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Marital Conflict and Pubertal Timing: Stress and Security as Mediators of Associations

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Marital Conflict and Pubertal Timing: Stress and Security as Mediators of Associations

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THESIS

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A thesis submitted in partial fulfillment of the
requirements for the degree of Master of Science in the
College of Arts and Sciences at the University of Kentucky

By

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Lexington, Kentucky

Director: Dr. Peggy S. Keller, Professor of Psychology

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ABSTRACT OF THESIS

Marital Conflict and Pubertal Timing: Stress and Security as Mediators of Associations

The timing of pubertal development has important mental and physical health consequences. Individuals who enter puberty off-time are at greater risk for psychological disorders, social difficulties, and physical morbidity. One variable associated with early pubertal development is marital conflict. Life History Theory proposes that marital conflict signals an unreliable environment and promotes advanced pubertal timing to enhance reproductive fitness. Such calibrations allow individuals to unconsciously invest more resources in reproduction, following a quantity over quality approach. Despite research supporting the role of marital conflict in early-onset puberty, research has struggled to find a mechanism for this relationship. The current study examined two possible mediators: emotional insecurity and cortisol levels in a sample of children aged 6-12 years from 2-parent families. Neither variable was supported as a mediator of this relationship. However, parental depression significantly predicted pubertal development for girls. Findings regarding the role of parental depression in the timing of girl's puberty support life history theory.

KEYWORDS: Emotional Security, Life History, Marital Conflict, Pubertal Timing

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__10/31/2013_____

Marital Conflict and Pubertal Timing: Stress and Security as Mediators of Associations

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Chapter One: Introduction

Consequences and Determinants of Off-time Pubertal Development

Early-onset of pubertal development has been associated with reduced self-esteem, increased rates of depression, and higher incidence of body image concerns and eating disorders in girls (Manuck et al., 2011). Social maladjustment is also more common among early developing girls, including increased drug use, school absence, earlier initiation of sexual intercourse, higher rates of teen pregnancy, and more deviant peer associations (Caspi & Moffitt, 1991; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Lanza & Collins, 2002; Manlove, 1997; Mezzich et al., 1997; Phinney, Jenson, Olsen, & Cundick, 1990; Simmons & Blyth, 1987; Stice, Presnell, & Bearman, 2001). Physical morbidities of early maturation include breast cancer, insulin insensitivity, uterine fibroid tumors, and all-cause mortality. For males, both early and late pubertal development is associated with psychopathology, school difficulties, and substance use, although these associations are less strong than those found for females (Archibald, Graber, & Brooks-Gunn, 2006). Improved understanding of environmental factors contributing to early pubertal timing is therefore critical.

A prominent explanation for pubertal timing involves body composition. Pubertal onset in females requires attainment of a high enough body fat percentage and the release of associated hormones, such as leptin (Archibald, Graber, & Brooks-Gunn, 2006). This has been suggested as both a source of individual differences as well as the "secular trend" of earlier onset puberty in females (Archibald, Graber, & Brooks-Gunn, 2006). In modern industrial societies, girls may obtain the required amount of body fat earlier in life, leaving puberty to be genetically determined. In this view, the environment plays

little to no role in determining the timing of puberty. As long as an individual can obtain the necessary adiposity, they will enter puberty at a genetically programmed time.

Although genetic factors have a strong influence on the timing of pubertal development, they do not explain all of the variance in pubertal timing. Psychosocial stress, particularly family structure and conflict, also appears to play a role. Steinberg (1988) hypothesized that emotional distance from parents led to earlier pubertal development. This distance was defined largely as a lack of monitoring and high rates of intense conflict. Maternal harshness predicts earlier onset of menarche for girls, even when controlling for genetics via mother's age at menarche; no significant effects were found for parenting practices on male pubertal timing in this study (Belsky et al., 2007). Kim and Smith (1999) found that marital conflict exposure and less parental monitoring were associated with earlier onset of menarche, and lower levels of closeness to mothers were associated with earlier spermarche. Along with marital conflict, father absence is one of the most consistent variables associated with early onset of menarche, possibly because it represents an extreme form of marital discord (Saxbe & Repetti, 2008).

Life History Theory and the Timing of Puberty

Why should the family environment play a role in determining pubertal timing? One possible answer can be found in Life History Theory (LHT), an evolutionary psychology theory. LHT proposes two possible reproductive strategies for pubertal timing: a slow, quality-oriented strategy and a fast, quantity-oriented approach (Del Giudice, 2009). In a hostile environment where individuals are at risk of dying young and unlikely to obtain secure parenting investment from mates, it would be adaptive to produce as many offspring as possible and to spend fewer resources on parenting than on

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reproduction. Early pubertal onset would serve this goal. On the other hand, if the environment is stable and parenting investments are likely, it is optimal for fitness to produce fewer, higher-quality offspring, and later pubertal timing would support this strategy. Psychosocial stressors in the family may represent a hostile environment that drives ontogeny towards a fast life history strategy (Belsky, Steinberg, & Draper, 1991). The family is one of the most salient sources of information regarding the stability of the environment and the likelihood of parental investment. For example, if an individual is raised without a father, he or she may expect less parental investment from future mates.

LHT has received significant empirical support. Koehler and Chisholm (2009) found that men who experienced high levels of early environmental stress were more likely to have more short-term relationships and a greater number of past sexual partners. Results of the study were less clear for female participants. LHT is also supported by documented associations between early menarche and earlier onset of sexual activity and higher rates of teen pregnancy among at-risk adolescents (Dunbar, Sheeder, Lezotte, Dabelea & Stevens-Simon, 2008; Kim & Smith, 1999). Thus, early pubertal timing is likely to produce more children across the lifespan. Interestingly, women with earlier maturation report that they will die sooner than their slower developing peers; consistent with the LHT prediction that early maturation is due to experiences of the world as dangerous (Chisholm, Quinlivan, Peterson, & Coall, 2005).

Physiological Stress Response and Puberty

Direct evidence for a mechanism through which environmental stress is associated with pubertal timing has generally been lacking. One possible explanation involves the psychophysiological response to stress, particularly the hypothalamic-pituitary-adrenal

axis (HPA). The HPA axis is one of the body's primary systems for responding to social stress (Chrousos & Gold, 1992), and HPA activity has implications for health that are relevant to reproduction. The primary effector molecules of the HPA axis are corticosteroids, such as cortisol (Loman and Gunnar, 2010). Cortisol is secreted in response to stress and serves to release energy to help the body meet the demands of stressors (Saplosky, Romero, & Munck, 2000). While the release of cortisol in the face of stress is adaptive, chronically altered cortisol levels may lead to dysregulation and maladaptive outcomes. Hypercortisolism has been posited as a biological mechanism by which environmental stress may trigger an earlier onset of puberty (Netherton, Goodyer, Tamplin, & Herbert, 2004).

Cortisol relates to BMI in a complex manner. Short-term increases in cortisol lead to increased lipolysis (Sapolsky, Romero, & Munck, 2000). However, HPA dysregulation and chronic stress are consistently associated with increased BMI (Spencer, & TIlbrook, 2011). Children raised in stressful families have a higher likelihood of obesity (Koch et al., 2008). HPA dysregulation appears to increase BMI through altered feeding habits, as well as altering energy storage processes within the body. As Peckett and colleagues (2011) explain, the immediate lipolytic properties of cortisol help the body prepare to meet the demands of stress. Stress creates increased metabolic demands on many body tissues, requiring the release of stored energy. Following this initial release of energy, the body should be motivated to increase food intake to replace lost energy stores. This leads to increased preference for comfort foods that are high in fat and sugar, as demonstrated in the laboratory following a Tier Social Stress Test (TSST). Participants showing higher cortisol responses to the TSST had the highest preferences for the "comfort" food (Epel,

Lapidus, McEwen, & Brownell, 2001). Treatment with cortisol leads to increased food

intake in humans, with the amount consumed influenced by the magnitude of the cortisol

response (George, Khan, Briggs, & Abelson, 2010).

The quality of family interactions is associated with HPA functioning in children.

