COPING WITH SOCIAL STRESS: IMPLICATIONS FOR PSYCHOPATHOLOGY IN ADOLESCENT GIRLS

By

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A THESIS PRESENTED TO THE GRADUATE SCHOOL
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To those who have always supported me and encouraged me to reach beyond my limits.

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This study investigated the impact of social stress on girls' psychosocial development during adolescence by integrating models of peer stress, pubertal timing, adrenocortical activity, and coping. Based on a sample of 111 girls (M_{age}=11.84, SD = .77), we explored whether patterns of cortisol, pubertal timing, and peer stress were associated with symptoms of internalizing distress and aggression. Additionally, we examined whether coping strategies and involuntary responses to stress mediated these associations. Cortisol reactivity was not associated with adjustment; however, pubertal timing and peer stress were. Coping and involuntary responses mediated the association between pubertal timing and peer stress and adjustment. Findings indicate that learning to effectively adapt to stress may help girls transition more successfully into adolescence.

CHAPTER 1
INTRODUCTION

Background

Adolescence is a period of rapid growth and change, both physically and psychologically. During the transition into adolescence, individuals must learn to manage the physical changes their bodies endure due to the onset of puberty, changes in social context, and changes in school environment (Graber & Brooks-Gunn, 1996). Such experiences inherently place young adolescents at heightened risk for experiencing a great number of new daily stressors (Brooks-Gunn, 1991; Ge, Conger, & Elder, 2001). Because the chance of encountering some type of environmental or social stressor is inevitable, young adolescents must learn to adapt and to cope with these life experiences in order to successfully navigate the transition into adolescence and eventually into early adulthood.

During the transition into adolescence, individuals experience more stressful events compared to older adolescents or children, which in turn is associated with an increase in such outcomes as depressive affect (Graber & Brooks-Gunn, 1996; Rudolph, 2002). Of these stressful events, off-time pubertal maturation and interpersonal stress within the context of peers pose particular threats to adolescents (Graber, Brooks-Gunn, & Archibald, 2005). According to Rudolph's model of interpersonal stress (2002), girls are at a particular risk for experiencing higher levels of peer stress and consequently psychological maladjustment compared to boys, due to the fact that peer relationships, specifically with close friends, are of greater importance to girls. In addition, some individuals exhibit disregulations in the production of cortisol in response to stressors which increases the risk for poor psychological outcomes. For example, variations in both basal activity and stress reactivity have been linked to internalizing and externalizing problems among young adolescents (Klimes-Dougan, Hastings, Granger, Usher, &

Zahn-Waxler, 2001). However, exposure to potentially stressful situations is not always linked to poor outcomes. The ability to cope with or respond effectively to stress relies heavily upon the ability to interpret and react appropriately to the stressor itself or to the physiological, emotional, and behavioral responses to the stressor. By integrating models of social and biological indicators of stress and by using a more comprehensive view of how adolescents respond to stressful experiences, this study explored what factors place some young adolescent girls at greater risk for maladjustment in the face of stressful life experiences.

Stress

Stress may be best conceptualized as a multidimensional concept that includes not only the stressor, but also the processing system, which includes the cognitive assessment of the stimuli, and the stress response (Compas, Orosan, & Grant 1993). Although conceptually stress may be a multilevel process, more often than not the experience of stress is quite automatic and usually occurs unconsciously. Moreover, not all experiences are equally stressful to individuals, thus making appraisals of potentially stressful experiences relevant (Lazarus & Folkman, 1984).

The transition into adolescence is marked not only by physical and sexual maturation, but also by increased exposure to new, many of them challenging, events (Brooks-Gunn, 1991; Ge et al, 2001; Graber & Brooks-Gunn, 1996). Although there is evidence that stress increases during early adolescence, the experience of particular events as stressful is not necessarily pervasive among all adolescents (Brooks-Gunn, 1991). Rather, the experience of an event as stressful is likely due to interactions among various changing life events (e.g., interactions among hormonal and physical changes, school transitions, peer group changes, and altering family dynamics) that influence an adolescent's interpretation of particular events as stressful (Brooks-Gunn, 1991).

One aspect of stress that seems to be particularly salient to adolescents is social stress. Social stress represents stimuli within the context of social interactions or relationships that

evoke emotional, physical, and psychological arousal in individuals. Social stress may be broadly characterized as experiences within a social context, including interactions with others (e.g., peer stress), as well as how one's own development (e.g., puberty) is experienced in a social context.

Peer stress

Peer stress may be best conceptualized as experiences within the peer context that exert significant pressure on an individual's ability to function adaptively. More concrete examples of peer stress include conflicts with close friends, being teased or hassled by other children, and being rejected by other children. Peer stress becomes increasingly salient as a threat to psychological well-being during adolescence due to the increasing amount of time spent with peers relative to other interpersonal relationships (La Greca, 2001). The relative salience of peer relationships and the impact of peer stress during the transition into adolescence are further supported by findings from Rudolph (2002) that demonstrated that levels of stress increase from preadolescence to adolescence independent of gender.

Increased exposure to interpersonal stress during childhood and adolescence has been linked to disorders and symptoms of psychopathology, namely anxiety, depression, behavior problems, and substance use (Coie, Dodge, & Kupersmidt, 1990; Hawker & Boulton, 2000). Specific aspects of peer stress, peer rejection and victimization, have been consistently linked to externalizing and internalizing problems in adolescents (Deater-Deckard, 2001). A study examining aggression and peer victimization as predictors of behavioral and emotional adjustment found that greater experiences of peer aggression predicted greater levels of both self-reported aggression and delinquency (Khatri, Kupersmidt, & Patterson, 2000). Another study, conducted by Storch, Nock, Masia-Warner, and Barlas (2003), found that among Hispanic

and African American children, overt peer victimization was positively associated with depressive symptoms and aspects of social anxiety. Additionally, relational victimization was uniquely associated with depressive and anxious symptoms for girls only. Recent work examining cross-national consistency in the relationship between being bullied and psychosocial adjustment found that being the recipient of bullying behaviors was linked to poor emotional adjustment, poor school adjustment and poor relationships with classmates (Nansel et al., 2004).

As indicated, girls experience higher levels of peer stress compared to boys during adolescence (Rudolph, 2002). This differential trend is likely due to the fact that peer relationships, specifically with close friends, are of greater importance to girls. It is not to say that boys do not have experiences that could be interpreted as stressful, but rather girls interpret comparable situations as more stressful and detrimental to their interpersonal relationships.

Pubertal timing

One aspect of social stress that is particularly salient to adolescents is the experience of pubertal changes and whether those changes reflect normative or off-time developmental trends within one's peer group. In order to better understand the impact of pubertal development on adolescents' overall experiences of stress and the relationship between puberty and psychopathology, it is useful to distinguish between pubertal status and pubertal timing. According to Graber, Peterson, and Brooks-Gunn (1996), pubertal status refers to the physical and physiological maturational levels of an individual at a given point in time, relative to the overall pubertal process. Pubertal timing, however, may be conceptualized as a social construction and best represents whether an individual's maturation is earlier, on-time, or later in comparison to same-age peers.

Research examining the relationship between pubertal maturation and changes in depressive or aggressive affect has found few links between pubertal status and negative outcomes (Brooks-Gunn, Graber, & Paikoff, 1994; Graber et al., 2005). In contrast, research focusing on the effects of pubertal timing has demonstrated that the stress and conflict elicited by one's off-time maturation is more strongly linked to psychological and behavioral maladjustment (Graber et al., 2005 for a recent review). Additionally, this research has also indicated that early maturing girls are particularly at risk for a variety of disorders or symptoms of disorders, including anxiety, depression, aggression, substance abuse, and eating disorders. A study by Graber, Seeley, Brooks-Gunn, and Lewinsohn (2004) examining the association between pubertal timing and psychopathology in young adulthood found that women who were early maturers were at unique risk for persistent difficulty and development of psychopathology during adolescence and young adulthood. Other studies examining the role of pubertal timing found that early maturing girls were at high risk for depressive symptoms, body dissatisfaction, aggression, and delinquency problems (Caspi, Lynam, Moffitt, & Silva, 1993; Ge, Brody, Conger, Simons, & Murry, 2002; Ge et al., 2001; Ohring, Graber, & Brooks-Gunn, 2002).

In order to explain the connection between hormonal and pubertal changes and negative affective and behavioral outcomes, Brooks-Gunn and colleagues (1994) proposed a model that included the possibility that social events or context, as well as the adolescent's perceptions of his or her pubertal processes, contribute to or function as mediators of hormonal effects on negative affect and behavior. In testing this model, Brooks-Gunn and Warren (1989) found that social events, not pubertal development, accounted for a good deal of variance in affect and behaviors. In terms of social processes, adolescents who mature early or late in comparison to their peers are placed in socially "deviant" categories compared with on-time peers (Petersen &

Crockett, 1985). Keeping in mind that girls as a group tend to mature a couple of years earlier than boys, early maturing girls are deviant in comparison to both other girls and boys, which places these girls at a heightened risk for maladjustment.

