Girls' Pubertal Development: An Examination of Predictors and Trajectories

By

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ABSTRACT

This dissertation used an evolutionary approach to examine the antecedents and outcomes to early pubertal development in girls in four major ethnic groups (i.e., European American, African American, Hispanic American, and Asian American). In the first study, logistic regressions were conducted to investigate the links between socioeconomic status (SES), parenting behaviors, and father absence to pubertal development across and within ethnic groups. SES and father absence predicted earlier pubertal development among European, African, and Hispanic Americans but not for Asian Americans. In the second study, growth curves were estimated for sexual outcomes across ethnic groups. Early developing European and African American girls had higher levels of sexual risk and pregnancy into adulthood. Puberty did not predict outcomes for Hispanic and Asian American girls. Overall, the findings from both studies revealed the importance of investigating childhood environmental contexts, puberty, and sexual health outcomes using an evolutionary perspective.

DEDICATION

This dissertation is dedicated to my parents, to my friends and graduate colleagues, and to Sandi and Scott.

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Introduction

Puberty is a critical life transition, where adolescents begin to mature sexually, cognitively, biologically, and socially (Piakoff, Brooks-Gunn, & Warren, 1991). During puberty, adolescents experience many changes including breast and pubic hair development, the onset of menstruation, hormonal changes, weight gain, skeletal development, and changes in fat distribution (Neistein & Kaufman, 2002). These changes affect the developing adolescent directly through body and hormonal changes and also indirectly through how their social environments react to them (Christopher, 2001). The interaction of puberty with the social and cultural environment has the potential to affect adolescents' development and has been associated with behavioral and emotional outcomes. Compared to girls who develop late, early developing girls are at increased risk for a variety of behavioral and emotional problems.

It is established across the literature that early developing girls, on average, are at increased risk for maladjustment. However, findings are less consistent for boys. Although puberty occurs for both boys and girls, hormonal fluctuations and the reactions from parents and peers on early pubertal development are different for boys and girls. Additionally, studies suggest that the antecedents and outcomes are unique for girls (Belsky, Steinberg, Houts, Friedman, DeHart, Cauffman, et al., 2007). Therefore, my research will focus on girls in order to gain a better understanding of the effects of early pubertal development on adjustment to aid in intervention efforts.

Early pubertal development has been identified as particularly problematic because of its continued association with adolescent maladjustment, including substance use, depression, early sexual behavior, and early and unintended pregnancy (Billy, Brewster & Grady, 1994; Bingham, Miller, & Adams, 1990; Deardorff, Gonzales, Christopher, Roosa, & Milsap, 2005; Dick, Rose, Viken & Kaprio, 2000; Hayward, Killen, Wilson, Hammer, Litt, Kraemer, et al., 1997; Manlove, 1997; Pinny, Jensen, Olsen, & Cundick, 1990; Romans, Martin, Gendall, & Herbison, 2003). Not only is early pubertal timing predictive of problems during adolescence for girls, it may also have long term implications for girls' mental and physical health into adulthood (Fleming & Offord, 1990). Thus, early sexual maturation can have important long-term implications for individuals as well as societal wellbeing.

An emerging question from the literature on pubertal development is whether the accelerating effect of puberty on sexual behavior is part of an underlying developmental process that begins before sexual maturation. Current evidence suggests that it does. For example, a few longitudinal studies have revealed that early negative family environments predicted earlier pubertal development. That is, negative family relationships (e.g., family nonsupportiveness, maternal harshness, father psychological disturbance, father absence) in early childhood predict more advanced secondary sexual characteristics and earlier menarche (Belsky et al., 2007; Ellis & Essex, 2007; Tither & Ellis, 2008). This relation holds true even when controlling for genetic and environmental confounds (Ellis, 2004).

According to modern evolutionary psychology theory, humans have evolved to be sensitive and responsive to the environment, and as a result children develop specific reproductive strategies (Draper & Harpending, 1982). These reproductive strategies are designed to maximize the opportunities for humans to pass on their genes. Theorists from an evolutionary ecological perspective hypothesize that in order for one to reproduce, effort must be allocated among 3 tasks, (1) growth and development, (2) mating, and (3) parenting. Although humans tend to allocate the greatest effort to growth and development, as shown by long periods of juvenile dependence and delayed sexual maturation, there is significant diversity in the ways that some human populations allocate effort to manage these tasks.

The evolutionary argument, therefore, is that girls respond with faster pubertal maturation when early environmental contexts are unstable and predict an uncertain future in intimate relationships (Belsky, Steinberg, & Draper, 1991; Ellis, 2004). This occurs for two reasons, (1) earlier maturation decreases the likelihood of the individual dying before reaching an age to bear offspring, and (2) there is a longer period in which to bear offspring because age at puberty and age at menopause are uncorrelated. Thus, unstable early environmental contexts accelerate maturation and influence child bearing (i.e., promiscuity). Alternatively, contexts that are supportive and nurturing delay puberty and sexual behavior. In turn, a greater investment is made in parenting. Specifically, when the environment is positive and protective, pubertal development is delayed. The

delay in development provokes a greater investment in the task of growth and development as opposed to mating or parenting.

Although this hypothesis explains current empirical findings, shortcomings exist in the literature. First, most of the research has primarily examined middle class, mostly well adjusted, European American girls (Stattin & Magnusson, 1990). Second, scholars have explored the primary childhood environment of parenting, specifically, negative parental behaviors. Given that SES varies by race/ethnicity, it is important to include SES as it influences both parenting behaviors and other childhood environments (McLoyd, 1990). Third, studies have not explored the long terms implications of early pubertal development. That is, studies have examined sexual risk and pregnancy at only one time point in early or middle adolescence. The current investigation addresses these limitations and seeks to increase our knowledge by expanding on parts of evolutionary theory to include minority adolescents, other aspects of parenting behaviors, SES, and long term outcomes. This dissertation is divided into two articles. In the first study, I examine variations in pubertal maturation by race/ethnicity. I also use SES, parenting behaviors (i.e., controlling parenting and parental warmth) and father absence to predict variation within and across racial/ethnic groups. In the second article, I examine how early pubertal maturation influences risky sexual behavior, STIs, and pregnancies from early adolescence into young adulthood. I also examine how the relation between early sexual maturation and sexual outcomes vary by race/ethnicity.

Study 1: Predicting Racial Variations in Girls Pubertal Maturation: The Role of SES, Father Absence, and Parenting

Literature Review

The onset of puberty is an important milestone for adolescent girls (Graber, Brooks-Gunn, & Warren, 1995). Pubertal maturation brings about changes on multiple levels, including hormonal, physical appearance, and social status (Neistein & Kaufman, 2002). Moreover, the timing of physical maturation is associated with various outcomes. Compared to on-time or late pubertal development, early pubertal development is associated with a number of undesirable outcomes. Earlier development places girls at increased risk for depression, antisocial behavior, using alcohol and illegal substances, early sexual debut, risky sexual behavior, and early or unintended pregnancy (Brooks-Gunn, Graber, & Paikoff, 1994; Petersen, Graber, & Sullivan, 1990; Silbereisen, Petersen, Albrecht, & Kracke, 1989). Given the implications that early pubertal development has on adolescent adjustment, it is important to identify its antecedents.

Recently, several studies have focused on the role of ethnicity in pubertal onset. Findings suggest that the prevalence of early sexual maturation varies by adolescents' ethnicity (Herman-Giddens, Slora, Wasserman, Bourdony, Bhapkar, Koch et al. 1997; Villarreal, Martorell, & Mendoza, 1989). Such that, ethnic minority adolescents are more likely to experience earlier maturation compared to European Americans.

There have been two hypotheses proposed to explain the ethnic variation in pubertal onset. The first hypothesis attributes ethnic differences in pubertal onset to differences in Body Mass Index (BMI). African American girls, on average, tend to have more body fat and, thus, higher BMIs (Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001). The fat associated with higher BMI is postulated as being linked to early secretion of hormones that would, in turn, accelerate pubertal development. The second explanation emphasizes the marked increase in stressors that some minorities face compared to European Americans (McLoyd, 1998). Specifically, exposure to chronic stressors (e.g., substandard living conditions, exposure to violence, racial discrimination, harsh parenting, and father absence) accelerate the sexual development and growth of minorities in order to increase reproductive success (Belsky, Steinberg, & Draper, 1991; Ellis, 2004).

Although an important line of research, key limitations to previous studies of pubertal maturation across ethnic groups exist. First, prior research has focused on African American and European American girls while ignoring pubertal maturation among other minority groups, such as Hispanic and Asian Americans. The studies that have included Hispanic girls typically have aggregated them with European American or African American groups when conducting analyses (Herman-Giddens et al., 1997). Additionally, Asian American girls have not been included in any comparative studies. The differences among adolescents of minority status are worthy of careful examination because of the possible differences in social and cultural environments in which they reside.

Second, the role of socioeconomic status (SES) has rarely been systematically investigated in conjunction with pubertal development. It is important to disentangle the effects of SES from those accredited to ethnicity[1] (McLoyd, 1990). It is possible that SES and not ethnicity account for the differences in puberty, as there are large disparities in the income of African American mothers and European American mothers. Minority families have disproportionately lower SES (Duncan, Brooks-Gunn, & Klebanov, 1994; U.S. Census Bureau, 2001). Furthermore, other factors associated with SES, such as parenting behaviors and father absence, have rarely been examined in association with pubertal development across ethnicities (McLoyd, 1990). Specifically, harsh and punitive parenting behaviors and father absence can create distress for children. Father absence is also more common among certain minorities than Caucasians (Population Reference Bureau, 2011). Inconsistent and harsh parenting have been associated with earlier pubertal maturation, however, these associations have only been studied in European American children (Belsky et al., 2007). Positive indicators of parenting have also rarely been used in studies. Theory indicates that positive parenting practices would predict later pubertal maturation. These parenting strategies may create distress for children leading to earlier sexual maturation.

The U.S. population includes a number of different ethnic groups but this study will focus on the four largest groups: European American, African American, Hispanic American, and Asian American (U.S. Census Bureau, 2001).

[1] While some studies have focused on race, I am choosing to use the term ethnicity to describe group differences.

Researchers have yet to examine differences in pubertal maturation in these four ethnic groups. Additionally, there has been no research examining the within group variation of pubertal timing. In light of the limitations previously stated, this study aims to investigate pubertal maturation by ethnic group, within each of the four ethnic groups, and as related to SES, father absence/presence and parenting behaviors.

Ethnic Differences in Pubertal Development

The prevalence of early sexual maturation, as most commonly measured by menarche (first menses), varies by adolescents' ethnicity. Although the overwhelming majority of research has focused primarily on well adjusted, middle class, European American girls (Stattin & Magnusson, 1990), in recent years the differences between European Americans and other groups have become better documented. Across different studies, the average age of menarche for European American girls has varied between 12.55-12.88 years (Chumlea, Schubert, Roche, Kulin, Lee, Himes et al., 2003; Herman-Giddens et al., 1997; Wu, Mendola, & Buck, 2002). For African American girls, the average age has been between 12.06-12.25 years (Chumlea et al., 2003; Herman-Giddens et al., 1997; Wu, Mendola, & Buck, 2002). Within each of these studies, the differences between European American girls and African American girls have been consistently statistically significant. For Hispanic Americans, the average age of menarche has ranged between 12.20-12.25 (Chumlea et al., 2003; Wu, Mendola, & Buck, 2002). Age at first menarche, therefore, has been closer for African

American and Hispanic American girls than either group was to European American girls.

The ethnic differences are not only reflected in menarcheal status but also in physical development. By eight years of age, 48% of African American girls, roughly 25% of Hispanic American girls, and less than 15% of European American girls have begun breast development (Chumlea et al., 2003; Herman-Giddens et al., 1997). Over 60% of African American and Hispanic American girls have pubic hair by age 10 compared to less than 30% of European American girls. Thus, consistent with menarche, African American and Hispanic American girls are closer in development than either two are to European American girls.

To date, Asian Americans have not been represented in studies of pubertal development. Therefore, it is impossible to know how Asian Americans compare to other ethnic groups in timing and tempo of pubertal maturation. Some studies originating in China, particularly Hong Kong, suggest the average age of menarche for Chinese girls is 13.17 years for urban girls and 13.83 years for rural girls (Hin, Chen, Su, Zhu, Xing, & LiJ-Y, 1992). The studies that examined secondary sexual characteristics in Chinese girls, started at too late an age to establish timing (i.e., age 8-12; Herman-Giddens, Slora, Hasemeier, & Wasserman, 1993). It is likely that pubertal development will differ for Chinese adolescents living in the U.S. Thus, a significant contribution of this study will be the examination of Asian American girls.

Mechanisms Influencing Differences in Pubertal Maturation

Theory and several studies provide evidence that pubertal maturation, particularly menarche, is sensitive to environmental and psychosocial stressors (Belsky et al., 2007; Belsky, Steinberg, Houts, & Halpern-Felsher, 2010; Graber, Brooks-Gunn, & Warren, 1995; Susman, Nottelman, Dorn, Inoff-Germain, & Chrousos, 1988). Belsky, Steinberg, and Draper (1991) proposed an evolutionary ecological model of pubertal maturation that linked childhood stressors, or stressful childrearing environments (e.g., harsh and controlling parenting, racism, disadvantaged neighborhoods), to earlier pubertal maturation. Alternatively, contexts that are supportive (e.g., parental warmth, parental supportiveness, adequate environmental resources) would predict later pubertal maturation. Their findings suggested that exposure to chronic stressors were associated with early pubertal onset and lower exposure to stressors was associated with later pubertal onset.

Ellis (2004) distinguished among three types of stressors. First were physical stressors that included socioeconomic status and poverty. The second type of stressor included socioemotional stressors such as lack of parental involvement and inconsistent or harsh parenting. The third type of stressor was father absence. Additionally, Moffitt, Caspi, Belsky, and Silva (1992) found support for an additive model of stress on girl's pubertal maturation. That is, multiple stressors increase the likelihood of early maturation. Consistent with Moffitt et al.'s (1992) additive model of stress, this study explores SES, father

absence, and parenting behaviors as stressors that could account for some of the differences in pubertal maturation across ethnicities.

In this study, SES, father absence, and parenting serve as proxy measures of psychosocial stress. Many levels of evolutionary psychology define stress as the ongoing conditions that require the use of coping strategies that, in turn, can undermine physiological functioning overtime (Belsky, Steinberg, & Draper, 1991; Ellis, 2004). These studies also refer to stressors as circumstances or events that cause stress. To be consistent with evolutionary psychology and previous research, this study will refer to SES, father absence and parenting behaviors as potential stressors for adolescent girls.