Levine (2005) hypothesized that normative HPA development required species-

normative parenting. Violations of these norms could therefore lead to HPA

dysregulation. Indeed, early childhood neglect is associated with higher morning cortisol

levels among internationally adopted children (Kertes, Gunnar, Madsen, & Long, 2008).

However, children placed in foster care for neglect exhibit lower morning cortisol levels

(Carlson, & Earls, 1997). HPA dysregulation is an important biological consequence of

early childhood maltreatment (Cicchetti, Rogosch, Gunnar, & Toth, 2010). Less extreme

adversity has also been associated with altered HPA functioning—typically, higher levels

of cortisol. Children of depressed or anxious parents show a heightened morning cortisol

response (Vreeburg et al., 2010). Inter-parental conflict has also been associated with

increased cortisol levels in children (Davies, Sturges-Apple, Cicchetti, & Cummings,

2008). Thus, family stress is related to maladaptive patterns of HPA axis activity, and

higher cortisol levels may explain why family adversity is associated with early pubertal

timing

Attachment Quality and Pubertal Timing

Another possible mediator is attachment security. Del Giudice (2009)

reconceptualized Attachment Theory in relation to LHT. He argues that the attachment

system allows individuals to unconsciously gauge the safety of the environment, with

potential consequences for mating strategies. Since secure attachment develops in the

context of consistent, sensitive care-giving, it is hypothesized to lead to a slower Life History strategy and later onset of puberty. In contrast, harsh and inconsistent parenting would lead to insecure attachment and a quick Life History strategy. In this conceptualization, one use of the attachment system is as a mechanism through which individuals gauge mortality risk and the availability of parental investments. Del Giudice's hypothesis is logical from an evolutionary perspective, but empirical support for it has been inconsistent (Bakersman-Kranenburg & van Ijzendoorn, 2009).

Emotional Security Theory

In addition to attachment security, children's emotional security about the marital relationship may play a key role in the timing of puberty. Emotional Security Theory (Cummings & Davies, 1994) proposes that children derive a sense of security from the stability of their parents' relationship. When exposed to severe inter-parental conflict, children display patterns of self-regulation that reflect their resulting emotional insecurity (Cummings & Keller, 2006). Children's insecurity in the interparental relationship is reflected through behavioral regulation of conflict exposure, emotional reactivity to conflict, and cognitive representations of the family as unstable. These reactions serve the short-term purpose of maintaining vigilance to potential threats to the family and reducing exposure to conflict when it occurs. Over the long term, however, emotional insecurity has been associated with increased internalizing and externalizing problems (Cummings & Davies, 2010), similar to children who enter puberty early. It is therefore proposed that children's emotional insecurity about the marital relationship will mediate associations between marital conflict and early pubertal timing.

The Present Study

The present study attempts to advance the existing literature on the environmental impact of pubertal timing by providing one of the first known tests of processes accounting for associations between family conflict and early pubertal timing. Figures 1-3 show the proposed models. Marital conflict was hypothesized to be related to early pubertal timing (Hypothesis 1; H1). This hypothesis was a replication of earlier research (Saxbe & Repetti, 2008). It was proposed that this association is mediated by emotional insecurity about the marital relationship and higher cortisol levels. Links between marital conflict and emotional insecurity are well-established (Cummings & Davies, 2010). Importantly, previous research has linked both marital conflict and parenting factors to changes in cortisol reactivity (Davies, Sturges-Apple, Cicchetti, & Cummings, 2008; Bugental, Martorell, & Barraza, 2003). These findings suggest a path from marital conflict to reduced emotional security and increased cortisol, leading to earlier onset puberty. Hypothesis two (H2) was that emotional insecurity would mediate the association between marital conflict and child pubertal development and between marital conflict and cortisol levels. Hypothesis three (H3) was that cortisol would mediate the association between marital conflict and child pubertal development, and between child emotional insecurity and pubertal development.

Figure 1
Child Report of Marital Conflict Predicting Pubertal Development
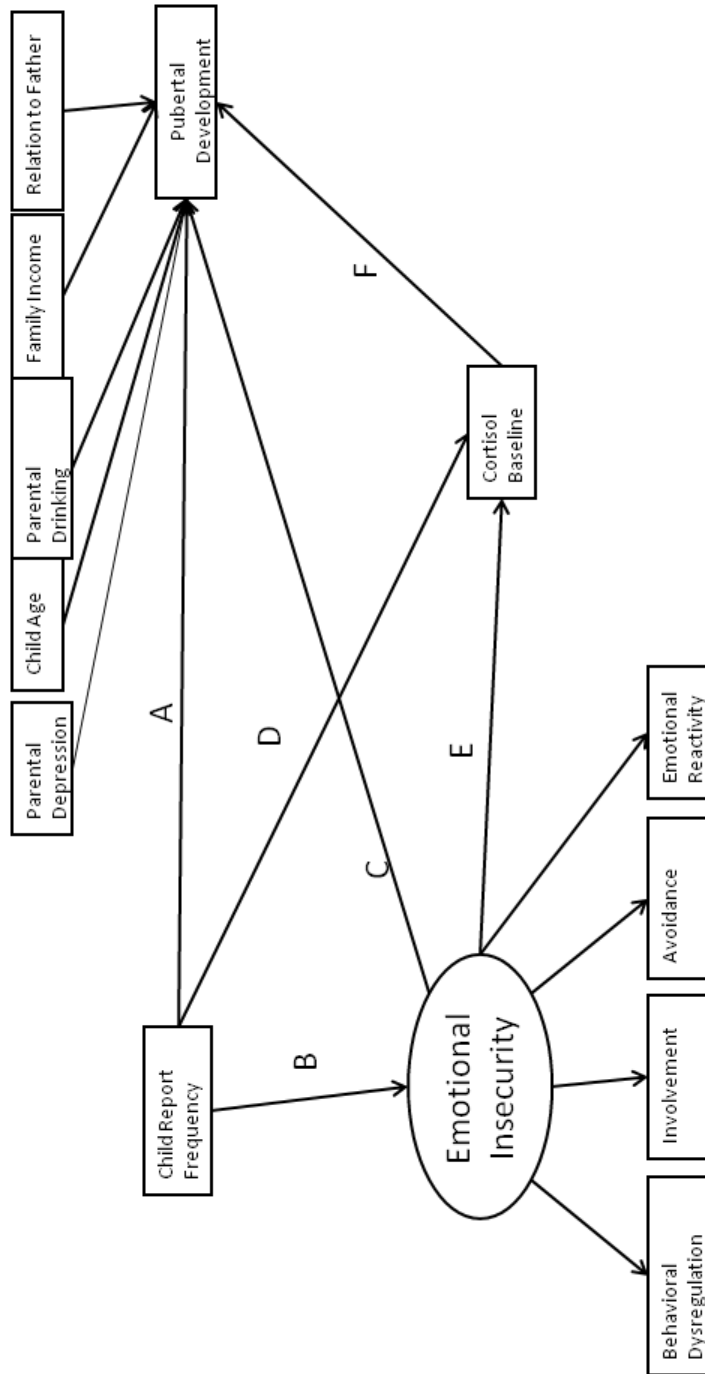
Figure 2
Parental Report of Marital Conflict (CTS) Predicting Pubertal Development
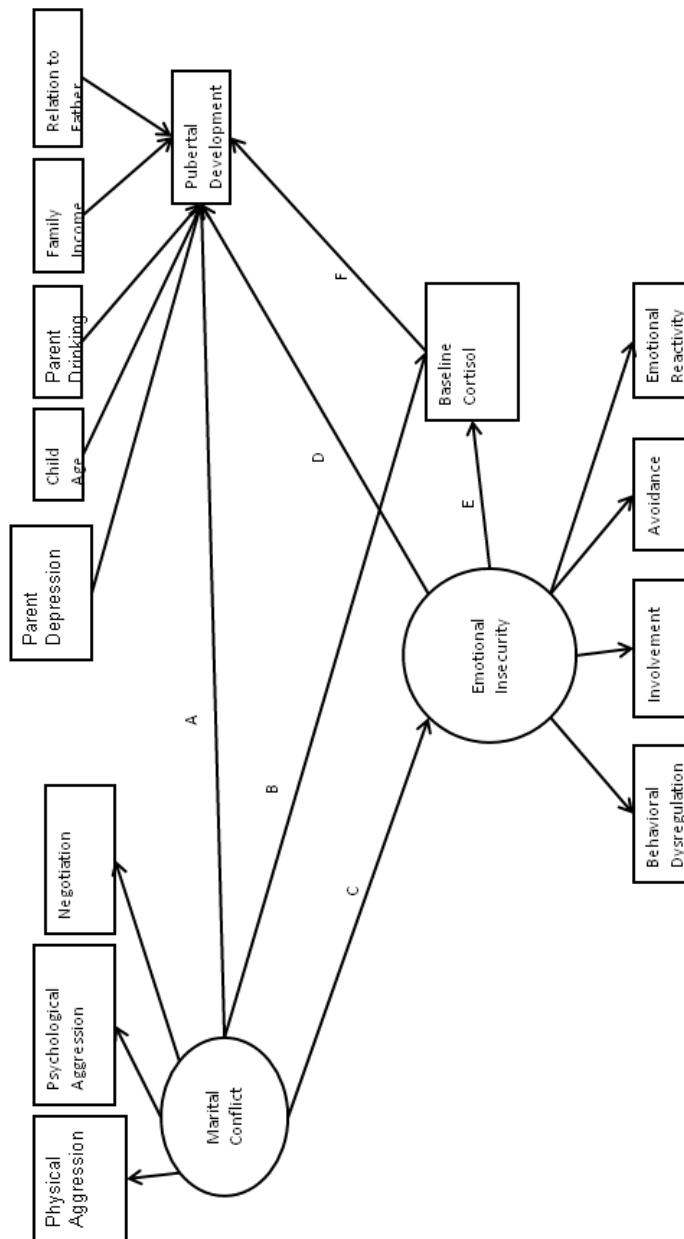
Figure 3

