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**REGULAR ARTICLE**

# Explaining the longitudinal association between puberty and depression: Sex differences in the mediating effects of peer stress

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## Abstract

This research investigated whether exposure to peer stress serves as one pathway through which pubertal development contributes to depression over time, differentially for girls and boys. Youth ( $N = 149$ ; 9.6–14.8 years) and their caregivers provided information at two waves, 1 year apart, on puberty (Wave 1), peer stress (occurring between Waves 1 and 2), and depression (Waves 1 and 2). Structural equation modeling analyses examined sex differences in the extent to which peer stress mediated the impact of pubertal status and timing on subsequent depression (i.e., tests of moderated mediation). Significant sex-moderated mediation was found for both pubertal status and timing. As indicated by moderate effect proportions, in girls, heightened peer stress partially mediated the longitudinal association between (a) more advanced pubertal status and depression; and (b) linear, but not curvilinear, pubertal timing (i.e., earlier maturation) and depression. This research contributes to our growing understanding of the interplay among physical, psychological, and social processes involved in the sex difference in adolescent depression.

Recent research implicates puberty, more so than age, in the emergent sex difference in adolescent depression (Conley & Rudolph, 2009; Hayward, Gotlib, Schraedley, & Litt, 1999). Yet, very little research explores why or how puberty influences depression, and the resulting sex difference that emerges in adolescence. The complex processes underlying the development of psychopathology necessitate research examining contributions from multiple systems (biological, psychological, and interpersonal) and the dynamic transactions between developing adolescents and their social contexts (Cicchetti & Rogosch, 2002; Lerner, 1987; Sroufe & Rutter, 1984). Following recent developments in research on the psychosocial context and effects of puberty (Compian, Gowen, & Hayward, 2009; Conley & Rudolph, 2009; Graber, Brooks-Gunn, & Archibald, 2005), the present study investigated sex differences in one possible pathway through which

puberty contributes to adolescent depression, namely, exposure to heightened peer stress.

## Pubertal Development and the Sex Difference in Depression

Research on pubertal development has revealed, beyond mere physical and biological changes, many psychological and social implications of puberty (Graber, 2003; Haynie & Piquero, 2006). There are several ways in which pubertal development might heighten risk for depression in particular. On a psychosocial level, pubertal changes confer negative psychological (e.g., poor body image) and social (e.g., exclusion, victimization) risks, which in turn predict depression. On a physical level, puberty entails bodily changes that mark a physical transition to adulthood at a time when adolescents are socially unprepared. On a biological level, puberty brings hormonal changes linked to depression (Angold, Costello, Erkanli, & Worthman, 1999; Susman, Dorn, & Chrousos, 1991). Just as pubertal hormones differ for girls and boys, the psychological and social effects of puberty vary by sex, which might influence the emerging sex difference in adolescent depression.

Pubertal *status*, one's stage of physical maturation, has been implicated in rising adolescent depression and the sex difference therein. More mature pubertal status in girls, but not in boys, is linked to higher rates of depressive disorders (Angold, Costello, & Worthman, 1998), as well as to higher levels of depressive symptoms and mood (Ge, Elder, Regnerus, & Cox, 2001; Hayward et al., 1999; Wichstrom, 1999). In one study,

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pubertal status fully accounted for the sex difference in adolescent depression (Ge, Conger, & Elder, 2001). Other research reveals that pubertal status, but not age, accounts for the sex difference in depression (Angold et al., 1998; Conley & Rudolph, 2009).

Pubertal *timing* (pubertal status relative to age) might have even stronger links with psychosocial adjustment (Negri, Fung, & Trickett, 2008). Earlier-developing adolescents might be underprepared for these changes, feel deviant and insecure about their difference, and lack social support from peers experiencing similar changes (Petersen, 1983; Ruble & Brooks-Gunn, 1982). On the other end of the spectrum, youth who develop later than their peers might feel left behind as their more-developed peers transition into adolescence. Thus, both earlier and later pubertal timing can heighten risk for psychosocial distress (Natsuaki, Biehl, & Ge, 2009; Weichold, Silbereisen, & Schmitt-Rodermund, 2003). Indeed, research links pubertal timing with depression, particularly in girls. Most consistently, earlier-maturing girls exhibit more depressive disorders, symptoms, and mood than their on-time or later-maturing peers (e.g., Conley & Rudolph, 2009; Ge et al., 2003; Negri et al., 2008; Patton et al., 2008). Later-maturing girls also experience psychological difficulties (Carter, Jaccard, Silverman, & Pina, 2009; Dorn, Susman, & Ponirakis, 2003; Natsuaki, Biehl, et al., 2009). Among boys, *later* maturation is associated with elevated depressive symptoms (Benjet & Hernández-Guzmán, 2002; Dorn et al., 2003; Huddleston & Ge, 2003; Weichold et al., 2003), and there is increasing evidence that earlier-maturing boys also exhibit more depression (Kaltiala-Heino, Kosunen, & Rimpela, 2003; Natsuaki, Biehl, et al., 2009; Negri et al., 2008). These findings suggest a curvilinear association between pubertal timing and depression for both girls and boys (Conley & Rudolph, 2009; for reviews, see Huddleston & Ge, 2003; Weichold et al., 2003).

