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Coping with Social Stress: Implications for Psychopathology in Young Adolescent Girls

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Abstract

This study investigated the impact of social stress on symptoms of psychopathology at the entry into adolescence (111 girls, Mage=11.84, SD=0.77). We examined whether peer stress and pubertal timing were associated with internalizing distress and aggression, and whether responses to stress and cortisol reactivity mediated or moderated these associations. Cortisol samples were collected from saliva samples during in-home visits, and the YSR was used to assess psychopathology. Interestingly, pubertal timing demonstrated a trend association with cortisol. Responses to stress mediated the association between social stress and symptoms of internalizing distress and aggression. Specifically, early maturers and girls with higher levels of peer stress exhibited more problematic responses to stress, in turn demonstrating higher levels of internalizing distress and aggression. Significant moderation effects also emerged. For example, early maturers who experienced higher levels of emotional/cognitive numbing in response to peer stress were at greater risk for aggression. Findings identify coping strategies that may be used in evidence-based programming to help girls transition more successfully into adolescence will be discussed.

Keywords

Peer stress; Coping; Pubertal timing; Internalizing; Aggression

Adolescence represents a period for developing skills—interpersonal as well as personal. However, as consistent with the diathesis-stress model, some individuals enter adolescence with prior vulnerabilities (poorer problem solving skills, less developed emotion regulation skills, difficulty with emotional expression, greater physiological reactivity) making it more challenging to adapt effectively to the quickly changing landscape of early adolescence (Igram and Luxton 2005). As time spent with peers increases during the transition into adolescence, so does the likelihood of experiencing negative peer interactions, such as victimization by peers (via both overt and relational aggression), interpersonal problems

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with a close friend, and being teased or hassled by one's peers. Based on prior vulnerabilities and prior experiences, adolescents often respond to such negative peer events with a range of reactions—anger, sadness, aggression, emotional suppression, etc. In turn, how an adolescent adapts to these experiences is often associated with the expression of emotional and behavioral problems (Compas et al. 2001).

Much of the research examining how children and adolescents manage and adapt to stressful experiences demonstrates that coping strategies or responses to stress, more broadly, may buffer the effects of stressful experiences, ultimately reducing the likelihood of subsequent psychosocial problems (Jaser et al. 2005; Wadsworth et al. 2005). However, the stress and coping literature also suggests that the stressful situation itself may influence the range and type of responses to stress, in turn impacting adjustment (Compas et al. 1999). Thus, it is important to recognize that there may be multiple pathways by which coping impacts adjustment.

Under the rubric of the diathesis-stress model, the present investigation examined the impact of the stress and coping process during early adolescence. Specifically, this study integrated multiple dimensions of stress (peer stress and early pubertal maturation) and multiple responses to stress (voluntary coping strategies and involuntary responses) in order to identify factors that place some young adolescent girls at greater risk for symptoms of psychopathology in the face of stressful life experiences. Our goal was to identify mechanisms through which social stress impacts psychopathology during the entry into adolescence. Specifically, we examined potential mediating and moderating effects of different responses to stress on the association between stress and symptoms of psychopathology (see Fig. 1).

Social Stress

It has been widely recognized that increased levels of stress are associated with a host of emotional and behavioral problems for children and adolescents (Grant and Compas 2003). Because of the increasing amount of time spent with peers and the importance of close interpersonal relationships during early adolescence, social stress becomes particularly salient as a threat to psychological well-being at this time (La Greca 2001). Moreover, levels of social stress, specifically in peer relationships, tend to increase from preadolescence to adolescence for both boys and girls; however, gender differences in levels of social stress typically begin to emerge during early adolescence with girls reporting higher levels of stress, suggesting that adolescence may represent a period of particular vulnerability for girls (Brooks-Gunn 1991; Ge et al. 2001).

Peer stress

Within the broader range of social stress, peer stress may be conceptualized as negative experiences with peers or friends that evoke emotional, physical, and psychological arousal and exert significant pressure on an individual's ability to function adaptively. Increased exposure to negative peer events, such as peer rejection and victimization, during childhood and adolescence has been linked to disorders and symptoms of psychopathology, namely anxiety, depression, behavior problems, and substance use (Coie et al. 1990; Deater-Deckard 2001; Nansel et al. 2004). Because of girls' tendencies to rely heavily on peers for emotional support and intimacy, the disruption in social networks and the shift of peer groups typical of the transition into adolescence are likely to create higher levels of negative peer events for girls compared to boys (Rudolph 2002).

Pubertal Timing

Another aspect of social stress that is particularly salient to adolescence is the experience of pubertal changes and whether those changes reflect normative or off-time developmental experiences within one's peer group; that is, whether one's pubertal maturation occurs earlier or later in comparison to same-age peers.

Although the physical changes associated with puberty may be perceived as a negative experience for some, being different from one's peers during early adolescence, whether it be physically or experientially, may be a stressor in and of itself. For example, early maturing girls may be fearful of eliciting negative responses from others for gaining weight or looking more adult-like. They may also develop concerns about not meeting certain social and sexual expectations from peers and family members. Regardless of whether or not these fears occur, the psychological, emotional, and cognitive arousal that the experience of early maturation elicits could be stressful and could negatively impact girls' psychosocial well-being. Supporting this idea, numerous recent studies have found that early maturation in girls has been linked to symptoms of depression, anxiety, and aggression (e.g., Caspi et al. 1993; Ge et al. 2002, 2001; Graber et al. 2004).

Overall, peer stress and early pubertal maturation are dimensions of social stress which pose particular challenges for girls. Hence, the present study focuses on girls' responses to stress in order to identify mechanisms that link social stressors to adjustment outcomes. Notably, few studies have examined the actual mechanisms that link peer stress or early pubertal timing and the development of psychopathology. Revisiting the discussion of social stress from the diathesis-stress perspective, experiencing multiple social stressors (i.e., peer stress and early maturation) simultaneously or cumulatively increases the risk of developing symptoms of psychopathology. For this reason, it is likely that early maturing girls who experience high levels of peer stress would be at greatest risk for developing symptoms of depression, anxiety, or aggression.

Coping and Responses to Stress

As discussed above, higher levels of stress put individuals at greater risk for emotional and behavioral problems. However, the effects of stress vary from person to person. It has been well established in the adult literature that how an individual copes with stress is often a strong indicator of psychosocial well-being. However, much less emphasis has been placed on understanding mechanisms of stress and coping in the realm of adolescent interpersonal relationships and how these processes impact psychopathology, suggesting a need for research examining these issues.

Voluntary and Involuntary Responses—Lazarus and Folkman (1984) define coping as efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person. Although this model identifies active responses to stress (i.e., coping), it does not consider the role of involuntary responses (e.g. physiological arousal, cognitive numbing, intrusive thoughts). As mentioned previously, some individuals enter adolescence with prior vulnerabilities that may hinder effective adaptation to stressful experiences. These vulnerabilities may include temperamentally based and conditioned cognitive, behavioral, and physiological reactions to stress that may or may not be within conscious awareness and are not under personal control, such as physiological arousal, intrusive thoughts, and rumination.

As a way of capturing both coping responses and prior vulnerabilities to stress, Compas and colleagues (2001) have proposed that examinations of coping and responses to stress include both *voluntary*/*active* (i.e., coping) and *involuntary* responses to stressful stimuli. In this

approach to voluntary or involuntary responses to stress, they also examine responses as engaging with or disengaging from the stressful experience. Responses to stress that qualify as *engagement* are directed at the stressor or stressful experience, and are often intended to influence or change the condition of the stressor or the emotions and cognitions about the stressor (e.g., problem solving, emotional expression, rumination, etc.). In contrast, *disengagement* refers to responses that are oriented away from the stressor or one's reaction to the stressful experience (e.g., denial, wishful thinking, cognitive numbing, etc.). Note that engagement and disengagement responses may be exhibited as both active coping strategies or involuntary responses to stress. Many researchers further differentiate voluntary engagement responses into two distinct subtypes of coping strategies: *primary control* and *secondary control* strategies (Compas et al. 1999; Rudolph et al. 1995). Primary control responses are aimed at maintaining or enhancing a sense of personal control over the stressor and/or one's emotions (Compas et al. 1999); example strategies include problem solving, emotional expression, and emotion regulation. In contrast, secondary control responses are intended to gain control indirectly by accommodating or adapting to the stressful event or context and include strategies such as acceptance, positive thinking, and cognitive restructuring (Compas et al. 1999).