Parental Report of Marital Conflict (CPS) Predicting Pubertal Development
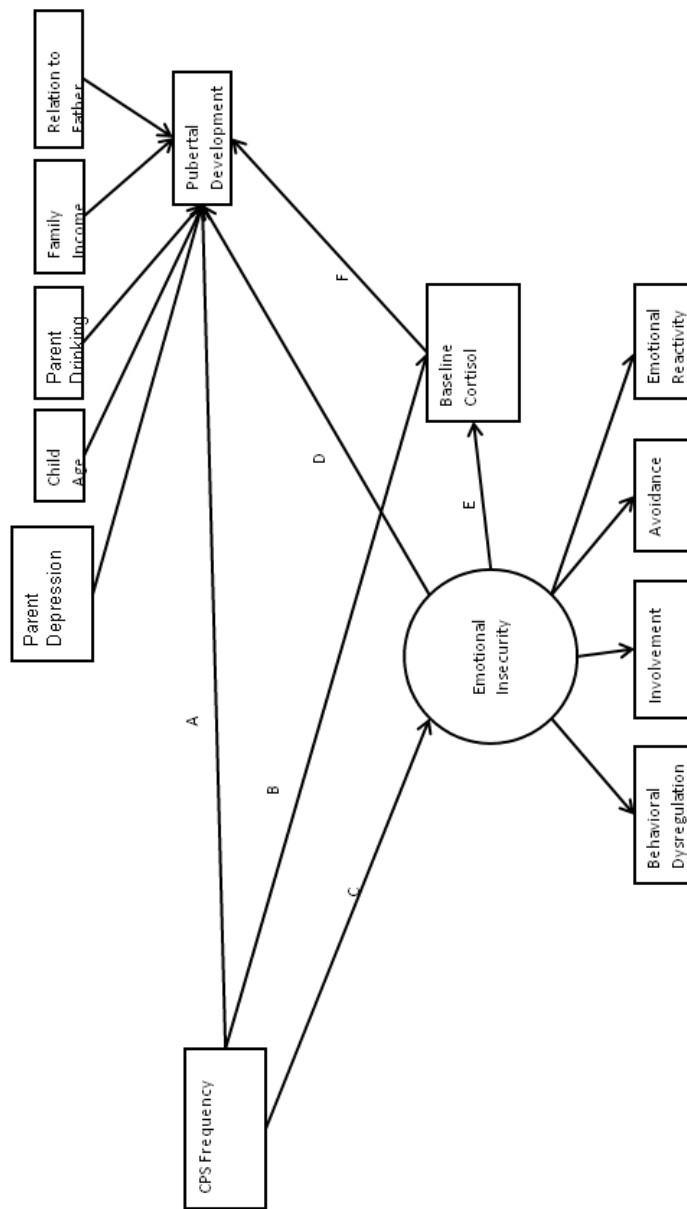
Chapter Two: Methods

Participants

      Participants were taken from two larger research projects and combined into one data set. Participants included 224 families. All participants were couples who had been cohabitating for at least three years and their children aged 6-12 years. Girls in the sample had a mean age of 8.57 years and boys had a mean age of 8.39 years. The subsample with cortisol data included 69 families and had a mean age of 8.35 years. Only one child per family participated. Families in the study were predominately Caucasian (84.4%) and had a median income of $55,000-74,999. Exclusion criteria included developmental delays, diagnosed sleep disorders, physical illness, and the use of medications.

Measures

Pubertal Development. Puberty status was measured through maternal report on the Pubertal Development Scale (PDS; Carskadon & Acebo, 1993). The PDS has separate forms for boys and girls. There are general questions regarding growth and development, as well as sex-specific items. The male form of the PDS consists of seven questions while the female form contains five items. The items are answered on a likert-type scale ranging from "barely started" (stage 1) to "seems complete" (stage 4). Mothers' report of pubertal development showed low reliability for girls in this study, α=.62 and acceptable reliability for boys in the study, α=.73. An overall pubertal development score will be calculated for all participants by averaging item scores.

      Marital Conflict. Both parents completed the Revised Conflict Tactics Scale (CTS-2) (Straus et al., 1996) and Conflict Properties Scale (CPS) (Kerig, 1996) while children completed the Children's Perception of Interparental Conflict Scale (CPIC)

(Grych, Seid, & Fincham, 1992). The CTS-2 is a measure of the frequency and severity of both physical and psychological aggression within couples during the past year. The CTS-2 shows high reliability and has been validated in multiple cultures. The measure has 39 items divided into two broad subscales for psychological and physical aggression. Participants' answers indicate the frequency at which the items have occurred, ranging from "This has never happened" to "More than 20 times in the past year," as well as an option for "Not in the past year, but it did happen before." Each parent completed the CTS-2 twice, once with regard to their own behavior and once with regard to their partner's behavior. The CTS-2 showed good reliability for female report of conflict, α=.85 and for male report of conflict, α=.82. The CPS conflict frequency subscale was utilized for this study. This subscale consists of two questions that ask each partner to rate how often they engage in both major and minor conflict episodes. The CPS showed acceptable reliability for female report, α=.72 and low reliability for male report of conflict α=.61. The CPIC measures relationship conflict from the child's perspective. The scale shows good validity and reliability. The CPIC consists of three subscales measuring conflict properties, threat, and self-blame. However, only the conflict properties subscale is relevant to the current investigation. The scale consists of 17 items which are answered "true," "sort of true," or "false." The CPIC had acceptable reliability in the study α=.78 The CPIC scores were treated separately from parent CTS-2 and CPS scores in analyses.

Child Emotional Insecurity. Emotional insecurity was measured using the Security in the Interparental Subsystem Scale (SIS; Davies et al., 2002). The SIS has both a child report (CR) and parent report (PR). Three SIS-CR subscales were used: (1) emotional reactivity to marital conflict (12 items, e.g., "When your parents have an

argument, do you get scared?"); (2) avoidance of marital conflict (5 items, e.g., "When your parents argue, do you try to stay far away from them?"); and (3) involvement in marital conflict (9 items, e.g., "When your parents argue, do you tell them to stop?"). The child rates items on a scale that includes "Yes," "sometimes," and "no." The SIS-CR in the study had excellent reliability, α=.95. The three subscales were used as indicators for a latent variable. Four SIS-PR subscales were used: (1) emotional reactivity to marital conflict, (2) avoidance of marital conflict, (3) involvement in marital conflict, and (4) behavioral dysregulation in response to marital conflict. The SIS-PR items are rated on a 5-point scale ranging from "Not at all like him/her" to "A whole lot like him/her." Mother report of emotional insecurity showed good validity, α=.85. The four subscales were used as indicators of a latent variable in analyses.