Socioeconomic Status. All girls in low SES stratums are exposed to more stressful conditions than those from higher SES (Jencks & Mayer, 1990; Kim & Smith, 1998). Multiple studies have shown that low income families are exposed to an astonishing number of environmental stressors (Allison, Burton, Marshall, Perez-Febles, Yarrington, Kirsh et al., 1999; Kim & Smith, 1998; Wilson, 1991, 1993). These stressors include more negative life events, higher interpersonal conflict, and limited access to social and health services as compared to those from higher SES. Given the differences in SES by ethnic group, it is surprising that few studies have examined SES and pubertal development by ethnic group (for exceptions see Obeidallah, Brennan, Brooks-Gunn, Kindon, & Earls, 2000 and Deardorff, Ekwaru, Kushi, Ellis, Greenspan, Mirabedi et al., 2010). Across studies, African Americans and Hispanic Americans reach physical maturation

and menarche faster than European American girls (Chumlea et al., 2003; Herman-Giddens et al., 1997; Wu, Mendola, & Buck, 2002).

Unfortunately, SES and ethnicity are confounded in the vast majority of studies. For example, Herman-Giddens et al.'s (1997) sample included a disproportional representation of low SES African Americans. In their study, over half of the African Americans were on Medicaid compared to only 10% of the European Americans. Medicaid is only available to low income individuals who cannot afford health insurance. Moreover, Lucky, Biro, Simbartl, Morrison, and Sorg (1997) and Brown, McMahon, Biro, Crawford, Schreiber, Similo et al. (1998) reported that 46.3% of African Americans in their sample reported incomes below $20,000 while only 17 % of the European American sample reported income below $20,000. Likewise, less than 24% of African American annual income was above $40,000, but over 50% of European Americans fell into this group. More African Americans fell into a lower income level than the European Americans. Thus, the differences in timing may be due to SES and not ethnic differences.

Obeidallah et al. (2000) provided some evidence for links between ethnicity, SES, and pubertal maturation. Significant differences in menarcheal onset existed between Hispanic and European American girls. Importantly, these differences disappeared after controlling for indicators of SES. This suggests that the differences in pubertal development may not be due to ethnicity but due to differences in SES. This finding highlights the importance of taking into account SES as an environmental stressor for girls.

Father Absence/Presence. Evidence also suggests that family structure predicts the timing of pubertal development. The majority of research on family structure and pubertal development has examined whether the father is present or absent (Ellis & Garber, 2000; Hetherington & Kelly, 2002). Father absence has been defined as the absence of the biological father from the home, usually before the onset of puberty. The results from studies examining father absence and puberty are very consistent; girls raised in father absent homes reach puberty several months earlier than girls raised in father present homes (Moffett et al., 1992; Wierson, Long, & Forehand, 1993). However, this relation has primarily been tested in European American, middle class girls. The relation does not always hold when ethnicity is considered. For instance, samples of African American girls growing up in father absent homes did not reach puberty at a different age than African American girls from two parent homes (Campbell & Udry, 1995; Rowe, 2000). This emphasizes the importance of including other ethnic groups in investigations of the effect of parent-child relations on pubertal development. The strength of my research is the inclusion of other ethnic groups and examining the within group variability.

Parenting Behaviors. The home environment is the context in which children have the most contact and has the greatest potential to impact child adjustment (Bronfenbrenner & Morris, 1998). Several studies have highlighted the importance of parenting behaviors in the onset of puberty (Belsky et al., 2007). Most investigations examining the parents' influence on pubertal development have tested Belsky, Steinberg, and Draper's (1991) hypothesis.

Belsky et al., (1991) argue that the home environment is shaped by extrafamilial stressors and supports, and that these influences are associated with the timing of sexual maturation. Specifically, they argue that early environmental stressors associated with the family predict two distinct life courses. The early family environment can either accelerate or delay pubertal maturation. There has been increasing evidence supporting this hypothesis.

Both longitudinal and retrospective studies have shown that negative parenting behaviors, such as coercion, conflict, and harshness, predict early pubertal development (Ellis & Garber, 2000; Moffitt et al., 1992; Garber, Brooks-Gunn, &Warren, 1995). However, it is not just indicators of family dysfunction that predict early pubertal development, indicators of positive family functioning also predict later pubertal development. For example, Ellis and colleagues found that the more affectionate and positive interactions that take place between mother and her child the later the age at first menarche (Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999). Nonetheless, one limitation to this area of research is that positive and negative aspects of the parent-child relationship have rarely been included in the same study. Prior work has revealed that positive and negative parent-child relationship qualities do not always predict the same child outcome (Petit, Bates, & Dodge, 1997). Additionally, research suggests that parent-child relationships have independent positive and negative dimensions and account for unique variance in child outcomes (Belsky, Hsieh, & Crnic, 1998; Hetherington & Clingempell, 1992). Thus, it is important to include both in studies of child outcomes.

The current investigation used parental warmth and behavioral control as indicators of parenting. Prior research, although limited, has found a relation between parental warmth and pubertal maturation, that is, greater parental warmth predicted later pubertal development (Ellis et al., 1999). Although behavioral control has traditionally been associated with positive outcomes, higher behavioral control has predicted higher BMIs for African American and Hispanic American girls (Fuemmeler, Yang, Costanzo, Hoyle, Siegler, et al., 2012). This is consistent with other research that has associated parental behavioral control with poor health outcomes (Thomas et al., 2008). Because increases in BMI are associated with pubertal maturation, this study examined behavioral control as a psychosocial stressor. From the adolescent's perspective, having strict parents might be considered stressor.

Although SES, father absence, and parenting behaviors were presented separately, theory would suggest that they are linked together. These stressors have bidirectional effects that may influence pubertal maturation and subsequent adjustment. That is, these independent predictors, SES, father absence, and parenting influence each other. Problems in one domain may exacerbate problems in the other domains. For example, researchers have posited that parenting styles are influenced by SES (Eccles, Furstenberg, McCarthy, Lord, & Geitze, 1993). That is, low SES and father absence contribute to maternal stress which can influence parenting styles. Moffitt et al. (1992) support this idea that each makes a contribution to stress but their also additive in nature.

Within Group Differences

Most studies examine differences between ethnic groups. However, it is also important to note that there is substantial variability within ethnic groups. For example, although European Americans have the highest overall median income, there is also a significant range in incomes across European American families (U.S. Census Bureau, 2001). There are similar patterns for African American, Hispanic American, and Asian Americans.

Additionally, there is variation within ethnic groups in parenting styles and how adolescents respond to these parenting behaviors. Specifically, European American adolescents respond well to a warm, reasoned, and collaborative approach to parenting (Steinberg, Mounts, Lamborn, & Dornbusch, 1991). Research findings on the effect of parenting style for African American and Asian American youth has been more inconsistent. In some studies, positive outcomes have been associated with less warm and more controlling parent-child relationships and in others warm and affectionate parenting (Steinberg, Lamborn, Darling, Mounts, & Dornbusch, 1994). Therefore, it is essential to examine both differences between ethnic groups as well as within each specific group.

The Current Investigation

The current research examined pubertal maturation within each ethnic group and across each ethnic group as predicted by SES, father absence/presence and parenting behaviors using the National Longitudinal Study of Adolescent Health (Add Health). More specifically, there were three goals of this study. The first goal was to test for differences in pubertal development among European

Americans, African Americans, Hispanic Americans, and Asian Americans. I hypothesized that African American and Hispanic American girls will enter puberty significantly earlier than European American and Asian American girls. The second goal was to test whether the stressors of SES, father absence, and behavioral control are positively related, and mother warmth is negatively related to pubertal development within each of these groups. The third goal was to examine whether there were differences across ethnic groups. All analyses included an indicator of BMI as it has been shown to be related to pubertal development (Kaplowitz et al., 2001). Add Health is a unique study that lends itself to the exploration of between and within variation among ethnic groups as it includes minority families with low and high SES in its sample. As discussed earlier, much of the research on the timing of pubertal development has sampled high SES European Americans and low SES ethnic minority youth. By using both high and low SES in each ethnic group I examined whether ethnic differences are related to differences in SES.

Method

Participants and Procedure

Data in this study came from Add Health; a multistage, stratified, school based sampling design (Bearman, Jones, & Udry, 1997). This dataset includes a large, nationally representative sample of adolescents in 7th through 12th grade. Add Health began with a school sample comprised of more than 90,000 students from 134 schools across the United States. A representative weighted sample of more than 20,000 adolescents was chosen to complete the in-home survey in

1995. Information on pubertal development, ethnicity, parenting behaviors, household structure, and control variables were collected from adolescents. The mother of some respondents also completed a questionnaire.

There were 10,342 girls who participated in the in-home survey. To obtain the desired sample three selection criteria were used. First, only girls between the ages of 11 and 16 were included because pubertal changes are usually complete by mid-adolescence (n = 3,648 dropped). By restricting the sample to early adolescence, this ensured that there would be variation in the pubertal measures because in late adolescence the pubertal transition would be complete. Third, the sample was limited to European American, African American, Hispanic American, and Asian American girls. This excluded adolescents who identified as American Indian or Native American (n = 51 dropped), other (n = 71 dropped), and those who marked multiple ethnicities (n = 770 dropped). Fourth, the sample contained only adolescent girls whose mothers completed the parent survey (n = 647 dropped). As a result, this study is based on 5,155 adolescent girls who reported menarcheal status (M_{age} = 14.57; SD = 1.20). The sample contained 3,268 (63%) European Americans, 1,313 (26%) African Americans, 364 (7%) Hispanic Americans, and 207 (4%) Asian Americans.

Measures

Pubertal development. Although menarche occurs late in the pubertal process, it is the most commonly used measure of pubertal development (Caspi, Lynam, Moffit, & Silva, 1993; Graber, Petersen, & Brooks-Gunn, 1996; Stattin & Magnusson, 1990). Thus, self-reported age at menarche, measured in whole years,

served as a proxy for early pubertal development. I constructed a binary variable indicating if girls were early (coded as 1) or on-time and late (coded as 0). Girls whose period had not arrived at the time of the assessment were coded as zero, an indication for on-time or late pubertal development. Early developing girls were those who reached menarche before age 12 (30% of the sample).

Ethnicity. Adolescents self-reported their race or ethnicity. Based on the answers to 2 questions, the categories of White, African American, Latina, and Asian American were created. Latina ethnicity was based on adolescents' answer to the question "Are you of Hispanic or Latino origin" (0 = *no*; 1 = *yes*). All others were created from the question "What is your race" (White; Black/African American; Asian or Pacific Islander).

Socioeconomic status. Two indicators of family SES were used. First, resident mothers' reported their highest level of education (0 = *did not graduate from high school*; 4 = *professional degree*). Mothers also reported on fathers highest level of education (0 = *did not graduate from high school*; 4 = *professional degree*). When two parents were in the household, the highest level of parent education was used. Second, mothers reported their total household income in 1994.

Father absence. The resident parent answered two questions (1) "Does {name}'s biological father live in this household" and, (2) "Did {name} ever live with her biological father?" These questions were combined to determine wither the biological father was ever present in the household (0 = *father present*; 1 = *father no longer resides in household*; 2 = *father never resided in household*).

Mother warmth. Adolescents reported on their mother's warmth using a 5-item scale. Two items included "How close do you feel to your mother" and "How much do you think she cares about you" (1 = *not at all*; 3 = *somewhat*; 5 = *very much*). The other three items included "Most of the time, your mother is warm and loving toward you," "You are satisfied with the way your mother and you communicate with each other" and "Overall, you are satisfied with your relationship with your mother" (1 = *strongly disagree*; 3 = *neither agree nor disagree*; 5 = *strongly agree*). These items were averaged together ($\alpha = .89$) with higher scores reflected more maternal warmth.

Behavioral control. Adolescents reported on their parents' behavioral control using seven items. These items used a 2 - point scale (0 = *no*; 1 = *yes*). Sample items include "Do your parents let you make your own decisions about what you wear," "Do your parents let you make your own decisions about the people you hang around with," and "Do you parents let you make your own decisions about the time you must be home on weekend nights?" Items were reverse coded and then summed so that a higher score indicated more behavioral control ($\alpha = .59$).

Body Mass Index. BMI was computed using adolescent self-reported height and weight. Weight in pounds was divided by squared height in inches and multiplied by 703 (Center for Disease Control and Prevention, 2003).

Plan of Analysis

The primary goals of this study were to investigate within and across group variation among European American, African American, Hispanic

American, and Asian American as well as to see if SES, parenting behaviors, and father absence predict this variation. To achieve these goals, preliminary analyses were conducted to examine patterns of missing data. To address the issue of missing data, Full Information Maximum Likelihood (FIML) was used to input values of missing variables. SAS was used to estimate logistic regression models in all analyses. Additionally, BMI was be used as a control in all analyses because body fat naturally increases during the pubertal transition (Halpern, King, Oslak, & Udry, 2005; Kaplowitz et al., 2001).

In order to test the first goal, that there would be differences among ethnic groups in pubertal development, five sequential logistic regression models were estimated. The first model examined only BMI as a predictor of pubertal development. The next four models examined differences across and within the four ethnic groups, European American, African American, Hispanic American, and Asian American on pubertal development above the effects of BMI. To compare pubertal development across ethnic groups, a series of models with dummy codes for ethnicity were estimated.

In each of the four models, one group was used as a reference group to test against each of the three other groups using dummy codes. For example, European American was the reference group and dummy codes were included to examine if African Americans, Hispanic Americans, and Asian Americans differed from European Americans. Similarly, African American was used as the reference group in one model and dummy codes were created for the remaining groups to determine if there were differences. Likewise, two additional models

were run for Hispanic Americans and Asian Americans. Thus, four separate models were estimated to account for all possible comparisons that are estimated in a traditional Analysis of Variance. In order to assess whether ethnicity predicted variation in pubertal development above and beyond BMI, the change in the chi-square from the first model (i.e., BMI only model) was compared to the remaining four models with the ethnicity dummy codes. That is, if the change in chi-square was significant then the model fit was improved with the addition of the ethnicity dummy codes (Tabachnick & Fidell, 2007).

The second goal investigated whether SES, parenting behaviors, and father absence predicted variation within each ethnic group. In order to address the second goal, four logistic regression models with interaction terms were run. These models allowed for examining *within* each group by looking at the main effects. The main effects would then represent the within group differences for each group, such as European Americans. Similar to the other models, dummy codes were created for each of the ethnic groups and each model used one group as the reference group. These models differ from the previous ones in that SES, parenting behaviors (warmth and behavioral control), and father absence/presence were used to predict variation in pubertal development. The main effects represent which predictors were associated with pubertal development within each ethnic reference group for each regression and correspond to goal two. The change in chi-square was examined for improvement in model fit.