This study explored the effects of pubertal timing within a social processes framework in order to expand on previous findings that have linked the social experience of being "off-time" and particular emotional or behavioral changes. As highlighted by Brooks-Gunn and colleagues (1994), social factors play a significant role in connecting the pubertal timing and negative affect or behavior. The way an individual interprets his or her timing is likely to influence the types of social interactions he or she experiences, and in turn is likely to influence potential affective or behavioral changes. Research has shown that early maturing girls consistently report poor quality of relationships at mid-adolescence and young adulthood (Graber et al., 2004). By examining how early maturing girls cope with or adapt to potentially stressful situations within the peer context, a better understanding of the mechanisms by which pubertal timing relates to maladjustment may emerge.

Biological correlates of stress

Under conditions interpreted as stressful, the limbic-hypothalamic-pituitary-adrenal (LHPA) axis in mammals (including humans) responds to this experience via a series of events that result in the secretion of glucocorticoids (cortisol in human and nonhuman primates, and corticosterone in rodents) from the adrenal cortex (Cicchetti & Walker, 2001). Prolonged exposure to environmental and internal stress, and in turn disregulations of cortisol levels, can affect psychological well-being. Past studies investigating the association between adrenocortical activity, stress, and psychosocial well-being found that variation in both the basal activity and in stress reactivity has been associated with psychopathology (Angold, 2003; Goodyer, Herbert,

Tamplin, & Altham, 2000; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001; Southwick, Vythilingam, & Charney, 2005). However, much of this literature has focused primarily on diurnal patterns of change and investigated cortisol reactivity in clinical populations (Oosterlaan, Geurts, Knol, & Sergeant, 2005; Van Bokhoven et al., 2005; Van de Wiel, Van Goozen, Matthys, Snoek, & Van Engeland, 2004). Therefore the question still remains as to *how* patterns of cortisol reactivity in normative samples of adolescents reflect experiences of stress, particularly social stress, and how these patterns of reactivity are associated with changes in negative affect and behavior.

For individuals who follow a typical day-night schedule, cortisol production peaks during the last few hours of sleep (Gunnar & Vazquez, 2001 for a review). Cortisol levels then decline rapidly throughout the morning and decrease gradually during the remainder of the day. When stress is induced, cortisol secretion initially increases. Following the onset of a stressor, cortisol 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. Following this peak, the negative feedback loop of the LHPA axis begins to reestablish homeostatic functioning by reducing the level of circulating cortisol.

Recent research examining the association between changes in daytime cortisol levels in response to social challenges (i.e., public speaking task or mother-child conflict task) has shown that adolescents and children tend to display increases in salivary cortisol approximately 20 to 30 min following exposure to a social challenge task, and then display a decrease in levels of cortisol (Granger, Weisz, & Kauneckis, 1994; Klimes-Dougan et al., 2001; Schmidt et al., 1999). In addition, findings from these studies have indicated that maintaining high levels of cortisol or

displaying strong task-related decreases are associated with higher levels of internalizing and externalizing behavior.

Findings such as those mentioned previously point to a link between stress response and psychopathology; however, activation of the L-HPA axis may not be directly linked to symptomatology. Additionally, there are few conclusive or consistent findings within the child and adolescent research about whether a true relationship between cortisol levels and psychopathology exists. Therefore, more research examining the association between cortisol and psychopathology is needed, especially within the adolescent female population.

Coping and Responses to Stress

Because of the increase in stressful experiences during the transition to adolescence (Brooks-Gunn, 1991), learning to adapt to the stressor and manage one's response to the stressful situation is crucial to leading a healthy and productive life. However, the effects of stress vary by individuals. Whereas most people fare quite well in the face of typically occurring stressful events, other individuals manifest symptoms of psychopathology, as mentioned previously. But the question therein lies, why do individuals demonstrate differential outcomes to stress exposure and how do these differences emerge? Coping strategies offer one of the mechanisms through which stress differentially affects individuals. Understanding the process of coping and how adolescents effectively or ineffectively react to stressful stimuli allows researchers to understand better why particular individuals adapt well or adapt poorly.

As suggested by Lazarus and Folkman (1984), coping involves efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person. Coping differs from general responses to stress in that coping is viewed as volitional or under the person's control. In addition, Lazarus and Folkman emphasize the role of cognitive processes in determining what is experienced as stressful and how one copes.

Theoretical frameworks of coping, such as the one developed by Lazarus and Folkman (1984), adequately target volitional responses to stress; however, few models have included involuntary responses to stress. One comprehensive model, developed by Compas, Connor, Saltzman, Thomsen, and Wadsworth (1999), includes both volitional and involuntary responses to stressful stimuli. According to this model, stress response can be characterized as being either effortful or involuntary. Effortful responses are cognitive and behavioral reactions to stress experienced as volitional and purposeful such as acceptance, denial, and avoidance. Involuntary responses, as defined by Compas et al. (1999), 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. In combination with the volitional responses to stress (i.e., coping), involuntary responses create a more representative model of how individuals respond to external and internal stressors throughout their lifespan.

Compas and colleagues (1999) further differentiated effortful and involuntary responses to stress by distinguishing between engagement and disengagement responses. Engagement responses are directed toward a stressor or one's reactions to the stressor and include approach responses. Disengagement responses are directed away from these targets and include avoidance responses. Given this model, an individual may respond to a stimulus in one or more of the following manners: effortful engagement (primary and secondary control), effortful disengagement, involuntary engagement, and involuntary disengagement. Connor-Smith et al. (2000) tested this model using a sample of adolescents and their parents. Findings indicated that not only was there adequate to excellent internal consistency for each subtype of response to stress (i.e., primary control, secondary control, disengagement coping, involuntary engagement,

and involuntary disengagement) but subscales of coping, specifically, mapped onto other standard measures of coping quite clearly. More important, however, was the finding that factors within this model of responses to stress correlated with internalizing and externalizing behavior. Thus, these exploratory analyses suggest that a model including both coping and involuntary responses adequately captured how adolescents adapt to stressors. Additionally, how one copes with stress was associated with emotional or behavioral outcomes.

Other studies examining the association between responses to stress and emotional and behavioral outcomes have consistently demonstrated that style of response mediates the relationship between stress, specifically family conflict and chronic pain, and anxiety, depression, and aggression (Jaser et al., 2005; Wadsworth, Raviv, Compas, & Connor-Smith, 2005). Extrapolating to other realms of stress, it is likely that an individual's type of response to social stress (e.g., peer stress) will mediate the relationship between social stress and psychopathology during adolescence. However, little empirical work has examined the effects of responses to stress on this relationship and even fewer studies have extended this research to examining how coping may serve as a mechanism through which biological indicators of stress (i.e., cortisol) and pubertal timing influence poor outcomes.

Proposed Study

This study investigated the impact of stress reactivity and social factors on negative outcomes from a bio-psychosocial perspective as a means to explain emotional and behavioral changes that occur during the transition into adolescence. By integrating several theoretical models that focus on the context of peer stress, pubertal timing, adrenocortical activity, and coping (Figure 1), this study aimed to provide a more comprehensive approach to understanding the pathways to adjustment. Within this framework, this study examined physiological and environmental indicators of social stress and how these indicators were associated with

symptoms of psychopathology, in particular internalizing distress and aggression. Additionally, this study explored whether responses to stress mediated this relationship. Specifically, the study examined the following questions:

Research question 1

- Are physiological and environmental indicators of social stress associated with symptoms of internalizing distress and aggression?

- Specifically, is biological stress reactivity (i.e., pattern of cortisol levels) associated with symptoms of psychopathology? Because of the exploratory nature of the study with respect to the findings associated with cortisol, it was predicted that pattern of reactivity was associated with symptoms of internalizing distress and aggression. However, specific predictions about the association between particular patterns of cortisol and behavioral outcomes were not made.

- Is pubertal timing associated with symptoms of internalizing distress and aggression? This study predicted that girls who reported early pubertal timing would demonstrate higher levels of internalizing distress and aggression compared to girls who reported being on-time or late.

- Are experiences of peer stress associated with symptoms of internalizing distress and aggression? It was hypothesized that girls who reported a greater number of stressful peer experiences would display more symptoms of internalizing distress and aggression.

Research question 2

- Do coping strategies mediate the association between biological and environmental indicators of stress and symptoms of psychopathology?

- Do coping strategies mediate the association between stress reactivity (i.e. cortisol patterns) and symptoms of internalizing distress and aggression? It was hypothesized that the 5 coping strategies or responses to stress (i.e., primary control, secondary control, effortful disengagement, involuntary engagement, and involuntary disengagement) would mediate the association between stress reactivity and symptoms of psychopathology. More specifically, it was predicted that effortful disengagement and involuntary disengagement would serve as mediators between stress reactivity and internalizing distress, whereas involuntary engagement would mediate the association between stress reactivity and aggression. It was expected that primary and secondary control coping strategies would mediate the association between stress reactivity and internalizing distress and aggression.