Child Cortisol. For 69 children, cortisol was measured through assays of four saliva samples collected in the laboratory. Samples were stored in a freezer at -20° Celsius. Samples were assayed at the Clinical Research Development and Operations Center of the Center for Clinical and Translational Research at the University of Kentucky. Assays were completed without modification to manufacturer's recommended protocol (Salimetrics, State College, PA). The test uses 25 µl of sample. The test has lower limit sensitivity of .007 µl/dl with a range of .007 to 3.0 µl/dl. The assays have average intra- and inter-assay coefficients of variation of less than 5% and 10%, respectively.

Procedure

The studies that participants were gathered from were approved by the University of Kentucky Institutional Review Board. The current section focuses only on the aspects

of those studies relevant to the proposed research. Informed consent and assent was obtained from all participants prior to the study. The family then participated in a 2.5-3 hour laboratory visit. During this visit, the parents completed questionnaires on computers in separate rooms. The child completed relevant questionnaires with the help of a research assistant. Saliva samples were collected via the passive drool method (Granger et al., 2007) after an acclimation period. Parents were instructed to prevent the child from eating within 40 minutes of the visit, and children were instructed to rinse their mouths with water before the saliva sample was collected.

Chapter Three: Results

Data Preparation

All variables were examined for univariate outliers. Any case that was beyond three standard deviations from the mean was considered an outlier. Less than 2.5% of cases were identified as outliers for all variables. In order to retain sample size, univariate outliers were trimmed to values equal to three standard deviations away from the mean. Variables were then examined for univariate normality, as an index of whether data were likely to meet the assumption of multivariate normality required by SEM. A skew-index of 2 or greater was determined to be a marker of significant non-normality. Using this criterion, 22 variables of interest were determined to be non-normal. This is expected given the non-normal distribution of aggressive behavior in the population. In order to accommodate the non-normality, models were fit in MPlus (Muthen & Muthen, 2011) using MLR estimation, which is robust against violations of the assumption of multivariate normality.

Analysis Plan

16 models were fit in order to test the study hypotheses. All models tested a direct effect between marital conflict and pubertal development. All models also tested a mediating path through emotional insecurity. The models differed in who was reporting marital conflict and emotional security. In models utilizing the CTS and CPS, mother and father reports were examined in separate models and child report of emotional security was used. In the models using child report of conflict via the CPIC, mother's report of emotional security was used. Models were also fit that utilized cortisol as a mediating

variable between marital conflict, emotional insecurity, and pubertal development. All models controlled for family income, child age, child relationship to the father, parental depression, and parental problem drinking. Where sample size allowed, separate models were fit for boys and girls, consistent with theory suggesting that girls may be more sensitive to family environments in regards to pubertal timing (Del Giudice, 2009). This was done for all models not including cortisol as a mediator. Due to small sample size, models utilizing cortisol as a potential mediator combined both boys and girls. Therefore, models utilizing cortisol also controlled for child gender, as well as the previously listed control variables. Criteria for a good fit included a non-significant $\chi2$, $\chi2$/df ratio below 2, a RMSEA value below .08 with a 90% confidence interval upper bound of .10, a CFI above .90, and an SRMR below .10 (Hooper, Coughlan, & Mullen, 2008). Models were determined to have acceptable fit if 3 of the 5 indices reached acceptable criteria, good fit if 4 of the indices reached criteria, and excellent fit if all criteria were met.

Descriptive statistics and correlations are presented in table 1. Fit indices for models are presented in table 2. Path coefficients are discussed only for models that evidenced acceptable or better fit.

Table 1
Bivariate Correlations and Descriptive Statistics

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Puberty														
2. Age	.57**													
3. Sex	-.26**	-.05												
4. Income	.00	.10	.05											
5. Child Relationship to Father	-.15*	-.15*	.09	-.04										
6. Race	.00	-.09	-.02	.22**	.01									
7. Father AUDIT	.00	-.03	-.02	.09	.04	-.05								
8. Mother AUDIT	.02	.06	.05	.12	.00	-.08	.59**							
9. Father Depression	-.07	-.06	-.06	-.19**	-.06	-.01	.01	.00						
10. Mother Depression	.08	-.14*	-.05	-.23**	-.06	-.03	-.08	-.10	.22**					
11. Father CPS Score	-.05	-.06	-.02	-.06	-.04	-.06	.02	-.03	.26**	.22**				
12. Mother CPS Score	.08	-.04	-.10	-.05	-.03	-.05	.02	-.13	.24**	.39**	.61**			
13. Mother CTS Negotiation Self	.04	-.02	.00	-.06	-.12	-.04	.10	.00	.23**	.29**	.42**	.52**		
14. Mother CTS Negotiation Partner	.06	.01	.00	-.05	-.12	.01	.08	-.01	.19**	.29**	.39**	.49**	.95**	
15. Father CTS Negotiation Self	.02	.01	-.03	-.06	-.13*	-.03	.04	.04	.25**	.22**	.53**	.40**	.43**	.42**
16. Father CTS Negotiation Partner	.00	.01	.02	-.01	-.14*	.09	.01	.01	.20**	.17*	.50**	.33**	.41**	.38**
17. Mother CTS Psychological Aggression Self	.05	-.13*	-.07	-.12	-.03	.07	-.01	-.03	.24**	.36**	.34**	.44**	.51**	.47**
18. Mother CTS Psychological Aggression Partner	.08	-.12	-.11	-.14*	-.06	.14*	.05	-.08	.25**	.36**	.35**	.53**	.55**	.52**
19. Father CTS Psychological Aggression Self	.02	-.04	-.02	-.06	-.08	.12	.05	-.01	.36**	.19**	.48**	.40**	.39**	.34**
20. Father CTS Psychological Aggression Partner	.03	-.07	-.03	-.11	-.05	.11	.08	.13	.39**	.25**	.49**	.37**	.43**	.37**
21. Mother CTS Physical Aggression Self	.15*	.06	-.13	-.10	-.01	.10	.04	-.01	.08	.19**	.18**	.13	.15**	.12
22. Mother CTS Physical Aggression Partner	.13	.01	-.01	-.14*	-.04	.11	.05	-.03	.15*	.32**	.05	.22**	.22**	.22**
23. Father CTS Physical Aggression Self	-.02	.02	.03	-.16*	-.04	.06	.03	-.02	.11	.13*	.12	.17*	.11	.13
24. Father CTS Physical Aggression Partner	.00	.04	-.05	-.18**	.01	-.13	.07	.06	.26**	.18**	.26**	.14*	.22**	.22**
25. CPICK Frequency	.01	.26**	.11	.12	.05	.06	.03	-.01	-.06	.15*	-.22**	-.23**	-.17*	-.15*

Table 1 continued

	15	16	17	18	19	20	21	22	23	24	25	26	27	28
26 CPICK Intensity	.03	.15*	.09	.11	.06	.04	-.15*	-.18**	-.01	-.17**	-.15*	-.13*	-.12*	-.14*
27. CSIS Emotional Reactivity	.01	.09	.00	.07	.09	.10	-.01	.03	-.07	-.08	-.06	-.13*	-.09	-.11
28. CSIS Involve	.15*	.25**	-.04	-.02	-.11	.09	-.10	.04	-.05	-.07	-.01	-.13	-.03	-.04
29. CSIS Avoid	.05	.06	.04	.04	.08	.02	.03	-.02	-.15*	-.10	-.06	-.07	-.07	-.08
30. SIS Emotional Reactivity	.24*	.11	.00	-.16*	-.02	-.01	-.12	-.10	.06	.28**	.05	.21**	.06	.06
31. SIS Involve	.06	-.12	.02	.00	.02	.00	.04	-.01	.01	.15*	.20**	.11	.15*	.15*
32. CSIS Avoid	.23*	.20**	.03	-.22**	-.15*	-.05	-.12	-.04	.07	.24**	.00	.22**	.06	.06
33. CSIS Behavioral Dysregulation	.05	.05	.05	.07	-.06	.12	.05	.03	.09	.16*	.06	.12	.11	.10
34. Cortisol Baseline	.09	.13	.07	.02	-.14*	-.01	.01	-.02	-.06	.12	.09	.17**	.13*	.12