### Social Processes Linking Puberty to Depression

Puberty is likely to affect adolescents' social worlds because it occurs within a social context. Pubertal development entails bodily changes that are held to close scrutiny by peers, amplified by the focus on social comparison and conformity in adolescence (Brooks-Gunn & Warren, 1989; Ruble & Brooks-Gunn, 1982). Further, many of the psychological effects of puberty depend on adolescents' reference to their peer group. In many Western cultures, for boys it is socially desirable to have the postpubertal physical form, whereas for girls it is not (Petersen & Crockett, 1985); thus, pubertal maturation has more negative psychological and social effects for girls compared to boys in general, and for earlier-developing girls and later-developing boys in particular (Felson & Haynie, 2002; Simmons, Blyth, & McKinney, 1983; Taga, Markey, & Friedman, 2006; Tobin-Richards, Boxer, & Petersen, 1983). For example, more advanced status and earlier timing are linked to girls' quantity and quality of friendships, friendship group composition (e.g., deviant peers, opposite-sex peers), and involvement in romantic and sexual relationships

(Cavanagh, 2004; Haynie, 2003). A growing body of research links girls' off-time development in *both* directions (earlier and later) to social disadvantages and stressors, including lack of close friendships (earlier and later puberty; Brooks-Gunn, Warren, Samelson, & Fox, 1986), low social support, acceptance, and popularity (later puberty; Brooks-Gunn & Warren, 1988; Michael & Eccles, 2003), and greater physiological reactivity to interpersonal conflict (earlier puberty; Smith & Powers, 2009). Furthermore, girls typically have a stronger depressive response to peer stress, or more broadly to interpersonal stress (Leadbeater, Blatt, & Quinlan, 1995; Oldenburg & Kerns, 1997; Rudolph, 2002; Rudolph & Hammen, 1999; Schraedley, Gotlib, & Hayward, 1999). In sum, this research suggests a curvilinear association between pubertal timing and stress in the peer domain, such that off-time development confers social disadvantages for girls, more so than for boys, which in turn might contribute to the sex difference in adolescent depression (Rudolph, 2009).

Although direct tests of such mediation pathways are limited, some research suggests that the impact of puberty on depression is at least in part due to psychosocial influences. For example, a recent study (Natsuaki, Klimes-Dougan, et al., 2009) revealed that earlier-maturing girls displayed heightened internalizing symptoms, in part because of their elevated sensitivity to interpersonal stress. In contrast, this mediation pathway did not hold up in boys because the association between interpersonal sensitivity and internalizing symptoms was nonsignificant. Other research found that earlier pubertal timing in girls predicted less adaptive responses to peer stress, which in turn predicted higher levels of aggression, but this model did not hold up for internalizing symptoms (Sontag, Graber, Brooks-Gunn, & Warren, 2008). A similar study (Graber, Brooks-Gunn, & Warren, 2006) revealed that the association between girls' earlier pubertal timing and depressive symptoms was mediated by emotional arousal.

Despite these important contributions, prior research examining mediational pathways linking puberty to adjustment suffers from several methodological limitations, including (a) concurrent designs; (b) the predominant use of symptom checklists to assess depression, which may not provide optimal discrimination among different types of psychopathology; and (c) limited assessment and operationalization of pubertal development (e.g., single-item assessments, dichotomous categories rather than continuous variables of pubertal timing; for two exceptions, see Lindberg, Grabe, & Hyde, 2007; Natsuaki, Klimes-Dougan, et al., 2009). The present study aimed to address these limitations and thus illuminate the longitudinal process linking puberty to depression in adolescence.

### Study Overview

A large body of research suggests that puberty contributes to adolescents' peer contexts and depression. Research also shows that peer stressors contribute to depression among adolescents, particularly girls (Hankin, Mermelstein, & Roesch, 2007; Rudolph, Flynn, Abaid, Groot, & Thompson, 2009).

Furthermore, the psychosocial effects of puberty appear to be more devastating for girls than for boys. This study extends prior research by examining whether puberty contributes to adolescent depression through heightened stress in the peer domain, differentially for girls and boys. Specifically, we hypothesized the following:

1. Sex would moderate the pathway from puberty to peer stress to depression.
2. Puberty would predict both peer stress and depression in the following ways:
  - a. More advanced *pubertal status* was expected to predict heightened subsequent peer stress and depression in girls (i.e., a positive linear association) but either to have no association, or to predict less subsequent peer stress and depression, in boys (i.e., a null or negative linear association).
  - b. In girls, both positive linear and positive curvilinear associations were expected from *pubertal timing* to subsequent peer stress and depression. In other words, earlier pubertal timing, and to a lesser extent, later timing, would predict more peer stress and depression over time. In boys, the opposite pattern was expected: later timing, and to a lesser extent, earlier timing, would predict more peer stress and depression over time (i.e., negative linear and positive curvilinear associations).
3. Peer stress would predict depression, above and beyond the contribution of puberty, in both girls and boys (but more strongly in girls).
4. The inclusion of peer stress in the longitudinal models would reduce the effects of puberty on depression, more strongly in girls than in boys.