In order to address the third goal, that there were differences across groups in the predictors, four logistic regression models with interaction terms were run.

These models were the same as the previous ones only here I focused on the interactions in order to examine and compare the different predictors across the four ethnic groups by examining interaction terms. Similar to the other models, dummy codes were created for each of the ethnic groups and each model used one group as the reference group. The main effects were entered followed by the interaction terms. The interaction terms were created using the predictors (i.e., SES, parenting behaviors, and father absence) and the dummy codes. Before creating the interaction terms each variable was centered to allow for interpretation.

Results

Descriptive statistics

As shown in Table 1 (see Appendix A), approximately 30% of adolescents entered puberty early. The largest percentage of early developing girls were African American (36%) followed by Hispanic Americans, European Americans, and Asian Americans (33%, 27%, and 22%, respectively).

Chi-square and ANOVA were used to examine differences across ethnic groups (significance levels and effect sizes reported in Table 1). Results from the chi-square analyses reveal that a larger proportion of African Americans and Hispanic Americans were classified as early developers and had a higher proportion of father absence than would be expected by chance. In contrast, a smaller proportion of European Americans and Asian Americans were classified as early developers and had a lower proportion of father absence than would be expected by chance.

Results from the ANOVA revealed that there were no significant comparisons for BMI and mother warmth [$F(3, 4687) = 2.06$, *ns*.; $F(3, 4537) = 2.37$, *ns*., respectively]. There were main effects for ethnic group when testing for differences in parent education [$F(3, 4642) = 59.34$, $p < .001$], income [$F(3, 4554) = 9.26$, $p < .001$], and behavioral control [$F(3, 4537) = 7.56$, $p < .001$]. Results of Tukey's posthoc tests revealed that of the six comparisons for parent education, all six were significant [p's < .01-.001]. Asian Americans had the highest parent education followed by European Americans, African Americans and Hispanic Americans. Additionally, Asian Americans had significantly higher income compared to all other ethnic groups [p's < .001]. For behavioral control, two of the six post hoc comparisons were significant. European Americans had lower behavioral control compared to African Americans [$p < .01$] and Hispanic Americans [$p < .001$]. All of these results should be interpreted with caution because the effect sizes were small with the exception of father absence.

Correlations among study variables for European Americans and African Americans are shown in Table 2 (see Appendix A). For European Americans, early pubertal development was associated with higher BMI [$r(3210) = .16$, $p < .001$] and father absence [$r(3210) = .07$, $p < .01$] and lower parent education [$r(3210) = -.06$, $p < .001$] as well as income [$r(3210) = -.04$, $p < 05$]. In the correlations for African Americans, early pubertal development was associated with a younger age [$r(1287) = -.14$, $p < .001$], higher BMI [$r(1287) = .07$, $p < .05$], and higher parent education [$r(1287) = .06$, $p < .05$]. As shown in Table 3, none of the indicators were significantly related to pubertal development for

Hispanic Americans. For Asian Americans, early pubertal development was associated with higher BMI [$r(202) = .15$, $p < .05$]. Although the correlations were statistically significant, some of these correlations are small, but statistically significant because of the large sample size.

Goal 1: Between Group Differences

The first goal of this study was to examine differences among ethnic groups in pubertal development. In order to test this goal five sequential logistic regressions were estimated. Results are reported in Tables 4-7 (see Appendix A) and show the regression coefficients, standard errors, odds ratios (OR), 95% confidence intervals (CI) for the odds ratios, model chi-square, and the change in the chi-square. In each table, Model 1 contained BMI only and the overall model was significant [$\chi^2 (1) = 52.30$, $p < .001$]. BMI was a significant predictor in all models, indicating that early development was associated higher BMI. The next four logistic regression models estimated differences in the four ethnic groups above and beyond BMI. To compare pubertal development dummy codes for each ethnicity were used. Table 4 shows the analyses using European Americans as the reference group. Examining Model 2 in Table 4 shows statistically significant improvement with the addition of the ethnicity dummy codes [$\Delta\chi^2 (3) = 31.00$, $p < .001$]. Specifically, African Americans were 1.75 times as likely to enter puberty early and Hispanic Americans were 1.52 times more likely to experience early pubertal development compared to European Americans after controlling for BMI. There was no significant difference between European Americans and Asian Americans.

In Table 5 (see Appendix A), African Americans were the reference group. After examining the change in chi-square, the model with the ethnicity dummy codes significantly improved overall model fit [$\Delta\chi^2$ (3) = 31.00, $p < .001$; Model 2]. Significant and negative betas indicated that European Americans and Asian Americans were .57 and .49 (i.e., less or half) as likely to enter puberty later compared to African Americans after controlling for BMI.

In the models using Hispanic Americans as the reference group (Table 6; see Appendix A), the addition of the dummy codes showed significant improvement [$\Delta\chi^2$ (3) = 31.00, $p < .001$; Model 2]. The only significant comparison after controlling for BMI was Hispanic Americans to European Americans, indicating that European Americans were .65 as likely to experience later pubertal development. In sum, Hispanic Americans were more likely to enter puberty early compared to European Americans.

The final model used Asian Americans as the reference group (Table 7; see Appendix A) showed statistically significant improvement over the BMI only model with the addition of the ethnicity dummy codes [$\Delta\chi^2$ (3) = 31.00, $p < .001$; Model 2]. In this model, African Americans were 2.06 times more likely to enter puberty early compared to Asian Americans.

Goal 2: Within Group Differences with Predictors

The second goal of this study was to examine if SES, parenting behaviors, and father absence predicted variation within each ethnic group. The results are reported in Model 3 of Tables 4-7 (see Appendix A). These logistic regression models were similar to the previous models except SES, parenting behaviors, and

father absence were added. Table 4 is the analysis for European American girls with Model 3 representing the within group differences. The change in chi-square was significant above the BMI only model [$\Delta\chi^2$ (18) = 73.55, $p < .001$]. After controlling for BMI, father absence was the only significant within group predictor. That is, European American girls with no father in the home were more than one and a quarter times more likely to enter puberty early than those with a father present.

Table 5 (see Appendix A) represents the analyses using African Americans as the reference group with Model 3 representing the within group differences. There was a statistically significant change in chi-square above the BMI only model [$\Delta\chi^2$ (18) = 73.55, $p < .001$]. Father absence was significant predictor for African American girls' pubertal timing, after controlling for BMI. That is, African American girls with no father in the home increased the likelihood of entering puberty early compared to African American girls whose father was present at least some of the time.

The results for the within group differences for Hispanic Americans are presented in Table 6, Model 3 (see Appendix A). There was significant improvement over the BMI only model [$\Delta\chi^2$ (18) = 49.96, $p < .001$]. Father absence was the only significant predictor. Specifically, Hispanic American girls were more likely to enter puberty early if they their father was no longer present in the home than Hispanic American girls whose fathers were present.

For Asian Americans, there was statistically significant change in chi-square above and beyond the BMI only model [$\Delta\chi^2$ (18) = 49.96, $p < .001$]. BMI

was the only significant within group predictor. Namely, girls who had higher BMIs were more likely to enter puberty early than girls with lower BMIs.

Goal 3: Across Group Differences

The third goal of this study was to examine if there were differences across groups in the predictors. To address this goal logistic regression models with interaction terms were estimated. The interaction terms compared the different predictors across the four ethnic groups. There were no significant differences for Hispanic Americans and Asian Americans as the reference group. There were two significant differences for European Americans and African Americans.

The statistically significant results are presented in Model 4 of Tables 4 and 5 (see Appendix A).When comparing the paths across European American and African American, there were differences for BMI and parent education. Higher BMI was associated with early pubertal development for European American girls but lower BMI was associated with early pubertal development for African American girls (Figure 1).

The interaction between parent education and ethnicity (i.e., European American and African American) was also significant. This indicates that higher parent education was associated with early pubertal development for African Americans but lower parent education was associated with early pubertal development for European Americans (Figure 2).

Discussion

Given that adolescents who enter puberty early are generally at higher risk for maladjustment (Billy, Brewster & Grady, 1994; Deardorff, Gonzales, Christopher, Roosa, & Milsap, 2005; Dick, Rose, Viken & Kaprio, 2000; Hayward, Killen, Wilson, Hammer, Litt, Kraemer, et al., 1997; Manlove, 1997; Romans, Martin, Gendall, & Herbison, 2003), understanding the antecedents of early pubertal maturation is imperative. Theory and prior research suggest that pubertal maturation is sensitive to environmental factors, and that girls have evolved to be sensitive to specific features of the environment (Belsky, Steinberg, & Draper, 1991; Ellis, 2004). That is, environments that are high in stress influence young girls to develop in a way that speeds up their maturation. Drawing from this evolutionary psychology perspective, the purpose of this investigation was to examine variation in pubertal development within and across each of the four major ethnic groups (i.e., European American, African American, Hispanic American, and Asian American) as predicted by SES, father absence, and parenting behaviors. Specifically, this investigation had three goals: a) to examine variation in pubertal development across four ethnic groups; b) to examine variation in pubertal development within each ethnic group by SES, father absence, and parenting behaviors; and c) to examine if these paths differed across ethnic groups.

Overall, this study found significant differences in pubertal development between ethnic groups. Consistent with previous research, African American and Hispanic American girls were more likely to enter puberty early compared to

European American girls (Chumlea et al., 2003; Herman-Giddens et al., 1997; Wu, Mendola, & Buck, 2002). Although a greater percentage of African American girls reported earlier timing, there were no statistically significant differences in pubertal maturation for African Americans and Hispanic Americans. One contribution of this study was that Asian American girls enter puberty significantly later than African American girls. Evolutionary psychology suggests that these differences might exist because of the differing exposure to psychosocial stressors. One primary goal of this study was to examine if exposure to specific stressors (i.e., SES, father absence, and parenting behaviors) influenced differences in timing within and across groups. This discussion will be organized around these stressors.

Socioeconomic Status

Results from extant research have suggested that ethnic differences in pubertal development might be due to SES. SES and ethnicity, however, are often confounded in this work (McLoyd, 1998). As discussed previously, studies have primarily used samples from low SES African Americans and high SES European Americans (Chumlea et al., 2003; Herman-Giddens et al., 1997; Wu, Mendola, & Buck, 2002). There were two indicators of SES used in this study, income and parental education. Results revealed that there were significant interaction between ethnicity and parent education on pubertal development. Such that, lower parent education for European American girls predicted earlier pubertal development whereas higher parent education for African American girls predicted earlier pubertal development.

Another interesting finding included that lower parent education was associated with accelerated pubertal maturation for European American girls. Higher SES was associated with later pubertal maturation. This finding is consistent with the few studies that have examined parent education as a predictor of secondary sexual characteristics in girls (Ellis & Essex, 2007). That is, higher SES was associated with low levels of secondary sex characteristics in previous studies and later menarche in this study. These findings indicate that low SES European American girls experience more stress. For example, lower SES homes are more likely to have a father absent from the home and poorer family relationships (Belsky, Steinberg, Houts, Halpern-Felsher, 2010). Additionally, low SES European American adolescents have reported more negatively impactful stressful life events and lower levels of perceived social support than did African American and Hispanic American adolescents (Prelow & Guarnaccia, 1997). Low parent education can be indicative of prolonged family stress. According to evolutionary theory, adolescents who experience higher levels of stress have accelerated pubertal maturation due to the perception of an unstable and unpredictable environment.

One counterintuitive finding was that for African Americans higher parent education was associated with earlier pubertal maturation. Potentially what might be occurring is that higher parent education for African American girls presents some different stressors unique to that group. For example, higher parent education might be associated with more racism and discrimination and more time isolated due to parent working hours or traveling (Brown, 2001; Gee, 2002;

Koplewicz, Gurian, & Williams, 2009; Luthar & Becker, 2002). Furthermore, higher SES may also be associated with weaker social support networks compared with lower SES families and communities, where using alternative caregivers maybe the norm (Hogan, Hao & Parish, 1990). Although these results seem contrary to past research on poverty, stress and health, other research on the family factors and pubertal development suggest that those may be important contributors to stress (Adler & Rehkopf, 2008; Belsky et al., 2007). When placed into an evolutionary perspective, racial discrimination, leaving a child isolated due to working hours, and weaker support networks could contribute to a stressful home environment for developing adolescents, and thus accelerate pubertal development.

However, one should be cautious when interpreting these results. First, the differences in parent education are not great. In terms of coding for parent education one indicates high school graduate and two indicates some college. For both early and on-time and late puberty parent education feel between high school and some college. This is relatively high parental education. These findings might have been significant due to the large sample size for European Americans and African Americans (n = 3,268, n = 1,313, respectively). Future studies should obtain samples of low and high SES European Americans and African Americans to examine the associated between SES and puberty.

Second, results from recent research have been mixed. For example, despite income predicting pubertal development in previous studies (Braithwaite, Moore, Lustig, Epel, Ong, Rehkopf, et. al., 2008; Deardorff et. al., 2010), this

study did not find any relation between income and menarche. Deardorff et al. (2010) found that higher income families with a father absent from the home predicted earlier pubertal development for African American girls. Braithwaite et al. (2008) found that parent education did not significantly predict early menarche. However, income predicted age at menarche for African American girls but only the highest income predicted late menarche for European Americans girls. Taken together, Braithwaite et al. (2008) and Dearforff et al. (2010) have highlighted the complexities of using indicators of SES in relation to pubertal maturation. Future research will need to further address parent education and income on pubertal development. Specifically, research will need to parcel out how SES is influencing stress levels and how these function differently in terms of ethnicity.

Father Absence

This study highlighted the link between father absence and early pubertal development. Interestingly, the mean-level differences in father absence map onto the differences present for puberty. For example, African Americans had the earliest pubertal development and the highest rates of father absence. Additionally, this study found that for European Americans, African Americans, and Hispanic Americans having no father in the home accelerated pubertal maturation within each group. This finding is consistent with a growing body of evidence that father absence plays a large role early pubertal development (Belsky et al., 2005; Belsky, Steinberg & Draper, 1991; Ellis & Garber, 2000; Deardorff et al., 2011; Moffett et al., 1992; Wierson, Long, & Forehand, 1993). When the

father is absent from the home, mothers are likely to be raising children on their own. Single mother families experience more stress than intact families. Specifically, single mothers have less money, longer work hours, less social support and involvement, and lower frequency of contact with friends and family compared to married mothers (Cairney, Boyle, Offord, & Racine, 2003). Children are sensitive to the environment and maternal stress (Hofferth & Sandberg, 2001) thus biologically, children may respond with accelerated development.