- Do coping strategies mediate the association between pubertal timing and symptoms of internalizing distress and aggression? It was expected that coping strategies would mediate the association between pubertal timing (i.e., early vs. on-time or late) and symptoms of psychopathology in the same way that was described for stress reactivity.

- Do coping strategies mediate the association between peer stress and symptoms of internalizing distress and aggression? It was hypothesized that coping strategies would mediate the association between peer stress and symptoms of internalizing distress and aggression in the same way that was described for stress reactivity and pubertal timing.

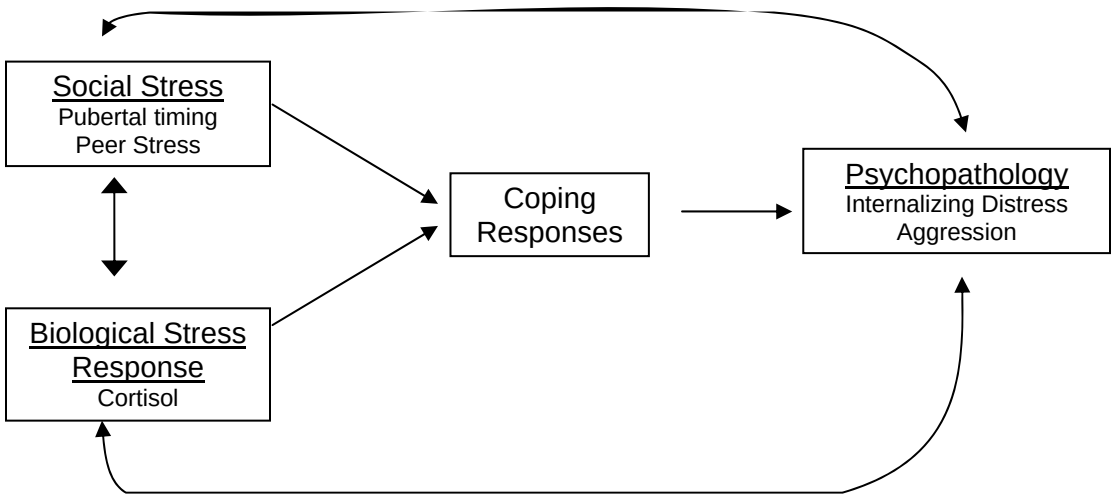


Figure 1-1. Bio-psychosocial framework

CHAPTER 2
METHODS

Participants

Participants were 111 adolescent girls (M_{age} = 11.84, *SD* = .77), who were drawn from a larger project, the Girls Health and Development project, that examined biological correlates of girls' social and emotional health as they made the transition to puberty. Approximately 15% of the girls were in 5th grade, 47% in 6th grade, 31% in 7th grade, and 2% in 8th grade. Of the 111 girls, 48% were Caucasian, 36% African American, and 16% Hispanic. Girls and their families were originally recruited in 1995-1996 from public schools in ethnically integrated, working-class and middle-class communities in the New York City area. The study included 138 girls at baseline; 111 of these girls participated in the final follow-up, approximately three years later. Analyses for the current study were completed using only girls who participated in the final follow-up.

Procedure

Girls and their families were recruited via their schools. Each time families participated, parents signed informed consent forms at their homes, on their own behalf and for their child's participation. Girls gave assent for their participation. Mothers and children completed several tasks (Appendix A) during a home visit and completed surveys during the 3 consecutive days following the home visit. Girls completed surveys on their own affect and behaviors. Surveys were picked up from the families' homes 3 days after the home visit. Girls received a gift (tote bag) for their participation and mothers were paid $75.

Measures

Demographic Data

Data concerning the characteristics of the participants were collected using standard items concerning age, household structure, and race/ethnicity. Girls reported their date of birth, and exact age was calculated from date of home visit. Mothers reported on family characteristics and child race/ethnicity. Race/ethnicity was collapsed into three categories: White, African American, and Hispanic. For regression analyses, two dummy coded variables were used: African American versus others and Hispanic versus other, with White as the omitted group.

Family SES was assessed using the Hollingshead Scale (Hollingshead, 1975), a scale based on survey data of social status tied to U.S. census categories. The standard scoring protocols were used for different household types. Mothers reported on the education and occupation of themselves and any other parent/caregiver in the household. For each head of household, education (coded on a 7-point scale from 1 = less than 7^{th} grade to 7 = graduate professional) was multiplied by a weight of 3 and occupation (coded on a 9-point scale from 1 = farm labor and menial service workers to 9 = higher executives and major professionals) was multiplied by a weight of 5. Thus the range of possible scores for family SES was 8 to 66 (Hollingshead, 1975). For one-parent households, the education score and occupation score were summed to yield an SES score. In two-parent families, one parent in paid employment, the family SES was derived by adding the mean of the parents' education scores to the occupation score for the employed parent. If both parents were employed, the family SES score was the mean of the parents' SES scores.

Biological Response to Stress

During the in-home visit, 5 saliva samples were collected to assess cortisol levels throughout a battery of tasks. See the Appendix A for a complete list of tasks. Approximately 15-20 min occurred between collections. The unit of measurement for cortisol assayed was µg/dl.

Saliva was collected via Salivettes. Girls kept the 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 (Granger et al., 1994; Gunnar & Vazquez, 2001; Hertsgaard, Gunnar, Larson, Brodersen, & Lehman, 1992; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). 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, McFadyen-Ketchum, Dodge, Pettit, & Bates, 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 -20ºC 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 3000 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.

Certain samples were excluded from analysis due to problems with the saliva sample. The lab processing the samples excluded participants' samples if the samples were insufficient to

process. Log transformations were applied to the cortisol data to reduce the large positive skew, which is consistent with standard procedures in cortisol research (Gunnar, 2000). However, original units were reported for ease of interpretation.

Pubertal Timing

Pubertal timing was based on self-reported age of menarche obtained during the home visit. A recent review of the use of self-reported menarche noted that accuracy of recall is reduced as the time interval between menarche and recall is increased (Coleman & Coleman, 2002). However, if assessed within a period of 1 year from menarche, girls can typically recall menarche within 1 month of the exact date. Given that the study's participants have recently experienced menarche, the use of self-report can be considered a valid measure.

Using self-reported age of menarche, girls were classified as either early-maturers or other (on-time or late). Cut-off ages by race (White, African American, and Latina) were derived from a large, normative study of puberty (Herman-Giddens et al., 1997). Based on mean ages of menarche reported by race in the Herman-Giddens study, girls that reported age of menarche ½ SD below the mean were categorized as early. All other girls were in the other group (on-time or late). According to an investigation of age at menarche for girls in the United States, age of menarche for Hispanic girls tends to fall between ages for non-Hispanic white girls and non-Hispanic black girls (Chumlea et al., 2003). Because the Herman-Giddens study only examined White and African American girls, estimates for early Latinas were calculated as ½ SD below the mean menarcheal age of the sample. Cut off ages for early maturing girls were: 12.28 y for White, 11.55 y for African American, and 11.97 y for Latina. Late maturation could not be determined due to the mean age of assessment (just under 12 y) because the mean age of menarche for each race as determined by Herman-Giddens et al. (1997) was greater than 12 years. Thirty five percent of the participants were classified as early maturers. Approximately

28% of White participants, 33% of African American participants, and 40% of Latina participants were early maturers.

Experiences of Peer Stress

Participants' experiences of peer stress were assessed as part of the survey administered in the days after the home visit using a stressful events checklist that was included in a version of the Responses to Stress Questionnaire that was adapted for this study (RSQ; Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000). The RSQ was developed to assess experiences of stressful events and responses to stress in adolescent populations. The checklist provided information about how many stressful experiences the participants had encountered since the start of the school year. The RSQ can be tailored to specific stressors; this form of the RSQ was tailored to assess peer stress. For the stressful events checklist, participants endorsed 9 items. Sum scores were calculated, with higher scores representing the experience of more stressful events. Peer stress, as discussed in the study, was operationalized as the number of stressful events a participant experienced.

Appraisal

Appraisal of stressful experiences was also assessed using the RSQ. Participants responded to a single item that assessed overall how stressful the problems endorsed in the stressful events checklist were for them on a likert scale (1 = not at all to 4 = very). Higher scores indicated that the participant felt the experiences to be more stressful.

