships and sexual activity (for reviews, see Paikoff & Brooks-Gunn, 1991; Susman & Rogol, 2004; Weichold et al., 2003). Moreover, developing earlier than one’s peers propels youth into these challenging social contexts before they are psychologically prepared to cope with their complexities. Early-maturing depressed youth are likely to be at particular risk for generating stress in their relationships because (a) they are especially likely to enter into risky social contexts (Ge, Conger, & Elder, 1996), and (b) they are navigating these challenges with personal resources and support systems that are already compromised by depressive symptoms and associated interpersonal deficits. In contrast, late-maturing youth seem to be buffered from the potential interpersonal stress-generation effect of depression. These youth do not have to simultaneously face stressors associated with changing physical development and changing social relationships; in fact, they likely face fewer social challenges than do early-maturing youth. Moreover, because of their younger physical appearance, adults may provide greater emotional support and scaffolding as they negotiate new relationships.

Beyond their involvement in challenging social contexts, other mechanisms may help to explain why early-maturing depressed youth generate interpersonal stress (or, alternatively, why late-maturing depressed youth are protected from this effect). Early maturation may foster psychological attributes that amplify the maladaptive interpersonal styles of depressed youth.
For example, heightened self-focus and a sense of insecurity or alienation resulting from their differing physical and social status may cause early-maturing depressed youth to engage in particularly high levels of reassurance seeking, thereby eliciting rejection, or to disengage from relationships, thereby leading to social isolation. Alternatively, biological processes associated with early maturation may intensify the stress-generation effect of depression. In light of the present focus on the psychological and social effects of puberty, this study assessed puberty through secondary sexual characteristics and other somatic markers. However, hormonal and morphological changes represent related but distinct indexes of pubertal development. Thus, it would be interesting to examine whether hormonal correlates of early maturation contribute to heightened risk for interpersonal stress generation in the context of depression.

It is notable that this study did not reveal sex differences in the interactive contribution of depression and early pubertal timing to interpersonal stress generation. Differentiating types of interpersonal stress (e.g., within intimate friendships vs. the broader peer group) may elucidate sex differences in the stress-generation process (Rudolph, 2002). Moreover, self-generated interpersonal stress may contribute more strongly to subsequent depression in girls than in boys (Hankin, Mermelstein, & Roesch, 2007). Thus, a self-perpetuating cycle may unfold across development wherein early-maturing depressed girls generate stress in their relationships, which perpetuates or exacerbates their depression. Examining such reciprocal transactions between depression and stress generation in early-maturing youth could shed light on the continuity of depression over time, the emergence of sex differences in depression, and the potential long-term adverse effects of early pubertal timing.

Overall, this research supports the importance of a developmentally informed approach that considers the complex interactions among development, social context, and depression. However, some limitations should be noted. First, although this sample included a reasonable percentage of youth who had experienced episodes of moderate to severe depression, these findings need to be replicated with more severely depressed youth. Second, despite some ethnic diversity, the sample size was not sufficient to test ethnic differences in the stress-generation process. Because puberty has different implications and consequences across ethnic groups (Hayward, Gotlib, Schraedley, & Litt, 1999), future research should investigate possible ethnic differences. Finally, this study investigated stress generation across a broad time span; it would be interesting to examine the relative short- versus long-term effects of depression and pubertal timing on stress generation.

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Figure 1.
The interactive contribution of depression and pubertal timing to interpersonal stress generation.
### Table 1

#### Descriptive Statistics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Girls</th>
<th></th>
<th></th>
<th></th>
<th>Boys</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>n</td>
<td>M</td>
<td>SD</td>
<td></td>
<td>n</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
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<td>82</td>
<td>12.37</td>
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<td></td>
<td>76</td>
<td>12.41</td>
<td>1.15</td>
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</tr>
<tr>
<td>W&lt;sub&gt;1&lt;/sub&gt; Pubertal Development Scale (Petersen, Crockett, Richards, &amp; Boxer, 1988)</td>
<td>76</td>
<td>2.54&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.79</td>
<td></td>
<td>69</td>
<td>1.99&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.61</td>
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<tr>
<td>W&lt;sub&gt;1&lt;/sub&gt; Tanner stages</td>
<td>81</td>
<td>2.97&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.19</td>
<td></td>
<td>70</td>
<td>2.65&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>W&lt;sub&gt;1&lt;/sub&gt; depression</td>
<td>82</td>
<td>0.67</td>
<td>1.43</td>
<td></td>
<td>76</td>
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<td>W&lt;sub&gt;1&lt;/sub&gt; dependent interpersonal stress</td>
<td>82</td>
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<td>76</td>
<td>3.51</td>
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<td>W&lt;sub&gt;2&lt;/sub&gt; dependent interpersonal stress</td>
<td>78</td>
<td>3.06</td>
<td>3.47</td>
<td></td>
<td>70</td>
<td>3.10</td>
<td>4.50</td>
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*Note.* W<sub>1</sub> = Wave 1; W<sub>2</sub> = Wave 2.

