DISENTANGLING THE IMPACT OF PRENATAL AND POSTNATAL EARLY LIFE STRESS ON PUBERTAL TIMING AND ADOLESCENT AGGRESSION

by

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Title: Disentangling the Impact of Prenatal and Postnatal Early Life Stress on Pubertal Timing and Adolescent Aggression

According to evolutionary-developmental theories, experiences of early life stress (ELS) accelerate child development to increase the chances of being able to thrive in a harsh environment. Children exposed to ELS often experience earlier pubertal timing and are at a greater risk of developing later risk behaviors in adolescence. However, the extent to which ELS at different sensitive periods (e.g., prenatal or postnatal ELS) predicts pubertal timing is not understood, nor are the mechanisms linking ELS to specific risk behaviors, such as aggression. The current study aims to add to the literature by simultaneously examining the impact of prenatal ELS (measured via perinatal risk factors) and postnatal ELS (measured via early environmental harshness) on pubertal timing, investigating the impact of both types of ELS on adolescent aggression through earlier pubertal timing, and exploring sex differences among these effects. Data collected on 561 adopted children and their adoptive parents and birth parents from infancy through age 15 were used to test the following research questions: 1) How do perinatal risks and early environmental harshness, both individually and in combination, predict pubertal timing?; 2) How do perinatal risks, early environmental harshness, and pubertal timing predict aggression?; and 3) What sex differences exist in the relations among

perinatal risks, early environmental harshness, pubertal timing, and aggression? Contrary to study hypotheses, results revealed no significant effect of perinatal risks or early environmental harshness on adolescent aggression. Pubertal timing did not mediate the link between either type of ELS on adolescent aggression. Additionally, sex was not a significant moderator of those results. Post hoc exploratory analyses showed that an unpredictable postnatal environment (a different operationalization of postnatal ELS) did significantly predict pubertal timing, but not adolescent aggression. This result was significant for males and females. These findings suggest that unpredictable ELS may be an important factor related to fast life history strategies, and that outcomes associated with fast life history strategies may not encompass aggression as a risk-taking behavior that can be explained with life history theory.

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CHAPTER I

INTRODUCTION

Early Life Stress

Decades of research on exposure to stress during childhood has shown that it can lead to myriad negative health outcomes (e.g., somatic diseases, addictions, mental health disorders, premature mortality; Felitti et al., 1998) and increased likelihood of engaging in risky behaviors (Wu et al., 2020) during adolescence and adulthood. Although early life stress (ELS) has been broadly defined in the literature as stress exposure during childhood (i.e., prenatal to age 18; Butler et al., 2017), there is evidence to suggest that ELS experienced earlier in development (e.g., the first five years of life) may have a larger impact on later risk behaviors because it is a time of heightened plasticity (Gunnar & Vazquez, 2006). For the current study, ELS will refer to stress experienced in utero until 5 years old.

Evolutionary-developmental theories suggest that early environmental cues can regulate physical development to attain evolutionary fitness goals (e.g., survival, reproduction, sexual selection; Hochberg & Belsky, 2013). Thus, ELS can lead to biological adaptations (e.g., stress reactivity, timing of puberty) that increase one's chance for evolutionary success in stressful environments (Gunnar & Vazquez, 2006), but can ultimately result in behaviors that are socially maladaptive (e.g., aggression and other risk behaviors; Hochberg & Belsky, 2013). More specifically, life history theory posits that early environments determine the type of *life history strategy* an individual develops (Belsky et al., 1991). Early experiences of supportive, nurturing, and predictable environments prepare the child for a long and predictable future that they can control,

which leads to the development of slow life history strategies. Individuals with slow life history strategies are more likely to have slower biological maturation, initiate sexual intercourse later than their peers, and give more effort to child-rearing. They also tend to have better future orientation, delay of gratification, self-regulation and show more aversion to risk (Belsky et al., 1991). In contrast, ELS prepares the child to anticipate a short, unpredictable future and early death through the development of a fast life history strategy. Those with fast life history strategies tend to have accelerated maturation, initiate sexual intercourse at relatively early ages, and devote less energy to parenting. They are also more likely to have difficulties considering future rewards and delaying gratification, and thus, are more likely to engage in impulsive and risky behaviors (Belsky et al., 1991; Del Giudice et al., 2016; Ellis et al., 2012). While most of the empirical literature in this area has been focused on linking fast life history strategies with sexual risk behaviors (e.g., Belsky et al., 2010), it is reasonable to assume that fast life history strategies may also be associated with other risky behaviors, such as aggression, since they share similar risk factors (e.g., impulsivity, favoring immediate rewards; Bjorklund & Hawley, 2014; Hochberg & Belsky, 2013).

Early Life Stress and Pubertal Timing

Pubertal maturation is considered a key biological indicator of different trade-offs in reproductive strategies (e.g., allocate resources towards early reproduction with many offspring or delaying reproduction to have fewer, evolutionary-fit offspring; see Ellis, 2004). In support of life history theory, many studies have documented that ELS calibrates pubertal timing (e.g., Belsky et al., 2007; Ellis & Essex, 2007; Henrichs et al., 2014; Sun et al., 2017), such that experiences of ELS are most often associated with

earlier onset of puberty (see Joos et al., 2018). These studies provide evidence to support the theory that ELS does trigger a developmental trajectory towards a fast life history strategy (Del Giudice et al., 2011) in which pubertal maturation is thought to be a key mechanism linking ELS to later risk behaviors in adolescence.

Much of the existing literature in this area has focused on the impact of ELS experienced after the child is born (i.e., postnatal ELS) and don't include measures of prenatal ELS (e.g., toxic exposures, pregnancy complications, maternal stress). However, investigating the impact of prenatal ELS on pubertal timing has become more popular in recent years. Studies have reported that maternal stress experienced during pregnancy is associated with earlier pubertal timing (Bräuner et al., 2021; Duchesne et al., 2017) and later behavioral and health problems in their children. Researchers have posited that prenatal ELS may lead to later problems through similar mechanisms as postnatal ELS (e.g., stress reactivity, early puberty; Belsky et al., 2015). There is also evidence to suggest prenatal and postnatal ELS may independently affect behaviors. For example, one study found that both prenatal and postnatal ELS significantly predicted increased adult mental health issues while controlling for one another (Herbison et al., 2017).

In contrast, some researchers have suggested that there may be different interactions between prenatal and postnatal environments that lead to slow vs. fast life history strategies. The prenatal period is a crucial time for establishing the foundational, biological processes that are thought to be altered by postnatal ELS (e.g., stress response systems and autonomic functioning; Hammock & Levitt, 2006). Further, prenatal stressors influence developmental plasticity related to an individual's ability to adjust to their postnatal environment (Pluess & Belsky, 2011). When prenatal and postnatal

environments match, children develop slow or fast life history strategies depending on whether they experience supportive or harsh environments, respectively. However, researchers have suggested that postnatal ELS might have a bigger impact than prenatal ELS on life history strategies since postnatal environments can result in epigenetic changes that modify stress response systems and life history strategies formed under prenatal conditions. Children exposed to high levels of prenatal ELS that grow up in a supportive postnatal environment are thought to develop a slow life history strategy (e.g., later pubertal timing, less risk taking; Pluess & Belsky, 2011). As mentioned above, this occurs through epigenetic modifications, caused by supportive environments, that recalibrate a child's heightened stress response system developed during exposures to prenatal ELS to support lower stress reactivity and slower life history strategies.

For children who experience low levels of prenatal ELS, but heightened exposure to harsh or unpredictable postnatal environments, the opposite occurs. The absence of prenatal risk exposure leads to downregulated stress response systems that are recalibrated by experiences of postnatal ELS to increase stress reactivity and result in fast life history strategies (e.g., earlier pubertal timing, more risk taking; Conradt et al., 2018). Empirical tests of interactions between prenatal and postnatal ELS are few, most likely due to difficulties associated with implementing causal designs to adequately uncouple prenatal and postnatal ELS. Most evidence linking prenatal and postnatal ELS to earlier pubertal timing and faster life history strategies comes from samples of children reared by their biological parents which confounds prenatal, postnatal, and heritable influences. Therefore, it is not yet understood how prenatal and postnatal ELS uniquely, and in combination, influence pubertal timing independent of heritable risk. Determining the

unique and interactive effects of prenatal and postnatal ELS on pubertal timing is an important next step to advance life history theories and will elucidate the extent to which different sensitive periods for stress exposure (i.e., pre- or postnatal) regulate pubertal development.

The Role of Pubertal Timing in the Relation between ELS and Aggression

As described above, ELS has been consistently associated with fast life history strategies. This link is supported by previous literature documenting that both ELS (Enoch, 2011; Wu et al., 2020) and early pubertal timing (Vaughan et al., 2015) are key predictors of later risky behaviors in adolescence (e.g., sexual risk behaviors, aggression, delinquency; see Hochberg & Belsky, 2013; Joos et al., 2018). Existing studies have also documented that ELS predicts pubertal timing (e.g., Ellis & Essex, 2007), and pubertal timing predicts later risk behaviors (e.g., Kogan et al., 2015). Although evolutionary developmental researchers view puberty as a major period for changing developmental trajectories, instead of an inherent risk, there are inconsistent findings among the few studies that have looked at ELS, pubertal timing, and adolescent risk behaviors concurrently. Some researchers have documented that pubertal timing is a key mechanism linking postnatal ELS to later risk behaviors (Belsky et al., 2010; Colich et al., 2020), while others found that pubertal timing does not mediate this relation (Kogan et al., 2015). Thus, it is still unclear how ELS and pubertal timing simultaneously influence adolescent risk behaviors.

The majority of studies examining the relation between ELS and pubertal timing on adolescent risk behaviors have focused on sexual risk behaviors (e.g., Belsky et al., 2010; James et al., 2012; Kogan et al., 2015) since there is a clear evolutionary purpose

(e.g., earlier sexual debut allows more time to reproduce). Less is known about how ELS and pubertal timing affect other risk behaviors, such as aggression or violence, which have a more indirect evolutionary purpose (e.g., social competition, mate selection). To my knowledge, the only study to compare the effects of ELS and pubertal timing on sexual risk behaviors vs. other risk behaviors during adolescence found that earlier pubertal timing mediated the association between ELS and sexual risk behaviors, but not other risk-taking behaviors (Belsky et al., 2010). However, there were only females in this sample and the composite of other risk-taking behaviors combined both substance use and aggressive behaviors which may have different evolutionary functions (e.g., social cooperation vs. social competition).