Father absence acts as a psychosocial cue of stress versus support in the family system. When the father is absent from the home, young girls get the impression that the family system is unstable and parental investment is minimal. Further, father absence also provides predictive cues for future male involvement later in life within relationships. In addition, adolescent girls gain increased exposure to unrelated males through the mothers dating relationships. Girls experienced earlier pubertal development when their childhood experiences included exposure to unrelated father figures, especially if that exposure was high in conflict (Ellis & Garber, 2000).

Despite the consistency in findings for father absence within each of the ethnic groups, father absence did not predict within group variation for Asian Americans. Perhaps because most Asian Americans in this sample (i.e., 74%) come from homes where the father is present, there was not enough variation or a large enough sample to detect an effect. Another possible explanation is that father absence is not influential for Asian Americans. Father absence may not have an impact because Asian Americans tend to live in mutigenerational

households where a related adult male lives in the home (Taylor et al., 2010). Future research will need to explore the possible role of father absence in pubertal maturation for Asian American girls.

Parenting Behaviors

Despite the consistent connections between parenting behaviors and pubertal development (Ellis & Garber, 2000; Moffitt et al., 1992; Garber, Brooks-Gunn, &Warren, 1995), the present investigation failed to find any associations between parenting behaviors and pubertal maturation. Across all ethnic groups, indicators of parenting behaviors were not associated with pubertal maturation. The expectation was that behavioral control would predict earlier puberty whereas parental warmth indicators would predict later pubertal development. There are two possible explanations for the lack of findings regarding parenting behaviors and pubertal maturation, one theoretical and one methodological.

First, one possible theoretical explanation is that these data were collected too late to capture differences. According to evolutionary psychology, it is the early rearing environment (i.e., under 7 years) that has the greatest impact in determining the timing of sexual maturation (Belsky, Steinberg, & Draper, 1991). Previous research has collected data on parenting behaviors and physical development in childhood with a younger aged sample. A few have collected parenting measures in early childhood, before age 6 and before pubertal development in adolescence (Ellis et al., 1999; Graber, Brooks-Gunn, & Warren, 1995). The data in the present study were collected in adolescence; perhaps too late to capture the role of parenting in pubertal development. Another study noted

that collecting family relationship and pubertal development data concurrently in adolescence did not show the same associations as when the measurement of family relationships was taken in childhood (Jorm, Christensen, Rogers, Jacomb, & Easteal, 2004). This is likely the case for parental warmth as warmth has been found to be related to timing of puberty in several other studies (Ellis et al. 1999; Graber, Brooks-Gunn, & Warren, 1995; Romans et al., 2003).

Second, the parenting measures, mother warmth and behavioral control might not be ideal measures for predicting pubertal maturation. In terms of indicators of negative parenting, behavioral control likely is not the best measure. Most studies examining parenting and pubertal maturation have used family conflict and coercion (Ellis & Garber, 2000; Moffitt et al., 1992; Garber, Brooks-Gunn, &Warren, 1995). Although some health psychologists have concluded that behavioral control is associated with higher BMIs and poorer health outcomes (Fuemmeler et al., 2012), many studies conducted by developmental and family psychologists suggest that behavioral control, especially when paired with high warmth, is advantageous for child development (Darling & Steinberg, 1993). Because this study used a secondary data set, the parenting measures, particularly behavioral control, were not the best indicators of family functioning.

Similarly, there may have been a lack of findings in parenting behaviors because this study used the menarche as a proxy measure for pubertal development. Often studies have used measures specific stages of physical development when assessing the relations between parenting behaviors and pubertal development (Belsky et al., 2007). This may be due to the young age at

which assessments were taken, that is, that menarche had not yet occurred in these samples. Thus to better understand the association between parenting and menarche, future research should include measures of physical development and begin with participants who are younger.

BMI

There was also a significant interaction between ethnicity and BMI and puberty. Consistent with prior research, a higher BMI predicts earlier pubertal development for European American girls. For African Americans a lower BMI predicted earlier development although the difference was very small. Several studies have linked higher BMIs with early puberty. This relation has been shown to be particularly strong for European American girls and a lesser degree to African American girls (Kaplowitz et al., 2001). While some studies have suggested that increases in body fat precipitate pubertal development, others suggest that genetic and environmental factors play a larger role African Americans (Wong, Nicolson, & Stuff, 1998). Some studies have noted that African Americans have a higher body fat percentage but that this doesn't necessarily predict puberty in all samples (Wolff, Berkowitz, Brower et al., 1999).

Limitations and Future Directions

There are several limitations to the present study. The primary limitation of this study was the concurrent measurement of the study variables. Because the variables were measured concurrently, it is difficult to determine causality and this may have contributed to the difficulty in detecting effects. This was particularly problematic in terms of measuring parenting behaviors. Additionally,

because this study used an existing data set, the measures for parenting behaviors were somewhat limited and might not have been the best assessment of positive or negative parenting. Future research should address better measures of parenting behaviors and measure them in early childhood, before age 6.

Moreover, because puberty was measured at only one time point, some girls may have entered puberty early but were coded as late in this study because they had not reached menarche in the first Wave of data collection. For example, an 11 year old adolescent girl could report that she had not experienced menarche but could potentially experience menarche the next day. In this study she should be coded as late however, her actual timing would be early. Because this study only used the first wave of data, we could not capture those girls who would be early but had not reached menarche yet. Furthermore, studies should include multiple markers of pubertal development (i.e., physical development, menarche, perception of timing) to examine if these associations vary with different indicators of puberty.

Another limitation is that the categorization for ethnicity was very broad. This study examined the four major ethnic groups; however, there is substantial variation within each group. For example, not all Hispanic Americans come from the same historical or cultural background. Conceivably, some of the lack in findings for Hispanic Americans and Asian Americans could because this study grouped everyone together when the groups are not homogenous. Future studies should continue to examine how these associations vary across and within groups especially for Hispanic American and Asian American girls.

SES is a complex concept that is designed from various indicators of social and economic standing. In this study I used two indicators of SES, income and parent education. Future studies should examine these indicators of SES separately and combined with other predictors of puberty because they might have different effects on the timing of pubertal maturation across ethnic groups. Recent studies have highlighted the complexities of these relations. Specifically, Deardorff et al. (2010) found interactions between income, father absence, and ethnicity. For example, father absence was a significant predictor for pubertal onset but only for high income African American families. Given the intricacy of these relations future studies will want to examine how puberty (andrenarche, secondary sexual characteristics, menarche, and perception of timing) varies by SES and ethnicity, and how these variables might interact with one another.

Conclusion

This study offers several contributions to literature on pubertal maturation. This study highlights the importance of investigating pubertal maturation and SES in various ethnic groups. Given the consistent results between early development and subsequent maladjustment, future studies need to explore these complex relations more in depth across ethnic groups (Billy, Brewster & Grady, 1994; Bingham, Miller, & Adams, 1990; Dick et al., 1997). The present study provided partial support for an evolutionary model of pubertal development. On one hand, parent education and father absence were significant predictors of early puberty. However, indicators of parenting were not significantly related to puberty, despite the consistency of findings in previous studies (Belsky et al., 2007; Ellis & Essex,

2007; Ellis & Garber, 2000; Garber, Brooks-Gunn, &Warren, 1995; Moffitt et al., 1992; Tither & Ellis, 2008). By examining these relations across as well as within groups, complex associations were revealed. Future research will want to continue to examine and parcel out these patterns.

Study 2: Trajectories of Sexual Risk and Pregnancy for Early Developing Girls

Literature Review

Puberty is the most salient marker of adolescent development. During this period of development, there is a transition from a biologically non-reproductive, infertile child to an individual who is capable of reproduction. While the sequence of physical changes related to puberty is predictable, the timing of these changes varies (Belsky, Steinberg, Houts, Friedman, DeHart, Cauffman et al., 2007). It has been well established in the literature that girls who enter puberty early are at increased risk for a variety of adverse outcomes including depression, delinquency, early substance use, and early sexual behavior (Belsky et al., 2007; Billy, Brewster & Grady, 1994; Deardorff et al., 2005; Hayward et al., 1997; Romans, Martin, Gendall & Herbison, 2003). Additionally, the developmental processes for boys and girls are different; thus, the antecedents and outcomes of early puberty are unique for girls (Belsky et al., 2007). Because these outcomes are far less consistent for boys and have generally negative outcomes for girls, this study focuses on girls' early pubertal development.

Belsky, Steinberg, and Draper (1991) hypothesize that the early environmental context contributes to girls' reproductive strategy. That is, early puberty is an adaptive, biological response to a stressful environmental context in childhood. Specifically, girls who grow up in an unfavorable environment will increase their reproductive success by physically maturing at an earlier age. Meaning, that individuals are matched with their environments to promote

survival and reproduction. By maturing earlier, girls begin sexual activity at an earlier age and facilitate reproduction. Although this may be biologically adaptive, it can also present risks for the developing adolescent girl. Early puberty has been shown to be particularly problematic for girls and is associated with a number of negative physical, emotional, and behavioral outcomes such as, early sexual debut, as well as early and unintended pregnancies (Billy, Brewster & Grady, 1994; Dick, Rose, Viken & Kaprio, 2000; Stattin & Magnusson, 1990).

Of all the outcomes that have been investigated, there has been particular interest in the sexual behavior of early developing girls. Early maturation increases the odds of early sexual debut, which itself is associated with engaging in risky sexual behavior such as inconsistent or incorrect condom use, multiple casual sexual partners, intercourse after drinking or using drugs, and intercourse with partners who have sexually transmitted infections (STIs; Boyer, Pollack, Becnel, & Shafer, 2008; Downing & Bellis, 2009; Phinny, Jensen, Olsen, & Cundick, 1990). Moreover, risky sexual behavior increases individual's risk of unintended or unwanted pregnancies (Dunbar, Sheeder, Lezotte, Dabelea, & Stevens-Simons, 2006; Waylen & Wolke, 2004). In terms of evolutionary theory, early maturation is adaptive because adverse environments signal that there are limited resources which increase the likelihood of having offspring that will survive and reproduce. In contrast, supportive environments delay sexual maturation in order to benefit from an extended period of development in which girls will have time to develop socio-competitive skills and resources necessary for successful competition in mating (Belsky, Steinberg, & Draper, 1991).

Although there are numerous studies showing that early puberty is associated with sexual risk (Downing & Bellis, 2009; Dunbar et al., 2006; Stattin & Magnusson, 1990; Waylen & Wolke, 2004), the question remains whether higher sexual risk, acquisition of STIs, and unwanted pregnancies continues into emerging adulthood particularly in relation to pubertal development. Additionally, it is unknown how the trajectories of risk vary by ethnicity.[2] This information could aid in prevention efforts. Using data from the National Longitudinal Study of Adolescent Health (Add Health), this study will examine the role of pubertal timing in longitudinal changes in sexual risk, acquisition of a STI, and pregnancies from early adolescence to young adulthood in girls. Moreover, this study will examine whether these trajectories vary by ethnicity using the four largest ethnic groups in the United States: European American, African American, Hispanic American, and Asian American (U.S. Census Bureau. 2001).

This study adds to the current body of literature by using a diverse sample of American girls. Typically, studies of sexuality, and especially studies of STIs and pregnancy, use clinic samples of girls who are seeking health care. Add Health is a nationally representative sample of girls who are not uniformally seeking health care. Additionally, this study uses a three wave longitudinal study and information on multiple dimensions of sexual risk to explore how ethnicity and puberty predict these outcomes.

[2] Some studies choose to use the term race, however, here I will be using the term ethnicity when discussing differences.

Effect of Puberty on Sexual Risk and Pregnancy

Belsky, Steinberg, and Draper (1991) hypothesized that the processes that accelerate puberty are part of a reproductive strategy. That is, early maturation encourages the early onset of sexual activity in the name of procreation. The onset of sexual activity is strongly linked to biological development; girls who mature early will initiate sexual intercourse at a younger age (Cavanagh, 2004; Stattin & Magnusson, 1990). The hormonal changes associated with puberty increase adolescents' sex drive, interest in sex, and level of arousal when exposed to sexual stimuli (Smith, Udry, & Morris, 1985). It might seem strange to conclude that early pubertal development leads to early pregnancy, especially given the availability and accessibility of contraceptives. However, young girls who enter puberty early are often cognitively unprepared for sexual activity. Early developing girls have a shorter length of time to acquire and strengthen coping skills (Ge, Conger, & Elder, 1996; Siegel, Aneshensel, Taub, Cantwell, & Driscoll, 1998). Moreover, these young girls may be too immature to deal effectively with the social sexual expectations from others, such as teenage boys, that are associated with physical maturation (Siegel et al., 1998).

There have been numerous studies linking early pubertal development with early sexual debut (Cavanagh, 2004; Stattin & Magnusson, 1990). Less frequently studied are the associations between early puberty and sexual risk, acquisition of STIs, and pregnancy. From the few studies that have examined these associations, all have found that girls who enter puberty early are at risk for getting pregnant at earlier ages compared to girls who mature later (Deardorff,

Conzales, Christopher, Roosa, & Millsap, 2005; Dunbar et al., 2008). Additionally, girls who perceive themselves to have more advanced physical maturity, as compared to their same aged peers, use condoms less, have a greater number of sexual partners, and engage in sexual intercourse while under the influence of alcohol or other drugs (Alan Guttmacher Institute, 2009; Halpern, Kaestle, & Hallfors, 2007; Moore, Miller, Glei, & Morrison, 1995). Related findings exist in the research on early sexual debut and other sexually risky behaviors. Specifically, girls who engage in early sexual intercourse (i.e., before 16 years of age) have more sexual partners, older sexual partners, and more frequent intercourse (Moore, Miller, Glei, & Morrison, 1995). In summary, young adolescent girls who enter puberty early are at increased risk for a variety of adverse sexual outcomes. The present study attempts to further this research by including multiple ethnic groups and examining behaviors over time.

Pubertal Development among Ethnic Groups

Perhaps one of the most significant stratifiers in American society is ethnicity. Girls' ethnicity is not only related to their physical traits but also the social group to which they belong. Individuals' social membership has a significant impact on their development (Johnson, Jaeger, Randolph, Cauce, Ward, & NICHD Early Child Care Research Network, 2003). Although ethnicity impacts individual development, few studies have examined how ethnicity alters the relations between pubertal development and sexually risky behavior, acquisition of STIs, and pregnancy. However, the literature on how girls' experience puberty suggests that there may be ethnic differences. Among the

studies reviewed, Hispanic American and European American girls react more negatively to the physical changes at puberty than African Americans (Brumberg, 1997; Ge, Elder, Regnerus, & Cox, 2001; Milkie, 1999). African Americans seem more resilient; they are less likely to hold negative self-perceptions about puberty (O'Sullivan, Meyer-Bahlburg, & Watkins, 2000).