Coping

Coping was also assessed using the RSQ. Adapted from the original RSQ for the purposes of this study, the measure consisted of 38 items rated on a 4-point likert scale (1 = not at all to 4 = a lot). The RSQ contains 5 subscales: primary effortful control, secondary effortful control, effortful disengagement, involuntary engagement and involuntary disengagement. In this study,

the primary effortful control subscale contained 7 items that assessed responses to stress categorized as emotional expression, emotional regulation, and problem solving (α = .80). The secondary effortful control subscale contained 4 items that measured responses to stress categorized as acceptance, positive thinking, and cognitive restructuring (α = .70). The effortful disengagement subscale contained 9 items and assessed denial, avoidance, and wishful thinking (α = .75). The involuntary engagement subscale contained 9 items, which assessed impulsive action, physiological arousal, and intrusive thoughts or rumination (α = .78). Finally, the involuntary disengagement subscale contained 9 items that measured inaction, emotional and cognitive numbing, and involuntary avoidance or fleeing (α = .76). High scores on each subscale indicated that participants were more likely to engage in these types of responses in the face of stress. Raw scores were reported as sum scores and used for reliability analyses. However, in order to control for overall responding bias, factor scores reflecting the proportion of total responses (e.g., sum of scores on primary control items/sum of all items) were used in analyzing the associations with internalizing distress and aggression as recommended by Connor-Smith and colleagues (2000).

Internalizing Distress

Internalizing distress was assessed during the home visit using the anxiety/depression subscale of the Youth Self Report (YSR/11-18) created by Achenbach (1991). The YSR for ages 11-18 is a self-report measure designed to measure competencies, adaptive functioning, and problems in youth and adolescents ages 11-18. It can be completed by those having a fifth grade reading level or administered orally. Participants were asked to report on behavior now or within the past 6 months and rated items on a likert scale (0 = not true, 2 = very true or often true). Scores for the anxiety/depression subscale were reported as sum scores. The anxiety/depression subscale contained 16 items, with a reliability of α = .83.

Aggression

Aggression was also assessed using the YSR/11-18 (Achenbach, 1991). Participants rated items on a likert scale (0 = not true, 2 = very true or often true). Participants were asked to report on behavior now or within the past 6 months. Scores for the subscale of aggression were reported as sum scores. The aggression subscale contained 19 items, with a reliability of $\alpha = .86$.

Analysis Plan

The goal of this study was to examine physiological and environmental indicators of social stress and how these indicators were associated with symptoms of psychopathology, specifically internalizing distress and aggression. Additionally, this study explored whether responses to stress mediated this association. First, attrition analyses were performed to determine if the sample used in this study differed on key variables from the original sample of participants recruited. Descriptive analyses were performed on key variables.

To address the first set of hypotheses, main effects were explored via ANCOVAs and hierarchical linear regression. As a means to explore the main effect of stress reactivity on internalizing distress and aggression, a combination of linear regression models and mixed models was used. Within the cortisol literature, researchers use multiple approaches to examine the association between stress reactivity (i.e., change in cortisol levels) and outcomes of interest. The computation of the area under the curve (AUC) is a frequently used method in endocrinological research and the neurosciences to explore information that is contained in repeated measurements over time (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). In the case of this study, AUC was employed to investigate task-dependent changes in cortisol level over the period of the home visit. $AUC_{cortisol}$ was calculated from zero and log transformations were applied to the $AUC_{cortisol}$ value for each participant to reduce the large

positive skew. To explore the main effects of stress reactivity, $AUC_{cortisol}$ was entered as the independent variable in a linear regression model.

Although frequently used in endocrinological research to capture patterns of change over time, the area under the curve as calculated from zero may neglect to capture idiosyncrasies of change that occur regardless of one's absolute levels of cortisol. Also, as indicated, calculating area under the curve requires data at each time point. Thus, in order to further explore potential associations between patterns of change in cortisol production and symptoms of internalizing distress and aggression, mixed effects models were used. These models addressed the question of whether one's pattern of production of cortisol over the 5 home visit samples changed in association with one's symptoms of psychopathology. In addition, mixed models do not require participants to have data at each time point. Thus, for this study, it was possible to estimate the desired associations with fewer than 5 cortisol samples, allowing for a greater total sample size.

Addressing the remaining components of the first set of research questions, the main effect of pubertal timing on internalizing distress and aggression was explored via a combination of ANCOVAs and a hierarchical linear regression model. The main effect of peer stress was explored using only a linear regression analysis.

Mediation was tested using the guidelines delineated by Baron and Kenny (1986). The mediators were primary control coping, secondary control coping, disengagement coping, involuntary engagement responses, and involuntary disengagement responses. Specifically, separate regression models tested the effects of the social stress predictors (peer stress, pubertal timing) or biological correlates of stress (cortisol) on each mediator and both internalizing distress and aggression. A full model was then tested with the social stress predictor and all the mediators 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. In all models, race and family SES was controlled for; in the models with peer stress as the predictor, appraisal was also controlled for.

Researchers comparing methods to test mediation have suggested that Baron and Kenny's (1986) method neglects to provide a direct estimate of the size of the indirect effect of the independent variable on the dependent variable (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Preacher & Hayes, 2004). In turn they suggest supporting research findings with a product of coefficients test, typically the Sobel Test, which states if *ab* (the indirect effect) is not significantly different from zero, there is no effect of mediation (Sobel, 1982). However, according to Preacher and Hayes (2004) this method is also misleading because there is reason to be suspicious of the use of the normal distribution for computing the *p* value for the Sobel test because the sampling distribution of *ab* may not be normal. To address this issue, Preacher and Hayes (2006) recommend bootstrapping the sampling distribution of *ab* and deriving a confidence interval with the empirically derived bootstrapped sampling distribution. This approach has been suggested as a way of circumventing the power problem introduced by asymmetries and other forms of non-normality in the sampling distribution of *ab* (Preacher & Hayes, 2004; Shrout & Bolger, 2002). Given this recommendation, a test of the indirect effect was performed to support mediation effects consistent with Baron and Kenny's (1986) guidelines.

CHAPTER 3
RESULTS

Attrition Analyses

Due to the loss of 19.5% of the sample of girls from baseline (n = 138) to the final follow-up 3 years later (n = 111), univariate analyses of non-response bias were conducted 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 families did not differ on variables used in this study.

Some participants were excluded from particular analyses because they did not have values for some variables. For analyses involving cortisol samples, some girls were excluded because their saliva samples were insufficient to assay cortisol, whereas others participated via phone interview and survey if they moved, and thus did not have a cortisol sample. For the exact number of reports for each variable see Table 3-1.

Descriptive Analyses

Descriptive information (i.e., M and SD) are shown in Table 3-1 and correlations among the variables of interest are shown in Table 3-2.

Contrary to the hypotheses, stress reactivity ($AUC_{cortisol}$) was not associated with internalizing distress or aggression ($r = .02$, ns, and $r = .12$, ns, respectively). In fact, only two variables demonstrated significant correlations with stress reactivity: pubertal timing ($r = .23$, $p < .05$), and involuntary engagement ($r = .27$, $p < .05$).

Pubertal timing also demonstrated significant associations with the outcomes. For the dichotomous pubertal timing variable (early vs. other), ANCOVAs (controlling for SES and race) were conducted, as the F-values, means, and standard deviations for the

effects may be more illuminating than correlations. As expected, girls who were early maturers reported more internalizing distress, $F(1, 104) = 4.83$, $p < .05$, with an average score of 6.63 ($SD = 4.76$) compared to other girls ($M = 4.57$, $SD = 4.44$), and more aggressive symptoms, $F(1, 104) = 4.54$, $p < .05$, with an average score of 8.77 ($SD = 6.39$) compared to other girls ($M = 6.29$, $SD = 4.96$).

Peer stress was positively associated with both internalizing distress ($r = .40$, $p < .01$) and aggression ($r = .23$, $p < .05$). Appraisal of the stressful experiences was positively associated with both peer stress ($r = .41$, $p < .01$) and internalizing distress ($r = .28$, $p \leq .05$).

As anticipated, the potential mediators of the association between biological and environmental indicators of social stress and adjustment demonstrated consistent associations with the outcomes of interest. Internalizing distress was positively associated with voluntary disengagement, involuntary engagement, and involuntary disengagement responses (Table 3-2). Additionally as predicted, internalizing distress was negatively associated with primary control strategies ($r = -.48$, $p \leq .01$), meaning that girls who used problem solving and emotional expression and regulation tactics to cope with peer stress reported fewer symptoms of anxiety and depression. The use of secondary control strategies, such as acceptance or cognitive restructuring, was also negatively associated ($r = -.45$, $p \leq .01$) with internalizing distress. This suggested that adolescent girls who attempted to manage stressful peer experiences by using positive self-talk or accepting these stressors as something from which to learn experienced fewer anxious and depressive symptoms than girls were less likely to use these strategies.

Similar to the findings for internalizing distress, aggression was positively correlated with involuntary engagement responses, and involuntary disengagement responses (Table 3-2). As hypothesized, primary control engagement coping and secondary control engagement coping were negatively correlated with aggression. The use of disengagement coping strategies, such as denial, avoidance, and wishful thinking, was not correlated with aggression.