From an evolutionary perspective, aggression is considered an adaptive response to early stressful environments. In particular, aggression can be used to achieve and maintain high social status, get access to resources, and increase mating opportunities during adolescence (Bjorklund & Hawley, 2014). Children who are exposed to ELS are more likely to develop behaviors associated with fast life history strategies, which may include using aggression to gain short-term rewards (e.g., increased social status; Bjorklund & Hawley, 2014; Hochberg & Belsky, 2013). This is directly in line with life history theory – fast life history strategies function to accelerate development in anticipation of a short life with few opportunities to thrive. In support of this, researchers have found that early pubertal timing is associated with greater phenotypical masculinization traits (e.g., higher body mass index, facial dominance, and bicep circumference) that are associated with social competition among males (Doll et al., 2016). Further, ELS is positively associated with adolescent aggression (e.g., Barnow &

Freyberger, 2003; Fonagy, 2004; Veenema, 2009; Winiarski et al., 2018). Unpredictable ELS during the first five years of life has also been shown to predict intimate partner violence perpetration during early adulthood, with relationship conflicts during adolescence serving as a mediating mechanism between ELS and perpetration (Szepsenwol et al., 2019).

It seems that similar risk factors and mechanisms that have been linked to fast life history strategies may be influencing the development of aggression during adolescence. Since both aggression and sexual risk behaviors can be conceptualized as an outcome of fast life history strategies and share similar evolutionary functions (e.g., social competition), it is reasonable to hypothesize that factors associated with the development of fast life history strategies related to sexual risk taking, including ELS and pubertal timing, would also be related to adolescent aggression. To date, there are no studies that have specifically examined the extent to which ELS and early pubertal timing predict aggressive behavior during adolescence. Investigating these relations will either provide initial evidence of whether life history theory can be expanded to include a broader range of adolescent risk behaviors or highlight the limits of life history theory in which it may only apply to sexual risk behaviors.

Sex Differences

As described above, there is a wealth of literature supporting the theory that ELS adjusts pubertal timing, and both affect later risk behaviors including aggression. However, the direction of the effect is not as well understood, especially in males. This is likely because there are countless studies investigating the link between ELS and pubertal timing in female samples, but many fewer that employ male samples. For females, ELS is

a key predictor of earlier pubertal timing (Belsky et al., 2007; Henrichs et al., 2014) and later risk behaviors (Belsky et al., 2015; Enoch, 2011). Some researchers have argued that associations among ELS, pubertal timing, and adolescent risk behaviors function similarly for males and females (Mendle & Ferrero, 2012; Sun et al., 2017; Wu et al., 2020). Others have suggested that associations among ELS, pubertal timing, and later risk behaviors exist only for females (Belsky et al., 2015; Ellis & Essex, 2007; Costello et al., 2007). There is also evidence to suggest that the direction of the associations among ELS, pubertal timing, and risk behaviors may differ based on sex. ELS has been associated with delayed puberty in males (Semiz et al., 2009), with earlier pubertal timing associated with increased prosocial behaviors in the home among male children (Carlo et al., 2012). These inconsistent findings highlight the importance of exploring sex differences in the associations among ELS, pubertal timing, and adolescent risk behaviors.

Even fewer studies exist that have investigated how sex influences the relation of different forms of ELS (e.g., prenatal and postnatal ELS) on pubertal timing and later problem behaviors. Studies of animal models have found that there are sex-specific epigenetic changes that may cause males to be more susceptible to the influence of prenatal ELS (Mueller & Bale, 2008), and evolutionary developmental researchers have also suggested that there are different biological mechanisms that are activated by prenatal stressors in males and females (see Hartman & Belsky, 2018). Sex has also been found to influence how prenatal and postnatal ELS affect later behaviors. In one study comparing the effects of experiencing prenatal and postnatal ELS on adult mental health, researchers found that, for males, prenatal ELS predicted later depression and anxiety

symptoms above and beyond the effect of postnatal ELS (Herbison et al., 2017). In contrast, postnatal ELS was more predictive of depression and anxiety symptoms among females (Herbison et al., 2017). While these studies suggest that sex differences may exist in how prenatal and postnatal ELS influence development, it is not clear how sex and type of ELS impact the development of fast life history strategies (i.e., early puberty, adolescent risk behaviors).

Sex differences in aggression have been documented for decades (e.g., Dodge et al., 2006; Tremblay et al., 1999), with sex differences in aggression appearing during the early toddler years (Baillargeon et al., 2007). In general, males are thought to display more physical aggression, whereas females tend to exhibit increased relational (i.e., social or indirect) aggression (Card et al., 2008). Studies have also shown that females are more likely to engage in bullying behaviors that are characterized by relational aggression, compared to males who show higher levels of direct bullying (Viding et al., 2009). Although sex differences exist in the presentation of aggressive behaviors, the evolutionary function of aggression might be the same for males and females. Relational aggression is thought to develop through evolved mechanisms that support mating strategies in females and functions to increase social status among peers and potential mates (Vaillancourt, 2013), which are the same functions associated with physical aggression in males (Archer, 2004; Bjorklund & Hawley, 2014). Thus, it may be that the developmental trajectory of aggression, within the life history framework, progresses similarly for males and females. Fast life history strategies have been directly linked to increased aggression and criminal behavior among adolescent males (Simmons et al., 2019). ELS (Mohapatra et al., 2010) and early maturation (Celio et al., 2006) have also

been linked to aggression among females, suggesting that factors associated with a fast life history strategy are predictive of female aggression. Overall, there is insufficient evidence to support or refute sex differences in evolutionary developmental models of adolescent aggression. It is imperative to investigate which, and to what extent, the associations among ELS, pubertal timing, and aggression are influenced by sex to fully understand evolutionary-developmental trajectories of adolescent risk behaviors.

CHAPTER II

SUMMARY AND HYPOTHESES

Determining the extent to which prenatal and postnatal ELS impact pubertal timing and examining how these factors, along with pubertal timing, influence adolescent aggression is a crucial next step to understand the limits of life history theory. Addressing these aims will elucidate whether exposure to ELS during prenatal or postnatal sensitive periods, or an interaction of the two, has the largest impact on pubertal development, as well as determine whether life history theory can be applied to other adolescent risk behaviors beyond sexual risk taking, such as aggression. Additionally, exploring how sex influences the relations among ELS, pubertal timing, and aggression is necessary to fully understand how these factors influence human development and how to appropriately apply evolutionary-developmental models to support prevention efforts that will fit with an individual's internal motivations to reduce adolescent risk behaviors.

Interestingly, both harsh and unpredictable environments have been used as measures of ELS, and are linked to pubertal timing, greater risky behavior, and faster life history strategies (Del Giudice et al., 2016; Ellis, 2004). As the main aim of the present study is to compare the effects of prenatal and postnatal ELS on facets of fast life history strategies (e.g., pubertal timing and risk behaviors), the current operationalizations of prenatal and postnatal ELS needed to be conceptually similar. This study uses data from the Early Growth and Development Study (EGDS; see Leve et al., 2019), an ongoing, prospective longitudinal adoption study. In this sample, the only available measure of prenatal ELS is a comprehensive perinatal risks index, which represents cumulative exposure to environmental hazards and is conceptualized as a measure of a harsh prenatal

environment (i.e., extrinsic morbidity-mortality experienced prenatally and immediately after birth; Ellis et al., 2022). Therefore, while there are measures of postnatal harshness and unpredictability, a priori hypotheses in the current study focus solely on postnatal environmental harshness, as it is more conceptually similar to the perinatal ELS measure (i.e., perinatal risks) available in the data set. The specific research questions and hypotheses to be addressed are:

1. How do perinatal risks and early environmental harshness, both individually and in combination, predict pubertal timing? Consistent with fetal programming theories (see Conradt et al., 2018), I hypothesize that early environmental harshness will predict earlier pubertal timing above and beyond the effect of perinatal risks. I also hypothesize that the interaction between perinatal risks and early environmental harshness will have the strongest effect on pubertal timing, such that high perinatal risks and greater environmental harshness will predict earlier pubertal timing.
2. How do perinatal risks, early environmental harshness, and pubertal timing predict aggression? In support of life history theory (Belsky et al., 1991), I hypothesize that both types of ELS will predict earlier pubertal timing and increased aggression, and that pubertal timing will mediate the association between ELS and aggression.
3. What sex differences exist in the relations among perinatal risks, early environmental harshness, pubertal timing, and aggression? Consistent with previous literature (e.g., Herbison et al., 2017; Mueller & Bale, 2008), I hypothesize that there will be sex differences in how perinatal risks and early

environmental harshness predict pubertal timing and later aggression. Specifically, I hypothesize that perinatal risks will have a larger effect for males on pubertal timing and adolescent aggression, whereas early environmental harshness will have a larger effect for females.

CHAPTER III

METHODS

Study Design and Sample

This study utilizes data from the Early Growth and Development Study (EGDS; see Leve et al., 2019), which is an ongoing, prospective longitudinal adoption study designed to investigate the interplay between heritable and environmental influences on various child outcomes. The EGDS sample was recruited through 45 adoption agencies based in the US and includes 561 adopted children (Cohort I n = 361; Cohort II n = 200) and their adoptive parents (562 adoptive fathers and 569 adoptive mothers), their birth mothers (n = 556), and birth fathers (n = 211). There were 321 (57.2%) male children in the sample and 240 (42.8%) female children. The average child age at adoption placement was 5.58 days (SD = 11.32, range = 0-91). Data collection began when the adopted child was approximately 3-6 months old, with repeated assessments conducted from infancy to age 13 for both cohorts, and age 15 for Cohort I. Assessments occurred approximately every 6 months for ages 3 months - 2.5 years, at age 4.5 years, once per year for ages 6-9 years, and at age 11, 13, and for Cohort 1, at age 15. Child, adoptive parent, and birth parent race and ethnicity is reported in Table 1. More information on EGDS sample demographics is reported in Leve et al. (2019).

Measures

Perinatal Risks. A comprehensive index of perinatal risks (developed in Marceau et al., 2016) was used to represent prenatal ELS. The perinatal risks index is a sum score of the frequency of experiencing obstetric complications experienced during pregnancy and immediately after birth. It includes pregnancy complications, neonatal complications,

substance use, exposure to toxins, and labor and delivery complications. These data were collected from prenatal care centers, delivery records, and home interviews of birth mothers at about 3 months postpartum on different prenatal risk exposures. Perinatal risk experiences were documented with the Life History Calendar method (Caspi et al., 1996) in which trained interviewers asked about exposure to substances during pregnancy. Interviewers directly asked about exposure to substance use and toxins during the interview, and pregnancy complications were measured via a pregnancy screener assessing medical aspects of pregnancy (e.g., weight change, blood pressures, frequency of doctor visits).

Table 1. *Child, adoptive parent, and birth parent race and ethnicity.*

Race/Ethnicity	Children N = 561	Adoptive Parents n = 1,113	Birth Mothers n = 556	Birth Fathers n = 211
	n (%)	*n (%)*	*n (%)*	*n (%)*
White	306 (55%)	1,009 (91%)	393 (70%)	148 (70%)
More than 1	100 (18%)	11 (1%)	26 (5%)	11 (5%)
Hispanic/ Latino	75 (13%)	22 (2%)	34 (6%)	19 (9%)
Black	74 (13%)	48 (4%)	75 (14%)	26 (12%)
American Indian/ Alaskan Native	3 (<1%)	1 (<1%)	14 (3%)	1 (<1%)
Asian	1 (<1%)	8 (<1%)	10 (2%)	1 (<1%)
Native Hawaiian/ Pacific Islander	1 (<1%)	3 (<1%)	1 (<1%)	1 (<1%)
Unknown	1 (<1%)	11 (1%)	3 (<1%)	4 (2%)

Note. n/N = number of observations.