The measurement of biochemical responses to stress offers an important complementary measure to the emotional, behavioral, and cognitive components of stress response. When stress or novelty is experienced, the Limbic-Hypothalamus-Pituitary-Adrenal (L-HPA) axis typically responds with an increase in cortisol secretion (Cicchetti and Walker 2001). Following the onset of a stressor, it usually takes about 10–15 min to produce a rise in circulating cortisol levels; about 20–30 min after the stressor, peak concentrations of cortisol are found in saliva (Klimes-Dougan et al. 2001). Following this peak, the negative feedback loop of the LHPA axis begins to reestablish homeostatic functioning by reducing the level of circulating cortisol (Gunnar and Vazquez 2001).

Mechanisms of Stress and Coping—The adult coping literature has suggested that coping strategies or general responses to stress take on a more trait-like quality and begin to reflect particular response styles as individuals transition into late adolescence and early adulthood, meaning that an adult is likely to respond to stressors at work in the same way he would to economic or interpersonal stressors (Compas et al. 1999). In contrast, the child and adolescent coping literature has argued that during childhood and early adolescence, the range of responses utilized are strongly influenced by the stressor itself, suggesting that coping strategies or responses to stress may serve as mediating factors between stress and psychopathology (Grant and Compas 2003). It has been suggested that in some cases adolescents may possess some of the mediating characteristics (e.g. coping strategies) prior to exposure to the stressor, but that the characteristic increases or decreases substantially in response to the stressor.

Researchers examining the impact of stress on coping strategies have found that continued exposure to either chronic stress or daily hassles taxes children's and adolescents' coping resources, reducing the ability to respond effectively to stressful situations, resulting in increases in symptoms of psychopathology (Graber and Sontag 2008). Wadsworth and colleagues (2005) examined mediating and moderating effects of coping on the association between poverty and psychopathology and found that coping strategies served as a mediator for children in the study. Interestingly, this pathway did not hold for the adults in the study; rather, coping strategies moderated the association between stress and psychopathology. Although these findings and other studies support the assertion that coping strategies during childhood and adolescence may be context-dependent and susceptible to influences directly from the stressor (Compas et al. 2001; Wadsworth et al. 2005), none of these studies have

focused exclusively on social stressors that may be most salient to adolescents (i.e., peer stress and pubertal timing).

Although research supports the notion that stress may impact the availability of coping resources and an adolescent's type of response, in turn increasing the risk for psychopathology (i.e., mediation as described above), children and adolescents do have prior tendencies toward certain stress responses (e.g. physiological arousal, anger, denial). When occurring in conjunction with high levels of stress, these prior response tendencies are likely to increase the risk for psychopathology. For example, a child who tends to experience increases in heart rate and feelings of anger when being teased by peers will likely be at risk for higher rates of aggression if faced with continual bullying at school. Or, a girl who tends to ruminate and has experienced puberty much earlier than her peers may be at risk for increased symptoms of depression. Scenarios such as these suggest that how an adolescent responds to stress may serve as a moderator of the association between peer stress and psychopathology, either attenuating or accentuating the outcome depending on the response exhibited.

Responses to stress are not only behavioral and psychological but also include physiological arousal linked to the activation of L-HPA axis. Some individuals exhibit dysregulations in cortisol production in response to stressors which has been linked to internalizing and externalizing problems among young adolescents (Klimes-Dougan et al. 2001). Recent research examining responses to social challenges (i.e., public speaking tasks, parent–child conflict discussion tasks, or peer-rejection simulations) has shown that adolescents and children who maintained high levels of cortisol or displayed strong task-related decreases demonstrated greater levels of internalizing and externalizing behavior (Granger et al. 1994; Klimes-Dougan et al. 2001; Schmidt et al. 1999). Although these studies have focused exclusively on cortisol reactivity in response to a specific task, other studies have found that chronic activation of the L-HPA system from continued exposure to stress may alter an individual's reactivity profile, increasing the likelihood of experiencing hypo- or hyper-secretion in response to future stressors (Gunnar and Vazquez 2001; Kendall-Tackett 2000). Exposure to stress again (e.g. peer stress) would then reinforce this reactivity profile. For example, some evidence suggests that early maturing girls exhibit different reactivity profiles compared to other girls, with early maturing girls more likely exhibit higher total cortisol responses and slower peak recovery (Graber et al. 2006; Stroud et al. 2004). This reactivity pattern then, in turn, may interact with stress exposure, increasing the likelihood of expressing symptoms of psychopathology.

Given that children or young adolescents may have difficulty self-reporting the experience of physiological arousal, the utilization of cortisol assays holds important clinical implications. Being able to recognize individuals with impairments in the negative feedback loop of the L-HPA axis may allow clinicians to better target children and adolescents at-risk for the development of internalizing and externalizing problems. However, it is important to note that within the child and adolescent literature, links between cortisol and symptoms of psychopathology have not been consistently found across all studies, especially in community rather than clinical samples (Klimes-Dougan et al., 2001). Therefore, additional research examining the association between cortisol reactivity and symptoms of psychopathology needed, especially within the adolescent population.

Goals and Hypotheses

The present investigation examined the impact of peer stress and pubertal timing on internalizing and aggressive symptoms in a community sample of young adolescent girls. Additionally, this study tested whether responses to stress, delineated by Compas and

colleagues (2001), and cortisol reactivity mediated and moderated these associations. Specifically, the following research questions were addressed:

Is social stress (peer stress and pubertal timing) associated with symptoms of psychopathology? Does the compound experience of early maturation and higher levels of peer stress increase the risk for psychopathology? It was hypothesized that early maturation and greater levels of peer stress would be associated with greater levels of internalizing distress and aggression. Additionally, it was expected that early maturers who experienced higher levels of peer stress would demonstrate greater levels of internalizing distress and aggression compared to other girls in the study.

Do responses to stress and cortisol reactivity mediate the association between social stress (peer stress and pubertal timing) and symptoms of psychopathology (Fig. 1a)? It was hypothesized that girls with high levels of peer stress early pubertal timing would demonstrate fewer voluntary engagement strategies (e.g., problem solving, emotion regulation), and in turn demonstrate higher levels psychopathology. It was also hypothesized that girls with high levels of peer stress or early pubertal timing would demonstrate more voluntary disengagement strategies and experience more involuntary responses (both engagement and disengagement types), in turn demonstrating higher levels of psychopathology. Finally, it was expected that girls with high levels of peer stress or early pubertal timing would exhibit high cortisol reactivity, in turn demonstrating higher levels of psychopathology.

Do responses to stress and cortisol reactivity moderate the association between social stress (peer stress and pubertal timing) and symptoms of psychopathology (Fig. 1b)? It was hypothesized that voluntary engagement strategies would buffer the negative effects of peer stress and early maturation, whereas, voluntary disengagement strategies, involuntary responses, and high cortisol responses would serve as risk factors for the development of internalizing distress and aggression.

Method

Participants and Procedure

Participants were 111 adolescent girls (M_{age}=11.84, *SD*= 0.77) drawn from a larger project that examined biological correlates of social and emotional health as girls made the transition to puberty. Girls and their mothers were originally recruited in 1995–1996 from public schools in ethnically integrated, working- and middle-class communities in the New York City area. A series of flyers describing the study were distributed to the girls in school and mothers were asked to give consent. Because flyers were passed out in multiple waves to the girls, a precise count of how many mothers received consent forms at home could not be established. However, of those who returned contact information, 85% enrolled in the study.

In this study, approximately 15% of the girls were in fifth grade, 47% in sixth grade, 31% in seventh grade, and 2% in eighth grade. Differences in time in school year when girls were enrolled in the original study in third grade and differences in tracking time for subsequent assessments resulted in greater variability in age and grade level in the present assessment. Of the 111 girls, 48% were Caucasian, 36% African American, and 16% Hispanic. Participants completed a battery of neutral and challenging tasks1 and a measure of their own affect and behavior during a home visit. Participants then completed an additional survey during the three consecutive days following the home visit that assessed peer stress

[1]A complete list of tasks is available from the author.

and coping. Girls received a gift (tote bag) for their participation and mothers were paid $75. Procedures were approved by the Institutional Review Board of Teachers College at Columbia University. Although girls participated in a prior assessment, coping and negative peer stress measures were only administered at this assessment, limiting the study to cross-sectional analyses.2

Measures

Demographic data—Girls reported their date of birth, and exact age was calculated from date of home visit. Participants' mothers reported on family characteristics and child ethnicity in an interview during the home visit. Child ethnicity was collapsed into three categories: White, African American, and Hispanic. For regression analyses, two dummy coded variables were used: African American versus other and Hispanic versus other, with White as the omitted group. Mothers also reported on the education and occupation of themselves and any other parent/caregiver in the household. Family socioeconomic status (SES) was scored using the Hollingshead Scale (Hollingshead, A. B. (1975). Four factor index of social status, Yale University, unpublished manuscript). The standard scoring protocols were used for different household types. The range of possible scores for family SES was 8 to 66. This sample had an average SES score of 38.30 (SD=13.81), which equated to a high school degree and employment as clerical workers, sales workers, or owners of small businesses.