## Method

### Participants

The present study involved the first two waves (Wave 1 and Wave 2) of a longitudinal investigation examining the development of depression during the adolescent transition (e.g., Conley & Rudolph, 2009; Rudolph et al., 2009; Rudolph & Troop-Gordon, 2010). Participants in the longitudinal study included 167 families drawn from a midsized midwestern city and several rural towns. Recruited youth for the longitudinal study had participated in schoolwide screenings using the Children's Depression Inventory (CDI; Kovacs, 1992). Youth who participated in these screenings represented approximately 80% of targeted participants. From the screening sample ( $n = 1,985$ ), we selected potential participants ( $n = 468$ ) along the range of the CDI, oversampling slightly at the high end without regard to sex (i.e., whereas 15.8% of the screening sample had CDI scores above 18, 20.3% of the participants we targeted for recruitment fell into this category). Participants from the screening sample were recruited for the longitudinal study based on CDI scores, a maternal caregiver in the home, and proximity to the university, until

the targeted sample was successfully recruited. Participants and nonparticipants in the longitudinal study did not differ in sex,  $\chi^2(1) = 0.39$ ,  $r = .04$ ,  $p = .53$ , ethnicity (White vs. minority),  $\chi^2(1) = 0.02$ ,  $r = .01$ ,  $p = .89$ , or depressive symptoms,  $t(280) = 1.11$ ,  $r = .07$ ,  $p = .13$ . Participants ( $M = 12.41$ ) were slightly younger than nonparticipants ( $M = 12.65$ ),  $t(275) = 2.28$ ,  $r = .14$ ,  $p = .012$ .

This research focused on a subsample of 149 youth (78 girls, 71 boys) who had relevant data on pubertal development, peer stress, and depression.<sup>1</sup> Among this subsample ( $M$  age = 12.38,  $SD = 1.24$ , range = 9.6–14.8; 77.2% White, 22.8% minority), socioeconomic status was diverse, with total family income below \$30,000 for 16.4% of the sample, and above \$75,000 for 18.5% of the sample. Of the original 167 participants, youth with complete data did not differ from those with missing data, in sex,  $\chi^2(1) = 0.40$ ,  $r = .08$ ,  $p = .53$ , ethnicity (White vs. minority),  $\chi^2(1) = 0.35$ ,  $r = .07$ ,  $p = .55$ , or any of the puberty, stress, or depression variables ( $ts < 1.63$ ,  $rs < .20$ ,  $ps > .05$ ), but those with complete data were slightly younger than those with missing data,  $t(165) = 2.18$ ,  $r = .26$ ,  $p = .015$ .

### Procedure

Youth and primary female caregivers completed 3- to 4-hr assessments with two interviewers, at baseline and 1 year later. At each assessment, families received a cash stipend, and youth received a gift certificate.

### Measures

Table 1 presents descriptive information for the measures.

*Assessment of pubertal status and timing.* Given this study's focus on how somatic changes associated with puberty influence depression, we followed precedent from past research by assessing secondary sex characteristics and other physical changes of puberty (Dubas, Graber, & Petersen, 1991; Ge, Elder, et al., 2001; Hayward, 2003; Petersen, Crockett, Richards, & Boxer, 1988). Participants completed two assessments of youths' pubertal status at Wave 1. The first measure consisted of a series of drawings illustrating the stages of pubertal development specified by Tanner (1969), and adapted by Morris and Udry (1980). Informants indicated which of the drawings in each group most closely matched youths' current stage of development.

Youths' self-ratings on the Tanner stages are significantly associated with clinician ratings on physical exams (Dorn, Susman, Nottelmann, Inoff-Germain, & Chrousos, 1990; Schlossberger, Turner, & Irwin, 1992; Shirtcliff, Dahl, & Pollak, 2009). In the present sample, youth and caregiver reports correlated well for girls' breast ( $r = .83$ ,  $p < .001$ ; 97% agreement within one category) and pubic hair ( $r = .68$ ,  $p < .001$ ;

1. Eight participants were missing Wave 1 puberty data, and an additional 10 participants were missing Wave 2 stress and/or depression data.

**Table 1.** Descriptive statistics and comparisons by sex

	Girls			Boys		
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>
W <sub>1</sub> pubertal status composite <sup>a</sup>	78	3.13	1.20	71	2.50	0.99
W <sub>1-2</sub> chronic peer stress	78	2.09	0.89	71	2.23	0.91
W <sub>1-2</sub> episodic peer stress	78	2.92	2.85	70	2.84	3.30
W <sub>1-2</sub> peer stress composite	78	-0.03	0.85	71	0.02	0.89
W <sub>1</sub> depression	78	0.63	1.36	71	0.61	1.19
W <sub>2</sub> depression	78	0.71	1.41	71	0.46	0.92
W <sub>2</sub> [W <sub>1</sub> ] residualized depression	78	0.12	1.05	71	-0.13	0.94

Note: W<sub>1-2</sub> peer stress consists of peer stress occurring between Wave 1 (W<sub>1</sub>) and Wave 2 (W<sub>2</sub>). W<sub>2</sub> [W<sub>1</sub>] residualized depression consists of W<sub>2</sub> depression, adjusting for W<sub>1</sub> depression (both measures over the past month).