Early Environmental Harshness. To assess harsh postnatal ELS, a parent-report latent variable was created to represent harsh environmental stressors during the first five years of life. The early environmental harshness variable consisted of three indicators: financial strain, marital harshness, and parent hostility symptoms. Financial strain was

assessed via a financial strain score from a large, family demographic survey where parents indicated the degree to which they have enough money to meet their needs. Data were collected at the 9-month, 18-month, 27-month, and 4.5-year time points from both adoptive parents. Marital harshness was assessed via the hostility subscale of the Behavior Affect Rating Scale (BARS; Conger, 1989; Melby et al., 1995) at the 9-month, 18-month, 27-month, and 4.5-year time points from both adoptive mothers and fathers. For the 9-month and 18-month assessment, parent reported on the hostility they have experienced from their partner. At the 27-month and 4.5-year assessment, parents reported on both the hostility they have experienced from their partner as well as hostility they have shown to their partner. Parent hostility symptoms were measured with the hostility dimension of the Symptom Checklist 90 Revised (Rauter et al., 1996) administered at ages 9 months and 4.5 years to both adoptive parents. Descriptive statistics from each indicator, tabulated by time of assessment and adoptive parent, are reported in Table 2. An average score across all available timepoints and adoptive parents was used for each indicator of early environmental harshness. Although there were missing responses at each timepoint (as shown in Table 2), taking the average of the available items (i.e., person mean imputation) when scales are highly correlated or the same (e.g., longitudinal data) to create a composite is acceptable and results in unbiased estimates and results (Heymans & Eekhout, 2019). Therefore, averages were computed using the available subscale scores.

Table 2. *Descriptive statistics for early environmental harshness subscales longitudinally by child age and adoptive parent (N* = 561).

	9 months				18 months			
	AP1		AP2		AP1		AP2	
Variable	*M (SD)*	*n*	*M (SD)*	*n*	*M (SD)*	*n*	*M (SD)*	*n*
Financial strain (FD)	3.63 (1.37)	521	3.60 (1.38)	498	3.64 (1.45)	520	3.49 (1.31)	491
Marital harshness (BARS)	23.67 (7.54)	539	26.69 (8.22)	517	25.23 (8.33)	503	28.78 (9.53)	484
Hostility symptoms (SC)	2.49 (1.91)	191[2]	2.45 (1.92)	181[2]				
	27 months				4.5 years			
	AP1		AP2		AP1		AP2	
	M (SD)	*n*	*M (SD)*	*n*	*M (SD)*	*n*	*M (SD)*	*n*
Financial strain (FD)	3.65 (1.41)	492	3.60 (1.33)	465	3.84 (1.43)	285[1]	3.62 (1.39)	272[1]
Marital harshness (BARS)	25.59 (7.50)	508	26.43 (7.91)	488	25.84 (8.18)	425	26.69 (7.86)	408
Hostility symptoms (SC)					1.59 (0.40)	282[1]	1.53 (0.40)	257[1]

Note. N/n = number of observations, AP1 = adoptive parent 1 (mostly mothers), AP2 = adoptive parent 2 (mostly fathers), *M* = mean, *SD* = standard deviation. FD = family demographics questionnaire, BARS = Behavior Affect Rating Scale, SC = Symptom Checklist 90. [1]Data only available for Cohort 1 (*n* = 361), [2]Data only available for Cohort 2 (*n* = 200).

Pubertal Timing. Pubertal timing was assessed at age 11 via the Pubertal Development Scale (PDS; Petersen et al., 1988). The PDS is a 5-item scale with different questions for males and females to characterize their secondary sex characteristics (e.g., growth spurt, body hair development, breast development and menarche in females, facial hair growth and voice change in males). Responses are on a 4-point scale: 1) development has not yet started, 2) development has barely started, 3) development has definitely started, and 4) development seems complete. Menarche was coded dichotomously based on whether menarche has started (4) or not (1). Items were averaged separately for females (including menarche) and males to give a continuous pubertal timing score representing level of pubertal development at age 11. A higher PDS score means that the adolescent is in later pubertal stages at age 11, compared to their same-sex peers. Pubertal timing was measured as a continuous score, rather than classified into pubertal stages, to better investigate the hypotheses concerning the impact of ELS on pubertal development in general. Specifically, subtle differences between adolescents in the same pubertal stages can be accounted for with this continuous pubertal timing score.

Aggression. Adolescent aggression was measured using the 18-item aggressive behavior subscale of the Child Behavior Checklist 6-18 version (CBCL; Achenbach & Rescorla, 2001). Adoptive mothers and fathers reported on their child's aggressive behavior at age 15 (for Cohort I only) on a 3-point scale (not true, sometimes true, very true). Responses were summed separately for mothers and fathers to compute the aggressive behavior subscales. To account for both adoptive parents' responses in the

current study, an average aggression score was computed across both adoptive parents for available items to create a single composite of adolescent aggression.

Covariates. The unique adoption design of the current study allows for investigation of unique and interactive effects of prenatal and postnatal ELS on pubertal timing since prenatal and postnatal environments are not confounded with heritable risk. Therefore, a retrospective report of pubertal timing among birth mothers and fathers collected when the child was 18 months old (Cohort II) and 4.5 years old (Cohort I) will be used to control for heritable risk. For the second research question, pre-pubertal aggression measured via the CBCL at age 8 (for Cohort 1) will be included as a covariate to control for the stability of aggression across development since prior work has shown that children who engage in aggressive behaviors are also more likely to show aggression during adolescence (Brame et al., 2001).

Exploratory Measures. The measures listed above for postnatal ELS were chosen because they represent early environmental harshness (e.g., marital harshness, family hostility, financial stress) rather than unpredictable ELS (e.g., family unpredictability, parent consistency) or harsh parenting or discipline variables. I chose to focus on harsh postnatal ELS measures to better compare the effect of a harsh prenatal environment (i.e., perinatal risks) with the effect of a harsh postnatal environment on pubertal timing and aggression. Additionally, measures of harsh parenting and/or discipline were originally excluded to attempt to reduce the influence of bidirectional effects (e.g., aggressive children causing harsh parenting/discipline). However, there is ample evidence to suggest that unpredictable ELS is associated with earlier pubertal timing and fast life history strategies (e.g., Ellis et al., 2009; Kogan et al., 2015; Zhang et

al., 2021). There is also literature documenting an association between harsh parenting and child aggression (see Labella & Masten, 2018), as well as between harsh parenting and pubertal timing (e.g., see Belsky et al., 2007; Pham et al., 2022).

To further investigate the association between ELS and pubertal timing and aggression, additional measures of unpredictable postnatal ELS and harsh parenting/discipline practices were included in *post hoc* exploratory analyses. There were four measures of unpredictable ELS examined: inconsistent discipline, marital instability, marital transitions, and parent inconsistency. Inconsistent discipline was measured using the inconsistent discipline subscale from the Alabama Parenting Questionnaire (Shelton et al., 1996) measured at 4.5 years. Marital instability was assessed via an abbreviated version of the Marital Instability Index (Booth & Edwards, 1983) administered to adoptive parents at age 9 months – age 4.5 years. A count of marital transitions was created using an unpublished measure of marital transitions administered at age 4.5 years that was developed to track changes (i.e., instability) in relationship statuses over the course of the study. Lastly, parent inconsistency was measured via the parental consistency subscale from an unpublished Discipline Questionnaire (see Pears et al., 2007) developed to measure discipline over the study years. The assessment at age 4.5 years was reverse coded to represent parent inconsistency; a high score indicates greater inconsistency among adoptive parents. For SEM models, unpredictable postnatal ELS was measured via a latent variable with 4 indicators: marital instability, marital transitions, parent inconsistency, and inconsistent discipline.

There were also four measures of harsh parenting or discipline included in exploratory analyses: corporal punishment, parental hostility, overreactive parenting, and

harsh discipline. Corporal punishment was assessed via the corporal punishment subscale on the Alabama Parenting Questionnaire (Shelton et al., 1996), administered at 4.5 years. Parental hostility was measured with the Iowa Family Interaction Rating Scales (Melby et al., 1989) at the 27 month and 4.5-year assessment. Adoptive parents reported on the hostility they experienced from their partner as well as the hostility they exhibited toward their partners. Overreactive parenting was assessed via the overreactivity subscale on the Parenting Scale (Arnold et al., 1993) administered at each time point from age 9 months to 4.5 years. Harsh discipline was measured with a harsh discipline subscale from the unpublished Discipline Questionnaire (see Pears et al., 2007) at age 4.5 years. As with main study variables, all measures of unpredictable ELS and harsh parenting/discipline were averaged across timepoint and adoptive parents to create a single composite for each construct.

Analytic Strategy

All statistical analyses were conducted in R version 4.0.3 (R Core Team, 2019). Prior to employing statistical models to test the study hypotheses, all regression assumptions were evaluated. Preliminary comparisons between male and female children on main study variables were analyzed with a series of Welch's *t*-tests. A one-way analysis of variance (ANOVA) was conducted to determine whether there were significant differences in main study variables by child race. All hypotheses were tested with structural equation models (SEM) with the *lavaan* R package version 0.6-7 (Rosseel, 2012). Robust Maximum Likelihood estimation (MLR) with robust (Huber-White) standard errors was used in all models as it is recommended for non-normal data (Lei & Wu, 2015). Missing data (presented in Tables 2 and 3) were analyzed using Little's

missing completely at random (MCAR; Little, 1988) test in the *naniar* R package version 0.6.1.9 (Tierney et al., 2021) to determine whether missing data in main study variables occurred completely at random. Results from these analyses are included for each test in the Results section. All models used full information maximum likelihood estimation to deal with missing data, since it has been shown to be effective at reducing potential bias in estimated SEM coefficients caused by missing values (e.g., with up to 25% missingness; Enders & Bandalos, 2001). Model fit was assessed according to formal guidelines of a comparative fit index (CFI) > .95; a root mean square error of approximation (RMSEA) < .08; a standardized root mean square residual (SRMR) < .08; a chi-square ratio < 2; and a chi-square different significance value of $p > .05$ (i.e., not significant; Byrne, 2011; Hu & Bentler, 1999; McDonald & Ho, 2002). Additional comparative model fit indices (e.g., Bayesian information criterion and Akaike information criterion) were evaluated without formal guidelines when comparing nested models. *Post hoc* power analyses were conducted based on the goal of obtaining a RMSEA representing good fit (.08) using the *semPower* R package (Moshagen & Erdfelder, 2016). Prior to testing the main study hypotheses, a confirmatory factor analysis (CFA) was analyzed to assess the measurement model of the latent early environmental harshness variable. As all indicators (financial strain, marital harshness, and hostility symptoms) generally approximated a normal distribution, the CFA model was conducted with the default maximum likelihood estimation. The latent variable was scaled by fixing the variance to 1.0 in the CFA and all SEM models. The measurement model was determined to fit well if it met the criteria described above for acceptable fit

and each indicator loaded significantly onto the latent factor. All reported coefficients are standardized unless otherwise stated.