1. Puberty
2. Age
3.Sex
4. Income
5. Child Relationship to Father
6. Race
7. Father AUDIT
8. Mother AUDIT
9. Father Depression
10.Mother Depression
11. Father CPS Score
12. Mother CPS Score
13. Mother CTS Negotiation Self
14. Mother CTS Negotiation Partner

	15	16	17	18	19	20	21	22	23	24	25	26	27	28
15. Father CTS Negotiation Self														
16. Father CTS Negotiation Partner	.89**													
17. Mother CTS Psychological Aggression Self	.29**	.27**												
18. Mother CTS Psychological Aggression Partner	.33**	.25**	.81**											
19. Father CTS Psychological Aggression Self	.49**	.40**	.44**	.51**										
20. Father CTS Psychological Aggression Partner	.48**	.41**	.54**	.51**	.79**									
21. Mother CTS Physical Aggression Self	.13	.11	.43**	.33**	.22**	.30**								
22. Mother CTS Physical Aggression Partner	.09	.06	.36**	.44**	.27**	.25**	.57**							
23. Father CTS Physical Aggression Self	.14*	.06	.15*	.26**	.37**	.35**	.13	.34**						
24. Father CTS Physical Aggression Partner	.27**	.15*	.28**	.30**	.31**	.48**	.34**	.23**	.66**					
25.CPICK Frequency	-.26**	-.20**	-.14*	-.20**	-.15*	-.21**	-.12	-.09	-.18**	-.19**				
26 CPICK Intensity	-.21**	-.11	-.15**	-.15**	-.15*	-.20**	-.10	-.10	-.17**	-.19**	.61**			
27. CSIS Emotional Reactivity	-.07	-.02	-.06	-.07	.05	.07	.00	.02	-.02	.03	.05	.10		
28. CSIS Involve	-.03	.05	.00	-.08	-.05	.01	.03	-.06	-.18**	-.05	.05	-.05	.30**	
29. CSIS Avoid	-.11	-.09	.01	.00	.06	.02	-.02	.01	-.04	-.06	.09	.07	.57**	.13
30. SIS Emotional Reactivity	.01	.05	.09	.09	.00	-.03	.01	.12	.03	-.06	-.01	-.02	-.10	-.01

Table 1 continued

31. SIS Involve	.01	.01	.19**	.20**	.07	.06	.19**	.14*	.09	.04	-.19**	-.16*	-.15*	-.17*
32. CSIS Avoid	.12	.09	.06	.08	-.04	-.08	.06	.16*	-.02	-.02	.03	.00	-.01	.06
33. CSIS Behavioral Dysregulation	.01	-.07	.07	.13*	.15*	.02	.05	.14*	.13	.06	.02	-.03	.02	.02
34. Cortisol Baseline	.09	.08	.07	.21**	.07	.09	.15*	.12	.11	-.06	-.03	-.09	-.02	.03

	29	30	31	32	33	34	M	SD
1. Puberty								
2. Age							7.51	2.37
3. Sex							8.50	1.87
4. Income							7.07	2.68
5. Child Relationship to Father								
6. Race								
7. Father AUDIT							9.37	5.18
8. Mother AUDIT							7.96	5.11
9. Father Depression							9.31	8.07
10. Mother Depression							9.28	8.43
11. Father CPS Score							8.04	2.93
12. Mother CPS Score							8.09	2.99
13. Mother CTS Negotiation Self							34.90	9.08
14. Mother CTS Negotiation Partner							33.83	9.11
15. Father CTS Negotiation Self							33.07	9.05
16. Father CTS Negotiation Partner							32.48	8.73
17. Mother CTS Psychological Aggression Self							4.24	3.85
18. Mother CTS Psychological Aggression Partner							3.95	3.92
19. Father CTS Psychological Aggression Self							4.19	3.90
20. Father CTS Psychological Aggression Partner							4.56	4.77
21. Mother CTS Physical Aggression Self							.16	.47
22. Mother CTS Physical Aggression Partner							.16	.49
23. Father CTS Physical Aggression Self							.11	.34
24. Father CTS Physical Aggression Partner							.22	.59
25. CPICK Frequency							7.65	2.11
26 CPICK Intensity							8.84	2.49
27. CSIS Emotional Reactivity							16.43	4.82
28. CSIS Involve							9.27	4.65
29. CSIS Avoid							5.38	2.76
30. SIS Emotional Reactivity	-.02						14.07	5.03
31. SIS Involve	.00	.23**					20.11	7.10
32. CSIS Avoid	.03	.65**	.02				15.15	5.17
33. CSIS Behavioral Dysregulation	.01	.18**	.05	.18**			9.37	3.26
34. Cortisol Baseline	.03	.11	.19**	.17*	-.04		.09	.06

Table 2
Model Fit Indices

	X^2	X^2/df	RMSEA	RMSEA 90% CI	CFI	SRMR
CPIC Intensity-PR Emotional Insecurity-Puberty Girls	77.24*	1.43	.06	.03-.09	.88	.07
CPIC Intensity-PR Emotional Insecurity-Puberty Boys	78.08*	1.45	.07	.03-.10	.71	.08
CPIC Frequency-PR Emotional Insecurity-Puberty Girls	73.00	1.35	.06	.01-.09	.91	.07
CPIC Frequency-PR Emotional Insecurity-Puberty Boys	71.13	1.32	.05	.00-.09	.80	.07
CPIC Intensity-PR Emotional Insecurity-Cortisol-Puberty	144.58*	2.16	.13	.10-.16	.41	.12
CPIC Frequency-PR Emotional Insecurity-Cortisol-Puberty	86.48	1.29	.07	.00-.10	.85	.08
Mother Self CTS-CR Emotional	85.19	1.25	.05	.00-.08	.92	.08

Table 2 continued

Insecurity-Puberty Girls Mother Self CTS-CR	99.17*	1.46	.07	.03-.09	.80	.08
Emotional Insecurity-Puberty Boys Mother Partner CTS-CR	81.86	1.22	.05	.00-.08	.94	.07
Emotional Insecurity-Puberty Girls Mother Partner CTS-CR	77.24	1.15	.04	.00-.07	.94	.07
Emotional Insecurity-Puberty Boys Mother Self CTS-CR	123.90*	1.51	.09	.05-.12	.72	.09
Emotional Insecurity-Cortisol-Puberty Mother Partner CTS-CR	123.90*	1.51	.09	.05-.12	..72	.09
Emotional Insecurity-Cortisol-Puberty Father Self CTS-CR	94.94*	1.40	.06	.03-.09	.89	.08
Emotional Insecurity-Puberty Girls Father Partner CTS-CR	76.00	1.12	.04	.00-.07	.94	.07
Emotional Insecurity-Puberty Boys Father Self CTS-CR	142.23*	1.67	.10	.07-.13	.64	.10
Emotional Insecurity-Cortisol-Puberty Father Partner CTS-	104.01*	1.58	.09	.06-.12	.73	.09

Table 2 continued

CR Emotional Insecurity-Cortisol-Puberty						
Mother CPS-CR Emotional Insecurity-Puberty Girls	62.12*	1.49	.06	.02-.10	.89	.08
Mother CPS-CR Emotional Insecurity-Puberty Boys	50.33	1.17	.04	.00-.08	.94	.07
Mother CPS-CR Emotional Insecurity-Cortisol-Puberty	94.61*	1.50	.09	.05-.12	.74	.09
Father CPS-CR Emotional Insecurity-Puberty Girls	49.01	1.14	.04	.00-.08	.96	.07
Father CPS-CR Emotional Insecurity-Puberty Boys	55.86	1.30	.05	.00-.09	.88	.07
Father CPS-CR Emotional Insecurity-Cortisol-Puberty	86.12*	1.48	.08	.04-.12	.72	.08

Model Results

The model utilizing maternal self-report of conflict behaviors was an acceptable fit for the data for boys. All factors loaded significantly for both the emotional insecurity and conflict latent variables. The hypothesized direct effect of marital conflict on pubertal development was not observed in this model, $B = .03$, $p = .47$. Emotional insecurity was also not significantly associated with boys' pubertal development, $B = -.02$, $p = .66$. Marital conflict also failed to predict emotional insecurity, $B = .01$, $p = .95$. The only variable in the model that was significantly associated with pubertal development was boys' age, $B = .41$, $p < .001$. The model was an excellent fit for the subsample of girls. All indicators loaded significantly on the latent variables of conflict and insecurity. Contrary to hypotheses, neither conflict nor emotional insecurity were significantly associated with girls' pubertal development, $B = .04$, $p = .35$ and $B = .03$, $p = .72$, respectively. Maternal report of depression was positively and significantly associated with pubertal development, $B = .06$, $p = .01$. Paternal report of depression was significantly and negatively associated with girls' pubertal development, $B = -.06$, $p = .01$.