<sup>a</sup>Variable differed significantly between sexes.

84% agreement within one category) development, and moderately well for boys' genital ( $r = .46, p < .01$ ; 78% agreement within one category) and pubic hair ( $r = .66, p < .001$ ; 79% agreement within one category) development, similar to other studies (e.g., Dorn et al., 1990). Youth and caregiver reports were averaged into consensual ratings, and then combined to form a single index of pubertal development ( $\alpha > 0.91$  for girls and boys;  $ps < .001$ ). The present sample included youth across the full range of Tanner stages: 31.4% of girls and 35.8% of boys fell between 1 and 2, 41.5% of girls and 46.2% of boys fell between 2 and 4, and 27.1% of girls and 18% of boys fell between 4 and 5.

The second measure, the Pubertal Development Scale (PDS; Petersen et al., 1988) assesses five physical aspects of pubertal development with Likert ratings (1 = *no development*, 2 = *development has just begun*, 3 = *development is definitely underway*, 4 = *development is complete*). The PDS has been well validated, with interitem reliability ranging from the 0.50s to the 0.80s (median  $\alpha = 0.71$  across three studies; Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Petersen et al., 1988; Tobin-Richards et al., 1983). The PDS also is moderately correlated with clinician ratings of the Tanner stages (Brooks-Gunn et al., 1987; Shirtcliff et al., 2009).

We scored the PDS using an established method that maps the measure's five pubertal indicators onto two pubertal indexes: adrenal and gonadal maturation. These two scores map onto clinician-rated Tanner stages, using a parallel 5-point scale, and correlate well with the underlying hormonal processes of puberty (see Shirtcliff et al., 2009). Youth and caregiver reports on these two PDS indexes were moderately correlated in the present sample ( $rs > .77$  for girls and  $> .57$  for boys;  $ps < .001$ ), and were averaged into consensual ratings. Following Shirtcliff et al. (2009), these two scores were then averaged to form a single index of pubertal development ( $\alpha > 0.85$  for girls and boys;  $ps < .001$ ).

### Creation of pubertal status and timing variables.

**Pubertal status.** Confirming the validity of Shirtcliff and colleagues' method (2009), scores from the Tanner ratings and the PDS correlated strongly with one another ( $rs = .86$  for girls and  $.72$  for boys;  $p < .001$ ), and thus were averaged to form an overall composite index of pubertal maturation ( $\alpha = 0.94$  for girls and  $0.86$  for boys;  $ps < .001$ ). Higher scores reflected *more advanced* pubertal status.

**Pubertal timing.** 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 age mates. 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; Steinberg, 1987; Susman & Rogol, 2004; Weichold et al., 2003).

**Assessment and coding of peer stress.** The Youth Life Stress Interview (Rudolph & Flynn, 2007), a revised version of the Child Episodic Life Stress and Chronic Strain Interviews (Rudolph & Hammen, 1999; Rudolph et al., 2000), assessed peer stress occurring between Wave 1 and Wave 2. This semi-structured interview elicits information from youth and their caregivers about the nature and intensity of chronic and episodic stress youth experienced over the past year.

Interviewers presented narrative information to a team of trained coders who had no knowledge of youths' diagnostic status or subjective response to the stress. Coders provided consensual ratings based on youth and caregiver reports. For chronic stress, coders rated the severity of stress on a 5-point scale: 1 = *no stress*, 2 = *mild stress*, 3 = *isolated stress*, 4 = *serious stress*, 5 = *severe stress*.<sup>2</sup> For episodic stress, coders rated the stressfulness or negative impact of each event, from 1 (*none*) to 5 (*severe*),<sup>3</sup> reflecting how stressful the event would be for a typical child in the described circumstances. Episodic peer stress scores were calculated as the total of the objective stress ratings for each peer event with a stress rating above 1.

To determine reliability, information from 41 interviews (including 160 episodic stress events) was presented to two teams of coders, who gave independent ratings. One-way random-effects intraclass correlation coefficients (ICCs) evi-

2. An example of a participant with a chronic peer stress rating of 1 (*no stress*): has many friends including four close friends; sees friends every day and engages in many social activities; is not lonely, teased, or under peer pressure; does not have arguments with friends. Example of a participant with a chronic peer stress rating of 5 (*severe stress*): moved from another state in the middle of the school year; made one friend at school but they broke up; feels lonely and gets teased every day.
3. An example of a peer event rated as 1.5 (lowest rating in sample; *no to mild stress*): grew apart from a friend, without any animosity, and while maintaining social contact in group settings. Example of a peer event rated as 4.5 (highest rating in sample; *serious to severe stress*): had a confrontational falling out with closest friend, resulting in ending the friendship and teasing by other peers.

denced high reliability for the chronic peer stress rating (ICC = 0.96) and the objective episodic stress rating (ICC = 0.90). The Cohen  $\kappa$  value for agreement on whether an event was peer-related was 1.00. As might be expected, stability was strong for chronic stress ( $r = .67, p < .001$ ) and moderate for episodic stress ( $r = .37, p < .001$ ). Confirming that chronic and episodic peer stress assess closely related aspects of peer stress, these two scores were moderately correlated ( $r_s = .53$  and  $.48$ , respectively, for Wave 1 and Wave 2,  $p_s < .001$ ). Thus, we computed a composite score of peer stress by averaging standardized scores on the two measures.