Research Question 1: How do perinatal risks and early environmental harshness, both individually and in combination, predict pubertal timing?

A conceptual model depicting this research question is presented in Figure 1. To address this research question, a series of SEM models were conducted. In the first model, perinatal risks and the early environmental harshness latent variable were included as independent variables predicting pubertal timing at age 11 to determine the unique effects of each type of ELS on pubertal timing. The second model included both perinatal risks and early environmental harshness as independent variables in addition to an interaction term of perinatal risks by early environmental harshness to examine whether early environmental harshness moderates the association between perinatal risks and pubertal timing.

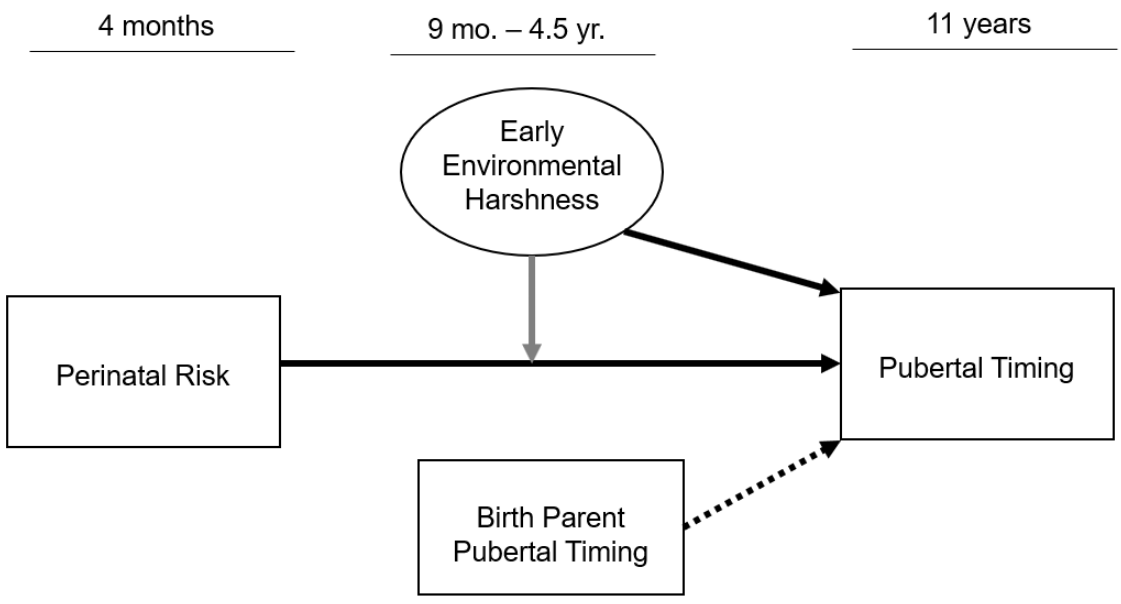

Figure 1. Conceptual model of research question 1.

To test the interaction between the observed perinatal risks variable and the latent early environmental harshness variable, a double mean centered product-indicator approach was used (see Schoemann & Jorgensen, 2021). Briefly, this involves mean centering each observed variable and indicator, calculating product indicators between each indicator and observed variable, and then mean centering each calculated product indicator. The double-mean centered product indicators are then used to create a latent interaction term that is a function of both independent variables and included as an additional predictor in the SEM model. Moderation was determined if the interaction term was statistically significant. In both models, perinatal risks was mean centered and early environmental harshness was a scaled latent variable (i.e., latent variance fixed to 1). Birth parent pubertal timing was included as a covariate in all models to control for heritable influences that contribute to pubertal timing. Fit statistics for the first model, excluding the interaction term, were used to determine model fit since fit indices for latent variable product indicator models are not appropriate to interpret (Schoemann & Jorgensen, 2021). This analysis employed the full sample of both cohorts (n = 561) and was appropriately powered to detect expected effects. Specifically, the results of a *post hoc* power analysis showed that the sample size of 561 is associated with a power of greater than 99.99% to reject an acceptable fitting model (df = 33, alpha = .05, RMSEA = .08).

Research Question 2: How do perinatal risks, early environmental harshness, and pubertal timing predict aggression?

To address this research question, a mediation model was specified in which pubertal timing mediated the effect of perinatal risks and early environmental harshness,

separately, on adolescent aggression (see Figure 2 for conceptual model). Two direct effects were modeled, one from perinatal risks to aggression and another from the latent early environmental harshness variable to aggression, to examine the extent to which each type of ELS has a direct effect on adolescent aggression. Consequently, two indirect effects through pubertal timing were also modeled, one for each type of ELS.

Birth parent pubertal timing was included as a covariate on adolescent pubertal timing to control for heritable risk, and child aggression (at age 8) was included as a covariate of adolescent aggression to control for pre-pubertal aggressive behavior patterns. In the larger study, there is only data available for Cohort 1 (n = 361) for aggression at age 8 and 15. Thus, this analysis only included Cohort 1 data. A *post hoc* power analysis revealed that the sample size of 361 is associated with a power of 99.98% to reject this model if it fit the data well (df = 29, alpha = .05, RMSEA = .08). Thus, this analysis was adequately powered with the current sample size.

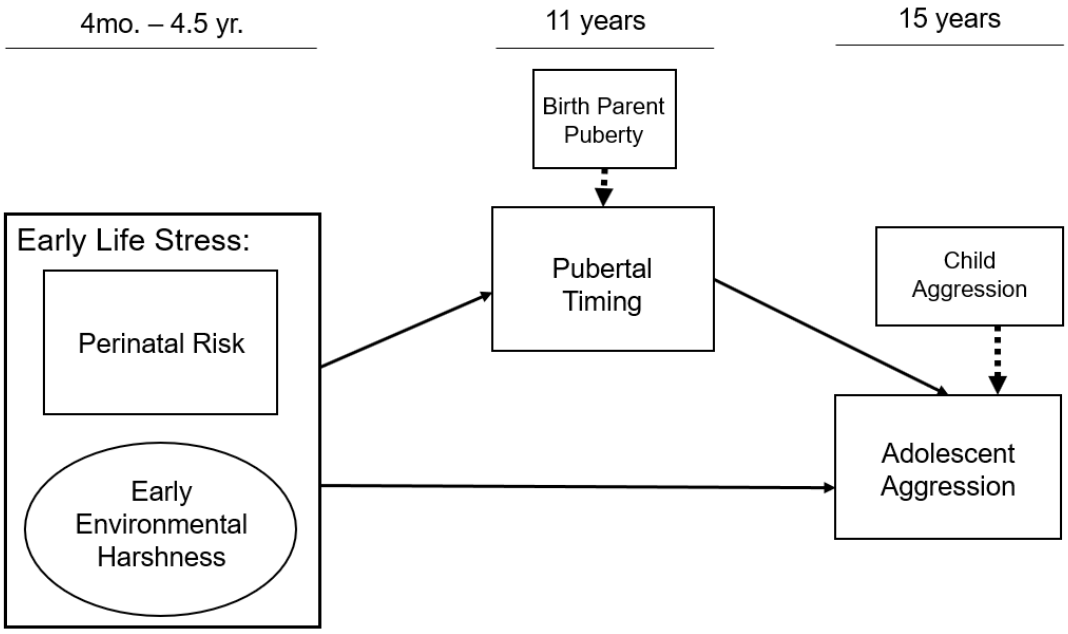

Figure 2. Conceptual model of research question 2.

Research Question 3: What sex differences exist in the relations among perinatal risks, early environmental harshness, pubertal timing, and aggression?

To address this research question, exploratory analyses were conducted to determine whether sex moderated the results of the first two research questions. A multiple group SEM analysis was conducted (see Kline, 2016) to evaluate whether there were significant sex differences in the statistical models from research question 1 and 2. The procedure was the same for both the moderation model (research question 1) and mediation model (research question 2). First, path coefficients in the models were freely estimated for both males and females. Then, main effect coefficients (e.g., regression coefficients and/or indirect effects) were constrained to be equal across sexes. Third, all regression coefficients and intercepts were constrained to be equal. In the last step, regression coefficients, intercepts, and factor loadings had equality constraints for males and females. Tests of nested model comparisons were then conducted via chi-square difference test to evaluate which model fit the data significantly better. Significant moderation (i.e., sex differences) was determined if the model with freely estimated group parameters fit the data significantly better than the constrained models. *Post hoc* power analyses revealed that with a sample size of 561 (males = 321, females = 240), power to detect a significant change in model fit for the latent interaction model (research question 1) is 99.81% (*df* difference = 16, alpha = .05, change in RMSEA from .08 to .10). Power for the mediation model (research question 2) is greater than 99.99% (*df* difference = 18, alpha = .05, change in RMSEA from .08 to .10) with the Cohort 1 sample of 361 (males = 207, females = 154). Therefore, these analyses were

appropriately powered to detect significant differences in model fit between male and females.

CHAPTER IV

RESULTS

Preliminary Analyses

Regression diagnostics. Distributions, relationships, and trends among study variables were visually inspected to check whether statistical assumptions were met (e.g., linearity, influential cases, distribution of residuals). Component residual plots showed the independent variables did have a linear relationship with the dependent variables. The assumption of multicollinearity was also met; no correlation coefficient between predictors was greater than 0.4 and all variance inflation factor (VIF) scores were below 2. The Durbin-Watson test was non-significant, and the test statistic (1.9) was in an acceptable range, indicating that the residuals were independent. There were three observations that were flagged as potentially influential cases (i.e., Cook's Distance greater than $4/n\text{-}k\text{-}1$), however, they all had low leverage values and model results did not differ significantly when the three observations were excluded. Since none of the potentially influential cases biased the results, all observations were retained for analyses. Both dependent variables, pubertal timing and aggression, were positively skewed (skew = 2.11 and 1.63, respectively) and had high kurtosis values (5.46, 2.90). Although a log transformation did approximate a normal distribution for both variables, model results did not differ between log transformed variables with maximum likelihood estimation (assumes normality) and raw, skewed variables with robust maximum likelihood estimation (robust to non-normality). Further, both pubertal timing at age 11 and adolescent aggression are considered to be truly positively skewed in the population. Therefore, raw values were included in all statistical analyses to better represent the

population distributions of these variables and estimate coefficients that are more meaningful and can be interpreted directly for each construct. Lastly, a plot of standardized residuals vs. fitted values (i.e., scale location plot) showed no clear signs of funneling. However, standardized residuals did seem to have a slight positive association with fitted values, and the Breusch-Pagan Test of non-constant error variance was significant, indicating heteroscedasticity was present. The assumption of homoscedasticity was met when aggression was log-transformed, suggesting the skewed outcome variables are the reason why there is non-constant error variance present. Since raw variables were used in all analyses, heteroscedasticity could have influenced the precision of model parameters and overestimate statistical significance.