Additionally, there are ethnic differences in the timing of menarche and sexual debut. African Americans and Hispanic Americans reach menarche earlier (Chumlea, Schubert, Roche, Kulin, Lee, Himes et al., 2003; Herman-Giddens, Slora, Wasserman, Bourdony, Bhapkar, Kock, et al., 1997; Wu, Mendola, & Buck, 2002) and transition to being sexually active earlier than European Americans (Cavanagh, 2004; Miller, Norton, Curtis, Hill, Schvanevedt, & Young, 1997; Upchurch, Levy-Storms, Sucoff, & Aneshensel, 1998). Evidence from China suggests that Asian American girls have the latest pubertal timing (Hin, Chen, Su, Zhu, Xing, & LiJ-Y, 1992), and studies from the United States suggest that Asian Americans have the latest sexual debut compared to other ethnicities (Upchurch et al., 1998).

Furthermore, African Americans and Hispanic Americans are less likely to use condoms during their first sexual intercourse than European Americans (Gibbs, 1998). This indicates that early intercourse has potentially different implications for the lives of African American and Hispanic American girls. Moreover, African Americans and Hispanic Americans adolescents have higher rates of fertility and higher rates of STIs (Centers for Disease Control, 2009; Hamilton, Sutton, & Ventura, 2003).There are also ethnic differences in minority

adolescents' beliefs about pregnancy and sexuality. For example, Hispanic American girls have the youngest desired age for first birth, and African American girls the youngest desired age for first sexual intercourse (East, 1998). This may be due, in part, to each group's differing social context and the prescribed norms of that context.

Researchers have often explored the association between puberty and sexual behavior for European American and African American adolescent girls, however, these relations have rarely been explored for Hispanic American girls and never explored for Asian American girls. Given the ethnic differences in pubertal timing, sexually risky behaviors, and fertility, there is evidence to suggest that ethnicity contributes to differences in the links among these variables. In the general literature on ethnicity, Hispanic American girls tend to fall between European Americans and African Americans on sexually related behaviors and perceptions (e.g., sexual debut and desired age at first birth), and the same is likely true here. For example, although Hispanic American girls have the earlier expectations of first birth and the higher adolescent fertility rates, they also possess strong family and community ties that might lessen the impact of early puberty (Center for Disease Control and Prevention, 2000; East, 1998).

The Present Study

The current study sought to examine the trajectories of sexual risk, acquisition of STIs, and unwanted pregnancy based on girls pubertal timing. A second goal of this study was to examine how these trajectories vary by ethnicity by using the four largest ethnic groups in the United States: European American,

African American, Hispanic American, and Asian American (U.S. Census Bureau. 2001). The hypotheses tested in this study were that: (a) early pubertal development predicted higher initial levels (i.e., intercept) of sexual risk, STIs and unwanted pregnancies within each ethnic group and, (b) early pubertal timing predicted increases of sexually risky behaviors over time (i.e., slope) within each ethnic group. Participants' Body Mass Index (BMI) was used as a control in all analyses because increased body fat has been associated with pubertal changes (Halpern, King, Oslak, & Udry, 2005; Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001). Additionally, parent education, mother warmth, and problem behavior were used as controls because these indicators have been associated with both girls' pubertal development and sexually risky behaviors in adolescence (Deardorff, Ekwaru, Kushi, Ellis, Greenspan, Mirabedi et al., 2010; Shrier, Emans, Woods, & Durant, 1997).

Method

Participants and Procedure

Data in this study came from Add Health, a representative sample of adolescents in 7th through 12th grade. Add Health is a multistage, stratified, school based sampling design (see Bearman, Jones, & Udry, 1997; Harris, Florey, Tabor, Bearman, Jones, & Udry, 2003). Add Health began with a school sample comprised of more than 90,000 students from 134 schools across the United States. These data were collected in the 1994-1995 school year. All of the students who completed the in-school survey were eligible for an in depth survey in the home. More than 20,000 adolescents were selected to complete the in-home

survey in 1995 (Wave I). About 15,000 respondents in the in-home sample were re-interviewed in 1996 (Wave II). A third wave (Wave III) interviewed more than 14,000 students in 2001-2002. Data for each of the three waves were collected by an interviewer using a Computer Assisted Personal Interview. Information on pubertal development, sexually risky behavior, STIs, pregnancy, and ethnicity were collected from adolescents.

Of the 7,816 adolescent girls who participated in all three waves of data collection, two selection criteria were used to obtain the current sample. First, because this study was designed to examine the effects of pubertal timing, only adolescent girls between the ages of 12 and 16 at Wave I who provided information on pubertal timing were included (n = 2,828 dropped). The sample was restricted to early adolescence in order to retain variability in the pubertal timing measure as most adolescents have completed puberty by age 16. Second, the sample was then restricted to adolescents who identified as European American, African American, Hispanic American, and Asian American at Wave I (n = 704 dropped). Girls who identified as (a) American Indian or Native American, (b) other, or (c) marked multiple ethnicities were excluded. As a result, this study is based on 4,011 adolescent girls. The mean age for Wave I, II, and III was 14.53 (SD = 1.22), 15.46 (SD = 1.26), and 20.90 (SD = 1.29), respectively. The sample contained 2,517 (63%) European American, 993 (25%) African American, 277 (7%) Hispanic American, and 223 (5%) Asian Americans.

Measures

Sexual risk behavior. A 3- item composite sexual risk behavior score was created to reflect the study participants' overall level of risk (Boyer et al., 2008). The items included the number of sexual partners, frequency of condom use (1 = *none of the time*; 4 = *all of the time*), and sexual intercourse under the influence of alcohol or drugs (0 = *never*; 4 = 5 *or more times*). Responses to each item were rescaled to range from 0 to 3 and recoded so that higher scores indicated elevated risk (Table 8; see Appendix B). The sexual risk behavior score is the sum of the rescaled scores (Table 10; see Appendix B).

STIs. The Add Health data set assessed whether an adolescent girl had ever had any of the following STIs. Girls self-reported if a doctor had ever told them that they had (a) Chlamydia, (b) syphilis, (c) gonorrhea, (d) HIV or AIDS, (e) genital herpes, (f) genital warts, (g) trichomoniasis, (h) bacterial vaginosis, and (i) non-gonococcal vaginitis. The score was the sum of the number of STIs reported.

Pregnancy. Girls self-reported the number of times they had been pregnant.

Pubertal timing. Girls self-reported their age at menarche. This served as a proxy for early pubertal development as it is the most commonly used measure of pubertal development (Caspi, Lynam, Moffit, & Silva, 1993; Graber, Petersen, & Brooks-Gunn, 1996; Stattin & Magnusson, 1990). Age at menarche, measured in whole years, was used to construct a binary variable indicating if girls were early (1) or on-time and late (0). Girls whose period had not arrived at the time of

the assessment were coded as zero, an indication for late pubertal development. Early developing girls were those who reached menarche before age 12 (29% of the sample).

Ethnicity. Dummy variables for European American, African American, Hispanic American, and Asian American were created from adolescents' responses at Wave I. Adolescents could report more than one race category but most only marked one. Those who marked multiple categories were coded according to the category they felt best described their racial background. Hispanic American ethnicity was based on the answer to the question "Are you of Hispanic or Latino origin?" Those who reported being Hispanic were coded as Hispanic regardless of race.

Age. Girl's age in years was calculated from their birth date and the date of the interview.

Controls. When examining pubertal development and sexually risky behaviors, it is important to control for BMI, parent education, mother warmth, and problem behavior as all have been associated with the constructs of interest. BMI was computed using adolescent self-reported weight in pounds and was divided by squared height in inches and multiplied by 703 (Center for Disease Control and Prevention, 2003). Parent education was created by taking the highest level of education from mothers or fathers (0 = *did not graduate from high school*; 4 = *professional degree*). Adolescents reported their mother's level of warmth using a 5-item scale with higher scores meaning higher levels of warmth (α = .86). Problem behavior was assessed using 7 items including (a) smoked

cigarettes; (b) drank beer, wine, or liquor; (c) got drunk; (d) raced on a bike, on a skateboard or roller blades, or in a boat or car; (e) did something dangerous because he or she was dared to; (f) lied to his or her parents or guardians; and (g) skipped school without an excuse (0 = *never*, 6 = *nearly everyday*; $\alpha = .75$).

Plan of Analysis

Using Age Instead of Wave

Typically, researchers who are interested in changes over time analyze data of people who are roughly the same age across multiple waves of data collection. However, there were considerable age variations within each wave because of the design of Add Health. In this study, data from Add Health consisted of adolescents aged between 12 and 16 years in Wave I who became 13 to 19 years in Wave II and 18 to 23 years in Wave III (Table 9; see Appendix B). Hence, Add Health is a cohort sequential design which is also known as an accelerated longitudinal design (Singer & Willett, 2003). This type of design is advantageous because cross sectional designs are prone to history effects, and longitudinal designs can be compromised by the repeated measurement of the same individuals.

Using wave instead of age for analysis in a cohort sequential or accelerated longitudinal design presents some methodological problems (Natsuaki, Biehl, & Ge, 2009). First, using wave ignores the substantial range of age variations. This becomes problematic when investigators interests are age sensitive or focused on development. Second, in Add Health, adolescents were

assessed at different points in time within a single wave. Thus, the ages at assessment vary markedly and analyzing by wave would ignore these variations.

Alternatively, one can analyze Add Health data using age at assessment. Add Health data consisted of limited repeated measures of several cohorts with overlapping measurements. That is, a long-term longitudinal study can be approximated by conducting several short term longitudinal studies of different age cohorts (Duncan, Duncan, & Hops, 1996). Several researchers have shown that using age instead of wave can correctly recover growth curves generated in the usual longitudinal design with one cohort (Duncan et al., 1996; Miyazaki & Raudenbush, 2000). Because I was interested in exploring trajectories of sexual outcomes from a developmental perspective, I used chronological age (rather than wave of data collection) to measure time in this analysis. As a result, the present study covers development from the ages of 12 to 23 years.

Hypothesis Testing

The purpose of the present study was to examine the change in sexual risk, acquisition of STIs, and unintended or unwanted pregnancies among early maturing girls into adulthood using growth curves. Additionally, the present study sought to examine whether puberty predicted the starting point and change in these outcomes within each ethnicity using the four largest groups in the United States: European American, African American, Hispanic American, and Asian American. The growth curves were estimated in MPlus version 5 using poisson models because the outcomes were count indicators (Muthén & Muthén, 1998-2004).

Before testing the hypotheses, it should be noted that traditional measures of fit (e.g., X^2, comparative fit index, and root mean square error of approximation) to evaluate growth curves cannot be used due to Add Health's design. Because Add Health is a cohort sequential or accelerated longitudinal design, the variance-covariance matrix of sexual outcomes for this dataset is incomplete as there are covariances that are missing. For example, respondents who were in the 12th grade at Wave I were not interviewed at Wave II. Given that the matrix is incomplete, structural equation modeling programs are unable to produce model fit indices except for the X^2. If model fit indices are not available, researchers suggest using the change in chi-square (Bollen & Curran, 2006). In the current study, all indictors of sexually risky behavior are count variables. Count variables do not provide X^2 but do provide log likelihoods. Comparisons across models were based on the likelihood ratio test (i.e., -2 times the likelihood ratio difference; -2LL; Bollen & Curran, 2006) which is distributed as a X^2 statistic, with degrees of freedom equal to the differences in degrees of freedom between the models compared.

Preliminary analyses were first conducted to examine patterns of missing data. To handle missing data, Full Information Maximum Likelihood (FIML) was used so that cases of missing data would be included.

Unconditional models. Before testing the hypotheses, a series of unconditional models were run to examine the trajectories of sexual outcomes to determine the appropriate trajectory across time. Separate models were run for sexual risk behavior, STIs, and pregnancies by ethnic group. The likelihood ratio

test, which is distributed as a X^2 was computed for the four unconditional models. In the first model, only the intercept mean was estimated. In the second model, the intercept variance was also estimated. In the third model, the linear slope mean and variance were also estimated. Finally, in the fourth model, the quadratic slope mean and variance were also estimated. To find the appropriate trajectory for each sexual outcome, the change in log likelihood between each model was examined. If the change between model 1 and model 2 was significant, then the model had a better fit when estimating the individual differences in the intercept compared to only the intercept mean. If the change between model 2 and model 3 was significant, then there was a linear change in the outcome. If there was a significant difference between model 3 and 4, then there was some curvature to the change over time in the outcome.

Conditional models. To test the hypotheses that the effect of pubertal timing on sexual outcomes would persist into young adulthood, latent growth curves were estimated using poisson models. The difference between these models and the previous ones was that pubertal timing was included in these models as a predictor. Separate models were run for each ethnic group and for sexual risk composite score, number of STIs acquired, and number of pregnancies. Additionally, BMI, parent education, mother warmth, and problem behavior were included as control variables to predict each growth curve as each has been shown to be related to pubertal timing and sexual behaviors (Halpern et al., 2005; Kaplowitz et al., 2001).

Results

Preliminary Analyses

The means, standard deviations and correlations for European Americans and African Americans are reported in Table 10 and Hispanic American and Asian American in Table 11 (see Appendix B). Puberty was positively associated with Wave 1 sexual risk [$r(2517) = .08$, $p < .001$], Wave 2 sexual risk [$r(2517) = .06$, $p < .01$], Wave 1 sexual risk [$r(2517) = .05$, $p < .05$], Wave 1 pregnancies [$r(2517) = .04$, $p < .05$], Wave 2 pregnancies [$r(2517) = .06$, $p < .01$], and Wave 3 pregnancies [$r(2517) = .11$, $p < .01$] for European Americans. That is, earlier pubertal development was associated with higher sexual risk, more STIs, and more pregnancies. For African Americans, puberty as not associated with sexual risk, STIs, and pregnancy at any wave [*r*'s = .01-.04, *ns*.]. Pubertal development was not associated with sexual risk, STIs, or pregnancy at any wave for Hispanic Americans [*r*'s = .00-.10, *ns*.]. For Asian Americans, there was one significant correlation between puberty and Wave 3 STIs [$r(223) = .17$, $p < .01$]. Meaning, earlier pubertal maturation was associated with higher STIs at Wave 3. All other variables were not associated with pubertal development [*r*'s = .04-.12, *ns*.]. These correlations are small but statistically significant, likely because of the large sample size.