Pubertal timing—Pubertal timing was based on self-reported age of menarche obtained during the home visit. Girls were classified as either early maturers or other (on-time or late). Because participation rates from each classroom and school were too low to provide informative comparisons to classmates, cut-off ages for pubertal timing for White and African American participants were derived from a large, normative study of puberty (Herman-Giddens et al. 1997). Girls that reported age of menarche one half SD below the mean for either White or African American age norms were categorized as early. All other girls were categorized as other (on-time or late). Because the Herman-Giddens study only examined White and African American girls, estimates for cut-offs for early menarche among Latinas were calculated as one half SD below the mean menarcheal age of the sample. Cut off ages for early maturation were: 12.28 years for White, 11.55 years for African American, and 11.97 years for Latina girls. According to an investigation of age at menarche for girls in the United States, the cut-off ages by ethnicity for early maturation were consistent with national trends, such that African American girls tend to experience menarche first, followed by Hispanic, then non-Hispanic White girls (Chumlea et al. 2003). Reports of breast and pubic hair development as reported by mother using the five-stage Tanner rating system (Marshall and Tanner 1969) were used to classify girls who had not yet experienced menarche. Again cut-off ages for White and African American participants were derived from the Herman-Giddens et al. (1997) study, such that girls who fell one half SD below the mean for a particular stage were classified as early; all others were classified as other. Thirty-four percent (*N*=38) of all participants were classified as early maturers and 66% (*N*=73) were either on-time or late. Approximately 34% of White, 33% of African American, and 39% of Latina participants were early maturers.

Peer stress—Girls completed the stressful events checklist in the Responses to Stress Questionnaire (RSQ; Connor-Smith et al. 2000). The negative peer events checklist asked

[2]Comparing the sample at the original assessment to the assessment used in this study, there was a loss of 19.5% of the sample of girls. The authors conducted univariate analyses of non-response bias to determine if the remaining sample differed on key demographic factors, pubertal development variables, and outcomes (depression, peer problems, internalizing and externalizing behavior) assessed at baseline. Results showed that participants did not differ on variables used in this study.

girls to indicate if they had experienced each of nine stressful events since the start of the school year. Example events included being teased or hassled by other kids, having someone stop being your friend, and being left out or rejected. In addition to the checklist, girls responded to one follow-up appraisal question that assessed how stressful the problems endorsed in the checklist were for them (1 = not at all, to 4 = very). Each item was summed to create a total checklist score. In order to calculate overall peer stress, the total checklist score was weighted by the appraisal score (checklist total×appraisal score) ranging from 0 to 36.

Coping and responses to stress—In order to assess both voluntary and involuntary response to stress, the RSQ (Connor-Smith et al. 2000) was used. The RSQ is designed to focus on specific stressors, in this case, peer stress. Girls rated items on a likert scale (1 = not at all, to 4 = a lot) that asked how they would respond to problems with peers indicated in the checklist. The RSQ contains five subscales, as named by Connor-Smith and colleagues (2000), which tap into both voluntary coping strategies (primary control, secondary control, and disengagement strategies) and involuntary responses (involuntary engagement and involuntary disengagement). For the present study, we used a slightly shorter form of the RSQ. The present form contained the same scales but fewer items per scale. As can be seen below, Chronbach's alphas were all ≥0.70 indicating that use of fewer items did not adversely impact the reliability of the scales. In the present study, the primary control scale contained seven items (e.g., "I try to think of different ways to change the problem or fix the situation") that assessed voluntary engagement responses to stress, and included items that assessed emotional expression, emotional regulation, and problem solving (α=0.80). The secondary control scale, which also assessed voluntary engagement strategies, contained 4 items (e.g. "I tell myself that everything will be all right") that measured responses to stress categorized as acceptance, positive thinking, and cognitive restructuring (α=0.70). The disengagement scale contained nine items (e.g., "I try to stay away from people and things that make me feel upset or remind me of the problem") that assessed denial, avoidance, and wishful thinking (α= 0.75). The involuntary engagement scale contained nine items (e.g., "I feel it in my body…my heart racing, my breathing speeding up, feeling sweaty or hot, or my muscles getting tight") that assessed impulsive action, physiological arousal, and intrusive thoughts or rumination (α=0.78). Finally, the involuntary disengagement scale contained nine items (e.g., "I don't feel anything at all, it's like I have no feelings") that measured inaction, emotional and cognitive numbing, and involuntary avoidance or fleeing (α=0.76).

Raw scores were reported as sum scores and used for reliability analyses. However, in order to control for overall responding bias, ipsative scores were used in analyzing the associations with internalizing distress and aggression as recommended by Connor-Smith and colleagues (2000). Connor-Smith and colleagues found, in general, that adolescents who experience more stress and demonstrate internalizing and externalizing problems have a tendency to endorse more coping strategies and responses to stress compared to other adolescents. Thus, there is a need to control for overall response rate when examining the effects of responses to stress. Ipsative scores make it possible to compare an individual's relative score on each subscale with his or her score on other subscales in the same measure. Because the score of each subscale is dependent on the score of the other subscales, problems arise when conducting factor analyses or structural equation modeling with multiple scales in the same model (Chan 2003). However, when used independently, ipsative scores allow researchers to compare general profiles of participants, which may ultimately be more informative than general associations (Cunningham et al. 1977). Higher scores on subscale represent a more dominant strategy in participant's response repertoire. That is, a high score on primary control means that a participant endorsed primary control strategies as a response to stress more often than the other responses.

Connor-Smith et al. (2000) tested the RSQ using a sample of adolescents and their parents. Findings indicated that not only was there adequate to excellent internal consistency for each subscale of stress response but subscales of voluntary coping strategies, specifically, mapped onto other standard measures of coping quite clearly. Although findings did not suggest that each subscale was completely orthogonal, the amount of shared variance, which ranged from 0% to 45%, suggested that each subscale should be examined independently (Connor-Smith et al. 2000).

Cortisol reactivity—During the in-home visit, five saliva samples were collected to assess cortisol levels throughout the battery of tasks mentioned previously. The challenging tasks (brief cognitive task, cold pressor task, and two interaction tasks with the mother) were designed to be stressful to adolescents and potentially elicit temporary physiological arousal. Approximately 15–20 min lapsed between collections, with the last three samples collected after the three stressful tasks. Saliva was collected via Salivettes. Girls kept a cotton roll in their mouths for 2 min, resulting in sufficient amounts of saliva for repeat assays. Girls ingested a small amount of sweetened Kool-Aid to stimulate saliva flow. Stimulating saliva flow with orally administered substances such as powdered drink mix was recommended protocol at the time this protocol was implemented (Schwartz et al. 1998). Subsequent to the development of this procedure, it was found that Kool-Aid may alter the results of the assay of cortisol from saliva samples (Schwartz et al. 1998). However, these effects were found to vary by assay kit used. The kit used in the present study was not highly susceptible to this problem. Saliva samples were stored in a refrigerator before being transported to Columbia-Presbyterian Reproductive Endocrinology Department in New York City, where they were stored in a freezer at −20°C until assayed for cortisol. Cortisol concentrations were determined using a radioimmunoassay adapted for measurements in saliva (Diagnostic Products Company). The lower detection limit for this assay was 0.02 μg/dl per 200 μl of saliva. Saliva samples were centrifuged at 3,000 rpm for 10 min. A 200 μl sample was used for duplicate analysis. All samples from a participant were analyzed in one assay run, with inter- and intra-assay coefficients of variations less than 5% and 3%, respectively. Missing data for cortisol occurred if samples had insufficient volumes of saliva to process.

In order to assess overall reactivity, area under the curve ($AUC_{cortisol}$) was calculated from zero across all five cortisol samples. Area under the curve measures the total area under the regression line that is created from the five samples of cortisol and thus provides an overall count of cortisol reactivity during the home visit.[3] Dysregulation of the LHPA axis would typically result in higher overall reactivity (higher values of $AUC_{cortisol}$). Log transformations were applied to the $AUC_{cortisol}$ to reduce the large positive skew, which is consistent with standard procedures in cortisol research (Gunnar 2000); however, original units were reported in Table 1 for ease of interpretation.