Tests of Main Effects

As indicated, in order to test whether or not physiological and environmental indicators of social stress were associated with symptoms of internalizing distress and aggression, a series of hierarchical regression analyses were performed. All analyses controlled for race/ethnicity (African American versus other, Hispanic versus other, White as omitted group) and family SES.

Biological Response to Stress

Controlling for race and family SES, $AUC_{cortisol}$ was not significantly associated with internalizing distress, $F(4, 65) = .59$, $p = .67$, or aggression, $F(4, 65) = .33$, $p = .86$, meaning amount of total stress reactivity over the 5 points of cortisol assessment were not associated with symptoms of internalizing distress or aggression. Sample sizes for these analyses were much lower than the main group of participants (N=111). This occurred because in order to calculate an $AUC_{cortisol}$ score, participants needed to have 4 out of the 5 samples and were not missing sample 1 or sample 5, in addition to needing race, SES, internalizing distress, and aggression scores.

An unrestricted covariance structure was used for each model and all continuous between-person variables were centered on the grand mean. Because race and family SES were shown not to be significant predictors of $AUC_{cortisol}$, they were not included in the

mixed effect models. Researchers claim that including covariates that do not exhibit associations with the dependent variable can generate spurious significant associations in models, and thus it is recommended to exclude covariates from the analyses that would otherwise be included in typical regression and ANCOVA analyses (Rovine, von Eye, & Wood, 1988; Weisberg, 1979). One set of analyses was performed for each set of symptoms (i.e., internalizing distress and aggression), resulting in 2 sets of analyses. However, because internalizing distress and aggression showed not to be significant predictors of participants' patterns of change in cortisol, results from the 2 sets of analyses will be discussed in combination.

In Model 1 (see Tables 3-3 and 3-4), the variance in cortisol was partitioned into within- and between-person variance; 41.18% of the variance was due to within-person variability and 58.82% was due to between-person variability. In Models 2 and 3, fixed effects were added in a systematic fashion and pseudo R^2 –statistics were calculated to determine the amount of variance these models accounted for relative to the baseline model.

In Model 2, time was included as a level-1 predictor of cortisol. Results demonstrated that time was a significant predictor of cortisol levels and accounted for 43% of the within-person variance. In Model 3, the effects of internalizing distress (Table 3-3) or aggression (Table 3-4) were added. Both internalizing distress and aggression were not significant predictors and did not account for any between-person variance. In addition, the interactions between time and internalizing distress and time and aggression were not significantly associated with cortisol levels. Thus, although results showed that cortisol levels changed over the 5 in-home collections, symptoms of

psychopathology were not associated with how cortisol levels changed over time. These results corroborate the findings from the area under the curve analyses.

Pubertal Timing

To test whether or not pubertal timing was associated with symptoms of internalizing distress and aggression, a series of hierarchical regression models was performed (Table 3-5). Controlling for race/ethnicity and SES, pubertal timing was significantly associated with internalizing distress, such that girls who were early maturers demonstrated higher levels of internalizing distress compared to other girls. Controlling for race/ethnicity and SES, pubertal timing was also significantly associated with aggression, such that girls who reported early maturation demonstrated higher levels of aggression compared to other girls. These findings were consistent with bivariate correlations reported in Table 3-2.

Peer Stress

As expected, results indicated that peer stress was significantly associated with both internalizing distress and aggression (Tables 3-7 and 3-8). Controlling for race, SES, and appraisal, higher levels of peer stress were associated with greater symptoms of internalizing distress and aggression.

Tests of Mediation

As indicated, a series of regression models were performed to test effects of mediation as specified by Baron and Kenny (1986), with subsequent examination of tests of mediation as proposed by Preacher and Hayes (2006).

Pubertal Timing

Figure 3-2-1 shows the model testing mediated pathways of pubertal timing (early vs. other) to internalizing distress. The standardized β coefficients are shown in the

figures. In all, 5 tests of mediation (one for each mediator) were performed for internalizing distress. As can be seen in Figure 3-2-1, there was evidence for full mediation of the effect of timing on internalizing distress via primary control and involuntary engagement responses. As per specifications of Baron and Kenny (1986), the coefficient of pubertal timing predicting internalizing distress was reduced and was no longer significant when the mediators were included in the model (i.e., β for pubertal timing to internalizing distress, when mediated by primary control, was reduced from .20, $p < .10$, to .08, ns, and was reduced from .20, $p < .10$, to .11, ns, when mediated by involuntary engagement). Results from Preacher and Hayes (2006) test of the indirect effect found that the indirect effect (ab = .11) of pubertal timing on internalizing distress through primary control was estimated to lie between .03 and .27 with 95% confidence. Also, the indirect effect (ab = .10) of pubertal timing on internalizing distress through involuntary engagement was estimated to lie between .03 and .20 with 95% confidence. For both indirect effects, mediation was said to occur because the indirect effects (ab) were significantly different from zero.

The tests of mediation demonstrated that early pubertal timing predicted less use of primary control strategies which subsequently predicted fewer symptoms of internalizing distress. Conversely, early timing predicted greater use of involuntary engagement responses, which subsequently predicted greater symptoms of internalizing distress. Pubertal timing was not associated with secondary control strategies, voluntary disengagement strategies, and involuntary disengagement responses.

Potential mediation effects on the association between pubertal timing and aggression were not examined. Although initial analyses demonstrated that pubertal

timing was significantly associated with aggression (Table 3-6), analyses for tests of mediation demonstrated that pubertal timing did not demonstrate a significant association with aggression ($\beta = .15$, $p = .14$). In the tests of mediation, listwise deletion was utilized, thus excluding participants who did not have scores for all the necessary variables needed to illustrate mediation (i.e., pubertal timing, internalizing distress, coping scores, and all covariates). This difference in analysis between the general regression and the tests of mediation accounts for the emergence of a significant association between pubertal timing and aggression in the original regression analysis and the non-significant association that emerged in the test of mediation.

Peer Stress

Figure 3-2 shows the model testing mediated pathways of peer stress to internalizing distress; and Figure 3-3 shows the model examining the association between peer stress and aggression. As shown in Figure 3-2 and 4, peer stress was positively associated with voluntary disengagement; however, because voluntary engagement strategies were not associated with either internalizing distress or aggression, mediation effects did not emerge. Peer stress was not associated with involuntary engagement and disengagement responses.

Two mediation effects emerged for both internalizing distress and aggression. As can be seen in Figure 3-2, there was evidence for partial mediation of the effect of peer stress on internalizing distress via primary control and secondary control strategies. The coefficient of peer stress predicting internalizing distress was reduced and was no longer significant when the mediators were included in the model (i.e., β for peer stress to internalizing distress, when mediated by primary control, was reduced from .35, $p < .01$, to .26, $p < .05$, and was reduced from .35, $p < .01$, to .25, $p < .05$, when mediated by

secondary control). Noteworthy was the fact that the association between peer stress and primary control was only a trend. However, using 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 = .10, CI (.02, .21), and for secondary control strategies, ab = .10, CI (.01, .23).

Figure 3-3 shows evidence for full and partial mediation of the effect of peer stress on aggression via primary control and secondary control strategies, respectively. The coefficient of peer stress predicting aggression was reduced and was no longer significant when the mediators were included in the model (i.e., β for peer stress to aggression, when mediated by primary control, was reduced from .25, $p < .05$, to .20, $p < .10$, and was reduced from .25, $p < .05$, to .17, $p = .13$, when mediated by secondary control). Supporting these findings, ab was significantly different from zero with 95% confidence for primary control, ab = .06, CI (.01, .16), and for secondary control strategies, ab = .08, CI (.01, .24). For tests of the indirect effect, ab was significantly different from zero, which demonstrated mediation. This means that greater peer stress predicted less use of primary and secondary control strategies which subsequently predicted fewer symptoms of internalizing distress and aggression.

Table 3-1 Means and standard deviations of adolescents' demographic information, health, stress, coping, and psychological symptoms

Variable	*N*	*M*	*SD*
Age	111	11.84	.78
SES	110	33.07	16.72
Cortisol sample 1	89	.17	.16
Cortisol sample 2	84	.14	.16
Cortisol sample 3	80	.12	.17
Cortisol sample 4	71	.09	.12
Cortisol sample 5	73	.10	.14
Cortisol AUC	72	9.72	10.20
Age of menarche	46	11.26	.87
Peer stress	98	2.60	1.96
Degree of hassle	96	2.16	1.00
Primary control	101	.23	.05
Secondary control	100	.13	.03
Effortful disengagement	101	.24	.05
Involuntary engagement	100	.20	.04
Involuntary disengagement	100	.19	.04
Internalizing distress	109	5.21	4.62
Aggression	109	7.07	5.54

Note. Coping and stress responses are reported as proportional scores.