Missingness mechanism. Little's MCAR test was significant (X^2 (42) = 77.70, p < .05). This indicates that missing data in this analytical sample was not MCAR. It is likely that the cause of missing data is either from regular attrition associated with the longitudinal nature of the study design and can be explained using other observed variables in the dataset (i.e., missing at random). There are few theoretical reasons to believe that the missing values were a result of the potential response of each corresponding variable (i.e., missing not at random). Therefore, the missing data in this sample is considered missing at random (MAR). Full information maximum likelihood estimation has been shown to produce unbiased estimates for SEM models with missing at random values (Enders & Bandalos, 2001). Further, statistical results did not differ significantly when models were conducted with list-wise deletion, compared to full information maximum likelihood. Thus, it is unlikely that missingness in this sample biased results.

Measurement model. The measurement model for early environmental harshness was saturated (i.e., $df = 0$). Thus, model fit indices are not applicable and not reported. For the early environmental harshness latent variable, factor loadings were significant for all indicators ($p < .001$). The standardized factor loadings were 0.21, 0.67, 0.51 for financial strain, marital harshness, and hostility symptoms, respectively. Although the standardized loading for financial strain was low (0.21), it was retained for all analyses because it was still a significant indicator and represents an external source of stress (i.e., outside the family) that is an important consideration for early pubertal timing and fast life history strategies (e.g., Belsky et al., 2015; Ellis & Essex. 2007) that is not captured in the other two indicators.

Sample characteristics. Descriptive statistics and bivariate correlations for study variables are presented in Table 3. The results from Welch's t-test revealed no significant differences between male and female children for perinatal risks, pubertal timing, aggression, or any early environmental harshness indicator (i.e., financial strain, marital harshness, and hostility symptoms; $p > .05$). Results from a series of one-way ANOVAs showed there were no differences by child race/ethnicity for any study variable (i.e., financial strain, marital harshness, hostility symptoms, perinatal risks, pubertal timing, and aggression; $p > .05$).

Table 3. *Descriptive statistics and bivariate correlations for study variables (N* = 561).

Variables	1	2	3	4	5	6	7	8
1. Financial strain	-							
2. Marital harshness	.14*	-						
3. Hostility symptoms	.11*	.34*	-					
4. Perinatal risks	.01	.05	.05	-				
5. Pubertal timing	.10	.03	-.04	.03	-			
6. Adolescent aggression (age 15)	.09	-.06	.13	-.07	.04	-		
7. Birth parent puberty	.01	.06	.01	.03	.02	-.02	-	
8. Child aggression (age 8)	.21*	.05	.25*	-.04	.06	.61*	.01	-
n	588	555	479	561	396	172[1]	493	247[1]
Mean	3.63	26.10	1.94	4.65	1.96	4.10	2.80	5.20
SD	1.14	6.66	1.12	3.18	1.09	4.33	0.73	4.59
Range	2-7.67	14.67-58.75	0-9	0-14	1-7	0-24	1-5	0-23.5

Note: N/n = number of observations, [1]Data only available for Cohort 1 (*n* = 361), *SD* = standard deviation. Range refers to the minimum and maximum observed values, *$p < .05$.

Research Question 1 Results: How do perinatal risks and early environmental harshness, both individually and in combination, predict pubertal timing?

The SEM with perinatal risks and early environmental harshness predicting pubertal timing had good model fit, χ^2 (df = 8) = 7.19, p > .05; CFI = .99; RMSEA = .01; SRMR = .03. Results did not support the hypothesis that perinatal risks and early environmental harshness would predict earlier pubertal timing. In the first model, excluding the interaction term, neither perinatal risks nor early environmental harshness significantly predicted pubertal timing (p > .05). Similarly, results from the second model showed no significant interaction between perinatal risks and early environmental harshness on pubertal timing (p > .05). Standardized coefficients for the interaction model are presented in Figure 3.

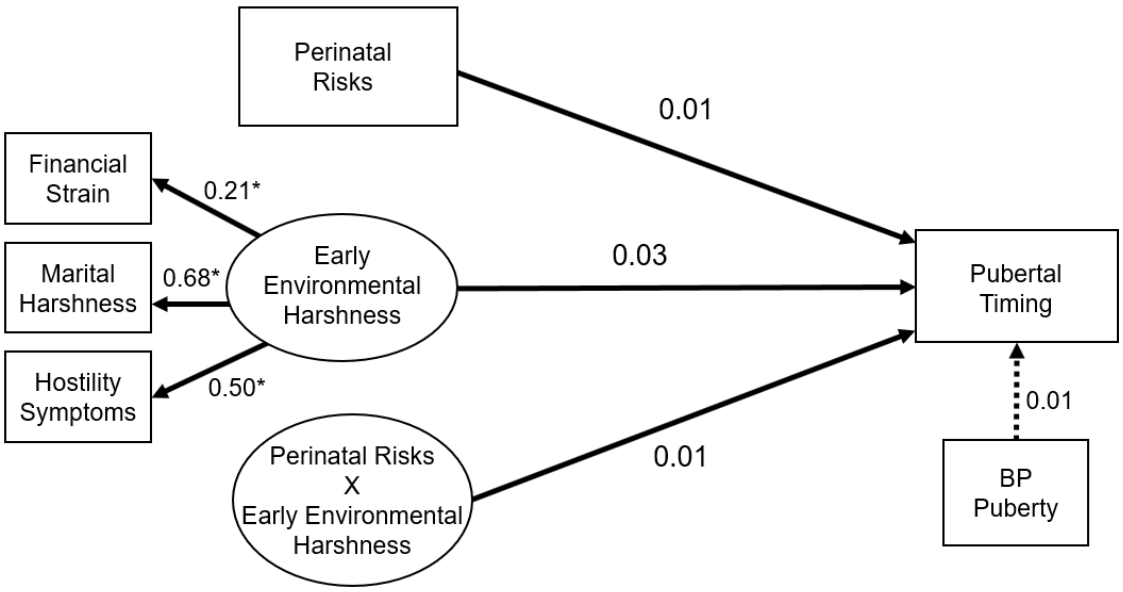

Figure 3. Model results for research question 1 interaction model, *p < .05.

Research Question 2 Results: How do perinatal risks, early environmental harshness, and pubertal timing predict aggression?

The SEM with pubertal timing mediating the relationship of both perinatal risks and early environmental harshness on adolescent aggression had acceptable model fit, χ^2 (df = 15) = 40.15, $p < .05$; CFI = .84; RMSEA = .07; SRMR = .07. However, results did not support the hypothesis that pubertal timing mediates the association between both types of ELS on adolescent aggression. The indirect effect of perinatal risks and early environmental harshness on adolescent aggression through pubertal timing was not significant ($p > .05$). Further, there were no significant direct effects from perinatal risks to aggression or from early environmental harshness to aggression. Interestingly, results showed a significant positive association between perinatal risks and pubertal timing (β = 0.12, $p < .05$). The full model results are presented in Figure 4.

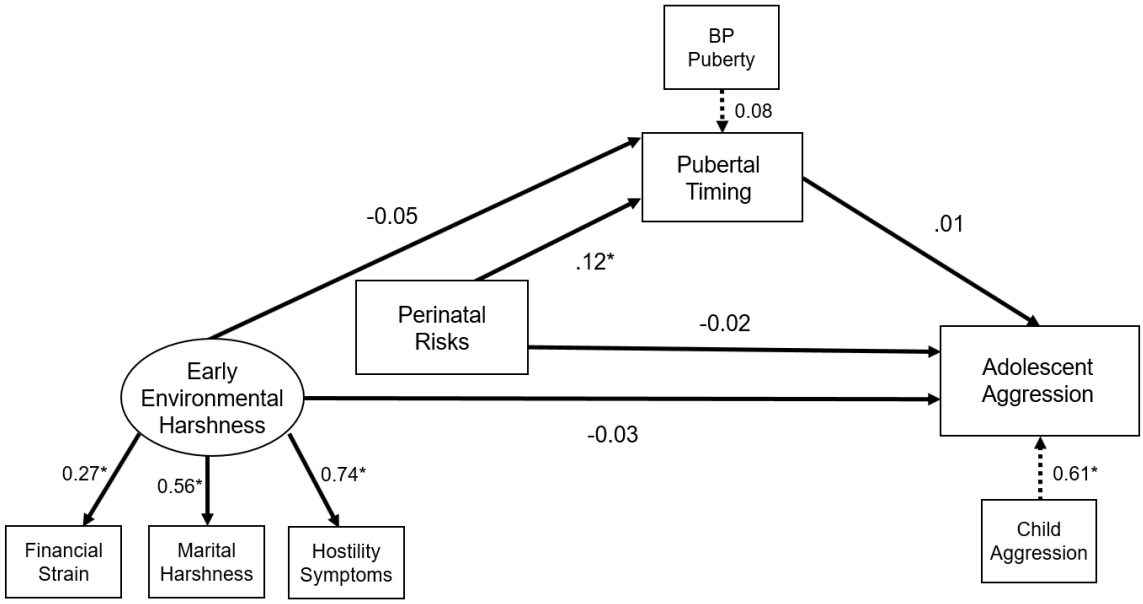

Figure 4. Model results for research question 2 mediation model, *$p < .05$.

Research Question 3 Results: What sex differences exist in the relations among perinatal risks, early environmental harshness, pubertal timing, and aggression?

Contrary to my hypotheses, exploratory results indicated that there were no significant sex differences in the previous model. A chi-square test of model comparisons (see Table 4) showed no significant change in model fit when model coefficients were freely estimated for males and females compared to when coefficients, intercepts, or loadings were constrained to be equal across groups ($p > .05$). This was true for the models predicting pubertal timing with perinatal risks and early environmental harshness (research question 1) and the model with pubertal timing mediating the relation between both types of ELS on adolescent aggression (research question 2). Although there were no significant sex differences, there were some interesting results when SEM models were conducted separately for males and females. For females, the early environmental harshness latent variable did not have significant factor loadings in the interaction model with perinatal risks and early environmental harshness predicting pubertal timing. Further, the financial strain indicator did not significantly load onto the latent environmental harshness variable for females in the model with pubertal timing mediating the link between both types of ELS and adolescent aggression. Interestingly, when the mediation model was estimated separately for males and females, results showed a significant direct path from early environmental harshness to aggression only for females. Full model results of the freely estimated models are presented in Figure 5 for the interaction model (research question 1) and Figure 6 for the mediation model (research question 2).