Hypothesis Testing: Growth Curves

One goal of this study was to test differences between ethnic groups. However, I was unable to conduct these tests. As noted below, the trajectories for each outcome were different for each ethnic group. For example, European

Americans, African Americans, and Hispanic Americans had the best fitting model when the linear slope was estimated. However, the best fitting model for Asian Americans did not contain a linear slope. Because the trajectories for each group did not include the same terms, comparing across them would create results that were not interpretable (Muthen, 2006). These results preclude the use of across group comparisons. As a result, all analyses are organized by ethnic group rather than outcome.

Unconditional models. For all outcomes in each ethnic group, there was no variability in ages 12 and 13 so the data were dropped from all models. Additionally, age 18 had very few observations, thus, it was dropped from the models. Separate models were run for each ethnic group and outcome (Tables 12-15; see Appendix B). Referring to Table 12 (see Appendix B), model 1 estimates the intercept mean only, model 2 also includes the intercept variance, model 3 includes the parameters from model 2 and the linear slope mean and variance, and model 4 the parameters from model 3 and the quadratic mean and variance. Any of the indicators that evidenced significant variance were predicted by puberty and the control variables in the conditional models. Graphs for each ethnic group are presented in Figures 3-6.

Table 12 (see Appendix B) and Figure 3 show the results of the unconditional models for European Americans. For sexual risk behavior and pregnancy, the best fitting model was one that included the linear slope mean and variance (Model 3 Table 12). In contrast, the best fitting model for STIs was one in which the quadratic change over time was also estimated. As shown in Figure

3, all outcomes increased overtime. However, the increase for STIs leveled off over time as indicated by the significant negative quadratic term. The variance estimates suggest there was significant individual variability in the intercept for all three outcomes. Sexual risk was the only outcome that had a significant slope variance indicating that there were significant individual differences in the change from 14 to 23 years.

The results for the unconditional models for African Americans are reported in Table 13 (see Appendix B). For both STIs and pregnancy, the best fitting model included the linear slope mean and variance. The best fitting model for sexual risk indicated that there was some curvature in the change overtime. As shown in Figure 4, the increases in sexual risk leveled off over time. For all outcomes, the intercept variance was significant meaning that there was significant individual variability in the intercept. For sexual risk, but not for STIs or pregnancy, the slope variance was statistically significant indicating that there were individual differences in change from 14 to 23 years.

Table 14 (see Appendix B) reports the unconditional models for Hispanic Americans. For sexual risk and pregnancy, the linear slope mean and variance had the best fitting model, indicating a linear growth from 14 to 23 years. For STIs, there was no significant improvement with the addition of the linear slope. Thus, the model fit best with only the intercept and intercept variance. As shown in Figure 5, there was significant increases overtime for sexual risk and pregnancy but not for STIs. The intercept variance was significant for both sexual risk and STIs meaning that there was individual variability in the intercept. Sexual risk

was the only model in which the slope variance was significant indicating that there were individual differences in change from 14 to 23 years.

Results for Asian American girls are reported in Table 15 (see Appendix B). The trajectories were different for each outcome. For sexual risk, the best fitting model included the linear slope mean and variance. These results indicate that there were significant increases in sexual risk overtime for Asian Americans. The intercept variance and slope variance were significant. Meaning that, there were individual differences in the intercept and change over time. However, the best model for STIs and pregnancy only included the intercept mean and the intercept mean and variance, respectively. Referring to Figure 6, there was no linear change for STIs and pregnancy. However; there was a linear change for sexual risk.

Conditional models. In the next step of the analysis, growth models were run using puberty to predict the intercepts and slopes for sexual risk, STIs, and pregnancies within each ethnic group. In all models, BMI, parent education, mother warmth, and problem behavior were used as control variables as they have been shown to influence puberty (Halpern et al., 2005). Puberty was treated as a time-invariant characteristic of the adolescent because puberty was assessed at the first Wave of data. Table 16 (see Appendix B) shows the models with puberty predicting the intercept and slope for each of the sexual outcomes by ethnic group.

For European Americans, puberty predicted the intercept and slope for all three outcomes (Table 16; see Appendix B). As shown in Figure 7, early

developing girls had higher initial rates of sexual risk, STIs, and pregnancy compared to on-time and late developing girls. For sexual risk, the effect of puberty on the slope was significant and negative, suggesting that early developing girls had a slower increase over time than on-time or late developing girls. For STIs and pregnancy, puberty did not predict the slope.

For African Americans, puberty predicted the trajectories for sexual risk and pregnancy (Table 16; see Appendix B). As shown in Figure 8, early developing African American girls had higher initial rates of sexual risk and pregnancy. Puberty had a significant and negative effect on the slope, meaning that early developing girls had slower increases over time compared to on-time or late developing girls (Figure 8). However, there was no significant effect for puberty on the slope for pregnancy. Additionally, there was no effect of puberty on the intercept or slope for STIs.

In the models examining sexual outcomes for Hispanic Americans, there was no significant effect of puberty on the intercept or slope for sexual risk (Table 16; see Appendix B). Additionally, there was no significant effect of puberty on the intercept for STIs. Because Hispanic Americans had no significant variation in the intercept for pregnancy, a conditional model was not estimated.

For Asian Americans, there was no significant effect of puberty on the intercept or slope for sexual risk (Table 16; see Appendix B). Conditional models were not run for STIs and pregnancy because there was no significant variation in the intercept for Asian Americans.

Discussion

Extant research has examined the role of early puberty in maladjustment among adolescents with special attention paid to sexual behavior. Evolutionary theory suggests that early environmental contexts contribute to girls' reproductive strategy (Belsky, Steinberg, & Draper, 1991; Ellis, 2004). That is, early stressful environments tend to accelerate pubertal timing to increase a girl's chances of reproductive success. Specifically, that early pubertal development would lead to an early sexual debut which, in turn, leads to higher sexual risk and subsequent pregnancies. Many studies have examined the association between early puberty and early sexual debut (Deardorff et al., 2005; James, Ellis, Schlomer, & Garber, 2012); however, few have investigated whether early puberty also leads to sexual risk taking, STIs, and early pregnancy. Additionally, to date, no studies have examined if girls who enter puberty early stay at increased risk into adulthood. Thus, the primary purpose of this investigation was to examine the role of puberty in the change in girls' sexual risk, acquisition of STIs, and pregnancies over time. Specifically, there were two goals to this study: (a) to examine if early pubertal development predicted higher initial levels (i.e., intercept) of sexual risk, STIs and pregnancies, and (b) to examine if early pubertal development predicted change over time (i.e., slope).

Puberty and Sexual Behavior

In regards to the first goal, this study found that early pubertal development, relative to on time and later pubertal development, was associated with higher *initial* levels of sexual risk, number of pregnancies, and STIs for

European Americans, as well as sexual risk and pregnancies for African Americans. This is consistent with previous research that has found relations between early pubertal maturation and sexual risk and early pregnancies (Deardorff et al., 2005; Downing & Bellis, 2009; Dunbar et al., 2006; Waylen & Wolke, 2004).

One problem with much of the research on sexual behavior in adolescence is that most studies are cross sectional or they are longitudinal studies that only use two time points (Belsky et al., 2010; James et al., 2012). Although few studies have examined early sexual maturation and sexual behavior, no studies have examined if higher risk associated with early pubertal development extends into adulthood. In this study, trajectories were modeled from 14 to 23 years of age. As a result, the second goal of this study was to examine if early developing girls continue to have greater sexual risk behavior into adulthood. Findings from this study partially supported this proposition. Specifically, early pubertal development predicted higher initial levels (i.e., the intercept), but did not predict accelerated increases over time consistently (i.e., slope) for European and African American girls. For sexual risk score, there was a significant negative effect of puberty on the slope; however, puberty did not predict the slope for pregnancy or STIs. This pattern suggests that girls who experienced puberty early were at elevated risk overtime because of their initial risk.

As adolescents enter puberty they experience hormonal changes that increase sex drive and interest in the opposite sex (Smith, Udry, & Morris, 1985; Talbert & Morris, 1986). This, in turn, leads to early sexual debut and

subsequently elevated sexual risk. The current findings supported these notions. Early developing girls are especially vulnerable because they may not have the social and psychological resources to translate knowledge about risks into action or to understand the consequences of risky sexual behavior (Steinberg, 2007). Thus, early maturation increases the likelihood of early sexual debut, which in turn, has been associated with inconsistent condom use, multiple sexual partners, intercourse after drinking or using drugs, and unwanted pregnancies (Boyer et al., 2008; Downing & Bellis, 2009; Dunbar et al., 2006; Waylen & Wolke, 2004).

In terms of change over time, the results of the present study did not support an evolutionary approach. The number of pregnancies increased over time for all girls. Evolutionary theory asserts that early maturing girls engage in riskier sexual behaviors as part of a reproductive strategy to increase the likelihood of bearing children. Meaning that, for girls, early maturation is meant to increase the likelihood of reproduction at an early age. However, according to this approach, girls would have a greater number of children in order to increase the likelihood of offspring survival. This suggests that pregnancy should increase for early developing girls. Instead, early developing girls slope increased at the same rate as on-time and late developing girls. One reason that we do not see an accelerating effect is that the highest risk of unintended pregnancy occurs among women 20 to 25 (Finer, 2011; National Health Statistics Reports, 2012). Early puberty predicts initial levels of pregnancy, as it has in multiple studies, but that as teens enter into early adulthood the risk is equal across all pubertal groups causing the slopes to be the same.

In contrast to pregnancy, early developing European and African American girls' sexual risk increased at a slower rate than on-time or late girls. There are two possible explanations for the results in this study. First, as girls age, they are able to acquire more cognitive skills to buffer some of the negative effects of early puberty. Higher cognitive maturity may enable girls to better understand the consequences of infrequent condom use and sexual intercourse after drugs or alcohol (Steinberg, 2007). This possible explanation is consistent with other studies that have found sexual risk levels off as youth move into adulthood (Fergus, Zimmerman, & Caldwell, 2007). Fergus et al. (2007) suggested that development of better decision making skills or the development of longer-term monogamous partnerships might be responsible for the decrease in sexual risk overtime. Additionally, experience with STIs or pregnancy might alter sexual risk. Those get pregnant or acquire an STI might alter their future behavior. However, the cognitive gains do not seem to be enough to completely diminish the risk created by early maturation as sexual risk was still higher for early developing girls.

Second, the decrease may reflect the combination of indictors that make up the sexual risk score. In this study, the sexual risk score was created using the number of sexual partners, frequency of condom use, and sexual intercourse after the influence of drugs or alcohol. Perhaps if each of these were modeled separately, the number of sexual partners might increase with other indicators decreasing. This would be consistent with other studies that have found that overall sexual risk decreased into adulthood even though the frequency of sexual

intercourse was increasing and condom use decreasing (Fergus, Zimmerman, Caldwell, 2007; Grunbaum, Kann, Kinchen et al., 2004).

One advantage of this study was that sexual risk and pregnancies were examined separately. The only study to examine the relations between pubertal development and sexual risk used a measure of risk that combined number of sexual partners, STI diagnosis, and ever been pregnant (Belsky et al., 2010). The current study is one of the first to link earlier timing of puberty, pregnancy and other sexual risk behaviors separately. When studies group pregnancies, STIs, and other risk behaviors together it is difficult to interpret results because you confound timing variables (i.e., first pregnancy) and risk variables (i.e., number of sexual partners). Results from this study suggest that puberty has differing effects on these outcomes. Future studies should examine measures of sexual risk separately to investigate if early puberty is only related to indicators of mating and parenting, as evolutionary theory asserts, or more general indicators of sexual risk.

Within Group Ethnic Differences

Above and beyond the findings from the primary goals, some interesting findings emerged in regards to the within group differences. One of the most interesting findings was that the trajectories were different for each group. For example, European Americans, African Americans, and Hispanic Americans had similar trajectories for pregnancy; model fit was improved when the linear slope was estimated. For Asian Americans, the model fit best when only the intercept variance was estimated. Within European and African Americans, early puberty

predicted a higher initial sexual risk; however, early developing girls experienced deceleration over time whereas on-time or late developing girls experienced steady increases over time (with no slowing). Additionally, early developing European and African American girls had higher initial number of pregnancies and that risk continued into adulthood. This study provides partial support for the evolutionary perspective. Early puberty predicted the intercept and slope for both European and African Americans.

Evolutionary theory would suggest that the same pattern of results should also emerge for Hispanic and Asian Americans; however, this was not the case in this study. Surprisingly, puberty did not predict the intercept or slope for any outcomes within Hispanic American and Asian American girls. Furthermore, Hispanic and Asian Americans had little within group variability. These results are consistent with previous research that suggests Hispanic American girls have the longest interval between first intercourse and use of contraceptives, are less likely to use contraceptives, are generally less informed about sex, and have one of the highest teen pregnancy rates (Deardorff et al., 2010; Raffaelli & Ontai, 2001).

There are two possible explanations for the findings regarding Hispanic Americans. First, perhaps there are cultural values that buffer the effect of puberty on initiation of sexual activity. For example, Hispanic Americans report higher levels of parental respect and familism (sense of obligation to family and connectedness with family) than other ethnic groups (Wahl & Eitle, 2010). Additionally, Hispanic Americans are more likely to live in multigenerational

homes with strong family and community ties which may be protective for sexual risk (Cohen & Casper, 2002; East, 1998; Taylor et al., 2010). Likewise, Hispanic American girls with strong Hispanic cultural orientations place a high value on sexual virtue which is associated with fewer sexual partners (Deardorff et al., 2010). Alternatively, because Hispanic American girls are least likely to use contraceptives and least likely to communicate with others about sexual behavior compared to European and African Americans, Hispanic American girls may be unlikely to accurately report their sexual experiences (Deardorff et al., 2010; Raffaelli & Ontai, 2001).

For Asian Americans, sexual risk, STIs, and pregnancy did not change over time and this group often did not exhibit significant within group variability in these outcomes either. This is consistent with other studies that have found Asian American adolescents are less likely to report romantic relationships and sexual activity compared to other ethnic groups (O'Sullivan, Cheng, Harris, & Brooks-Gunn, 2007). Asian American adolescents are less likely to tell others that they have engaged in sexual activity, report fewer opposite sex interactions, and have less social support for dating (Grunbaum, Lowry, Kann, & Pateman, 2000).