Symptoms of psychopathology—Internalizing distress and aggression were assessed during the home visit using the anxiety/depression and aggression subscales of the YSR (Achenbach 1991). Participants were asked to report on behavior now or within the past 6 months and rated items on a likert scale (0 = not true, to 2 = very true or often true). Scores for the anxiety/depression and aggression subscales were reported as sum scores. The anxiety/depression subscale contained 16 items, with a reliability of α=0.83. The aggression subscale contained 19 items, with a reliability of α=0.86. In addition to good reliability

[3]In addition to $AUC_{cortisol}$, the authors conducted preliminary multilevel analyses to examine whether or not patterns of change over time in cortisol were associated with symptoms of psychopathology. Results indicated that *pattern of change* was not associated with internalizing distress or aggression, suggesting that the multi-level analyses added no additional information to the study beyond what $AUC_{cortisol}$ demonstrated. A complete description of the multi-level analyses is available upon request.

demonstrated in this study, excellent validity and reliability has been established for the YSR in studies of clinical and community samples (Achenbach 1991).

Analytic Plan

To address the first set of hypotheses, the associations between social stress and symptoms of psychopathology were explored via analysis of covariances (ANCOVAs; for pubertal timing) and hierarchical linear regressions (for peer stress). Additionally, the interaction between pubertal timing and peer stress were examined using hierarchical regression, with pubertal timing dummy coded as early (1) versus on-time and late (0). To address the second set of hypotheses, mediation was tested using the guidelines delineated by Baron and Kenny (1986). Specifically, separate regression models tested the effects of the social stress (peer stress and pubertal timing) on each mediator (the five RSQ subscales and cortisol reactivity) and both internalizing distress and aggression. A full model was then tested with the social stress predictor and the mediator entered on the same step. For mediation to occur, the predictor must have a significant association with the mediator and the outcome variable; *and*, in the full model, the mediator must predict the outcome variable and the size and significance of the association between the predictor and the outcome variable must be reduced. Subsequent tests of the indirect effect were conducted. Although benefits arise from examining mediation via structural equation modeling (i.e., the ability to examine multiple mediators and predictors simultaneously), the use of ipsative scores and sample size limitations restrict analyses to the aforementioned approach. Finally, in order to address the third set of hypotheses, moderation was tested, examining potential interactions between social stress (pubertal timing or peer stress) and responses to stress (the five RSQ subscales and cortisol reactivity). Participants were excluded from analyses on a list-wise basis if they had missing data for a particular variable. All analyses controlled for age.

Results

Descriptive Analyses

Descriptive information (i.e., M and SD) for all variables is shown in Table 1 and correlations among the variables of interest are shown in Table 2. When examining the bivariate correlations among the variables of interest, several general themes emerged. Although the majority of participants were about the same age during assessment, results demonstrated a mild association between age and coping strategies and involuntary responses to stress, such that older girls were more likely to use fewer adaptive strategies and exhibit more involuntary responses (Table 2). Thus, subsequent analyses controlled for age. Results also demonstrated that most of the responses to stress were mildly to moderately correlated, ranging in absolute value from 0.19 to 0.64, suggesting that these responses tap similar but not completely overlapping constructs.

Finally, the association between pubertal timing and stressful peer events was examined. Preliminary analyses demonstrated no difference in the number of peer events experienced when comparing early maturers to the other girls in the study (F (1,95)=0.33, p=0.57). Interestingly, when examining each item of the stress checklist individually, early maturers differed from other participants on two items. Early maturers were more likely to endorse “having someone stop being your friend” (F (1,96)=3.62, p=0.06) and “feeling pressured to do something” (F (1,96)=35.17, p=0.03). Because timing was not associated with the full peer stress scale (negative events total × appraisal score), effects of peer stress and pubertal timing on symptoms of psychopathology were examined separately.

Preliminary analyses indicated that ethnicity and family SES were not significantly associated with internalizing distress or aggression. In turn, they were not included as

covariates in the subsequent analyses. Including covariates that do not exhibit associations with the dependent variable can generate spurious significant associations in models, and thus we excluded covariates from these analyses that would otherwise be included in typical regression and ANCOVA analyses (Rovine et al. 1988; Weisberg 1979).

Tests of Main Effects

As indicated, in order to test whether peer stress and pubertal timing were associated with symptoms of internalizing distress and aggression, a series of hierarchical regression analyses and ANCOVAs were performed, respectively.

Peer stress—After controlling for age, peer stress was associated with four of the five responses to stress: primary control (β=−0.23, $p<0.05$; $\Delta R^2=0.21$), secondary control (β=−0.24, $p<0.05$; $\Delta R^2=0.10$), disengagement (β=0.18, $p<0.10$; $\Delta R^2=0.05$), and involuntary engagement (β=0.17, $p<0.10$; $\Delta R^2=0.05$). Peer stress was not significantly associated with cortisol reactivity (β=−0.01, $p=0.92$).

In addition, higher levels of peer stress were associated with greater symptoms of internalizing distress (F (2,93)= 7.03, $p<0.001$; Total $R^2=0.13$; $\Delta R^2=0.12$; β=0.34, $p<0.001$) after controlling for age. However, contrary to expectation, peer stress was not significantly associated with aggression (F (2,93)=2.09, $p=0.13$).

Pubertal timing—Analyses indicated that pubertal timing was significantly associated with two of the five subscales from the RSQ and cortisol reactivity. After controlling for age, early maturers demonstrated a lower ratio of primary control strategies (problem solving, emotion regulation) to other responses (F (1,98)=5.01, $p<0.05$, partial $\eta^2=0.05$; M=0.22, SD=0.05) when compared to other girls (M=0.24, SD=0.05). In addition, early maturers demonstrated a higher ratio of involuntary engagement responses (F (1,97)=4.07, $p<0.05$, partial $\eta^2=0.04$; M=0.21, SD=0.03) when compared to other girls (M=0.19, SD=0.04). These findings suggest that early maturers used problem solving and emotional regulation strategies (primary control) less often when dealing with peer stress and experienced involuntary responses such as rumination and physiological arousal (involuntary engagement) more often compared to on-time or late maturers. Controlling for age, results indicated a trend association between pubertal timing and $AUC_{cortisol}$ (F (1,69)=2.87, $p<0.10$, partial $\eta^2=0.04$), with early maturers exhibiting higher overall reactivity (M_{AUC}= 12.24, SD=12.13) compared to on-time and late maturers (M_{AUC}=8.21, SD=8.66). Finally, a series of ANCOVAs were performed to test whether or not pubertal timing was associated with symptoms of internalizing distress and aggression. After controlling for age, timing was not significantly associated with internalizing distress (F (1, 106)= 2.22, $p=0.14$), however, it was significantly associated with aggression (F (1,106)=4.92, $p<0.05$, partial $\eta^2=0.04$). Early maturers reported more aggressive symptoms, with an average score of 8.81 (SD=6.27) compared to other girls (M=6.17, SD=4.93).

Although it was expected that early maturing girls with higher peer stress would exhibit greater levels of internalizing distress and aggression compared to other girls, results demonstrated a non-significant interaction between peer stress and pubertal timing when examining both internalizing distress (β=0.04; $p=0.70$) and aggression (β =0.08; $p=0.43$), when controlling for age. In addition to these findings, initial analyses indicated that levels of peer stress did not differ significantly by pubertal timing, and peer stress and pubertal timing were not similarly associated with internalizing distress and aggression. Based on these findings it was assumed that peer stress and pubertal timing function as two independent constructs. Thus, the remainder of the analyses will examine these two aspects of social stress independently.

Tests of Mediation

As indicated, mediation was examined via a series of regression models as specified by Baron and Kenny (1986), with subsequent significance tests of mediation. Specifically, Preacher and Hayes (2006) recommend deriving a confidence interval with an empirically derived bootstrapped sampling distribution for the indirect effect (*ab*), which is typically used in such tests as the Sobel test (Sobel 1982). All models controlled for age.

Peer stress—Previous analyses established significant associations with both voluntary engagement coping strategies (primary control and secondary control) and involuntary engagement (e.g., arousal). Thus, these responses to stress were examined as potential mediators. In addition, because analyses demonstrated that peer stress was not associated with aggression, mediation was only examined with internalizing distress as the outcome.

The standardized β coefficients are shown in the figures. As shown in Fig. 2, there was evidence for partial mediation of the effect of peer stress on internalizing distress via both voluntary engagement coping strategies (primary control and secondary control), as well as involuntary engagement (e.g. arousal). The coefficient of peer stress predicting internalizing distress was reduced in magnitude and overall significance when the mediators were included in the models. Preacher and Hayes' (2006) test of the indirect effect supported the finding of partial mediation, demonstrating that *ab* was significantly different from zero with 95% confidence for primary control, *ab*= 0.10, *CI* (0.02 to 0.22), secondary control strategies, *ab*=0.09, *CI* (0.03, 0.20), and involuntary engagement, *ab*=0.06, *CI* (0.01 to 0.15). Results indicated that more peer stress was associated with the tendency to use fewer primary control (problem solving, emotion regulation skills, and emotional expression) and secondary control (acceptance, positive thinking, and cognitive restructuring) strategies in comparison to other coping strategies, which subsequently was associated with greater symptoms of internalizing distress. In addition, greater peer stress was associated with the tendency to experience involuntary engagement (rumination, physiological arousal, intrusive thoughts, etc.) which was subsequently associated with elevated levels of internalizing distress. Contrary to expectation, the indirect effect of peer stress on internalizing distress via involuntary engagement (e.g., intrusive thoughts and physiological arousal) was not significantly different from zero, *ab*=0.03, *CI* (−0.002 to 0.11), indicating no mediation.