Table 4. *Chi-square nested model comparison results to explore sex differences.*

Model	Comparison	*df*	df_{diff}	χ^2	χ^2_{diff}	*p*
RQ1 Interaction Model						
1. Free parameters		47		1,795.8		
2. Constrained paths	Model 1 vs. Model 2	50	3	1,796.0	0.2	.93
3. Constrained paths and intercepts	Model 1 vs. Model 3	53	6	1,802.0	6.2	.50
4. Constrained paths, intercepts, and loadings	Model 1 vs. Model 4	63	16	1,813.1	17.3	.56
RQ2 Mediation Model						
1. Free parameters		30		55.61		
2. Constrained paths	Model 1 vs. Model 2	39	9	60.64	5.03	.69
3. Constrained paths and intercepts	Model 1 vs. Model 3	46	16	78.69	23.08	.06
4. Constrained paths, intercepts, and loadings	Model 1 vs. Model 4	48	18	79.85	24.24	.14

Note. RQ = research question, *df* = degrees of freedom, df_{diff} = change in degrees of freedom, χ^2 = chi-square statistic, χ^2_{diff} = chi-square difference, *p* = p-value.

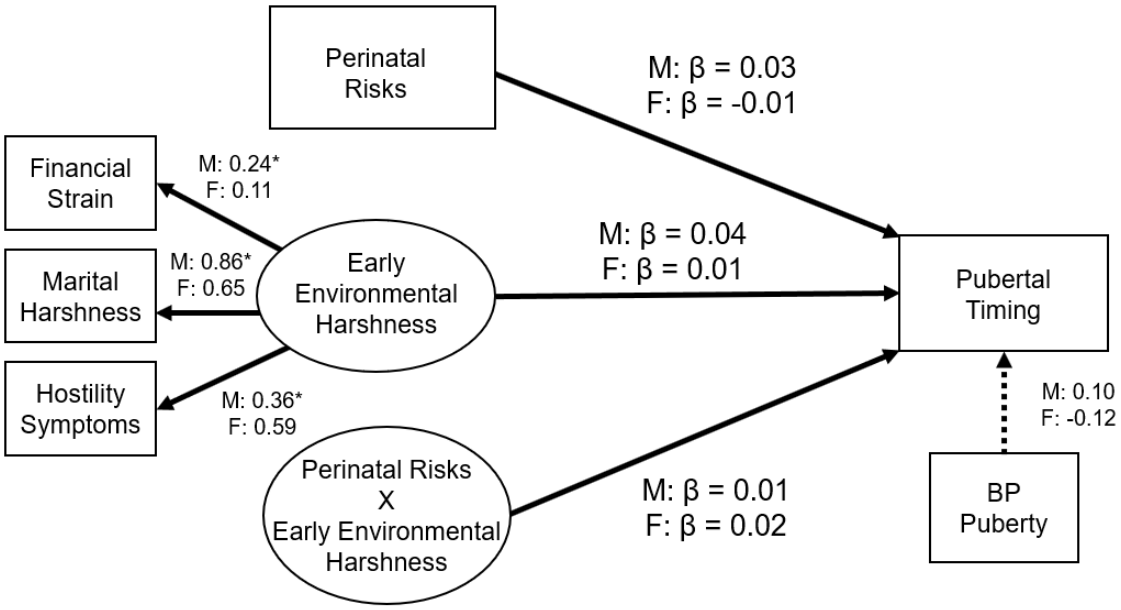

Figure 5. Model results by sex for research question 1 interaction model, p < .05.

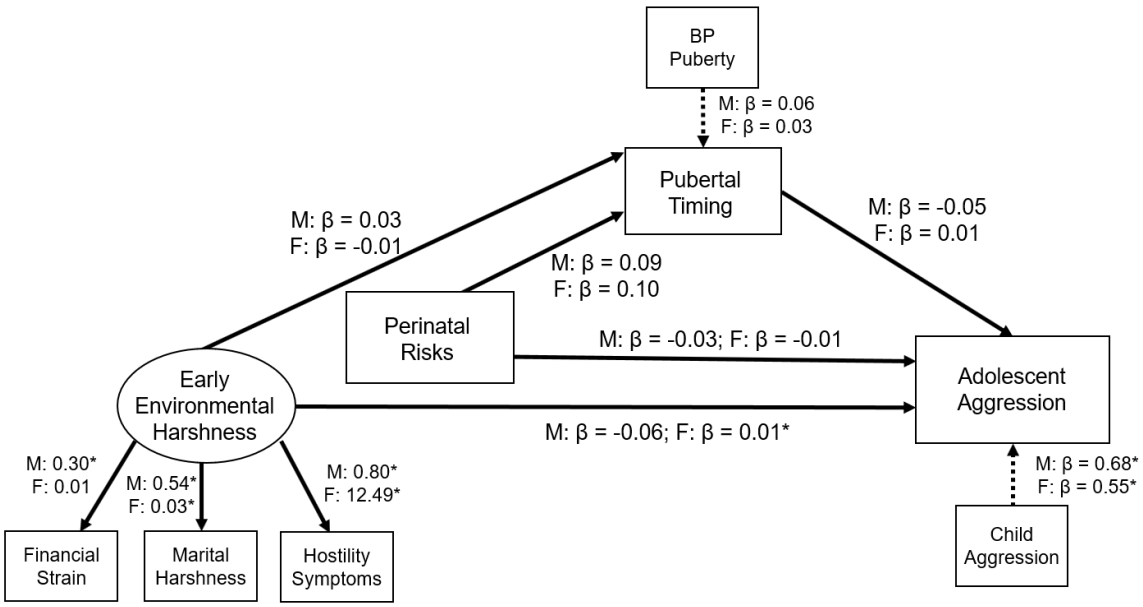

Figure 6. Model results by sex for research question 2 mediation model, $p < .05$.

Exploratory Results

Since neither perinatal risks nor early environmental harshness significantly predicted pubertal timing or aggression, additional *post hoc* exploratory analyses were conducted to explore the relation between other types of ELS (e.g., unpredictable ELS, harsh parenting and discipline; see Alternative Measures in Methods section above) on pubertal timing and aggression. Bivariate correlations and descriptive statistics for alterative measures and study outcomes are presented in Table 5. Out of all the alternative measures, only marital instability ($r = .13$) and marital transitions ($r = .21$) were significantly correlated with pubertal timing ($p < .05$). There were only three significant correlates of adolescent aggression (age 15), parent hostility ($r = .18$), overreactive parenting ($r = .22$), and child aggressive behavior ($r = .61$; $p < .05$). For child aggression (age 8), there were more significant associations observed. Child aggression was significantly correlated with corporal punishment ($r = .23$), parent

hostility (r = .44), overreactive parenting (r = .33), harsh discipline (r = .27), and inconsistent discipline (r = .19; $p < .05$).

Unpredictable ELS. The results from the bivariate correlations suggested that measures of unpredictable ELS (e.g., marital instability, marital transitions) are associated with pubertal timing, compared to measures of environmental or parenting harshness. To formally test whether unpredictable ELS predicts pubertal timing, a SEM was conducted. Controlling for heritable risk (i.e., birth parent puberty), pubertal timing was regressed onto a latent unpredictable ELS variable with four indicators: marital instability (standardized loading = .54), marital transitions (.44), parent inconsistency (.59), and inconsistent discipline (.42). The measurement model had acceptable fit, χ^2 (df = 2) = 15.36, $p < .05$; CFI = .91; RMSEA = .10; SRMR = .04. The full SEM model had decent model fit, χ^2 (df = 9) = 31.82, $p < .05$; CFI = .85; RMSEA = .07; SRMR = .05. As the purpose of this analysis was to explore the relation between unpredictable ELS and pubertal timing, rather than build a good fitting model, no actions were taken to improve model fit. Results from the SEM model revealed that unpredictable ELS was a significant predictor of pubertal timing (standardized β = .19, $p < .05$). This relationship was not significantly moderated by sex. Results from additional models with aggression as the outcome showed that unpredictable ELS did not significantly predict child or adolescent aggression.

Table 5. *Descriptive statistics and bivariate correlations for alternative postnatal ELS measures (N* = 561).

Variables	1	2	3	4	5	6	7	8	9	10	11
1. Corporal punishment	-										
2. Parental hostility	.33*	-									
3. Overreactive parenting	.42*	.65*	-								
4. Harsh discipline	.49*	.45*	.50*	-							
5. Inconsistent discipline	.14*	.36*	.36*	.36*	-						
6. Marital instability	-.05	.17*	.08	.01	.16*	-					
7. Marital transitions	-.10*	.04	-.03	-.07	.16*	.30*	-				
8. Parent inconsistency	.04	.20*	.17*	.20*	.32*	.28*	.18*	-			
9. Pubertal timing	-.05	.01	.01	-.07	.04	.13*	.21*	-.06	-		
10. Child aggression	.23*	.44*	.33*	.27*	.19*	-.03	.07	.03	.06	-	
11. Adolescent aggression	.14	.18*	.22*	.15	.14	-.10	.07	.06	.04	.61*	-
n	450	520	556	452	451	555	538	438	396	247[1]	172[1]
Mean	1.30	9.59	2.00	1.49	2.02	5.95	1.34	1.65	1.96	5.20	4.10
SD	0.33	2.48	0.48	0.30	0.38	1.70	0.84	0.65	1.09	4.59	4.33
Range	1-2.5	5-23	1-3.8	1-3	1.2-3.2	5-16.67	0-6	1-4.5	1-7	0-23.5	0-24

Note: N/n = number of observations, [1]Data only available for Cohort 1 (*n* = 361), *SD* = standard deviation. Range refers to the minimum and maximum observed values, *$p < .05$.

Harsh parenting. As seen in the bivariate correlation results (main and exploratory, Tables 3 & 5), there are harsh postnatal environment and parenting measures that are correlated with aggression, compared to unpredictable measures. This is especially relevant for aggression at age 8. A series of linear regression analyses were conducted to explore which correlates of aggression are strongest. When child aggression (age 8) was regressed on all the correlated variables (i.e., corporal punishment, parental hostility, overreactive parenting, harsh discipline, inconsistent discipline, marital harshness, and financial strain), the only parental hostility was significant (unstandardized $\beta = 0.10$, $p < .05$). The next model examined the extent to which parent hostility, overreactive parenting, and child aggression were associated with adolescent aggression (age 15). Child aggression was significantly associated with adolescent aggression (unstandardized $\beta = 0.11$, $p < .05$) over and above the effect of both harsh parenting variables. Results from models with pubertal timing as the outcome showed that harsh parenting was not significantly associated with pubertal timing.

Sex differences. To further examine how sex influences the associations among ELS, pubertal timing, and aggression, exploratory correlation tables were tabulated by child sex. Correlations among alternative measures of ELS (e.g., harsh parenting and unpredictable ELS) are presented for males and females in Tables 6 and 7, respectively. There were 11 bivariate associations that were different, based on statistical significance of the correlation coefficient, between males and females. For males, corporal punishment was significantly correlated with child aggression ($r = .28$, $p < .05$), This relation was non-significant among females. Interestingly, pubertal timing was significantly associated with child aggression for males ($r = .19$, $p < .05$), but not females.