Table 3-2 Intercorrelations among demographic information, health, stress, coping, and psychological symptoms

	1	2	3	4	5	6	7	8	9	10	11	12
1. SES	---											
2. Cortisol AUC	-.02	---										
3. Pubertal Timing	-.06	.23*	---									
4. Peer Stress	.05	-.05	.02	---								
5. Degree of Hassle	.16	.06	-.09	.41**	---							
6. Primary Control	.11	-.13	-.22*	-.23*	-.18†	---						
7. Secondary Control	-.03	.20	-.05	-.27**	-.20†	.48**	---					
8. Vol. Disengagement	-.05	-.09	.07	.19	.04	-.59**	-.26**	---				
9. Inv. Engagement	.03	.27*	.21*	.17	.26*	-.44**	-.57**	-.19†	---			
10. Inv. Disengagement	-.08	-.11	.06	.13	.10	-.62**	-.64**	.01	.35**	---		
11. Internalizing Distress	.10	.02	.21*	.40**	.28**	-.48**	-.45**	.19†	.40**	.42**	---	
12. Aggression	-.04	.12	.21*	.23*	.13	-.34**	-.41**	-.05	.46**	.43**	.56**	---

Note. † $p < .10$; * $p < .05$; ** $p < .01$

Table 3-3 Mixed effects models examining the association of internalizing distress and cortisol

	Parameter	Model 1	Model 2	Model 3
Fixed Effects				
Initial status				
Intercept	γ_{00}	-1.12***	-0.96***	-0.96***
		(0.04)	(0.04)	(0.04)
Internalizing Distress	γ_{01}			-.002
				(0.01)
Rate of change				
Intercept	γ_{10}		-0.09***	-0.09***
			(0.01)	(0.01)
Time X Internalizing	γ_{11}			-0.003
Distress				(0.002)
Variance Components				
Level 1				
Within-person residual	e_{ij}	0.07***	0.04***	0.04***
		(0.02)	(0.004)	(0.004)
Level 2				
In initial status	μ_{0j}	0.10***	0.11***	0.12***
		(0.65)	(0.02)	(0.02)
In rate of change	μ_{1j}		0.006***	0.005***
			(0.002)	(0.002)
Pseudo R^2 and Goodness of Fit				
R^2_{ε} (within person variance)			0.43	0.00
R^2_0 (variance in initial status)			0.00	0.00
R^2_1 (variance in rate of change)			0.00	0.17
Deviance (-2LL)		260.4	135.1	153.4
ΔDeviance (-2LL)			125.3***	-18.3

Note. *** $p < .001$

Table 3-4 Mixed effects models examining the association of aggression and cortisol

	Parameter	Model 1	Model 2	Model 3
Fixed Effects				
Initial status				
Intercept	γ_{00}	-1.12***	-0.96***	-0.96***
		(0.04)	(0.04)	(0.04)
Aggression	γ_{01}			-.002
				(0.01)
Rate of change				
Intercept	γ_{10}		-0.09***	-0.09***
			(0.01)	(0.01)
Time X Aggression	γ_{11}			-0.003
				(0.002)
Variance Components				
Level 1				
Within-person residual	e_{ij}	0.07***	0.04***	0.04***
		(0.02)	(0.004)	(0.004)
Level 2				
In initial status	μ_{0j}	0.10***	0.11***	0.12***
		(0.65)	(0.02)	(0.02)
In rate of change	μ_{1j}		0.006***	0.005***
			(0.002)	(0.002)
Pseudo R^2 and Goodness of Fit				
R^2_{ε} (within person variance)			0.43	0.00
R^2_0 (variance in initial status)			0.00	0.00
R^2_1 (variance in rate of change)			0.00	0.17
Deviance (-2LL)		260.4	135.1	155.3
ΔDeviance (-2LL)			125.3***	-20.2

Note. *** $p < .001$

Table 3-5 Pubertal timing predicting internalizing distress

Variable	R^2	ΔR^2	*B*	*SE B*	β
Step 1	.006				
African American			-.10	.99	-.01
Latina			-.07	1.30	-.01
Family SES			.02	.03	.08
Step 2	.051*	.045*			
African American			-.19	.97	-.02
Latina			-.21	1.28	-.02
Family SES			.03	.03	.09
Pubertal Timing			2.12	.95	.21*

Note. * $p < .05$

Table 3-6 Pubertal timing predicting aggression

Variable	R^2	ΔR^2	*B*	*SE B*	β
Step 1	.028				
African American			1.06	1.17	.09
Latina			-.84	1.55	-.06
Family SES			-.04	.04	-.11
Step 2	.068*	.04*			
African American			.95	1.15	.08
Latina			-1.00	1.52	-.07
Family SES			-.04	.04	-.10
Pubertal Timing			2.40	1.13	.20*

Note. * $p < .05$

Table 3-7 Peer stress predicting internalizing distress

Variable	R^2	ΔR^2	B	$SE\ B$	β
Step 1	.08				
African American			.07	1.08	.01
Latina			.48	1.41	.04
Family SES			.01	.03	.03
Degree of Hassle			1.29	.47	.28**
Step 2	.173**	.094**			
African American			.08	1.02	.01
Latina			.50	1.35	.04
Family SES			.01	.03	.03
Degree of Hassle			.64	.51	.14
Peer Stress			.81	.26	.38**

Note. ** $p < .01$

Table 3-8 Peer stress predicting aggression

Variable	R^2	ΔR^2	*B*	*SE B*	β
Step 1	.031				
African American			.72	1.26	.06
Latina			-.15	1.66	-.01
Family SES			-.03	.04	-.09
Degree of Hassle			.75	.57	.14
Step 2	.082*	.051*			
African American			.73	1.23	.06
Latina			-.13	1.62	-.01
Family SES			-.03	.03	-.09
Degree of Hassle			.20	.61	.04
Peer Stress			.68	.31	.25*

Note. * $p < .05$

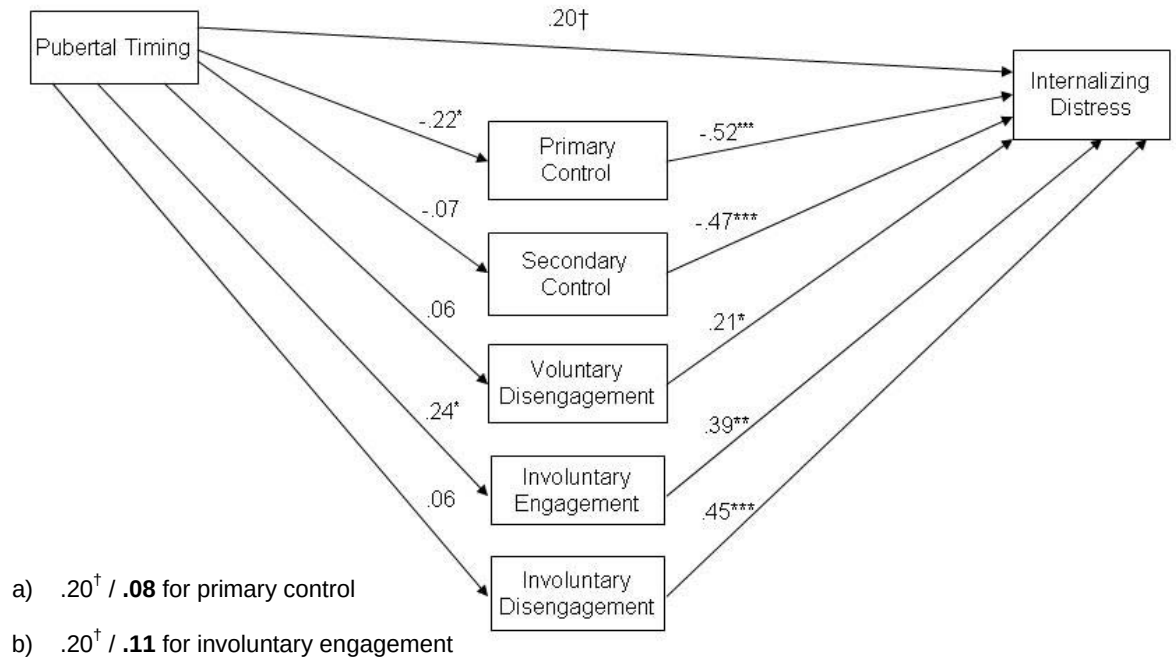
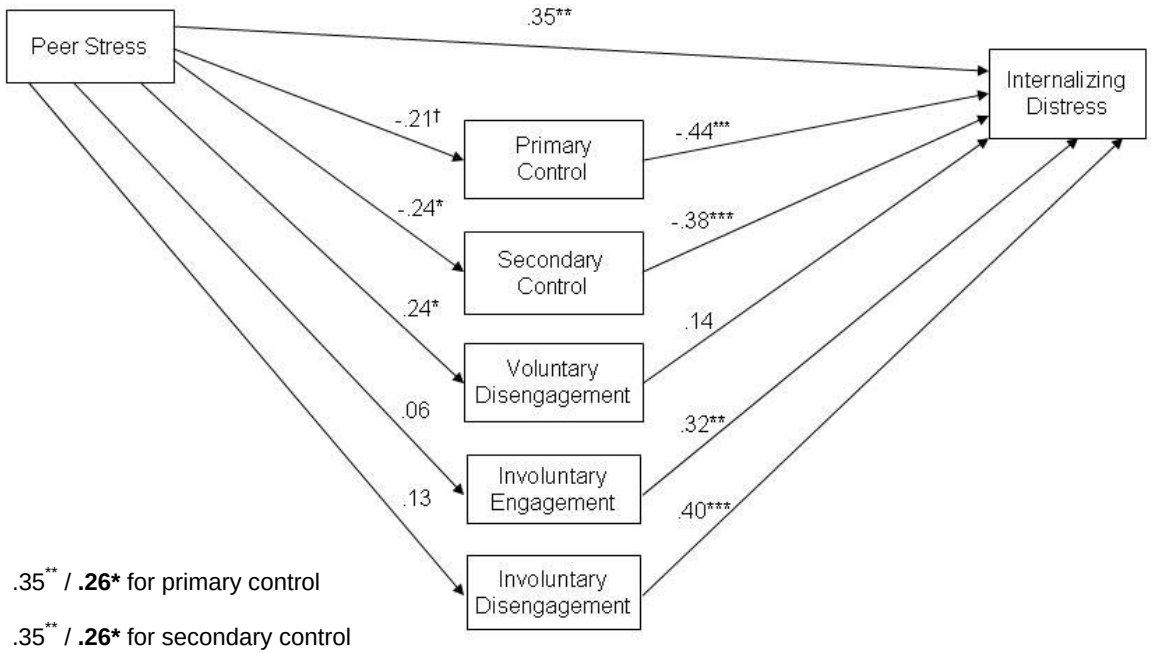