Pubertal timing—Figure 3 shows the models testing the mediated pathways of pubertal timing (early vs. other) to symptoms of aggression. Previous analyses established significant associations of pubertal timing with primary control (e.g., problem solving and emotion regulation), and involuntary engagement (e.g., arousal). In addition, because analyses demonstrated that pubertal timing was not associated with internalizing distress, mediation was only examined with aggression as the outcome.

As can be seen in Fig. 3, similar to the findings for peer stress, there was evidence for full mediation of the effect of timing on aggression via primary control (e.g., problem solving) and involuntary engagement (e.g., arousal) responses. The coefficient of pubertal timing predicting aggression was reduced and was no longer significant when the mediators were included in the model. Preacher and Hayes' (2006) test of the indirect effect supported the finding of mediation, demonstrating that *ab* was significantly different from zero with 95% confidence for primary control, *ab*=0.07, *CI* (0.02 to 0.18) and involuntary engagement, *ab*=0.09, *CI* (0.03 to 0.20).

The tests of mediation demonstrated that early pubertal timing was associated with the tendency to use fewer primary control strategies (problem solving, emotion regulation skills, and emotional expression) in comparison to other strategies which subsequently was associated with greater symptoms of aggression. In addition, early maturation was

associated with the tendency to experience involuntary engagement responses (rumination, physiological arousal, intrusive thoughts, etc.) in comparison to other involuntary responses, which was associated with greater symptoms of aggression.

Tests of Moderation

All five of the subscales of the RSQ and cortisol reactivity were examined as potential moderators between the two indicators of social stress (peer stress and pubertal timing) and symptoms of internalizing distress and aggression. Separate multiple regression analyses were run, containing either peer stress or pubertal timing, one type of stress response, and their interaction to test whether responses to stress moderated the association between social stress and symptoms of psychopathology. All interactions were computed using centered variables. In addition, all analyses controlled for age. Only those interactions that emerged as significant are reported below.

Peer stress—As indicated, there was no main effect of peer stress on aggression. However, examination of the interactions suggested a different story (see Fig. 4). Analyses identified significant interactions of peer stress with primary control ($\beta=0.23$, $p<0.05$; $\Delta R^2=0.05$) and with involuntary disengagement ($\beta=-0.25$, $p<0.01$; $\Delta R^2=0.05$) predicting aggression. As indicated by the significant interaction between primary control and peer stress, adolescent girls who were more likely to use problem solving, emotional expression and regulation to deal with peer stress than other strategies demonstrated the hypothesized positive association between stress and aggression. Interestingly, however, girls who used these coping strategies the least did not exhibit the expected association between peer stress and aggression, demonstrating higher levels of aggression, regardless of the level of peer stress.

As indicated by the interaction between involuntary disengagement (inaction, emotional/cognitive numbing, and involuntary avoidance) and peer stress, girls who were least likely to experience involuntary disengagement compared to other responses demonstrated a positive association between peer stress and aggression, whereas girls who experienced involuntary disengagement more often than the other responses demonstrated higher levels of aggression, regardless of the level of peer stress.

Pubertal timing—Analyses indicated one significant interaction between pubertal timing and involuntary disengagement ($\beta=0.26$, $p<0.01$; $\Delta R^2=0.07$) when examining aggression. No other significant interactions emerged. Results suggested that early maturers demonstrated a stronger association between involuntary disengagement and aggression ($\beta=0.75$, $p<0.001$; $\Delta R^2=0.46$) compared to on-time and late maturers ($\beta=0.29$, $p<0.05$; $\Delta R^2=0.08$). This suggested that early maturers who have a tendency to experience inaction and emotional and cognitive numbing more often than other responses to peer stress are likely to demonstrate high levels of aggression, whereas girls who are on-time or late demonstrate fairly low levels of aggression even under higher levels of peer stress (see Fig. 5).

Discussion

The goal of this study was to examine the association between social stress and symptoms of psychopathology from an integrated bio-psychosocial perspective and to expand upon the current understanding of how biological and social factors work in combination to influence emotional and behavioral problems during adolescence. Recent studies examining the impact of social stress on adjustment during adolescence have begun to explore how stress responses influence this relationship; however, most of these studies have focused on the direct association between stress responses and adjustment (see Compas et al. 2001 for

review). The present study demonstrated that *how* an adolescent responds to social stress may account for the association between stressful experiences and emotional and behavioral problems during adolescence.

Peer Stress

As expected, girls who experienced more peer stress reported higher levels of internalizing distress (Deater-Deckard 2001; Nansel et al. 2004; Rudolph 2002). Moreover, voluntary coping strategies mediated this association. Specifically, girls who experienced more peer stress typically used fewer effective strategies, such as problem solving, positive thinking, and emotional regulation, in relation to other responses to stress. In addition, girls who experienced more peer stress engaged in disengagement strategies (e.g., denial and wishful thinking) more often compared to other strategies. As no moderating effects were found for the association between peer stress and internalizing distress, findings suggest that peer stress may lead to poor coping skills rather than that it interacts with poor coping skills; poor coping skills (e.g., using problem solving and emotion regulation less often and engaging in denial and wishful thinking more often), in turn, were associated with greater levels of internalizing distress. Because the study is cross-sectional, it is not possible to determine whether or not higher levels of peer stress overwhelmed girls making them less likely to use better skills or whether girls who engaged in poorer coping strategies (in comparison to better strategies) were more likely to experience negative peer events due to their poor social competence. Longitudinal studies are needed to illuminate this issue.

Initial findings suggested peer stress was not associated with aggression, which was contrary to current research examining aggression during adolescence (Coie et al. 1990; Deater-Deckard 2001). However, analyses examining moderating effects found that primary control strategies (problem solving and emotional expression and regulation) and involuntary disengagement responses (inaction, emotional and cognitive numbing, etc.) moderated the association between peer stress and aggression. Specifically, peer stress was positively associated with aggression for girls who were high on primary control or for girls low on involuntary disengagement. Interestingly, girls who were either low on primary control or high on involuntary disengagement were consistently high on aggression regardless of their levels of peer stress. Although it appears that higher levels of peer stress pose a risk for the development of aggressive behavior, this finding may not generalize to adolescents who display problematic profiles of responses to stress. Perhaps these girls are more representative of adolescents with more severe conduct problems, suggesting that the stress and coping process may function differently for adolescents demonstrating more severe psychopathology. Future research examining stress and coping processes among adolescents with conduct disorder and other more pathological traits is needed.

Pubertal Timing

Consistent with prior research, early pubertal timing was linked to higher levels of aggression (see Graber 2003; Weichold et al. 2003 for reviews). However, based on findings from this study, this association may be partially accounted for by the way early maturing girls deal with stress. The use of primary control strategies (i.e., problem solving, getting advice from others, and emotional expression and regulation) has been shown to attenuate the effects of stressful experiences, whereas, the experience of involuntary engagement (i.e., impulsive action, physiological arousal, and intrusive thoughts or rumination) has been shown to intensify the effects of stress on the individual (Compas et al. 1999; Wadsworth et al. 2005). In the present study, results demonstrated that early maturers used fewer primary control strategies and reported more involuntary engagement (rumination, intrusive thoughts, physiological arousal), which in turn may have accounted for greater levels of aggression. The fact that these responses to stress served as mediators and not moderators

suggests that early maturation may predispose young adolescent girls to interpret and react to negative peer events differently from other girls, ultimately putting them at risk for aggressive behavior. For example, given the fairly restricted age range of the sample, early maturing girls were, for the most part, more advanced in their development than other girls. As such, the physiological changes of puberty may explain why earlier maturation was predictive of responses to stress that were assessed in part by physiological arousal. This finding is consistent with a prior study that found that higher emotional arousal mediated the pathway between early maturation in girls and adjustment (in that case depressive symptoms; Graber et al. 2006). It has also been shown that stressful home environments is associated with early maturation in girls; hence, it may also be that stressful family factors influence responses to stress that girls develop over time (Belsky et al. 2007).