Table 6. *Descriptive statistics and bivariate correlations for alternative postnatal ELS measures among male children (n =* 321*).*

Variables	1	2	3	4	5	6	7	8	9	10	11
1. Corporal punishment	-										
2. Parental hostility	.42*	-									
3. Overreactive parenting	.46*	.65*	-								
4. Harsh discipline	.57*	.43*	.52*	-							
5. Inconsistent discipline	.14*	.31*	.36*	.29*	-						
6. Marital instability	-.02	.12*	**.04**	-.01	**.10**	-					
7. Marital transitions	-.07	.01	-.04	-.12	**.06**	.33*	-				
8. Parent inconsistency	.09	.21*	**.20***	.20*	.33*	.27*	.13*	-			
9. Pubertal timing	-.01	-.05	-.08	-.05	-.02	**.06**	.19*	-.04	-		
10. Child aggression	**.28***	.39*	.30*	.21*	**.07**	-.12	**-.07**	.01	**.19***	-	
11. Adolescent aggression	.08	.16	**.19**	.11	**.02**	-.10	.15	.01	.11	.64*	-

Note: Bolded values indicate coefficients that have difference statistical significance between male and female children, *$p < .05$.

Table 7. *Descriptive statistics and bivariate correlations for alternative postnatal ELS measures among female children (n =* 240*).*

Variables	1	2	3	4	5	6	7	8	9	10	11
1. Corporal punishment	-										
2. Parental hostility	.18*	-									
3. Overreactive parenting	.36*	.63*	-								
4. Harsh discipline	.40*	.49*	.48*	-							
5. Inconsistent discipline	.14	.43*	.36*	.44*	-						
6. Marital instability	-.10	.23*	**.14***	.02	**.24***	-					
7. Marital transitions	-.15*	.11	-.02	-.02	**.28***	.27*	-				
8. Parent inconsistency	-.03	.17*	**.10**	.20*	.30*	.30*	.25*	-			
9. Pubertal timing	-.09	.07	.11	-.10	.12	**.23***	.24*	-.07	-		
10. Child aggression	**.17**	.48*	.35*	.33*	**.35***	.11	**.22***	.06	**-.01**	-	
11. Adolescent aggression	.21	.21	**.25***	.18	**.29***	-.10	-.01	.11	.01	.57*	-

Note: Bolded values indicate coefficients that have difference statistical significance between male and female children, *$p < .05$.

Among females, inconsistent discipline ($r = .35$) and marital transitions ($r = .22$) were significantly associated with child aggression ($p < .05$). Further, overreactive parenting ($r = .25$) and inconsistent discipline ($r = .29$) were significantly correlated with adolescent aggression among females ($p < .05$). There was also a significant association between marital instability and pubertal timing for females ($r = .23$, $p < .05$). None of these associations were significant among males.

Separate regression analyses were conducted to explore which sex-specific correlates had the strongest association with child aggression. When child aggression was regressed onto all significant correlates for females (i.e., parental hostility, overreactive parenting, harsh discipline, inconsistent discipline, and marital transitions), only parental hostility significantly predicted female aggression at age 8 (unstandardized $\beta = .10$, $p < .05$). Among males, pubertal timing (unstandardized $\beta = .50$) was still associated with child aggression even after accounting for corporal punishment, parental hostility, overreactive parenting, and harsh discipline ($p < .05$).

CHAPTER V

DISCUSSION

The purpose of this study was to investigate relations among perinatal risks, early environmental harshness, pubertal timing, and adolescent aggression in a longitudinal sample of adopted children. The present study advances the current literature by utilizing an adoption design to isolate the unique effects of prenatal and postnatal stressors on pubertal timing and aggression within a life history framework. Further, this is one of the first studies to investigate sex differences in how perinatal risks and early environmental harshness relate to pubertal timing and adolescent aggressive behavior.

Summary of Main Results

Contrary to the hypothesis for research question 1, analyses revealed no significant effects of perinatal risks or early environmental harshness on pubertal development at age 11. Further, there was no significant interaction between perinatal risks and early environmental harshness on pubertal timing in this sample. These results are in contrast with previous literature documenting an association between prenatal ELS and earlier pubertal timing (e.g., Bräuner et al., 2021; Duchesne et al., 2017), as well as a positive association between postnatal ELS and pubertal development (e.g., Colich et al., 2020; Henrichs et al., 2014).

The current study also examined the extent to which perinatal risks and early environmental harshness predict adolescent aggression, as well as whether pubertal timing mediated the association between both types of ELS and aggression. Results did not support the study hypotheses. There were no significant direct effects from perinatal risks or environmental harshness on adolescent aggression. This contradicts prior

research that shows a positive association between both prenatal ELS (Buchmann et al., 2014) and postnatal ELS (e.g., Belsky et al., 2010; Winiarski et al., 2018) on aggressive behavior. The current results also showed there was no significant mediation from either type of ELS to aggression through pubertal timing. This is consistent with prior work revealing that pubertal timing only mediates the link between ELS and sexual risk behaviors and not the link between ELS and other risk behaviors such as substance use or aggression (Belsky et al., 2010). These results suggest that life history theory may only be relevant to explain development of sexual risk behaviors that are directly related to evolutionary success, rather than adolescent risk behaviors more broadly. Interestingly, there was a small, significant association between perinatal risks and pubertal timing within the mediation model. However, since this relation was not significant in the first analysis with pubertal timing as the outcome (research question 1) and the magnitude is small within the mediation model, this result should be interpreted with caution.

The final aim of this study was to explore sex differences in the relations among perinatal risks, early environmental harshness, pubertal timing, and aggression that were examined in the first two research questions. Contrary to the hypothesis, there were no significant sex differences among these factors that could potentially explain the null findings from the first two models. There was no significant effect of perinatal risks or early environmental harshness on pubertal timing for male or female children, and pubertal timing did not mediate the effect of either type of ELS on adolescent aggression. Although there was a statistically significant association between early environmental harshness and adolescent aggression for females, not males, this effect does not appear to be practically meaningful (i.e., it is very small in magnitude) and seems to be mostly

driven by adoptive parents' hostility symptoms rather than all types of early environmental harshness (see factor loadings in Figure 6).

There are a few different potential explanations for the current results across all three research questions. First, the contradictory results could be due to disproportionate levels of prenatal and postnatal ELS compared to the general population. Previous studies using this sample have documented that adoptive households have substantially higher levels of socioeconomic status and backgrounds compared to birth parents (see Leve et al., 2019), suggesting that this sample may have lower levels of postnatal ELS than in the general population. Further, this sample included those from domestic adoptions placed within the first 90 days of birth, suggesting they have experienced fewer stressors (i.e., less risk) associated with lengthy adoption processes can be present in at risk adoption samples. This sample has also been shown to have experienced higher levels of perinatal risks compared to the national average (Marceau et al., 2016). These differences in ELS measures and experiences, coupled with the low variability observed for pubertal timing, could explain why there were no significant associations found among perinatal risks, early environmental harshness, pubertal timing, and adolescent aggression in the current study (e.g., not enough information available to estimate a pattern or relation).

Second, the observed null results could be due to the way perinatal risks and early environmental harshness were measured. Since the current study involved secondary data analysis, the available measures of ELS may not be the best way to conceptualize prenatal and postnatal ELS as it relates to fast life history strategies. Many studies that have documented an association between ELS and pubertal timing include measures of extreme environmental harshness (e.g., experiences of violence). Maternal exposure to

stressful life events during pregnancy (Bräuner et al., 2021) and prenatal maternal stress associated with experiencing a natural disaster (Duchesne et al., 2017) have been found to lead to earlier pubertal timing in girls. For postnatal ELS, studies show that pubertal timing and subsequent adolescent externalizing behaviors (e.g., aggression) is related to extreme threat-based stressors (e.g., physical/sexual abuse, exposure to domestic violence, neighborhood violence) rather than deprivation related stressors (e.g., financial insecurity/poverty and neglect; Colich et al., 2020). Additionally, postnatal ELS has been shown to lead to early pubertal timing only when many different adversities are experienced (e.g., 5+ adversities; Henrichs et al., 2014). More recently, Dinh et al. (2022) showed that only childhood exposure to violence and poor health, not general environmental harshness or unpredictability, predicted pubertal timing. Therefore, the associations between ELS and pubertal timing, and between ELS and aggression, could depend on the type and/or severity of the environmental stressor. The current measures do not represent these more extreme harsh types of external stressors which could explain the null effects of perinatal risks and early environmental harshness on both pubertal timing and aggression.

Lastly, it is possible that the current results are not biased due to sample and measurement restrictions and are an accurate representation of the limits to life history theory. This may be especially true for the non-significant mediation of pubertal timing between ELS and adolescent aggression, as this result is consistent with prior research suggesting the mediating effect of pubertal timing is restricted to adolescent sexual risk behaviors (e.g., Belsky et al., 2010). Thus, it is likely that outcomes associated with fast life history strategies do not encompass broader risk behaviors, such as aggression, but

are limited to sexual risk behaviors directly related to reproductive evolutionary success. Findings also revealed that child aggression was a significant predictor of adolescent aggression in all relevant models. This could mean that there are other risk factors, related to life history theory or not, associated with child aggression that were not considered in the current model. Child aggression peaks around age 10, and children with aggression are more likley to show aggression in adolescence (Brame et al., 2001). Therefore, it is possible that child aggression fully mediates the association between ELS and adolescent aggression, which would explain the null findings between ELS and adolescent aggression in models controlling for pre-pubertal aggression.

Summary of Exploratory Results

Initially, only harsh postnatal environmental stressors were examined so the results for postnatal ELS could be compared to the effects for prenatal ELS, which includes measures more indicative of a harsh prenatal environment than unpredictability. However, since results showed that neither harsh prenatal ELS nor harsh postnatal ELS predicted pubertal timing or aggression, additional exploratory analyses were conducted to investigate the relation between other types of ELS (e.g., unpredictable ELS and harsh parenting) on pubertal timing and aggression. Observed patterns among bivariate correlations of unpredictable ELS, harsh parenting and discipline, pubertal timing, and aggression suggested that unpredictable ELS measures were associated with pubertal timing, rather than harsh environmental or parenting factors. This motivated an additional analysis to test whether unpredictable ELS predicted pubertal timing above and beyond the effect of heritable risk (i.e., birth parent pubertal timing).