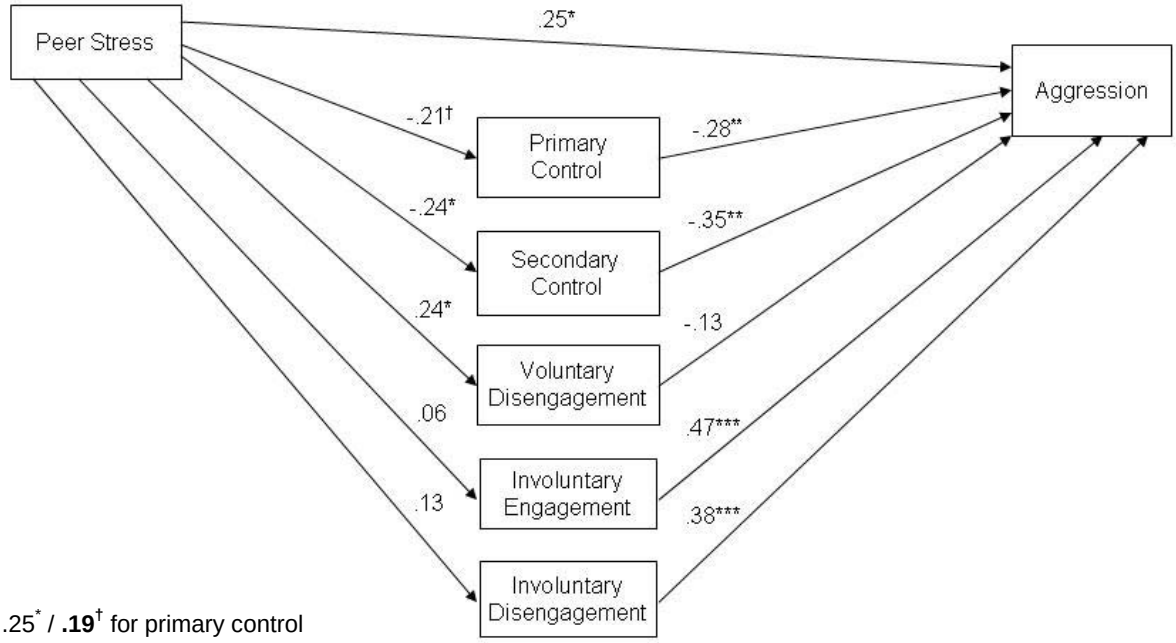
Figure 3-1. Path model for pubertal timing to internalizing distress. Each pathway controls for race and family SES. At the top of the model, the β value was calculated for the pathway between pubertal timing and internalizing distress. On the left side of the model, the β values were calculated separately for each pathway for pubertal timing to each potential mediator. The β values on the right side of the model were calculated with all variables (covariates, predictors, and mediators) entered simultaneously into the model. (a) On the right side, change in β values for pathway between pubertal timing and internalizing distress when primary control is simultaneously entered; (a) On the right side, change in β values for pathway between pubertal timing and internalizing distress when involuntary engagement is simultaneously entered. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$



a) .35** / **.26*** for primary control

b) .35** / **.26*** for secondary control

Figure 3-2. Path model for peer stress to internalizing distress. Each pathway controls for race, family SES, and degree of hassle. At the top of the model, the β value was calculated for the pathway between peer stress and internalizing distress. On the left side of the model, the β values were calculated separately for each pathway for peer stress to each potential mediator. The β values on the right side of the model were calculated with all variables (covariates, predictors, and mediators) entered simultaneously into the model. (a) On the right side, change in β values for pathway between peer stress and internalizing distress when primary control is simultaneously entered; (b) On the right side, change in β values for pathway between peer stress and internalizing distress when secondary control is simultaneously entered. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$



a) .25* / **.19**† for primary control

b) .25* / **.17** for secondary control

Figure 3-3. Path model for peer stress to aggression. Each pathway controls for race, family SES, and degree of hassle. At the top of the model, the β value was calculated for the pathway between peer stress and aggression. On the left side of the model, the β values were calculated separately for each pathway for peer stress to each potential mediator. The β values on the right side of the model were calculated with all variables (covariates, predictors, and mediators) entered simultaneously into the model. (a) On the right side, change in β values for pathway between peer stress and aggression when primary control is simultaneously entered; (b) On the right side, change in β values for pathway between peer stress and aggression when secondary control is simultaneously entered. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

CHAPTER 4
DISCUSSION

Although much is known about stress and coping during adolescence, few studies have examined the influence of coping strategies on the relationship between social stress and adjustment. More specifically, relatively little is known as to how adolescents cope with being "off-time" with respect to puberty, and how responses to being off-time influence psychosocial outcomes (Compas et al., 1999; Connor-Smith et al., 2000; Ge et al., 2001; Jaser et al., 2005; Klimes-Dougan et al., 2001). The goal of this study was to examine the association of biological and psychosocial indicators of stress and 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 negative outcomes during adolescence.

This study examined the effect of peer stress on adjustment and why the impact of peer stress on adjustment varies among young adolescent girls. Consistent with prior research, girls who experienced more peer stress reported higher levels of internalizing distress and aggression (Deater-Deckard, 2001; Nansel et al., 2004; Rudolph, 2002). Moreover, as expected, coping strategies, specifically primary and secondary control strategies, mediated both of these associations. Specifically, girls who experienced more peer stress used fewer primary and secondary control strategies, which are typically considered to be adaptive and effective means of coping, such as problem solving, positive thinking, emotional regulation, etc. This association, in turn, accounted for greater levels of internalizing distress and aggression. Recently 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 or examined how coping and stress responses moderated the association between stressful life events and adjustment

(Connor-Smith, et al., Hampel & Petermann, 2006). Building upon prior research, the present study focused specifically on peer stress and demonstrated that how an adolescent responds to peer stress *mediates* the association between stressful experiences and adjustment during adolescence. These findings are important because they provide a more thorough understanding of how stressful experiences with peers impact adolescents' psychosocial well-being.

This study also examined the effect of pubertal timing on adjustment. As anticipated, girls who were early maturers, compared to those who were on-time or late, demonstrated higher levels of internalizing distress and aggression. This finding is consistent with prior research that has demonstrated that experiencing puberty earlier than one's peers may place girls at a heightened risk for a variety of behavioral and emotional problems (Graber, 2003; Weichold, Silbereisen, & Schmitt-Rodermund, 2003). However, based on findings from this study, this association may be partially accounted for by the way girls deal with being early maturers. The use of primary control strategies (i.e., emotional expression, emotional regulation, and problem solving) has been shown to attenuate the effects of stressful experiences, whereas, the use 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, Raviv, Compas, & Connor-Smith, 2005). In this study, these two responses to stress mediated the association between early pubertal timing and internalizing distress. Meaning, early maturers used fewer primary control strategies and more involuntary engagement strategies, which in turn accounted for greater levels of internalizing distress.