Mothers' report of paternal conflict tactics was then used in a model predicting pubertal development from marital conflict and child's report of emotional insecurity. This model was an excellent fit to the data for the sample of boys. Fathers' marital conflict tactics were marginally associated with boys' pubertal development, $B = .08$, $p = .07$. Emotional insecurity was not associated with pubertal development, $B = -.03$, $p = .27$. Boys' age was significantly associated with boys' pubertal development, $B = .42$, $p < .001$. Marital conflict was not associated with emotional insecurity, $B = -.14$, $p = .45$.

The model was an excellent fit for the girl sample as well. Contrary to hypotheses, marital conflict was not associated with pubertal development, B = .03, p = .36. Emotional security was also not associated with pubertal development, B = .04, p = .69. Marital conflict was not associated with emotional insecurity in this model, B = .05, p = .26. Girls' pubertal development was positively associated with age, B = 1.07, $p < .001$, and maternal report of depression, B = .06, p = .02. Paternal depression was associated with earlier pubertal status, B = -.06, p = .01.

The model predicting pubertal development from paternal self-report of conflict was a good fit to the data for boys. Marital conflict was marginally associated with pubertal development, B = .08, p = .09, but not with boys' self-report of emotional insecurity, B = .12, p = .50. Emotional insecurity did not significantly predict pubertal development in boys, B = -.04, p = .16. Pubertal development among boys was associated only with child age, B = .40, $p < .001$. Models utilizing paternal self-report of conflict tactics were an acceptable fit to the sample of girls. Contrary to hypotheses, marital conflict did not predict pubertal status, B = -.03, p = .55, or emotional insecurity, B = .04, p = .43. Emotional insecurity also failed to predict pubertal development, B = .05, p = .56. Pubertal development was significantly associated with maternal depression, B = .07, p = .004, and child age B = 1.07, $p < .001$. Paternal depression was marginally associated with girls' pubertal development, B = -.05, p = .07.

The model predicting pubertal development from paternal report of mothers' conflict behaviors was an excellent fit to the sample of boys. However, none of the hypothesized effects were significant. Marital conflict did not significantly predict pubertal development, B = .04, p = .48, or emotional insecurity, B = .20, p = .21.

Emotional insecurity was not associated with pubertal development, B = -.04, $p = .20$.

Child age was the only variable in the model significantly associated with boys' pubertal development, B = .42, $p < .001$. The model was an acceptable fit for girls. All of the proposed mediational paths were non-significant. Marital conflict was not significantly associated with pubertal development, B = .01, $p = .94$, or emotional insecurity, B = .06, $p = .38$. Emotional insecurity was also not associated with pubertal development, B = .04, $p = .60$. Pubertal development was positively associated with child age, B = 1.07, $p < .001$, and maternal depression, B = .07, $p = .005$. Father report of depression was associated with delayed pubertal development, B = -.05, $p = .03$.

Models were then fit examining the role of marital conflict and emotional insecurity using maternal report of conflict frequency from the CPS. This model was an excellent fit for boys. However, none of the proposed mediational paths were significant. Frequency of marital conflict did not predict pubertal development, B = .03, $p = .60$, or emotional insecurity, B = -.37, $p = .09$. Boys' emotional insecurity was not significantly associated with pubertal development, B = -.03, $p = .26$. This model did not meet criteria for acceptable fit for girls.

The model using paternal report of conflict frequency was a good fit for the sample of boys. The proposed mediational paths did not reach significance. Father report of marital conflict did not predict pubertal development, B = .04, $p = .46$, or emotional insecurity, B = -.2, $p = .25$. Emotional insecurity was also not associated with pubertal development, B = -.04, $p = .24$. The model was an excellent fit for girls. However, the hypothesized pathways did not reach significance. Marital conflict was not associated with pubertal development, B = -.01, $p = .18$, or emotional insecurity, B = .08, $p = .26$.

Child report of emotional insecurity was not associated with pubertal development, B = .06, p = .49. Pubertal development was significantly associated with maternal depression, B = 07, p = .002, and child age, B = 1.07, p < .001. Paternal depression was marginally associated with pubertal development, B = -.04, p = .09.

Models were then fit using child-report of marital conflict via the CPICK and maternal report of child emotional insecurity. The CPICK subscales did not load significantly onto a latent variable for conflict, so separate models were fit utilizing the frequency and intensity subscales. The model using conflict intensity was a good fit for boys. The proposed main effect of conflict on pubertal development was not supported, B = -.09, p = .26. Conflict was also not associated with emotional insecurity, B = .06, p = .72. Emotional insecurity was associated with pubertal development in the hypothesized direction, B = .09, p = .03. Child age was significantly associated with girls' pubertal development, B = 1.04, p < .001, as was maternal depression, B=.04, p=.048, and paternal depression, B = -.05, p = .02. The model utilizing child-report of conflict frequency was a good fit for girls. The hypothesized main effect of marital conflict on puberty was not found, B = .01, p = .95. Marital conflict was also not associated with emotional insecurity, B = -.03, p = .90. Emotional insecurity did predict pubertal development in the hypothesized direction, B = .08, p = .04. Pubertal development was also associated with child age, B = 1.02, p < .001, maternal depression, B = .05, p = .04, and paternal depression, B = -.05, p = .02.

Child report of conflict intensity predicting pubertal development was not an acceptable fit to the data for boys. Models using girls' report of conflict frequency were a good fit to the data. The proposed mediational paths were not supported. Marital conflict

was not associated with pubertal development, B = .01, *p* = .90, or emotional insecurity, B = -.06, *p* = .74. Emotional insecurity was not associated with girls' pubertal development, B = .04, *p* = .16. Child age was the only significant predictor of pubertal status in this model, B = .39, *p* < .001.

Models were then fit with the subsample of participants for whom baseline cortisol data were available. This yielded a sample of 69 families. However, none of these models reached acceptable fit. Attempts were made to improve model fit by respecifying the models, including the removal of latent variables. However, likely due to the small sample size available for these models, acceptable model fit was not achieved. As such, path coefficients were not interpreted and are not presented here.

Chapter Four: Discussion

Summary of Hypotheses and Results

The current study examined emotional insecurity and cortisol levels as potential mediators in the relationship between marital conflict and children's pubertal status. These hypotheses were not supported by the data. However, parental depression was found to consistently predict child pubertal development.

None of the models tested replicated the main effect of marital conflict on pubertal timing, as found in previous studies, in a sample of early adolescent children, when controlling for race, income, age, relationship to the father, parental depression, and parental alcohol use (e.g. Belsky et al., 2007; Kim & Smith, 1999). Two models showed a trend for this relationship among boys, but given the number of models fit, this should be considered weak support at best. One potential explanation for the failure to replicate this finding may be the sample size utilized. The current study used a sample of 218 families, divided by child gender into a sample of 107 boys and 111 girls. While this is not a small sample, it is quite a bit smaller than many previous studies examining the effect of psychological stressors on pubertal timing. Given the small effect size generally found in studies examining marital conflict and pubertal timing, the current study may have been underpowered to detect this relationship. This may be especially true for the models that utilized the CPICK and CPS to measure conflict as these measures provided subscales of intensity and frequency of conflict, but not specific tactics and other components of conflict that may influence children's interpretation of parental disagreement. Another possible reason for the failure to replicate previous findings is the age of the current sample. The mean age of girls in the sample was 8.57 years, with a

range from 6-12. This may have created a restricted range of pubertal development, making it difficult to detect any relationship between marital conflict and pubertal development. This problem would have been exacerbated in the sample of boys, who develop at a later age. However, life history theories posit that boys are less likely to be sensitive to the family environment in regards to pubertal development (Del Giudice, 2009; Belsky et al., 2007). Therefore, the lack of support for this hypothesis amongst the sample of boys is still consistent with life history theories.