*Assessment and coding of depression.* Interviewers administered the Schedule for Affective Disorders and Schizophrenia for School-Age Children—Epidemiologic Version 5 (Orvaschel, 1995) to youth and their caregivers, to assess youth depression at Wave 1 and Wave 2. Consensual diagnoses were assigned using a best-estimate approach (Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005; Klein, Ouimette, Kelly, Ferro, & Riso, 1994) to combine youth and caregiver reports.

For each period and type (e.g., major depression, dysthymia) of depression (both diagnosable episodes and subclinical symptoms), interviewers used the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision; *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 impairment, 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 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 and/or the presence of symptoms from multiple categories (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. 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, Eberhart, Hammen, & Brennan, 2006). Depression ratings demonstrated strong interrater reliability (one-way random-effects ICC = 0.97, based on independent coding of 42 interviews) and high stability over time ( $r = .67, p < .001$ ).

At Wave 1, 37 youth (24.8%) had some depression (i.e., a score above 0 for at least one type of depression), and 15 of these youth (10.1%) had a clinical diagnosis of depression or dysthymia. At Wave 2, 40 youth (26.8%) had some depression, and 12 of these youth (8.1%) had a diagnosis. Scores on the depression summary index ranged from 0 to 7 at Wave 1, and 0 to 6 at Wave 2.

### Results

As reflected in Table 1, girls' pubertal status was significantly more advanced than boys',  $t(147) = 3.47, r = .28, p < .001$ . This difference, along with the absence of a sex difference in age,  $t(147) = .23, r = .02, p = .82$ , is consistent with the fact that pubertal maturation occurs earlier in girls than in boys. There were no sex differences in any other variables ( $t_s < 1.52, r_s < .12, p_s > .065$ ). The absence of sex differences in depression and peer stress is likely due to the fact that these sex differences tend to emerge during middle adolescence (about age 13; e.g., Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Ge, Lorenz, Conger, Elder, & Simons, 1994; Rudolph & Hammen, 1999), and nearly two-thirds of the present sample was younger than 13 years old.

Table 2 presents intercorrelations among the study variables. As expected, more advanced pubertal status and earlier pubertal timing generally correlated with peer stress and depression in girls. In contrast, this pattern was not as strong

**Table 2.** Correlations among puberty, peer stress, and depression in girls and boys

	1	2	3	4	5	6
1. W <sub>1</sub> pubertal status composite	—	.66***	.20†	.22†	.22†	.13
2. W <sub>1</sub> pubertal timing	.75***	—	.22†	.27*	.35**	.28*
3. W <sub>1-2</sub> peer stress composite	-.20†	-.08	—	.39***	.51***	.42***
4. W <sub>1</sub> depression	-.21†	-.17	.46**	—	.75***	.23*
5. W <sub>2</sub> depression	-.03	.17	.58***	.48***	—	.82***
6. W <sub>2</sub> [W <sub>1</sub> ] residualized depression	.14	.33**	.23†	-.33**	.67***	—

Note: Correlations in girls are presented above the diagonal, and correlations in boys are presented below the diagonal.  $N_s = 78$  girls and 71 boys. W<sub>1-2</sub> peer stress consists of peer stress occurring between Wave 1 (W<sub>1</sub>) and Wave 2 (W<sub>2</sub>). W<sub>2</sub>[W<sub>1</sub>] residualized depression consists of W<sub>2</sub> depression, adjusting for W<sub>1</sub> depression (both measures over the past month). † $p < .10$ . \* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$ .

or consistent in boys. Furthermore, among both girls and boys, peer stress was strongly associated with depression. Notably, however, residualized depression was more strongly associated with peer stress in girls than in boys.

#### Overview: Data analysis strategy

Structural equation modeling analyses were conducted via LISREL 8 (Jöreskog & Sörbom, 1996) to examine sex differences in the pathway from puberty to peer stress to depression across a 1-year period. All path models used covariance matrices as input with maximum likelihood estimation, which is robust to moderate violations of nonnormality (Bollen, 1989). Path models were estimated separately for pubertal status and timing. Residualized depression (Wave 2 adjusting for Wave 1) was used as the final outcome variable to examine changes in depression over the past year. This allowed for a rigorous test of longitudinal effects, while also conserving degrees of freedom and increasing statistical power (Cohen & Cohen, 1975). Two steps were taken for each analysis.

First, to test hypotheses about differences in mediation between girls and boys, we used multigroup path analysis to impose cross-group equality constraints on the magnitude of path coefficients across groups (Jöreskog & Sörbom, 1996). We also used a cross-group algebraic constraint to provide a direct test of sex-moderated mediation by constraining the product of the path coefficients composing the indirect effect to be equal in girls and boys. Given a “saturated” baseline model with perfect goodness of fit, a significant multigroup chi-square value signified that the coefficients being constrained equal were in fact different across groups, whereas a nonsignificant multigroup chi-square value signified that the coefficients being compared did not differ across groups.