Unpredictable ELS, representing marital instability, marital transitions, parent inconsistency, and inconsistent discipline experienced in the first five years of life, did significantly predict earlier pubertal timing at age 11 for both males and females. This is somewhat in line with previous research documenting a relation between unpredictable and harsh environments on pubertal timing (Klopack et a., 2020; Kogan et al., 2015), however most studies combine across both harsh and unpredictable measures and do not investigate separate effects of both types of ELS. Similarly, life history theory does not make a distinction between potential separate effects of unpredictable and harsh types of ELS on life history outcomes (e.g., Ellis et al., 2009). In a recent study comparing different environmental ELS on facets of life history theory (e.g., pubertal timing, numerous reproductive success outcomes), researchers found that only harsh environments (i.e., exposure to violence) predicted pubertal timing; there was no significant effect of unpredictable ELS on puberty (Dinh et al., 2022). Additionally, research on antecedents of pubertal timing often employ solely female samples (e.g., Belsky et al., 2015; Ellis & Essex, 2007). Studies that compare effects among males and females are inconsistent, some suggest the effects are similar both sexes (e.g., Sun et al., 2017), while others have documented important differences (e.g., Semiz et al., 2009). These findings highlight a need for more research comparing the effects of harsh and unpredictable ELS on life history trajectories, especially concerning pubertal timing, among both males and females.

The exploratory results also suggest that child aggression (age 8) is related to harsh parenting and discipline measures, and not unpredictable ELS. Further, correlation results from the main analyses revealed that measures of early environmental harshness

were also associated with child aggression, but not adolescent aggression. Therefore, environmental and parenting harshness may be key factors related to child aggression, but not adolescent aggression or pubertal timing. Not surprisingly, the strongest correlate of adolescent aggression was child aggression, and child aggression remained a significant predictor of adolescent aggression even when controlling for harsh parenting factors. These findings are supported by previous literature showing that harsh environmental stressors lead to the development of aggression and violence (Fongay, 2004) and that ELS is associated with child aggression (Jonson-Reid et al., 2010). Since child aggression peaks around age 10 and is a strong predictor of later adolescent aggression (Brame et al., 2001), future investigations on the development of aggression through a life history lens should include younger samples of children. Further, studies aimed at elucidating specific factors of adolescent aggression need to account for levels of child aggression and partial out factors associated with child aggression to understand unique predictors, mechanisms, and differences in child and adolescent aggressive development.

To further explore how environmental and parenting harshness related to aggression during childhood, analyses were conducted to determine which environmental factor had the strongest effect on child aggression. Results showed that parental hostility predicted child aggression over and above the effects of any other harsh ELS variable. This result is supported by previous work documenting that hostile parenting predicts child aggression over and above other family measures (e.g., low income, early childbearing, single parent household; Côté et al., 2007). The link between parental hostility and child aggression can be explained with social learning theory (Bandura,

1973), in which children learn that hostile behaviors can be an effective way to meet their goals, and thus adopt aggressive behaviors during interactions with others. Similarly, social information processing theory suggests that parental hostility can teach children to develop a hostile attribution bias (e.g., tendency to interpret ambiguous social cues as negative or threatening), which causes children to feel unduly provoked and respond with aggression (Crick & Dodge, 1994). Future studies should aim to examine potential mechanisms, social and evolutionary, that may account for the relation between parental hostility and child aggression.

The final exploratory analyses were conducted to examine sex differences in the exploratory results. Results did show different patterns of significant associations between males and females. No measure of unpredictable ELS or harsh parenting was correlated with male pubertal timing. Among females, greater marital instability in adoptive families was associated with earlier pubertal timing at age 11. This result is consistent with prior literature suggesting ELS is associated with earlier pubertal timing in females (e.g., Ellis & Essex, 2007), as well as literature showing no relation between harsh parenting and pubertal timing for males (Klopack et al., 2020). However, other studies have documented a significant relation between harsh and unpredictable ELS and male puberty (Kogan et al., 2015), suggesting further work is needed to identify how ELS functions differently for males and female life history strategies and determine whether ELS is a significant predictor of male pubertal timing.

For aggression, both corporal punishment and pubertal timing were correlated with child aggression for males, but not females. Surprisingly, pubertal timing was the only significant correlate of child aggression for males after accounting for different

types of harsh parenting practices (i.e., corporal punishment, parental hostility, overreactive parenting, and harsh discipline). As child aggression (age 8) was assessed prior to pubertal timing (age 11), this result could indicate there is a potential extraneous consideration that could contribute to both of these factors among males. Within the life history framework, it is possible that the development of stress regulation systems (e.g., HPA axis and associated regulatory processes) could explain both increased child aggression and earlier pubertal timing among males (see Joos et al., 2018). According to the adaptive calibration model (Del Giudice et al., 2011), stress response systems are calibrated by early environments to function in high or low stress conditions. Thus, stress response systems are considered an underlying mechanism linking ELS to changes in pubertal development. Subsequent research should aim to explore how ELS, stress regulation processes, and child aggression operate concurrently to influence pubertal timing, especially among males.

In contrast with the results for males, measures of unpredictable ELS (i.e., inconsistent discipline and marital transitions) were associated with child aggression for females, but not for males. However, out of all the significant correlates of female aggression at age 8, parental hostility remained the strongest predictor of female aggression. This is the same pattern of results that were observed for the full sample, and the results for males when not considering pubertal timing, suggesting the developmental trajectory of aggression, especially related to familiar factors, might function and progress similarly for both sexes. This is in contrast with prior work documenting sex differences in the antecedents of aggressive behavior (Archer, 2004), and further

highlights the need for simultaneously evaluating both males and females when investigating the causes or development of aggression.

Strengths and Limitations

The current study has several strengths. First, although the sample size was slightly smaller than the average sample size of many large-scale investigations of life history theory (e.g., Belsky et al., 2010; Colich et al., 2020; Dinh et al., 2022; Kogan et al., 2015), each analysis was appropriately powered to detect significant small effects. Thus, it is not likely that the presence of null results in tested models is due to an issue with power, but perhaps other differences in the analytical sample (e.g., adopted children, including both parent reports with male and female children) or confounding variables that were not assessed in the current study (e.g., parent sex, stress regulation systems). Second, it was possible to statistically compare the effect of perinatal risks and early environmental harshness on pubertal timing and aggression because of the complex adoption design that separates postnatal rearing environments from potentially confounding prenatal and heritable influences. There are few study designs that allow this opportunity to investigate unique effects of prenatal and postnatal ELS. Relatedly, the inclusion of birth parent puberty as a control for child pubertal timing was another strength of this study. It afforded the opportunity to investigate how ELS predicted child pubertal timing after accounting for heritable risk which is a common extraneous confound in previous literature. Third, this is one of the few studies to conceptualize adolescent aggression as a specific life history outcome associated with ELS and pubertal timing. Thus, these results add to a small, but growing, literature which suggests life history theory may only be applicable for sexual risk behaviors. Another important

strength of this study was the inclusion of both male and female children, and more specifically, investigating significant sex differences in how ELS and pubertal timing relate to aggression. Many studies have included only female or male samples when investigating factors related to pubertal timing or aggression and were not able to compare how relations differ based on sex.

The present study also had several limitations that could have influenced the results. As mentioned above, the way that ELS, both perinatal risks and early environmental harshness, was assessed may not be the most ideal way to measure ELS as it relates to fast life history strategies. Since this study utilized data from a larger study that was not focused on life history theory, only available measures of early environmental harshness could be considered, and there was only one measure of perinatal risks available to capture prenatal ELS. Further, this sample of adoptive parents has been shown to have lower levels of postnatal ELS compared to the general population. Therefore, these ELS measures may not represent environmental stressors that are harsh enough to trigger a fast life history strategy. Another limitation of this study was that data was only available for one cohort for measures of aggression, therefore, results from the full sample could not be estimated. However, power analyses did reveal that there was adequate power to detect small to medium effects with Cohort 1 data. An additional limitation of the current study was the inability to control for potential bidirectional effects (e.g., aggressive children causing harsh parenting). While I tried to reduce the influence of bidirectional effects by including only measures of harsh environmental ELS, rather than harsh parenting, in main analyses, there was no way to statistically model or control for bidirectional effects since measures of environmental

harshness were combined across all early life timepoints to represent early environmental harshness within the first five years of life. However, since there were no substantial effects from the main analyses, there are likely no bidirectional results that would have been revealed if they were modeled. Another limitation to note is that this study did not control for adoption openness (i.e., amount of contact, and communication between birth and adoptive parents) which is a common covariate in gene-environment interplay studies using the EGDS adoption sample and could be related to postadoption adjustment that may result in lower levels of postnatal ELS (Ge et al., 2008). Finally, all ELS measures were parent report, and pubertal timing and aggression measures were self-report.

Future Directions

Future research should continue to examine different ELS factors that relate to pubertal timing and aggression. Future studies should investigate both unpredictable and harsh measures of ELS during the prenatal and postnatal period and the extent to which each factor leads to pubertal timing and aggression among both males and females. Subsequent research should also aim to address the limitations of the current study. The current results highlight the need for further research to disentangle the unique effects of prenatal and postnatal ELS on pubertal timing and adolescent risk behaviors. Future studies should include measures of prenatal and postnatal ELS with varying levels of severity and frequency (e.g., extreme harsh and unpredictable stressors). Studies focused on examining the application of life history theory for adolescent risk behaviors should assess different types of outcomes and employ statistical models to directly compare the effect of accelerated life history strategies on different types of adolescent risk behaviors. Future studies should also simultaneously compare the effects of unpredictable ELS and

harsh ELS on pubertal timing and aggressive behavior, while exploring sex differences in those associations.

The exploratory results from the current study also highlight important associations that future work should aim to reproduce and build on. Since there were no statistically significant differences among factors related to pubertal timing and aggression between males and females, future studies should not exclude males in studies of pubertal timing or exclude females in studies of aggression, unless there is a justified and reported reason to do so. On the other hand, exploratory associations revealed there are different significant factors related to female aggression compared to male aggression. Future studies should aim to reproduce these effects and investigate potential mechanisms that could explain significant effects. Specifically, the current study found a strong association between hostile parenting and child aggression. Future investigations should explore potential intervening mechanisms that could account for that link (e.g., stress response systems). Future research could also explore different mechanisms that could explain associations between child aggression and pubertal timing among males, unpredictable ELS and pubertal timing among both males and females, or between child aggression and adolescent aggression. All of which were significant exploratory results in the current study.

Conclusion

The current study contributes to the literature by highlighting potential limits of life history theory. Specifically, neither perinatal risks nor early environmental harshness predicted pubertal timing at age 11, and pubertal timing did not mediate the association between either type of ELS and adolescent aggression. These results were consistent

among female and male children. The null results from the current study may indicate that life history theory is limited to sexual risk behaviors that have a direct evolutionary function (e.g., reproductive success), and does not expand to broader risk behaviors that have a more indirect evolutionary purpose. Exploratory results revealed that, although there are different factors that relate to pubertal timing and aggression for males and females, these sex differences were not statistically significant. In addition, unpredictable ELS was the only type of ELS that significantly predicted earlier pubertal timing for males and females. Future work is needed to reproduce these findings and further examine the link between different types of ELS (i.e., harsh and unpredictable; prenatal and postnatal) and factors and/or mechanisms of accelerated life history strategies (e.g., pubertal timing, different risk behaviors, stress response systems).

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