Contributions and Limitation

The original contribution of the current study was the examination of emotional insecurity and cortisol as potential mediators in the relationship between marital conflict and pubertal development. While one model did show a significant association between girls' emotional insecurity and pubertal development, the majority of models did not. As such, findings should be considered weak support for the hypothesized relationship. This may support Del Giudice's hypothesis that the attachment system mediates this relationship (2009). However, the interpretation of null results is problematic. As discussed above, issues of sample size and potentially restricted range of pubertal development in the current sample makes interpretation of these null results particularly problematic. More studies are needed with larger samples and a wider age range in order to examine the potential roles of emotional insecurity and cortisol in pubertal development.

Parental depression was consistently associated with pubertal development for girls in the current study. To our knowledge, this is a novel finding in the life history literature on puberty. However, this finding was not based on an a priori hypothesis, so

care should be taken in interpreting it. Specifically, mothers' depression scores predicted girls' advanced pubertal development while fathers' depression was associated with later development. The finding regarding mothers' depression may be consistent with Steinberg's finding that maternal distance was associated with advanced pubertal status for girls (1988). It is likely that depressed mothers are more distant in their interactions with daughters than are non-depressed mothers. The finding that paternal depression is associated with reduced pubertal development is less consistent with life history theories. It would seem logical that a depressed father would be less likely to invest in child care or relationship maintenance. Life history theories would then predict that this would lead to advanced pubertal development for girls, as investment and the environment would be viewed as unstable. It is possible that depressed fathers are present more often than non-depressed fathers, which may bias individuals towards an interpretation of investment as high, leading to a slower life history strategy and delayed pubertal development. However, prior research suggests that depressed fathers engage in fewer positive parenting behaviors and increased negative parenting. In a meta-analytic review, only 1 study was found in which depressed fathers engaged in increased positive parenting (Wilson & Durbin, 2010). However, it is possible that the mere presence of the father signals investment to girls, even if the majority of interactions are negative, and that depressed fathers are present more often than non-depressed fathers. A follow-up study utilizing fathers' parenting behaviors as well as total time spent with children would help to answer this question. Another possibility is that depressed fathers provide inconsistent clues for the prediction of future environmental states. In situations in which organisms are provided with inconsistent environmental cues, it may be optimal for their fitness to

delay making any decision between growth and reproduction and to continue sampling from the environment in order to avoid an organism-environment mismatch (Frankenhuis & Panchanathan, 2011). This extended environment sampling time may be reflected in the reduced development of daughters of depressed fathers. This hypothesis would need to be tested empirically. Given the cross-sectional nature of the current study, issues of directionality should be considered, as well. It is possible that as girls become older, fathers are less likely to feel depressed. This has been supported in a longitudinal study that found that older fathers reported fewer depressive symptoms than younger fathers (Lee, Fagan, & Chen, 2012). Care should be taken in extrapolating from this study, however, as the sample included fathers much younger than those in the current sample. However, it may support hypotheses that as fathers age they develop skills and relationships that may protect against depression. Support for this possibility may also be derived from Kessler and colleagues who found that depression rates are higher for men under 45 than those over 45 (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). Therefore, older fathers in this sample may exhibit lower levels of depression while being more likely to have older daughters who exhibit more pubertal development.

The current study should be considered in light of its limitations. A major limitation was the sample size. The study proposed a mediational model for a small main effect and was thus underpowered. This was particularly problematic in the models that attempted to examine the role of cortisol in the relationship between marital conflict and pubertal development, as the small sample made it impossible to fit models that were an acceptable fit to the data. Previous research examining the relationship between marital conflict and pubertal timing have generally utilized a sample size over 300 (e.g. Belsky,

Houts, & Pasco Fearon, 2010). The current study was also cross-sectional in nature. This prevents conclusions about the directionality of the associations that were found. The study may have also been limited by the low reliability of the PDS in the current sample, particularly for girls. Low reliability can bias estimates in a manner that would make it more difficult to detect significant effects. The largest limitation of the current study was likely the age of the children studied. The girls in the study ranged from 6-12 years old. This means that there was likely a restriction of range in the pubertal development of girls, which would reduce the ability to detect significant relationships between marital conflict, emotional insecurity, and pubertal development. This restriction of range may have been exacerbated by using parental report of pubertal development, as subtle changes that may signal early development may have gone unnoticed by parents.

Conclusions

The current study examined the roles of emotional insecurity and cortisol as mediating variables in the relationship between marital conflict and pubertal timing. While one model did show a significant relationship between emotional insecurity and marital conflict, the general pattern of findings did not support the hypothesized relationship. The finding that parental depression was significantly associated with girls' pubertal development does provide support for the life history hypothesis that environmental cues may be utilized to guide physical development in order to maximize fitness. To our knowledge, the finding that parental depression is associated with pubertal development amongst girls is a novel finding, advancing the literature on life history theory.

References

Archibald, A.B., Graber, J.A., & Brooks-Gunn, J. (2006). Pubertal processes and physiological growth in adolescence. In Adams, G.R., & Berzonsky, M.D. (Eds) *Blackwell handbook of adolescence.* Oxford.: Blackwell Publishing.

Bakersman-Kranenburg, M.J., & van Ijzendoorn, M.H. (2011). Differential susceptibility to rearing environment depending on dopamine-related genes: New evidence and a meta-analysis. *Development and Psychopathology, 23,* 39-52.

Belsky, J., Jonassaint, C., Pluess, M., Stanton, M., Brummett, B. & Williams, R. (2009). Vulnerability genes or plasticity genes?. *Molecular Psychiatry, 14*, 746-754.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*(4), 647-670.

Belsky, J., Steinberg, L.D., Houts, R.M., Friedman, S.L., Dehart, G., Cauffman, E., Roisman, G.I., Halpern-Fisher, B.L., & Susman, E. (2007). Family rearing antecedents of pubertal timing. *Child Development, 78,* 1302-1321.

Bugental, D., Martorell, G. A., & Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones and Behavior*, *43*(1), 237-244.

Carlson, M., & Earls, F. (1997). Psychological and neuroendocrinological sequelae of early social deprivation in institutionalized children in Romania. In C. Carter, I. Lederhendler, B. Kirkpatrick (Eds.) , *The integrative neurobiology of affiliation* (pp. 419-428). New York, NY US: New York Academy of Sciences.

Carskadon, M.A., & Acebo, C. (1993). A self-administered rating scale for pubertal development. *Journal of Adolescent Health, 14,* 190-195.

Caspi, A., & Moffitt, T. E. (1991). Individual differences are accentuated during periods of social change: The sample case of girls at puberty. *Journal of Personality and Social Psychology*, *61*(1), 157-168.

Chisholm, J.S., Quinlivan, J.A., Peterson, R.W., & Coall, D.A. (2005). Early stress predicts age at menarche and first birth, adult attachment, and expected lifespan. *Human Nature, 16,* 233-265.

Chrousos, G.P., & Gold, P.W. (1992). The concept of stress and stress system disorders. *JAMA: The Journal of the American Medical Association, 267,* 1244-1252.

Cicchetti, D., Rogosch, F. A., Gunnar, M. R., & Toth, S. L. (2010). The differential impacts of early physical and sexual abuse and internalizing problems on daytime cortisol rhythm in school-aged children. *Child Development*, *81*(1), 252-269.

Cummings, E.M., & Davies, P.T. (1994). *Children and marital conflict: The impact of family dispute and resolution.* New York: Guilford Press.

Cummings, E.M., & Davies, P.T. (2010). *Marital Conflict and Children*. New York: Guilford Press.