Second, when evidence was found for significant sex-moderated mediation, mediation was tested within girls and boys based on the parameters obtained from each single-group path model. Several indicators were examined to evaluate the degree of mediation (Baron & Kenny, 1986; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Shrout & Bolger, 2002). Each *total effect* (i.e., puberty to depression, without peer stress in the model) was compared to the corresponding *direct effect* (i.e., puberty to depression, with peer stress in the model). To quantify the strength of mediation, each *indirect effect* was examined for size, significance (Sobel, 1982, 1986), and ratio to the total effect (i.e., *effect proportion mediated* [ $P_M$ ], using standardized estimates). When the total effect was smaller than the direct effect (i.e., suppression), the effect proportion was not calculated. Finally, the strength of the paths from puberty to peer stress, and peer stress to depression, was examined.<sup>4</sup>

4. We report two-tailed  $p$  values based on unstandardized coefficients divided by their standard errors. Thus, a standardized coefficient can be nonsignificant even if it is larger than a smaller standardized coefficient that is statistically significant. As measures of effect size, we report Pearson  $r$  (for between-group differences in means and frequencies, and differences in maximum-likelihood chi-square values), and standardized regression coefficients (for direct, indirect, and total effects). In some cases, the

#### Pubertal status

The model for pubertal status predicted 18% of the variance in depression in girls (medium effect size) but only 9% in boys (small effect size; see Figure 1). Confirming the a priori hypothesis, a test of sex-moderated mediation indicated that the indirect effect of pubertal status on depression via peer stress was significantly different in girls ( $\beta = 0.08$ ,  $p = .10$ ) and boys ( $\beta = -0.05$ ,  $p = .17$ ),  $\chi^2(1, N = 149) = 6.04$ ,  $r = .20$ ,  $p = .014$ . Multigroup path analysis using equality constraints revealed that the path coefficient linking pubertal status to peer stress significantly differed in girls and boys,  $\chi^2(1, N = 149) = 5.95$ ,  $r = .20$ ,  $p = .015$ . Although the effects within sex did not reach significance, more advanced pubertal status predicted more peer stress in girls ( $\beta = 0.20$ ,  $p = .069$ ) but less peer stress in boys ( $\beta = -0.20$ ,  $p = .089$ ). There was no significant sex difference in the strength of the path coefficient linking peer stress to depression,  $\chi^2(1, N = 149) = 1.52$ ,  $r = .10$ ,  $p = .22$ .

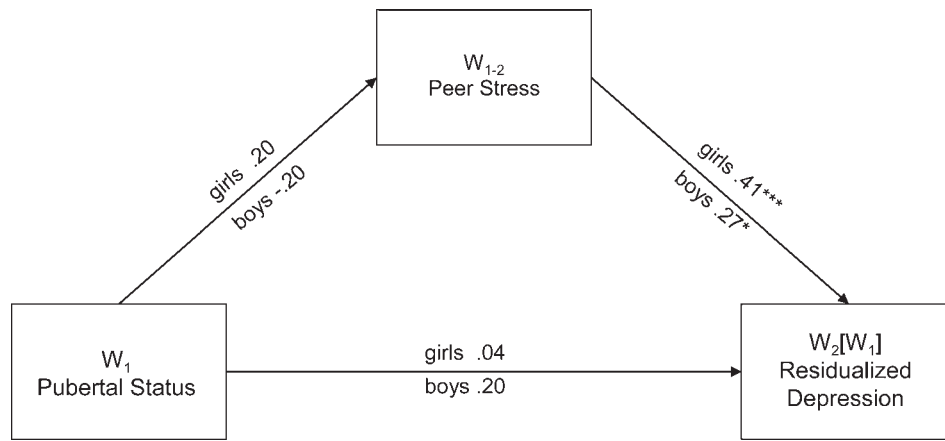
Within girls, pubertal status had a stronger effect on depression when stress was not included (total effect  $\beta = 0.12$ ,  $p = .27$ ) than when it was included (direct effect  $\beta = 0.04$ ,  $p = .70$ ) in the model; this indirect effect explained 67% of the total effect ( $P_M = 67\%$ ). Within boys, pubertal status had a stronger effect on depression when stress was included (direct effect  $\beta = 0.20$ ,  $p = .094$ ) than when it was not included (total effect  $\beta = 0.15$ ,  $p = .23$ ) in the model, suggesting that peer stress slightly suppressed the effect of pubertal status on depression.<sup>5</sup>

#### Pubertal timing

The model for pubertal timing predicted 21% of the variance in depression in girls (medium effect size) and 18% in boys (medium effect size; see Figure 2). Confirming the a priori hypothesis, a test of sex-moderated mediation indicated that the indirect effect of linear timing on depression via peer stress was significantly different in girls ( $\beta = 0.09$ ,  $p = .071$ ) and boys ( $\beta = -0.02$ ,  $p = .53$ ),  $\chi^2(1, N = 149) = 4.35$ ,  $r = .17$ ,  $p = .038$ . Contrary to predictions, a test of sex-moderated mediation indicated that the indirect effect of curvilinear timing on depression via peer stress was nonsignificant,  $\chi^2(1, N = 149) = 0.52$ ,  $r = .04$ ,  $p = .47$ . Thus, we did

sum of the direct and indirect effects does not equal the total effect because of rounding error.