<sup>a</sup>Means differ at *p* < .001.

<sup>b</sup>Means differ at *p* < .10.
Table 2
Levels of Depression Within Developmental Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Age group</th>
<th>Pubertal status group</th>
<th>Pubertal timing group</th>
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<td>1</td>
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<td>3</td>
</tr>
<tr>
<td>n</td>
<td>26</td>
<td>27</td>
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<tr>
<td>M depressive symptoms</td>
<td>0.58</td>
<td>0.93</td>
<td>0.52</td>
</tr>
<tr>
<td>% with depression</td>
<td>11.5</td>
<td>18.5</td>
<td>10.3</td>
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<tr>
<td>% with symptoms</td>
<td>19.2</td>
<td>18.5</td>
<td>10.3</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>M depressive symptoms</td>
<td>0.78</td>
<td>0.48</td>
<td>0.42</td>
</tr>
<tr>
<td>% with depression</td>
<td>11.1</td>
<td>8.0</td>
<td>4.2</td>
</tr>
<tr>
<td>% with symptoms</td>
<td>25.9</td>
<td>12.0</td>
<td>16.7</td>
</tr>
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</table>

Note. M depressive symptoms = mean depressive symptoms on continuous rating scale; % with depression = percentage of youth with a depression diagnosis; % with symptoms = percentage of youth with moderate or diagnostic-level symptoms. For each developmental index, 1 = bottom third of sample; 2 = middle third of sample; 3 = top third of sample.
### Table 3

Correlations Among the Variables by Sex

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
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<tr>
<td>1. W1 age</td>
<td>—</td>
<td>.74***</td>
<td>.00</td>
<td>.04</td>
<td>.11</td>
<td>.15</td>
</tr>
<tr>
<td>2. W1 pubertal status</td>
<td>.64***</td>
<td>—</td>
<td>.68***</td>
<td>.23*</td>
<td>.25*</td>
<td>.31**</td>
</tr>
<tr>
<td>3. W1 pubertal timing</td>
<td>.00</td>
<td>.77***</td>
<td>—</td>
<td>.30**</td>
<td>.25*</td>
<td>.29**</td>
</tr>
<tr>
<td>4. W1 current depression</td>
<td>−.12</td>
<td>−.19</td>
<td>−.15</td>
<td>—</td>
<td>.30**</td>
<td>.24*</td>
</tr>
<tr>
<td>5. W1 dependent stress</td>
<td>.01</td>
<td>.12</td>
<td>.15</td>
<td>.14</td>
<td>—</td>
<td>.30**</td>
</tr>
<tr>
<td>6. W2 dependent stress</td>
<td>−.16</td>
<td>.09</td>
<td>.25*</td>
<td>.27*</td>
<td>.42***</td>
<td>—</td>
</tr>
</tbody>
</table>

**Note.** W1 = Wave 1; W2 = Wave 2. Correlations for girls are above the diagonal; correlations for boys are below the diagonal. Correlations among age, pubertal status, and pubertal timing are reported in Conley and Rudolph (in press).

* \( p < .05. \\
** \( p < .01. \\
*** \( p < .001. \\
**** \( p < .0001. \\

Table 4
Predicting W2 Interpersonal Stress Generation From W1 Depression, Development, and Depression × Development Interactions

<table>
<thead>
<tr>
<th>Wave 1 predictors</th>
<th>Chronological age</th>
<th>Pubertal status</th>
<th>Pubertal timing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β</td>
<td>t</td>
<td>ΔR²</td>
</tr>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interpersonal stress</td>
<td>.35</td>
<td>4.54</td>
<td>***</td>
</tr>
<tr>
<td>Depression</td>
<td>.22</td>
<td>2.89</td>
<td>**</td>
</tr>
<tr>
<td>Development</td>
<td>−.01</td>
<td>−.14</td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression × Development</td>
<td>−.09</td>
<td>−1.22</td>
<td>.01</td>
</tr>
</tbody>
</table>

Note. βs and ts represent standardized coefficients and t statistics at the final step. ΔR² represents percentage of variance accounted for at each step. For each analysis, df = 4,143.

* p < .05.
** p < .01.
*** p < .001.