Cummings, E., & Keller, P. S. (2006). Marital Discord and Children's Emotional Self-Regulation. In D. K. Snyder, J. Simpson, J. N. Hughes (Eds.), *Emotion regulation in couples and families: Pathways to dysfunction and health* (pp. 163-182). Washington, DC US: American Psychological Association.

Davies, P. T., Forman, E. M., Rasi, J. A., & Stevens, K. I. (2002). Assessing children's emotional security in the interparental relationship: The Security in the Interparental Subsystem Scales. *Child Development*, *73*(2), 544-562.

Davies, P. T., Sturge-Apple, M. L., Cicchetti, D., & Cummings, E. (2008).

Adrenocortical underpinnings of children's psychological reactivity to interparental conflict. *Child Development*, *79*(6), 1693-1706.

Del Giudice, M. (2009). Sex, attachment, and the development of reproductive strategies. *Behavioral and Brain Sciences*, *32*(1), 1-21.

Dunbar J, Sheeder J, Lezotte D, Dabelea D, Stevens-Siimon C. (2008). Age at menarche and first pregnancy among psychosocially at-risk adolescents. *American Journal of Public Health, 98*, 1822–1824.

Efron, B. & Tibshirani, R.J. (1998). *An introduction to the bootstrap*. New York: Chapman & Hall/CRC.

Ellis, B.J., & Boyce, W.T. (2011). Differential susceptibility to the environment: Toward an understanding of sensitivity to developmental experiences and context. *Development and Psychopathology, 23,* 1-5.

Ellis, B.J., Shirtcliff, E.A., Boyce, W.T., Deardorff, J., & Essex, M.J. (2011). Quality of early family relationships and the timing and tempo of puberty: Effects depend on biological sensitivity to context. *Development and Psychopathology, 23*, 85-99.

Epel, E., Lapidus, R., McEwen, B., & Brownell, K. (2001). Stress may add bite to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology*, *26*(1), 37-49.

Frankenhuis, W. E., & Panchanathan, K. (2011). Individual differences in developmental plasticity may result from stochastic sampling. *Perspectives on Psychological Science*, *6*(4), 336-347.

George, S. A., Khan, S., Briggs, H., & Abelson, J. L. (2010). CRH-stimulated cortisol release and food intake in healthy, non-obese adults. *Psychoneuroendocrinology*, *35*(4), 607-612.

Graber, J. A., Lewinsohn, P. M., Seeley, J. R., & Brooks-Gunn, J. (1997). Is psychopathology associated with the timing of pubertal development?. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(12), 1768-1776.

Granger, D. A., Kivlighan, K. T., Fortunato, C., Harmon, A. G., Hibel, L. C., Schwartz, E. B., & Whembolua, G. (2007). Integration of salivary biomarkers into developmental and behaviorally-oriented research: Problems and solutions for collecting specimens. *Physiology & Behavior*, *92*(4), 583-590.

Grych, J. H., Seid, M., & Fincham, F. D. (1992). Assessing marital conflict from the child's perspective: The Children's Perception of Interparental Conflict Scale. *Child Development*, *63*(3), 558-572.

Hooper, D., Coughlan, J., & Mullen, M. (2008). Structural equation modeling: guidelines for determining model fit. *Articles*, 2.

Kerig, P.K. (1996). Assessing the links between interparental conflict and child adjustment: The conflicts and problem-solving scales. *Journal of Family Psychology, 10,* 454-473.

Kertes, D. A., Gunnar, M. R., Madsen, N. J., & Long, J. D. (2008). Early deprivation and home basal cortisol levels: A study of internationally adopted children. *Development and Psychopathology*, *20*(2), 473-491.

Kessler, R. C., McGonagle, K. A., Swartz, M., Blazer, D. G., & Nelson, C. B. (1993). Sex and depression in the National Comorbidity Survey I: Lifetime prevalence, chronicity and recurrence. *Journal of Affective Disorders*, *29*(2), 85-96.

Kim, K., & Smith, P.K. (1999). Family relations in early childhood and reproductive

development. *Journal of Reproductive and Infant Psychology, 17,* 133-148.

Koch, F., Sepa, A., & Ludvigsson, J. (2008). Psychological stress and obesity. *The Journal of Pediatrics*, *153*(6), 839-844.

Koehler, N., & Chisholm, J.S. (2009). Early psychosocial stress affects men's relationship length. *Journal of Sex Research, 46,* 366-374.

Lanza, S. T., & Collins, L. M. (2002). Pubertal timing and the onset of substance use in females during early adolescence. *Prevention Science*, *3*(1), 69-82.

Lee, Y., Fagan, J., & Chen, W. Y. (2012). Do Late Adolescent Fathers Have More Depressive Symptoms Than Older Fathers?. *Journal of Youth and Adolescence*, *41*(10), 1366-1381.

Levine, S. (2005). Developmental determinants of sensitivity and resistance to stress. *Psychoneuroendocrinology*, *30*(10), 939-946.

Loman, M. M., & Gunnar, M. R. (2010). Early experience and the development of stress reactivity and regulation in children. *Neuroscience and Biobehavioral Reviews*, *34*(6), 867-876.

Manlove, J. (1997). Early motherhood in an intergenerational perspective: The experiences of a British cohort. *Journal of Marriage and the Family*, *59*(2), 263-279.

Manuck, S.B., Craig, A.E., Flory, J.D., Halder, I., & Ferrell, R.E. (2011). Reported early family environment covaries with menarcheal age as a function of polymorphic variation in estrogen receptor-α. *Development and Psychopathology, 23,* 69-83.

Mezzich, A., Tarter, R. E., Giancola, P. R., Lu, S., Kirisci, L., & Parks, S. (1997). Substance use and risky sexual behavior in female adolescents. *Drug and Alcohol Dependence*, *44*(2-3), 157-166.

Muthen, L. K., & Muthen, B. O. (2011). Mplus User's Guide. Sixth Edition. Los Angeles, CA: Muthén & Muthén.

Netherton, C., Goodyer, I., Tamplin, A., & Herbert, J. (2004). Salivary cortisol and dehydroepiandrosterone in relation to puberty and gender. *Psychoneuroendocrinology*, *29*(2), 125-140.

Peckett, A.J., Wright, D.C., & Ridell, M.C. (2011). The effects of glucocorticoids on adipose tissue lipid metabolism. *Metabolism, 60,* 1500-1510.

Phinney, V. G., Jensen, L. C., Olsen, J. A., & Cundick, B. (1990). The relationship between early development and psychosexual behaviors in adolescent females. *Adolescence*, *25*(98), 321-332.

Sapolsky, R.M., Romero, L.M., & Munck, A.U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews, 21,* 55-89.

Saxbe, D.E., & Repetti, R.L. (2008). Brief report: Fathers' and mothers' marital relationship predicts daughters' pubertal development two years later. *Journal of Adolescence, 20,* 1-9.

Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods*, *7*(4), 422-445

Simmons, R. G., & Blyth, D. A. (1987). *Moving into adolescence: The impact of pubertal change and school context*. Hawthorne, NY US: Aldine de Gruyter.

Spencer, S.J, & Tillbrook, A. (2011). The glucocorticoid contribution to obesity. *Stress,*

14, 233-246.

Steinberg, L.D. (1988). Reciprocal relation between parent-child distance and pubertal maturation. *Developmental Psychology, 24,* 1-7.

Stice, E., Presnell, K., & Bearman, S. (2001). Relation of early menarche to depression, eating disorders, substance abuse, and comorbid psychopathology among adolescent girls. *Developmental Psychology*, *37*(5), 608-619.

Straus, M. A., Hamby, S. L., Boney-McCoy, S., & Sugarman, D. B. (1996). The revised Conflict Tactics Scales (CTS2): Development and preliminary psychometric data. *Journal of Family Issues*, *17*(3), 283-316.

Vreeburg, S. A., Hartman, C. A., Hoogendijk, W. G., van Dyck, R., Zitman, F. G., Ormel, J., & Penninx, B. H. (2010). Parental history of depression or anxiety and the cortisol awakening response. *The British Journal of Psychiatry*, *197*(3), 180-185.

Wilson, S., & Durbin, C. E. (2010). Effects of paternal depression on fathers' parenting behaviors: a meta-analytic review. *Clinical Psychology Review*, *30*(2), 167-180.

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