5. To address the potential confounding effects of age when analyzing the impact of pubertal status on stress and depression, we estimated two additional path models: (a) one in which we added age as a second exogenous predictor along with pubertal status, with links from both age and pubertal status to stress and depression (and from stress to depression); and (b) the other in which we omitted pubertal status from the model and used age as the only exogenous predictor of stress and depression, with a link from stress to depression. Results revealed that the effects originally found for pubertal status were unchanged when controlling for the effects of age for both girls and boys. Moreover, age had no significant direct effects on either stress or depression for girls or boys when dropping pubertal status from the model. These findings strongly suggest that the effects we have identified for pubertal status are not due to age per se.



**Figure 1.** Peer stress as a mediator of the effect of pubertal status on subsequent depression in girls and boys. \* $p < .05$ . \*\*\* $p < .001$ .  $N = 149$  (78 girls, 71 boys).

not test for mediation in the path from curvilinear timing to peer stress to depression.

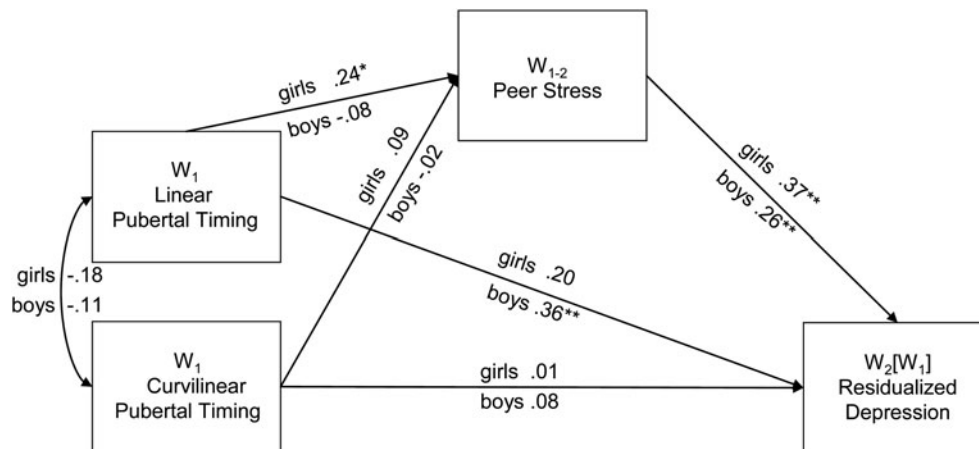
Multigroup path analysis using equality constraints revealed that the path coefficient linking linear timing to peer stress marginally differed in girls and boys,  $\chi^2(1, N = 149) = 3.57, r = .15, p = .059$ . Notably, earlier pubertal timing predicted more peer stress in girls ( $\beta = 0.24, p = .037$ ) but the association was nonsignificant, and in the opposite direction, in boys ( $\beta = -0.08, p = .52$ ). There was no significant sex difference in the strength of the path coefficient linking peer stress to depression,  $\chi^2(1, N = 149) = 1.21, r = .12, p = .14$ .

Within girls, linear pubertal timing had a stronger effect on depression when stress was not included (total effect  $\beta = 0.29, p = .011$ ) than when it was included (direct effect  $\beta = 0.20, p = .067$ ) in the model; this indirect effect explained 31% of the total effect ( $P_M = 31\%$ ). Within boys, pubertal timing had a stronger effect on depression when stress was included (direct effect  $\beta = 0.36, p = .001$ ) than when it was not included (total effect  $\beta = 0.34, p = .004$ ) in the model, sug-

gesting that peer stress slightly suppressed the effect of pubertal timing on depression.

### Discussion

Following the principles of developmental psychopathology and recommendations of developmental scientists (Cicchetti, Rogosch, & Toth, 1994; Graber, 2003; Hayward & Sanborn, 2002; Susman & Rogol, 2004), this research took a contextualized approach to understanding the developmental processes linking sex and puberty to depression. Consistent with predictions, structural equation modeling analyses confirmed that (a) sex moderated the pathway from puberty to peer stress to depression; (b) the associations between puberty (status and linear timing) and depression were in opposite directions for girls and boys; (c) peer stress predicted depression, above and beyond the contribution of puberty, in both girls and boys; although they did not significantly differ, these paths were consistently stronger in girls than in boys; (d) consequently, the indirect



**Figure 2.** Peer stress as a mediator of the linear and curvilinear effects of pubertal timing on subsequent depression in girls and boys. \* $p < .05$ . \*\* $p < .01$ .  $N = 149$  (78 girls, 71 boys).



effects of puberty and depression were in opposite directions in girls and boys; and (e) in girls, peer stress accounted for 67% of the effect of pubertal status on depression, and 31% of the effect of pubertal timing on depression; in contrast, the mediated effect proportions were not calculated in boys due to suppression effects. In sum, this research elucidated sex differences in one pathway through which puberty influences depression, via heightened stress in the peer domain.