Although main effects of pubertal timing were demonstrated for both internalizing distress and aggression, these effects were reduced in the full regression (i.e., when stress response factors were included). The main effect of pubertal timing on internalizing distress was reduced,

but remained statistically significant, thus allowing for mediation effects of stress responses to be examined, as recommended by Baron and Kenny (1986). However, the main effect of pubertal timing on aggression was reduced to the point that it was no longer significant, hindering tests of mediation. According to Baron and Kenny (1986), mediation effects cannot be tested if a significant effect between the independent variable (pubertal timing) and the dependent variable (aggression) does not exist. The drop in significance was likely due to the exclusion of participants that did not have responses to stress scores. Five of these participants were early maturers and 5 of them were on-time or late. Proportionally, there was a greater drop in the number of early maturers included in the analyses. For this reason, it likely became more difficult to demonstrate significance. Because the inability to test mediating effects of coping and stress responses on the association between pubertal timing and aggression was likely due to an attrition effect, future research should revisit this issue in order to demonstrate whether or not using particular coping strategies benefits early maturers.

Although an abundance of research exploring the relationship between pubertal timing and maladjustment exists, few studies have attempted to explore exactly *how* pubertal timing relates to adjustment and how girls cope with the social and emotional ramifications of being earlier than their peers (Brooks-Gunn et al., 1994; Caspi et al., 1993; Ge et al., 2002; Graber et al., 2005). The findings from this study are important because they demonstrate that some of the association between early timing and adjustment may be accounted for by how adolescents cope with or react to the stress of being "off-time."

Although examining the influence of individual coping strategies and stress responses is useful and informative, future research exploring coping and peer stress should also examine coping *styles* and how they impact adjustment. Recent research exploring coping has suggested

that individuals have a tendency to respond to stress using a particular set of coping strategies and involuntary responses rather than responding with one strategy (Reinhard, Wolff, & Wadsworth, 2006). Research that focuses on one type of stress response may be missing the big picture of how individuals deal with stress. Thus, it may be informative to understand how these different combinations of strategies (i.e., coping styles) either attenuate or exaggerate the effects of peer stress.

Contrary to expectation, this study failed to demonstrate an association between stress reactivity and symptoms of internalizing distress and aggression; as a result, potential mediating effects of coping strategies could not be explored. This study found no connection between *how* cortisol levels changed over the course of a home visit (that included a variety of tasks) and symptoms of psychopathology. Much of the current literature has found associations between cortisol reactivity and psychopathology, but mostly 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 stress reactivity and internalizing symptoms, but were unable to find clear associations between stress reactivity and aggression. It was only until adolescents displayed clinical levels of aggression that a significant association with stress reactivity emerged. Thus, because most of the girls in this study displayed low or at most marginally high levels of internalizing distress and aggression, associations between stress reactivity and symptoms of psychopathology may not have emerged. In turn, further research examining adrenocortical activity in normative adolescent samples should be conducted in order to provide

a more thorough understanding of whether or not a reliable connection between stress reactivity and symptoms of psychopathology exists.

Some of the inconsistency in findings may be due to analytic differences. Most studies examining stress reactivity in child and adolescent samples utilize reactivity categories (i.e., increased dramatically, remained stable, decreased slightly, etc.) to explore stress reactivity and adjustment (Granger, et al., 1994; Klimes-Dougan, et al., 2001; Schmidt et al., 1999). This study utilized area under the curve and mixed modeling to explore patterns of change. There exists a debate as to what method is most appropriate to utilize when exploring adrenocortical activity and adjustment. Thus further research exploring the method of AUC and mixed modeling in different age groups of both boys and girls is necessary to understand the implications of using these methods of analysis.

This study has taken informative steps in understanding better how social stress influences psychosocial development; however, some limitations of the study exist. The examination of stress and coping was based solely on adolescent girls, rather than both boys and girls. Although researchers have suggested that social stress may be more detrimental to adolescent girls' psychosocial well-being (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, and whether particular coping strategies are more problematic for one gender versus the other. In addition, the study focused on early adolescence, particularly 12 year olds, which prevented exploration of developmental changes in coping and stress responses over the course of adolescence and the impact of late pubertal timing. Additionally, due to the fact that data were only collected at a single time point, the direction of associations between social stress, coping, and psychopathology were inferred from theoretical perspectives and previous research

exploring stress, coping and adjustment. Thus, results should be interpreted with some caution. For example, it is unclear if girls who utilize particular types of coping strategies such as primary and secondary control are more socially skilled and experience less peer stress. A final limitation was the fact that participants were drawn from a large metropolitan area, limiting generalization to adolescents in suburban and rural areas.

Despite the limitations, results of this study provide useful information regarding the identification of specific coping strategies and stress responses that mediate the relationship between stress and internalizing distress, and aggression. These findings speak to skills that could be taught in programs that emphasize how to effectively adapt to the social and psychological demands of transitioning into adolescence. Although the present study focuses on negative outcomes, potential also exists to understand better what factors promote positive development (i.e., prosocial behavior, academic success, positive self-esteem) in the face of stressful social situations. Understanding these factors and how they affect psychosocial development may ultimately expand our current understanding of resilience in the face of stress.

APPENDIX A
HOME DATA COLLECTION PROCEDURE AND SALIVA SAMPLE COLLECTION

1. Consents

2. Specimen and questionnaire delivery details

3. Saliva #1

4. Maze task

5. Neighborhood/peer questions

6. Saliva #2

7. Cold pressor task #1

8. Cold pressor questions

9. Cold pressor task #2

10. Cold pressor questions

11. Disagreement Questions

12. Saliva #3

13. Daily routines/ nutrition/ sleep questions

14. Physical exam

15. Menstruation & social activities questions

16. Saliva #4

17. Peer issues interaction task

18. Disagreement interaction task

19. Saliva #5

APPENDIX B
MIXED EFFECTS MODELS EXAMINING THE IMPACT OF TIME AND SYMPTOMS OF PSYCHOPATHOLOGY ON PATTERNS OF CORTISOL PRODUCTION

Table B1. Impact of time and internalizing distress on patterns of cortisol

Model	Level-1 Model	Level-2 Model	Composite Model
A	$Cortisol_{ij} = \pi_{oi} + e_{ij}$	$\pi_{0i} = \gamma_{00} + \mu_{0j}$	$Cortisol_{it} = \gamma_{00} + (e_{ij} + \mu_{0j})$
B	$Cortisol_{ij} = \pi_{oi} + \pi_{1i} Time_{ij} + e_{ij}$	$\pi_{0i} = \gamma_{00} + \mu_{0j}$	$Cortisol_{it} = \gamma_{00} + \gamma_{10} Time_{ij} +$
		$\pi_{1i} = \gamma_{10} + \mu_{1j}$	$e_{ij} + \mu_{0j} + \mu_{1j} Time_{ij})$
C	$Cortisol_{ij} = \pi_{oi} + \pi_{1i} Time_{ij} + e_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}(Anx/Dep - \overline{Anx/Dep}) + \mu_{0j}$	$Cortisol_{it} = \gamma_{00} + \gamma_{10} Time_{ij} +$
		$\pi_{1i} = \gamma_{10} + \gamma_{11}(Anx/Dep - \overline{Anx/Dep}) + \mu_{0j}$	$\gamma_{01}[(Anx/Dep - \overline{Anx/Dep})(Time_{ij}) +$
			$(e_{ij} + \mu_{0j} + \mu_{1j} Time_{ij})$

Note. (A) unconditional means model (partitions variance into between and within variance); (B) unconditional growth model (do cortisol levels vary over time); (C) examines effect of internalizing distress on initial status and rate of change of cortisol

Table B2. Impact of time and aggression on patterns of cortisol

Model	Level-1 Model	Level-2 Model	Composite Model
A	$Cortisol_{ij} = \pi_{oi} + e_{ij}$	$\pi_{0i} = \gamma_{00} + \mu_{0j}$	$Cortisol_{it} = \gamma_{00} + (e_{ij} + \mu_{0j})$
B	$Cortisol_{ij} = \pi_{oi} + \pi_{1i} Time_{ij} + e_{ij}$	$\pi_{0i} = \gamma_{00} + \mu_{0j}$	$Cortisol_{it} = \gamma_{00} + \gamma_{10} Time_{ij} +$
		$\pi_{1i} = \gamma_{10} + \mu_{1j}$	$(e_{ij} + \mu_{0j} + \mu_{1j} Time_{ij}$
C	$Cortisol_{ij} = \pi_{oi} + \pi_{1i} Time_{ij} + e_{ij}$	$\pi_{0i} = \gamma_{00} + \gamma_{01}(Agg - \overline{Agg}) + \mu_{0j}$	$Cortisol_{it} = \gamma_{00} + \gamma_{10} Time_{ij} +$
		$\pi_{1i} = \gamma_{10} + \gamma_{11}(Agg - \overline{Agg}) + \mu_{1ij}$	$\gamma_{01}[(Agg - \overline{Agg})(Time_{ij}) +$
			$e_{ij} + \mu_{0j} + \mu_{1j} Time_{ij})$

Note. (A) unconditional means model (partitions variance into between and within variance); (B) unconditional growth model (do cortisol levels vary over time); (C) examines effect of aggression on initial status and rate of change of cortisol

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