Additionally, Asian Americans have some protective factors within their culture. Asian Americans are more likely to live in multigenerational homes and have higher SES (Cohen & Casper, 2002; East, 1998; Taylor et al., 2010). Furthermore, there may be cultural values unique to Asian Americans that are protective against and risky sexual behaviors. Parental respect is a cultural value often associated with many Asian cultures. Studies indicate that adolescents with

higher parental respect report less risk behaviors, presumably because they are less likely to disobey rules and maintain family cohesion (Shih, Miles, Tucker, Zhou, & D'Amico, 2012). These protective factors may buffer the negative effects of early puberty on sexual behavior and could be the reason for little change and within group variability in the current study. Future studies will want to include cultural indicators to assess cultural differences.

Limitations and future directions

There are limitations in the present study. First, from a theoretical standpoint, future research will need to investigate more than just sexual risk and pregnancy. Specifically, Belsky, Steinberg, and Draper (1991) highlight that early pubertal maturation accelerates sexual activity and orients the developing girl to unstable pair bonds and lower levels of parental investment. In this study, girls who matured early had higher sexual risk and pregnancy rates that continued into adulthood but this is only one part of the picture. This study was unable to examine if early maturation is associated with aspects of mate selection and parental investment as the evolutionary approach would suggest. For example, future studies will want to investigate attitudes and indictors of good parenting or investment in child rearing. If indicators of pair bonds and parenting are not associated with pubertal maturation then perhaps the theory will need to be revised.

The measure of puberty was another limitation. First, because puberty was measured at only one time point, this study may have failed to capture all early developing girls. In this study some of the 11 year old girls may have entered

puberty after these data were collected and should have been classified as early developers. However, because there was only one data point, these girls would have been classified as late because they were premenarcheal. Second, although age at menarche has been shown to be a valid and reliable measure of pubertal development (Caspi, Lynam, Moffit, & Silva, 1993; Graber, Petersen, & Brooks-Gunn, 1996), menarche occurs late in the pubertal process. Because many of the physical and hormonal changes have occurred prior to menarche, obtaining menarcheal status indicates that a girl has reached an advanced level of pubertal development. Studies should use multiple markers of pubertal development (i.e., physical development, menarche, perception of timing). Using multiple markers of pubertal development allows researchers to make more accurate conclusions considering different indicators are only moderately related.

Another limitation was the smaller sample size of Hispanic and Asian American girls. The combination of the cohort-sequential design of AddHealth coupled with the small sample size for Hispanic and Asian Americans resulted in not having any Hispanic Americans and Asian Americans at particular ages (Hispanic American $n = 277$; Asian American $n = 223$). It is difficult to distinguish whether the lack of findings in these groups is due to protective factors unique to Hispanic and Asian Americans (e.g., late pubertal development, self-esteem, positive family relations, and community ties) or whether the sample sizes were too small to capture variability. It is possible that different trajectories might have emerged for Asian Americans and Hispanic Americans with a larger sample that would increase the number of data points at all ages and variability

within each group. These relations should be retested with a larger sample of Hispanic and Asian Americans to confirm this assertion.

A final limitation was the lack of biological tests for STIs. Often studies of STIs are clinical samples of girls seeking health care with biological data (Boyer, Shafer, Wibbelsman, Seeberg et al., 2000; Liau, DiClemente, Wingood, Crosby et al., 2002). One strength of these studies is the collection of biological data. The strength of the present study was the use of a nationally representative sample however; girls may be reluctant to report STIs due to the stigma surrounding infections. The stigma associated with STIs might have led to low reporting rates among adolescents as STIs can influence self-percepts, fear, and feelings of unworthiness (East, Jackson, O'Brien, & Peters, 2012). These same feelings and stigma might not translate to teen pregnancy or risky sexually behaviors because there is greater acceptance of these occurrences. Future studies should use biological samples with STIs.

Conclusion

Overall, this investigation found that early developing European American and African American girls engaged in more risky sexual behavior and have higher rates of teen pregnancy than on-time or late developing girls. Moreover, early puberty predicted higher initial rates of sexual risk and pregnancies for European American and African American girls. However, no significant relations were found for Hispanic or Asian American girls. To date, this study was the first to examine how puberty is related to various aspects of sexual risk over into early adulthood within ethnic groups. Taken as a whole these findings

present a unique contribution to the study of puberty and sexual risk using an evolutionary approach

The current study suggests that prevention and intervention efforts should be aimed at girls entering puberty, particularly European American and African American girls. Given that menarche occurs late in the pubertal process, it would be advantageous to give sexual health information earlier than 14 when risk is already beginning to elevate. Puberty is directly related to sexual risk and pregnancy in early adolescence for European American and African American girls thus intervention efforts may target these groups more extensively. Future research should continue to examine if puberty is related to sexual risk for Hispanic and Asian Americans. If cultural values lessen the impact of early puberty, programs should be created to strengthen those values.

General Conclusions

In 1991, Belsky and colleagues proposed an evolutionary model in which psychosocial stressors were responsible for pubertal maturation and future reproductive strategy. Belsky's (1991) predictions were unique considering that the field had largely studied differences in puberty only in terms of weight and BMI, exercise, and nutrition. Specifically, Belsky and colleagues (1991) proposed that early childhood environments that are higher in stress would result in earlier pubertal maturation in order to lengthen the time and opportunity to reproduce. Thus according to their theory, early developing girls would initiate sexual activity earlier, have unstable relationships, and have low levels of parental involvement/investment. Alternatively, childhood environments that are low in stress would predict later pubertal maturation, later sexual activity, stable relationships, and higher levels of parental investment. However, past research and this dissertation provide inconsistent support for some of the tenants of this theory. This suggests that at the very least, more research needs to be conducted on certain aspects of the model.

In terms of support for Belsky and colleagues' (1991) model, consistent results between parenting behaviors and timing of pubertal maturation, in that, negative parenting predicts earlier maturation and positive parenting predicts later pubertal maturation (Belsky et al., 2007; Belsky, et al., 1998; Belsky, et al., 2010; Ellis, et al., 1999; Ellis & Garber, 2000; Graber, Brooks-Gunn, & Warren, 1995; Hetherington & Clingempell, 1992; Moffitt et al., 1992; Petit, Bates, & Dodge, 1997). Additionally, there is support for the associations between timing of

puberty and sexual debut, sexual risk, and timing of first pregnancy (Belsky et al., 2007; Deardorff et al., 2005; Downing & Bellis, 2009; Dunbar et al., 2006; Stattin & Magnusson, 1990; Waylen & Wolke, 2004). That is, early pubertal maturation has predicted early sexual debut, higher sexual risk, and early pregnancies. These studies have used methodologically sound investigations that provide reasonable support for these portions of Belsky and colleagues (1991) theory.

However, in this dissertation, I found that father absence and parent education were predictors of early pubertal maturation. Although other studies have highlighted the importance of parenting behaviors, my findings suggests that other stressors might be just as important. Moreover, as scholars study other indicators of the process of pubertal maturation, a more complex picture seems to emerge. Researchers using other indicators of stress (i.e., SES and father absence) found differing relationships between these variables in predicting pubertal maturation. These results are further complicated when examining ethnicity. For example, Deardorff and colleagues (2012) found father absence predicted earlier development in higher income families but not in lower income families; these results were stronger for African American girls. The process for European and African Americans may be similar, but studies will need to parcel out the effects of SES as research has largely used samples of high SES European Americans and low SES African Americans (Chumlea et al., 2003; Herman-Giddens et al., 1997; Wu, Mendola, & Buck, 2002).

Additionally, it is possible that SES and father absence have direct effects on puberty and indirect effects though parenting strategies. In this dissertation, I

found that SES and father absence had direct effect in pubertal timing; however, there might be indirect effects of SES and father absence through parenting behaviors. The present studies did not examine indirect effects and future studies should explore this possibility. Moreover, other aspects of the child's environment may influence puberty directly and indirectly. These may include daily hassles, neighborhoods, and community resources. Research has found that these are indicators of stress and are differentially associated with development across ethnic groups (Allison et al., 1999; Kim & Smith, 1998; Wilson, 1991, 1993). Adding these additional stressors might explain more of the variation in pubertal maturation and future sexual behavior. Future research will need to examine a larger variety of childhood contexts and their direct and indirect effects on pubertal maturation.

Furthermore, one of the tenants of Belsky and colleagues (1991) theory is that early pubertal development predicts unstable pair-bonds and future parenting behaviors. This assumption implies that early pubertal maturation would have effects that should be seen into early adulthood. Evidence for these associations is weak. This dissertation was one of the first to examine the long term outcomes of pubertal maturation. In this dissertation puberty predicted initial levels of sexual risk and pregnancy; however, sexual risk decelerated overtime and there was no effect of puberty on the slope for pregnancy. In addition, the only study to have examined theoretically relevant variables of mating and parenting failed to find associations between number of sexual partners, incidence of sexual infidelity, preference for a mate who displays indictors of good parenting and good genes,

and attitudes toward investment in children (Hoier, 2003). Taken together, these findings suggest that there may not be associations between puberty and future behaviors and attitudes into adulthood, as predicted by Belsky and colleagues (1991).

The primary problem with much of this research is that it does not address the underlying biological mechanisms. It has been theorized that the hypothalamic-pituitary-adrenal (HPA) axis, which is responsible for the regulation of stress, might play an important role in puberty, particularly andrenarche (i.e., maturation of the adrenal androgen production; Chisholm, Burbank, Coall, & Gemmiti, 2005; Dorn, Hitt, & Rotenstein, 1999). Research by Dorn and colleagues suggest that there is support for the physiological processes. Dorn and colleagues (1999) found that girls with premature adrenarche had higher production of cortisol relative to those without premature adrenarche. This indicates that girls with premature adrenarche have exaggerated reactivity of the HPA axis, suggesting that there is an underlying biological mechanism that connects stress and early andrenarche. This is also consistent with research that has found higher behavioral problems in girls with premature andrenarche (Dorn, Hitt, & Rotenstein, 1999). Caution should be noted as this line of research is in its infancy and contains some considerable methodological limitations. The research conducted to date in this area has been cross-sectional and longitudinal studies are needed to examine cortisol secretion before and after puberty. Moreover, measurement of pubertal hormone concentrations is difficult. Specifically, the current assay methods for detecting the hormones associated with puberty are not

sensitive enough for prepubertal children (Dorn, Dahl, Woodward, & Biro, 2006). Future research needs to address this underlying process.

Another major issue is that this theory focuses on individual level adaptation and largely ignores group level processes that might explain some of the variations between ethnic groups. Most psychologists do not differentiate between individual and group level adaptation, although Darwin hypothesized that adaptation occurs at various levels (Wilson, Van Vugt & Gorman, 2007). This perspective was cited as particularly relevant to cultural psychology considering that we are all humans but have adapted differently to survive in vastly different environments. Culturally humans evolve distinct adaptations to the local pressures and environment much quicker than genetic evolution alone (Boyd & Richerson; 2009; Wilson, 2005). Learning as a group allows human populations to acquire information over generations and leads to cultural evolution of highly adaptive behaviors. Perhaps group level adaptation might provide a basis for examining how cultural adaptation might play in the effects of (a) cortisol on accelerating pubertal development, (b) entering puberty early and, (c) early puberty on future sexual behaviors. As the Belsky and colleagues (1991) theory is written, there is currently no differentiation between individual and group level processes.

Given the findings in this dissertation, it is possible group level processes in adaptation are occurring. Findings for Hispanic and Asian Americans as groups were largely different than the other groups (although they were not directly compared in the second study). It is likely that there are some cultural variables

that account for some of these differences that might be better understood using a multilevel approach. Specifically, there are cultural values that are specific to Hispanic and Asian Americans including, familism, respect, and sexual virtue that could protect against early puberty and risk behavior (Cohen & Casper, 2002; Deardorff et al., 2010; East, 1998; Grunbaum, et al., 2000; Wahl & Eitle, 2010; Taylor et al., 2010). However, as Hispanic and Asian American adolescents become more acculturated to the mainstream American culture, their sexual attitudes and behaviors may become more consistent with European American norms. Perhaps as later generation Hispanic and Asian Americans, adopt the cultural norms, the same relations between stress, puberty and sexual behavior might emerge as European Americans and African Americans.

There is a larger proportion of Hispanic and Asian Americans that are more recent immigrants compared to European and African Americans. For Hispanic and Asian Americans, primary immigration occurred after the 1960's (U.S. Census Bureau, 1993). Data from AddHealth were collected in the 90s and in the 90s the majority of Asian Americans (66%) in 1990 were born in foreign countries (U.S. Census Bureau, 1993). Because more Hispanic and Asian Americans might be recent immigrants, they may have stronger associations with their cultural values regarding sexuality. Additionally, it is important to note that Hispanic and Asian Americans are very broad pan-ethnic groups, meaning, there is considerable cultural variation within these broad groups. For example, people of Asian ancestry differ in terms of national origin, language, nativity, religion, and customs. In the United States, 24% of the Asian population is Chinese, 20%

Filipino, followed by 11 to 12% each Japanese, Korean, and Vietnamese (U.S. Census Bureau, 2010). Similarly, of the Hispanic American population in the U.S. in 1990, 59% were Mexican, 10% Puerto Rican, 4% Cuban, and the rest Dominican, Central American, and South American. These groups have different historical backgrounds that might influence how quickly they acculturate to the dominant culture.

Implications

This dissertation, combined with the results from extant research, have some important implications for clinical practice. Family relationships and early pubertal development are significant stressors for adolescent girls. It places them at greater risk for poor sexual outcomes that persist into adulthood. Considering the role that the family plays in predicting pubertal maturation, intervention or prevention with the family might be a place to start. Because pediatricians, child psychologists, and nurse practitioners are often the first to learn of difficult family situations, they might be in the best position to discuss pubertal maturation and sexual health with both parents and adolescents. It is particularly important that intervention happen as early as possible considering that early menarche is associated with early onset of regular menstrual cycles. It is only approximately one year before a regular cycle begins if menarche occurs before 12 and four and a half years for a regular cycle if menarche occurs after 13 (MacMahon et al., 1982). Additionally, I found that puberty predicted initial levels of risk that carried over into adulthood which also reinforces how vital it is to reach adolescents early, before puberty. Furthermore, because adolescents are unlikely

to receive effective sex education in the school, clinical intervention becomes even more important. Often adolescents do not receive any information on puberty or sexual health until most adolescents are late, if not completed, in the pubertal process. Many schools also promote abstinence when these programs have no empirical evidence to suggest they are effective.