Results also demonstrated a significant interaction between pubertal timing and involuntary disengagement (inaction, emotional/cognitive numbing, etc.) when examining aggression. On average, the tendency to exhibit involuntary disengagement more often than other responses was associated with higher levels of aggression. However, when comparing early maturers to other girls, results showed that the magnitude of association between involuntary disengagement and aggression was more than double that of the association exhibited by other girls and accounted for 46% of the variance in aggression for early maturers. Results demonstrate that early maturing girls who typically respond to peer stress with inaction, emotional and cognitive numbing, and involuntary fleeing are more likely than their peers to exhibit high levels of aggression. These findings clearly illustrate the interactive impact of prior vulnerabilities (poor responses stress) and stressful social experiences (early pubertal timing) on the expression of symptoms of psychopathology.

Notably, this study did not replicate previously established links between early maturation and internalizing distress (Graber et al. 2005). Past research has demonstrated that dramatic increases in internalizing distress in girls typically does not occur until mid-adolescence, resulting in a truncated range of symptoms when sampling early adolescent populations (see Costello et al. 2006, for a recent review). Thus, the fact that the majority of girls in this study were just entering adolescence ($Median_{age}$= 11.84) may have contributed to the absence of association between timing and internalizing distress.

Cortisol Reactivity

Contrary to expectation, cortisol reactivity was not associated with peer stress and symptoms of psychopathology. As previously indicated, cortisol-psychopathology links have mostly been found in samples reporting borderline or clinical levels of internalizing and externalizing behaviors (Angold 2003; Klimes-Dougan et al. 2001; Oosterlaan et al. 2005). In a study similar in design to the current study (i.e., normative adolescent samples exposed to social challenge tasks), Klimes-Dougan and colleagues (2001) demonstrated links between cortisol reactivity and internalizing symptoms, but were unable to find clear associations between cortisol reactivity and aggression. It was only among adolescents who displayed clinical levels of aggression that a significant association with cortisol reactivity emerged. Because most of the girls in this study displayed low or at most marginally high levels of internalizing distress and aggression, associations between cortisol reactivity and symptoms of psychopathology may not have emerged.

Interestingly, this study found a trend for early maturers to exhibit higher overall cortisol reactivity compared to other girls, suggesting that early maturers may have a more difficult time self-regulating once physiological arousal ensues. Results also found that cortisol reactivity was positively associated with involuntary engagement. Because one aspect of involuntary engagement is a self-report measure of physiological arousal, this finding suggests that adolescents may be able to reliably report on their own experiences of arousal.

This finding, coupled with the fact that the physiological measure of involuntary reactivity (i.e., $AUC_{cortisol}$) was not associated with psychopathology, holds important implications for the use of self-report when assessing physiological responses to stress. Given that the association between adrenocortical activity and psychopathology among adolescents is still not clear, it may be important to include self-report measures of physiological arousal along with physiological measures.

Limitations and Clinical Implications

This study has taken steps toward understanding how social stress influences the development of emotional and behavioral problems; however, some limitations of the study exist. The current study was limited to participants' self-report of peer stress, coping, and psychopathology. Only cortisol reactivity and pubertal timing were assessed via other methods. Additionally, because assessments were obtained concurrently the direction of associations were inferred from theoretical perspectives and previous research exploring stress, coping and psychopathology. For these reasons, results should be interpreted with some caution. For example, it is unclear if girls who tend to be more aggressive and engage in more involuntary responses to stressful experiences elicit more negative responses from peers, rather than peer stress triggering involuntary responses (e.g. physiological arousal), which in turn trigger aggressive behavior. Another limitation was that this investigation was based solely on young adolescent girls. Although researchers have suggested that social stress may be more detrimental to adolescent girls' psychosocial wellbeing (Rudolph 2002), it would be beneficial to examine further if gender differences during early adolescence emerge with respect to how girls and boys cope with social stress, whether particular coping strategies are more problematic for one gender versus the other, and whether certain coping strategies become more prominent throughout adolescence. Furthermore, this study did not directly explore how early maturers cope with the social and emotional ramifications of maturing earlier than their peers. Future studies exploring the links among peer events, pubertal timing, and symptoms of psychopathology should incorporate indicators of social stress that directly target the experience of maturing earlier than one's peers.

Despite the limitations, results speak to the importance of understanding how responses to stress impact symptoms of psychopathology. Findings of this study highlight the importance of considering both the active and involuntary aspects of stress response when addressing emotional and behavioral problems of adolescents. More specifically, the findings speak directly to the utilization of the cognitive-behavioral therapy approach by teachers, counselors, and clinicians to help teens learn to deal with potentially stressful experiences and minimize symptoms of psychopathology. Encouraging adolescents to modify potentially ineffective cognitive, behavioral and emotional reactions to stress (e.g., training on the use of problem solving tactics, importance of emotional expression, etc.) and teaching them to recognize and regulate involuntary responses such as physiological arousal and intrusive thoughts may help ease the psychological demands of transitioning into adolescence and ultimately reduce the likelihood of experiencing internalizing and externalizing problems. As suggested by the findings in this study, this treatment approach may be especially helpful to those adolescents exhibiting higher levels of aggression and a reduced tendency to engage in active and effective coping strategies as a means to manage involuntary responses to stress.

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References

Achenbach, TM. Manual for the Youth Self-Report and 1991 Profile. Burlington: Department of Psychiatry, University of Vermont; 1991.

Angold A. Adolescent depression, cortisol, and DHEA. Psychological Medicine. 2003; 33:573–581. Medline. [PubMed: 12785459]

Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. Journal of Personality and Social Psychology. 1986; 51:1173–1182. Medline. [PubMed: 3806354]

Belsky J, Steinberg LD, Houts RM, Friedman SL, DeHart G, Cauffman E, et al. Family rearing antecedents of pubertal timing. Child Development. 2007; 78:1302–1321. Medline. [PubMed: 17650140]

Brooks-Gunn, J. How stressful is the transition to adolescence for girls. In: Colten, ME.; Gore, S., editors. Adolescent stress: causes and consequences. New York: Aldine de Gruyter; 1991. p. 131-149.

Caspi A, Lynam D, Moffitt TE, Silva PA. Unraveling girls' delinquency: Biological, dispositional, and contextual contributions to adolescent misbehavior. Developmental Psychology. 1993; 29:19–30.

Chan W. Analyzing ipsative data in psychological research. Behaviormetrika. 2003; 30:99–121.

Chumlea WC, Schubert CM, Roche AF, Kulin HE, Lee PA, Himes JH, et al. Age at menarche and racial comparisons in US girls. Pediatrics. 2003; 111:110–113. Medline. [PubMed: 12509562]

Cicchetti D, Walker EF. Stress and development: Biological and psychological consequences. Development and Psychopathology. 2001; 13:413–418. Medline. [PubMed: 11523841]

Coie, JD.; Dodge, KA.; Kupersmidt, JB. The role of poor peer relationships in the development of disorder. In: Asher, SR.; Coie, JD., editors. Peer rejection in childhood. New York: Cambridge University Press; 1990. p. 274-305.

Compas, BE.; Connor, JK.; Saltzman, H.; Thomsen, AH.; Wadsworth, M. Getting specific about coping: Effortful and involuntary responses to stress in development. In: Lewis, M.; Ramsay, D., editors. Soothing and stress. Mahwah: Erlbaum; 1999. p. 229-256.

Compas BE, Connor-Smith JK, Saltzman H, Thomsen AH, Wadsworth M. Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. Psychological Bulletin. 2001; 127:87–127. Medline. [PubMed: 11271757]

Connor-Smith JK, Compas BE, Wadsworth ME, Thomsen AH, Saltzman H. Responses to stress in adolescence: measurement of coping and involuntary stress responses. Journal of Consulting and Clinical Psychology. 2000; 68:976–992. Medline. [PubMed: 11142550]

Costello EJ, Foley DL, Angold A. Ten-year research update review: The epidemiology of child and adolescent psychiatric disorders: II. developmental epidemiology. Journal of the American Academy of Child and Adolescent Psychiatry. 2006; 45:8–25. Medline. [PubMed: 16327577]

Cunningham WH, Cunningham ICM, Green RT. The ipsative process to reduce response set bias. Public Opinion Quarterly. 1977; 41:379–384.

Deater-Deckard K. Annotation: Recent research examining the role of peer relationships in the development of psychopathology. Journal of Child Psychology and Psychiatry. 2001; 42:565–579. Medline. [PubMed: 11464962]

Ge X, Brody GH, Conger RD, Simons RL, Murry VM. Contextual amplification of pubertal transition effects on deviant peer affiliation and externalizing behavior among African American children. Developmental Psychology. 2002; 38:42–54. Medline. [PubMed: 11806701]

Ge X, Conger RD, Elder GH. Pubertal transition, stressful life events, and the emergence of gender differences in adolescent depressive symptoms. Developmental Psychology. 2001; 37:404–417. Medline. [PubMed: 11370915]

Graber, JA. Puberty in context. In: Hayward, C., editor. Gender differences at puberty. New York: Cambridge University Press; 2003. p. 307-321.