#### *Peer stress as a mediator between puberty and depression*

The present research provides evidence that peer stress partially accounts for the association between puberty and depression in girls but not in boys. In models of pubertal status and timing, the longitudinal association between puberty and depression was more strongly mediated by peer stress in girls than in boys. Specifically, peer stress accounted for a moderate to large amount (i.e., 31% to 67%) of the association between puberty and depression in girls but not in boys. Furthermore, puberty and peer stress together explained a moderate proportion of the variance in depression in girls, and a smaller proportion of the variance in boys. These findings provide evidence that, at least in girls, puberty partially contributes to depression in adolescents because it triggers stress in peer relationships, which in turn heightens depression. The pattern of stronger findings in girls is consistent with research highlighting girls' tendencies toward interpersonal sensitivity, social-evaluative concerns, and social approval-based self-evaluations, which often contribute to psychological distress (Crocker & Wolfe, 2001; Cross & Madson, 1997; La Greca & Lopez, 1998; Rudolph, Caldwell, & Conley, 2005; Rudolph & Conley, 2005).

The observed pathways in the present study suggest the developmental unfolding of transactions between adolescents and their social contexts (Cicchetti & Toth, 1994; Lerner, 1985; Sameroff, 1987). Specifically, adolescents' personal and physical characteristics (including sex, pubertal status, and pubertal timing) hold particular values and meanings, and evoke certain social responses. Depending on particular personal characteristics (e.g., being an earlier-developing girl), these social consequences can take the form of social exclusion, teasing, lowered social status, restricted friendships, or other forms of peer stress. These stressful peer experiences, in turn, feed back into the developing adolescent's adjustment, reflected in the depressive reactions that often accompany stress in the peer domain (Rudolph et al., 2000). Furthermore, by virtue of their pubertal development and timing, adolescents also seek out particular social relationships and environments (Brooks-Gunn et al., 1986; Magnusson, 1988). Thus, adolescents both select and shape their social contexts in ways that contribute to their subsequent developmental trajectories (Lerner, 1987; Scarr & McCartney, 1983; Steinberg, 1995).

#### *Contributions and future directions*

The present study contributes to our growing understanding of the interplay among physical, psychological, and social

processes involved in the sex difference in adolescent depression. This research also offered various methodological strengths in comparison to past research in this area. At the same time, there are areas for improvement in future research. An important contribution of the present study is its longitudinal design, which is particularly significant in testing mediational models and for tracking development over time. The study's sample included a broad age range. However, many of the youth were still undergoing pubertal changes at Wave 2. This is particularly true for boys, who develop later than girls (Tanner, 1969). Future research with even broader age ranges can establish whether the current pattern of findings continues to hold later in adolescence.

Further, this study used a multiple-informant assessment approach and interview methods that are less subject to informant bias (Rudolph & Hammen, 1999; Rudolph et al., 2000). First, as recommended by Hayward (2003), we assessed multiple aspects of pubertal development. Two separate measures of pubertal development, from two informants, were converted into a composite index that has demonstrated reliability with both clinician-rated assessments and the underlying hormonal processes of puberty (Shirtcliff et al., 2009). However, future research is needed to determine whether the same pattern of findings would emerge for other assessments of puberty, such as hormone levels. Second, the in-depth semistructured life stress interview assessed both chronic and episodic peer stress, both of which are important contributors to psychological adjustment (Compas, 1987). Third, depression was assessed with a semistructured diagnostic interview, which provides optimal discrimination among different types of psychopathology. Despite recent emphasis on continuous assessments of psychopathology (Brown & Barlow, 2005; Hankin et al., 2005), and evidence that stress–depression links are similar for clinical and subclinical levels of depression (Shih et al., 2006), additional research with larger samples should confirm whether the present findings can be replicated when predicting categorical diagnoses of depression.

Finally, although this study confirmed significant contributions of puberty and peer stress to adolescent depression, many of the effect sizes were medium, or even small. Given the complex and dynamic nature of adolescent development, there are likely several other contributors to the emergence of sex differences in depression. For example, theory and research underscore the value of integrative models that consider physical and biological, cognitive, affective, and contextual processes (Cyranski, Frank, Young, & Shear, 2000; DeRose, Wright, & Brooks-Gunn, 2006; Graber et al., 2005; Hilt & Nolen-Hoeksema, 2009; Hyde, Mezulis, & Abramson, 2008; Rudolph, 2009). The present research focused specifically on physical and social contributions within the peer domain. Future research is still needed to integrate various theoretical perspectives and research findings into a comprehen-

sive developmental model of the sex difference in depression.

### Summary and implications

These findings contribute to a growing body of research that demonstrates the role of social context in the sex-differentiated pathway between puberty and depression. Specifically, puberty and peer stress are two aspects of adolescent development that have a powerful influence on the development of depression, particularly in girls. More broadly, these findings suggest that there are longitudinal, transactional associations between developing adolescents and their social contexts, ultimately contributing to the rising rates of depression, and the sex difference therein, during adolescence.

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