Graber, JA.; Brooks-Gunn, J.; Archibald, AB. Links between girls' puberty and externalizing and internalizing behaviors: moving from demonstrating effects to identifying pathways. In: Stoff, DM.; Susman, EJ., editors. Developmental psychobiology of aggression. New York: Cambridge University Press; 2005.

Graber JA, Brooks-Gunn J, Warren MP. Pubertal effects on adjustment in girls: Moving from demonstrating effects to identifying pathways. Journal of Youth and Adolescence. 2006; 35:391–401.

Graber JA, Seeley JR, Brooks-Gunn J, Lewinsohn PM. Is pubertal timing associated with psychopathology in young adulthood? Journal of the American Academy of Child and Adolescent Psychiatry. 2004; 43:718–726. Medline. [PubMed: 15167088]

Graber, JA.; Sontag, LM. Internalizing problems during adolescence. In: Lerner, RM.; Steinberg, L., editors. Handbook of adolescent psychology. 2nd Edn. New York: Wiley; 2008. in press.

Granger DA, Weisz JR, Kauneckis D. Neuroendocrine reactivity, internalizing behavior problems, and control-related cognitions in clinic-referred children and adolescents. Journal of Abnormal Psychology. 1994; 103:267–276. Medline. [PubMed: 8040496]

Grant KE, Compas BE. Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. Psychological Bulletin. 2003; 129(3):447–466. Medline. [PubMed: 12784938]

Gunnar, MR. Early adversity and the development of stress reactivity and regulation. In: Nelson, CA., editor. The effects of adversity on neurobehavioral development: The Minnesota Symposia on Child Psychology. Vol. 31. Mahwah: Erlbaum; 2000. p. 163-200.

Gunnar MR, Vazquez DM. Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. Development & Psychopathology. 2001; 13:515–538. Medline. [PubMed: 11523846]

Herman-Giddens ME, Slora EJ, Wasserman RC, Bourdony CJ, Bhapkar MV, Koch GG, et al. Secondary sexual characteristics and menses in young girls seen in office practice: a study from the pediatric research in office settings network. Pediatrics. 1997; 99:505–512. Medline. [PubMed: 9093289]

Igram, RE.; Luxton, DD. Vulnerability-stress models. In: Hankin, BL.; Abela, JRZ., editors. Development of psychopathology: a vulnerability-stress perspective. Thousand Oaks: Sage; 2005. p. 32-46.

Jaser SS, Langrock AM, Keller G, Merchant MJ, Benson M, Reeslund K, et al. Coping with the stress of parental depression II: adolescent and parent reports of coping and adjustment. Journal of Clinical Child and Adolescent Psychology. 2005; 34:193–205. Medline. [PubMed: 15677293]

Kendall-Tackett KA. Physiological correlates of childhood abuse: chronic hyperarousal in PTSD, depression, and irritable bowel syndrome. Child Abuse and Neglect. 2000; 24:799–810. Medline. [PubMed: 10888019]

Klimes-Dougan B, Hastings PD, Granger DA, Usher BA, Zahn-Waxler C. Adrenocortical activity in at-risk and normally developing adolescents: Individual differences in salivary cortisol basal levels, diurnal variation, and responses to social challenges. Development & Psychopathology. 2001; 13:695–719. Medline. [PubMed: 11523855]

La Greca, AM. Friends or foes? Peer influences on anxiety among children and adolescents. In: Silverman, WKAT.; Treffers, PDA., editors. Anxiety disorders in children and adolescents: research, assessment and intervention. Cambridge: Cambridge University Press; 2001. p. 159-186.

Lazarus, RS.; Folkman, S. Stress, appraisal, and coping. New York: Springer; 1984.

Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in girls. Archives of Disease in Childhood. 1969; 44:291–303. Medline. [PubMed: 5785179]

Nansel TR, Craig W, Overpeck MD, Saluja G, Ruan WJ. Cross-national consistency in the relationship between bullying behaviors and psychosocial adjustment. Archives of Pediatric & Adolescent Medicine. 2004; 158:730–736. Medline.

Oosterlaan J, Geurts HM, Knol DL, Sergeant JA. Low basal salivary cortisol is associated with teacher-reported symptoms of conduct disorder. Psychiatry Research. 2005; 134:1–10. Medline. [PubMed: 15808285]

Preacher, KJ.; Hayes, AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in simple and multiple mediator models. 2006. Manuscript submitted for publication, may be accessed at: http://www.comm.ohio-state.edu/ahayes/indirect2.pd.

Rovine, MJ.; von Eye, A.; Wood, P. The effect of low covariate criterion correlations on the analysis-of-covariance. In: Wegmen, E., editor. Computer science and statistics: Proceedings of the 20th symposium of the interface; Alexandria, VA: American Statistical Association; 1988. p. 500-504.

Rudolph KD. Gender differences in emotional responses to interpersonal stress during adolescence. Journal of Adolescent Health. 2002; 30:3–13. Medline. [PubMed: 11943569]

Rudolph K, Dennig MD, Weisz JR. Determinants and consequences of children's coping in the medical setting: Conceptualization, review, and critique. Psychological Bulletin. 1995; 118:328–357. Medline. [PubMed: 7501740]

Schmidt LA, Fox NA, Sternberg EM, Gold PW, Smith CC, Schulkin J. Adrenocortical reactivity and social competence in seven year-olds. Personality and Individual Differences. 1999; 26:977–985.

Schwartz EB, Granger DA, Susman EJ, Gunnar MR, Laird B. Assessing salivary cortisol in studies of child development. Child Development. 1998; 69:1503–1513. Medline. [PubMed: 9914636]

Sobel, ME. Asymptotic intervals for indirect effects in structural equations models. In: Leinhart, S., editor. Sociological methodology 1982. San Francisco: Jossey-Bass; 1982. p. 290-312.

Stroud, LR.; Papandonatos, GD.; Williamson, D. Sex differences in the effects of pubertal development on responses to a corticotropin-releasing hormone challenge: the Pittsburgh Psychobiologic Studies. In: Dahl, RE.; Spear, LP., editors. Adolescent brain development: vulnerabilities and opportunities. New York: New York Academy of Sciences; 2004. p. 348-351.

Wadsworth ME, Raviv T, Compas BE, Connor-Smith JK. Parent and adolescent responses to poverty-related stress: tests of mediated and moderated coping models. Journal of Child and Family Studies. 2005; 14:283–298.

Weichold, K.; Silbereisen, RK.; Schmitt-Rodermund, E. Short-term and long-term consequences of early versus late physical maturation in adolescents. In: Hayward, C., editor. Gender differences at puberty. New York: Cambridge University Press; 2003. p. 241-276.

Weisberg HI. Statistical adjustments and uncontrolled studies. Psychological Bulletin. 1979; 86:1149–1164.

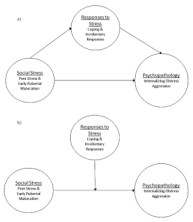

Fig. 1.

Conceptual models of stress and coping mechanisms via **a** mediation and **b** moderation pathways

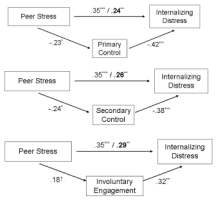

Fig. 2.

Path model for stressful peer events to internalizing distress. Each pathway controls for age. The *bolded β value to the right of the backslash* represents the association between peer stress and internalizing distress, controlling for the mediator. For the model including primary control, total $R2=0.30$, and F (3, 91)=13.07, $p<0.001$. For the model including secondary control, total R2=0.51, and F (3, 91)=10.53, $p<0.001$. For the model including involuntary engagement, total $R2=0.47$, and F (3, 91)=8.62, $p<0.001$. †$p<0.10$, *$p<0.05$, **$p<0.01$, ***$p<0.001$

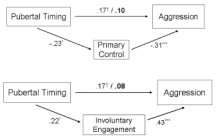

Fig. 3.

Path models for pubertal timing to aggression. Each pathway controls for age. The *bolded β value to the right of the backslash* represents the association between pubertal timing and aggression, controlling for the mediator. For the model including primary control, total $R2$=0.16, and F (3, 96)=5.98, p<0.01. For the model including involuntary engagement, total $R2$=0.22, and F (3, 95)=39.04, p<0.001. Mediation could not occur for cortisol, given that it was not significantly associated with aggression. †p<0.10, *p<0.05, **p<0.01, ***p<0.001

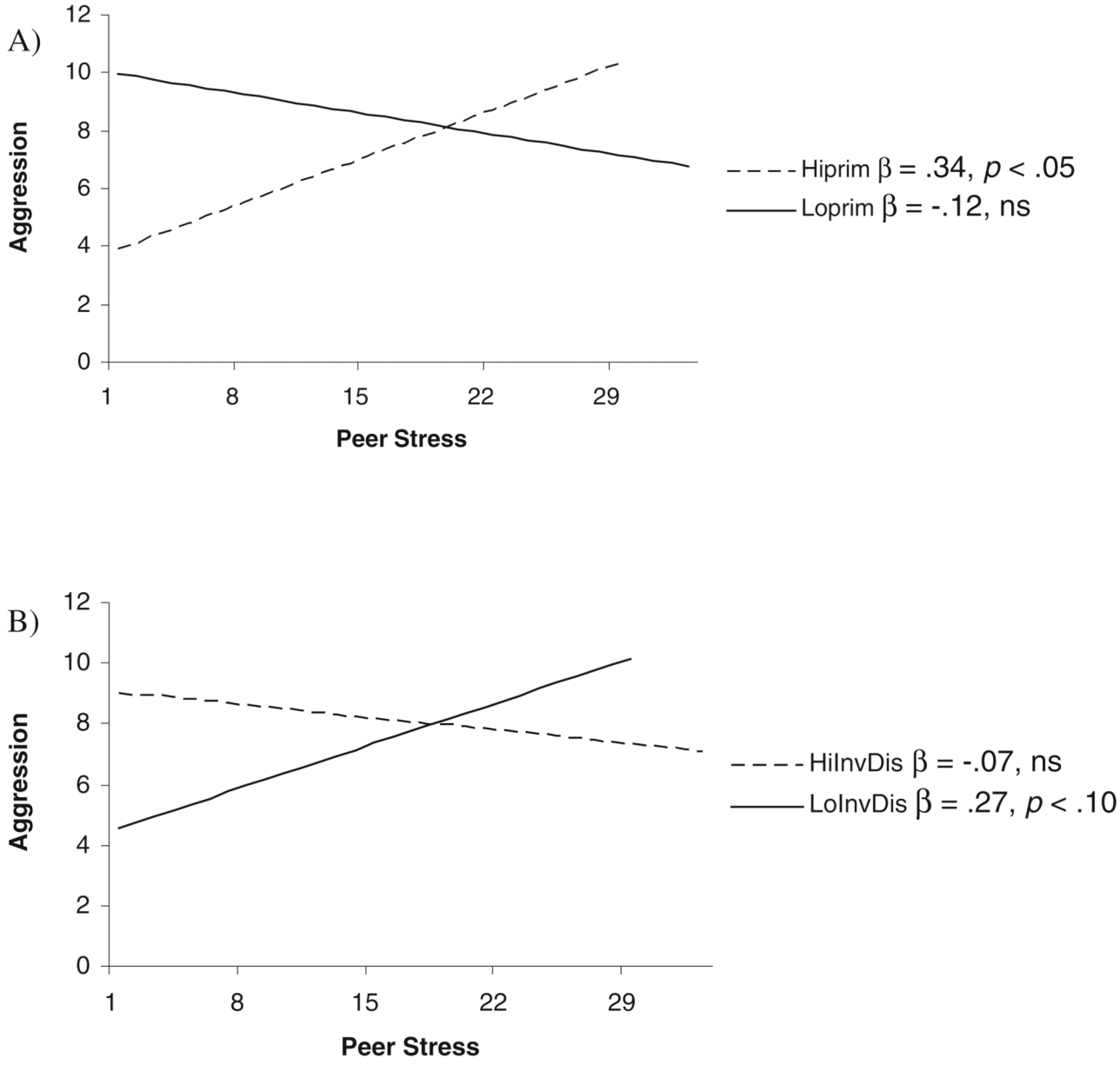

Fig. 4.

The following figures represent the interaction between peer stress and responses to stress, when examining aggression. **A** Interaction between peer stress and primary control; and **B** interaction between peer stress and involuntary disengagement. Beta weights are presented for each subgroup for the association between peer stress and aggression

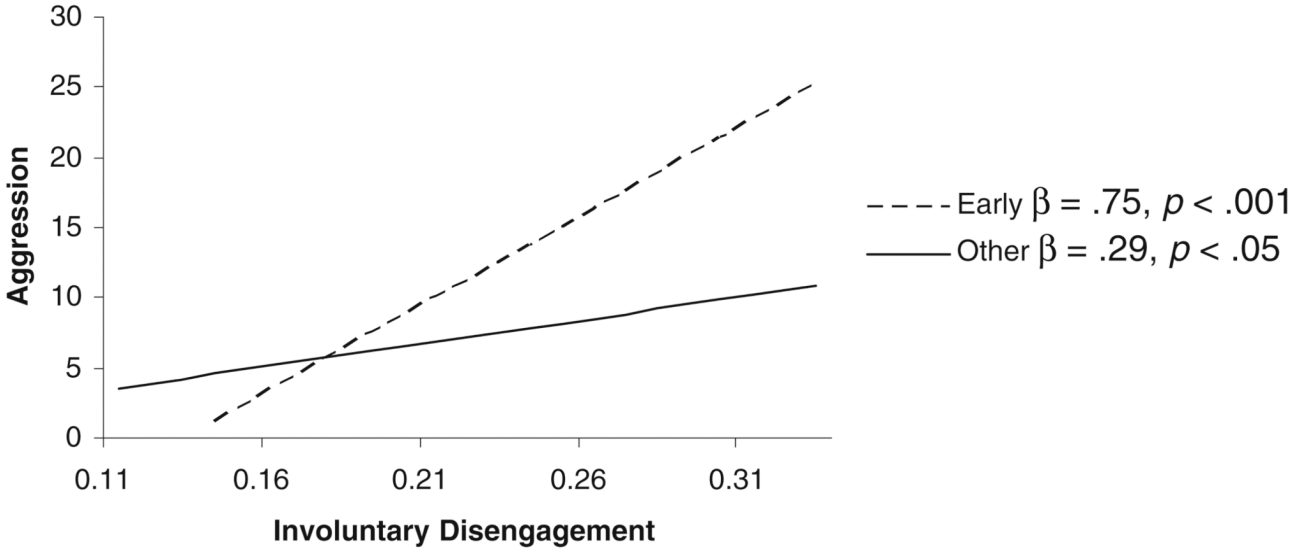

Fig. 5.

The following figure represents the interaction between pubertal timing and involuntary disengagement, when examining aggression. Beta weights are presented for early maturers and other (on-time and late) girls for the association between involuntary disengagement and aggression

Table 1

Means and Standard Deviations of Adolescents' Demographic Information, Health, Stress, Coping, and Psychological Symptoms

Variable	*N*	*M*	SD
Age	111	11.84	0.78
SES	110	38.30	13.81
Peer stress	97	6.41	6.67
Stressful peer events	98	2.60	1.96
Appraisal	97	2.16	1.00
Responses to stress			
Primary control	101	0.23	0.05
Secondary control	100	0.13	0.03
Disengagement	101	0.24	0.05
Involuntary engagement	100	0.20	0.04
Involuntary disengagement	100	0.19	0.04
Cortisol AUC	72	9.72	10.20
Internalizing distress	109	5.21	4.62
Aggression	109	7.07	5.54

Responses to stress are reported as ratio scores.

Table 2

Intercorrelations Among Demographic Information, Pubertal Timing, Stress, Coping, and Psychological Symptoms

	1	2	3	4	5	6	7	8	9	10	11	12
1. Age	–											
2. SES	−0.18*	–										
3. Pubertal Timing	0.11	−0.08	–									
4. Peer Stress	0.14	0.05	−0.06	–								
5. Primary Control	−0.01	0.19*	−0.22**	−0.23**	–							
6. Secondary Control	−0.20**	0.11	−0.02	−0.26**	0.48***	–						
7. Disengagement	−0.25**	−0.09	0.04	0.14	−0.59***	−0.26***	–					
8. Inv. Engagement	0.28***	−0.12	0.21**	0.21**	−0.44***	−0.57***	−0.19*	–				
9. Inv. Disengagement	0.22**	−0.10	0.06	0.15	−0.62***	−0.64***	0.01	0.35***	–			
10. Cortisol AUC	0.09	−0.07	0.21*	0.002	−0.13	−0.20	−0.09	0.27**	−0.11	–		
11. Internalizing Distress	0.14	0.02	0.16*	0.35***	−0.48***	−0.45***	0.19	0.40***	0.42***	−0.03	–	
12. Aggression	0.19**	−0.08	0.23**	0.12	−0.34***	−0.41***	−0.05	0.46***	0.43***	0.12	0.56***	–

* $p<0.10$,

** $p<0.05$,

*** $p<0.01$