Future Directions

The two empirical articles in this document did have some limitations that future research will want to explore. First, measures of early childhood contexts should be collected at earlier ages than they were here. Evolutionary theory posits that it is the first 3-7 years of life that will determine the reproductive strategy. Thus, these measures were collected too late and are probably the cause for a lack of findings in the first paper. Additionally, future studies should explore the role of other environmental contexts (i.e., neighborhoods) in determining pubertal timing and possible ethnic differences in development. Stressors outside family relationships (i.e., SES) have been shown in this study to be associated with pubertal development and future studies should continue to explore these. Future work will also want to examine if the process for accelerating puberty for girls is similar to boys.

Evidence from this study could not address this important theoretical question: what are the physiological mechanisms that underlie early experiences to early sexual maturation and reproductive strategy. This is perhaps the most important area that needs to be addressed. Research in this area is difficult because of methodological limitations that need to be overcome. There are some

newer methods for the hormones associated with puberty at young ages however, these methods are not widely used or distributed. Although research has been limited, some preliminary work with humans suggests that prepubertal girls have higher cortisol levels. Additional research in rats also provides support and has found that maternal grooming of newborns helps regulate stress and delays the onset of puberty and sexual behavior (Cameron, Fish, & Meaney, 2008). However, it is unclear whether the processes are the same in rats and humans.

Given that these data came from a secondary source, this study could not address the genetic components of pubertal development. Future studies should incorporate these measures to control for genetic variation in pubertal maturation. Although some studies have incorporated control variables to account for genetic influences, all of these are still correlational and cannot rule out selection effects. The field will need to use experimental designs to test for the causal pathways from family environments to pubertal timing. This could potentially be done by including interventions that aim to change family relationships by promoting more positive relationships.

Conclusion

Taken as a whole, this dissertation partially supported an evolutionary approach to the study of pubertal development. The results support some propositions proposed by Belsky and colleagues (1991) however, there remains much to be learned. In this dissertation, I examined within and across ethnic group differences in the antecedents of pubertal development and within ethnic group differences in the trajectories of sexual risk. Overall, this study found

variations within and across ethnic groups that warrant further research particularly for Hispanic and Asian Americans who may or may not have similar developmental processes. Future work should focus on (a) understanding the biological mechanisms underlying the stress and puberty assertion, (b) focus more on the ethnic variations in these processes, and (c) determining whether the theory should extend into adulthood.

References

Adler, N.E. & Rehkopf, D.H. (2008). U.S. disparities in health: Descriptions, causes and mechanisms. *Annual Review of Public Health, 29*, 235-252. doi: 10.1146/annurev.publhealth.29.020907.09

Alan Guttmacher Institute (2009). Facts in brief: Teen sex and pregnancy in the first months of intercourse. Retrieved from: www.agi-usa.org/pubs/fb_teen_sex.pdf

Allison, K. W., Burton, L. M., Marshall, S., Perez-Febles, A., Yarrington, J., Kirsh, L. B., & Merriwether-DeVries, C. (1999). Life experiences among urban adolescents: Examining the role of context. *Child Development, 70,* 1017–1029. doi:10.111/1467-8624.00074

Bearman, P.S., Jones, J., & Udry, J.R. (1997). *The national study of adolescent health: Research design*. Retrieved from www.cpc.unc.edu/ projects/addhealth/design.html.

Belsky, J., Hsieh, K.-H., & Crnic, K. (1998). Mothering, fathering, and infant negativity as antecedents of boys' externalizing problems and inhibition at age 3 years: Differential susceptibility to rearing experience? *Development and Psychopathology, 10,* 301–319. doi: 10.1017/S095457949800162X

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development, 62*, 647-670. doi: 10.2307/1131166

Belsky, J., Steinberg, L.D., Houts, R.M., Friedman, S.L., DeHart, G., Cauffman, E., et al. (2007). Family rearing antecedents of pubertal timing. *Child Development, 78*, 1302-1321. doi: 10.1111/j.1467-8624.2007.01067.x

Belsky, J., Steinberg, L.D., Houts, R.M., & Halpbern-Felsher, B.L. (2010). The development of reproductive strategy in females: Early maternal harshness, earlier menarche, increased sexual risk taking. *Developmental Psychology, 46*, 120-128. doi: 10.1037/a0015549

Berry, H.E., Shillington, A.M., Peak, T., & Hohman, M.M. (2000). Multi-ethnic comparison of risk and protective factors for adolescent pregnancy. *Child and Adolescent Social Work Journal, 17*, 79-96. doi: 10.1023/A:1007554122283

Billy, J.G., Brewster, K.L., & Grady, W.R. (1994). Contextual effects on the sexual behavior of adolescent women. *Journal of Marriage and the Family, 56,* 387-404. doi: 10.2307/353107

Bingham, C.R., Miller, B.C., & Adams, G.R. (1990). Correlates of age at first sexual intercourse in a national sample of young women. *Journal of Adolescent Research, 5*, 18-33. Doi: 10.1177/074355489051004

Bollen, K. A., & Curran, P. J. (2006). *Latent curve models: A structural equation perspective. Wiley series in probability and mathematical statistics*. New York: Wiley.

Boyer, C.B., Pollack, L.M., Becnel, J.N., & Shafer, M.A. (2008). Relationships among sociodemographic markers, behavioral risk, and sexually transmitted infections in U.S. female Marine Corps recruits. *Military Medicine, 173*, 1078-1084. Retrieved from: http://login.ezproxy1.lib.asu.edu/login?url=http://search.proquest.com.ezproxy1.lib.asu.edu/docview/621624636?accountid=4485

Boyer, C.B., Shafer, M., Wibbelsman, C.J., Seeberg, D., Teitle, E., & Lovell, N. (2000). Associations of sociodemographic, psychosocial, and behavioral factors with sexual risk and sexually transmitted diseases in teen clinic patients. *Journal of Adolescent Health, 27*, 102-111. doi: http://login.ezproxy1.lib.asu.edu/login?url=http://search.proquest.com.ezproxy1.lib.asu.edu/docview/621930892?accountid=4485

Braithwaite, D., Moore, D.H., Lustig, R.H., Epel, E.S., Ong, K.K., Rehkopf, D.H., Wang, M.C. et al. (2008). Socioeconomic status in relation to early menarche among black and white girls. *Cancer Causes Control, 20*, 713-720. doi: 10.1007/s10552-008-9284-9

Brooks-Gunn, J., Graber, J. A., & Paikoff, R. L. (1994). Studying links between hormones and negative affect: Models and measures. *Journal of Research on Adolescence, 4*, 469–486. doi: 10.1207/s15327795jra0404_2

Bronfenbrenner, U. & Morris, P.A. (1998). The ecology of developmental processes. In W. Damon & R.M. Learner (Eds.), *Handbook of child psychology: Volume 1: Theoretical models of human development* (pp.993-1028). Hoboken, NJ: John Wiley & Sons inc.

Brown, T. N. (2001). Measuring self-perceived racial and ethnic discrimination in social surveys. *Sociological Spectrum, 21*, 377–392. doi: 10.1080/027321701300202046

Brown, K.M., McMahon, R.P., Biro, F.M., Crawford, P., Schreiber, G.B. et al. (1998). Changes in self-esteem on Black and White girls between the ages of 9 and 14 years: The NHLBI growth and health study. *Journal of Adolescent Health, 23*, 7-19. doi: 10.1016/S1054-139X(97)00238-3

Brumberg, J. (1997). *The body project: An intimate history of American girls*. New York: Vintage.

Cameron, N.M., Fish, E.W., & Meaney, M.J. (2008). Maternal influences on sexual behavior and reproductive success of the female rat. *Hormones and Behavior, 54*, 178-184. doi: 10.1016/j.yhbeh.2008.02.013

Campbell, B.C. & Udry, J.R. (1995). Stress and age at menarche of mothers and daughters. *Journal of Biosocial Science, 27*, 127-134. doi: 10.1017/S0021932000022641

Cairney, J., Boyle, M., Offord, D.R. & Racine, Y. (2003). Stress, social support and depression in single and married mothers. *Social Psychiatry and Psychiatric Epidemiology, 38*, 442-449. doi: 10.1007/s00127-003-0661-0

Caspi, A., Lynam, D., Moffit, T., & Silva, P. (1993). Unraveling girls' delinquency: Biological, dispositional, and contextual contributions to adolescent misbehavior. *Developmental Psychology, 29*, 19–30. doi: 10.1037/0012-1649.29.1.19

Cavanagh, S.E. (2004). The sexual debut of girls in early adolescence: The intersection of race, pubertal timing, and friendship group characteristics. *Journal of Research on Adolescence, 14*, 285-312. doi: 10.1111/j.1532-7795.2004.00076.x

Center for Disease Control and Prevention (2003). *Assessing your weight*. Retrieved from http://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/childrens_bmi_formula.html

Centers for Disease Control and Prevention (2009). *Tracking the hidden epidemics: Trends in STDs in the United States, 2000.* Atlanta, CA: Division of Sexually Transmitted Diseases, United States Department of Health and Human Services. Retrieved from: http://www.cdc.gov/std/Trends2000/trends2000.pdf

Chisholm, J.S., Burbank, V.K., Coall, D.A., & Gemmiti, F. (2005). Early stress: Perspectives from developmental evolutionary biology. In B.J. Ellis & D.F. Bjorklund (Eds.), *Origins of the social mind: Evolutionary psychology and child development* (pp. 76-107). New York: Guildford.

Christopher, F.S. (2001). *To dance the dance: A symbolic interactional exploration of premarital sexuality*. Mahway, NJ: Lawrence Erlbaum.

Chumlea, W.C., Schubert, C.M., Roche, A.F., Kulin, H.E., Lee, P.A., Himes, J.H. et al. (2003). Age at menarche and racial comparisons in US girls. *Pediatrics, 111*, 110-113. doi: 10.1542/peds.111.1.110

Cohen, P.N. & Casper, L.M. (2002). In whose home? Multigenerational families in the United States, 1998-2000. *Sociological Perspectives, 45,* 1-20. doi:

Costello, D.M., Swendsen, J., Rose, J.S. & Dierker, L.C. (2008). Risk and protective factors associated with trajectories of depressed mood from adolescent to early adulthood. *Journal of Consulting and Clinical Psychology, 76,* 173-183. doi: 10.1037/0022-006X.76.2.173

Duncan, G. J., Brooks-Gunn, J.,&Klebanov, P.K. (1994). Economic deprivation and early childhood development. *Child Development, 65,* 296–318. doi: 10.2307/1131385

Deardorff, J., Ekwaru, J.P., Kushi, L.H., Ellis, B.J., Greenspan, L.C., Mirabedi, A., Landaverde, E.G. et al. (2010). Father absence, body mass index, and pubertal timing in girls: differential effects by family income and ethnicity. *Journal of Adolescent Health, 48,* 441-447. doi: 10.1016/j.jadohealth.2010.07.032

Deardorff, J., Gonzales, N.A., Christopher, F.S., Roosa, M.W., & Milsap, R.E. (2005). Early puberty and adolescent pregnancy: The influence of alcohol use. *Pediatrics, 116,* 1451-1456. doi: 10.1542/peds.2005-0542

Dick, D.M., Rose, R.J., Viken, R.J., & Kaprio, J. (2000). Pubertal timing and substance use: Associations between and within families across late adolescence. *Developmental Psychology, 36,* 180-189. doi: 10.1037/0012-1649.36.2.180

Downing, J., & Bellis, M.A. (2009). Early pubertal onset and its relationship with sexual risk taking, substance use and anti-social behavior: A preliminary cross sectional study. *BMC Public Health, 9,* 446-457. doi:10.1186/1471-2458-9-446

Draper, P. & Harpending, H. (1982) Father absence and reproductive strategy: An evolutionary perspective. *Journal of Anthropological Research, 38,* 255-273. Retreived from: http://www.jstor.org/stable/3629848

Dunbar, J., Sheeder, J., Lezotte, D., Dabelea, D., & Stevens-Simons, C. (2006). Age at menarche and first pregnancy among psychosocially at-risk adolescents. *American Journal of Public Health, 98,* 1822-1824. doi: 10.2105/AJPH.2007.120444

Duncan, S.C., Duncan, T.E., & Hops, H. (1996). Analysis of longitudinal data within accelerated longitudinal designs. *Psychological Methods, 1,* 236-248. doi: 10.1037/1082-989X.1.3.236

East, L., Jackson, D., O'Brein, L. & Peters, K. (2012). Stigma and Stereotypes: Women and sexually transmitted infections. *Collegian, 19*, 15-21.

East, P.L. (1998). Racial and ethnic differences in girls sexual, marital, and birth expectations. *Journal of Marriage and the Family, 60*, 150-162. doi:10.2307/353448

Eccles, J.S., Furstenberg, F., McCarthy, K., Lord, S. & Geitze, L. (1993). *How parents respond to risk and opportunity in moderate to high risk neighborhoods.* Retrieved from: http://www.rcgd.isr.umich.edu /garp/presentations/eccles93f.pdf

Ellis, B.J. (2004). Timing of pubertal maturation in girls: An integrated life history approach. *Psychological Bulletin, 130*, 920-958. doi:10.1037/0033-2909.130.6.920

Ellis, B.J., & Essex, M.J. (2007). Family environments, adrenarche, and sexual maturation: A longitudinal test of a life history model. *Child Development, 78*, 1799-1817. doi: 10.1111/j.1467-8624.2007.01092.x

Ellis, B.J. & Garber, J. (2000). Psychosocial antecedents of variation in girls pubertal timing: Maternal depression, stepfather presence, and marital and family stress. *Child Development, 71*, 485-501. doi: 10.1111/1467-8624.00159

Ellis, B.J., McFadyen-Ketchum, S., Dodge, K.A., Pettit, G.S., & Bates, J.E. (1999). Quality of early family relationships and individual differences in the timing of pubertal maturation in girls. *Journal of Personality and Social Psychology, 77*, 387-401. doi: 10.1037/0022-3514.77.2.238

Fuemmeler, B.F., Yang, C., Costanzo, P., Hoyle, R.H., Siegler, H.C., Williams, R.B. et al. (2012). Parenting styles and body mass index trajectories from adolescence to adulthood. *Health Psychology, 31*, 441-449. doi: 10.1037/a0027929

Garcia Coll, C., Crnic, K., Lamberty, G. & Wasik, B.H. (1996). An integrative model for the study of developmental competencies in minority children. *Child Development, 67*, 1891-1914. doi: 10.2307/1131600

Ge, X., Conger, R.D., & Elder, G.H. (1996). Coming of age too early: Pubertal influences on girls vulnerability to psychological distress. *Child Development, 67*, 3386-3400. doi: 10.2307/1131784

Ge, X., Elder, G.H., Regnerus, M., & Cox, C. (2001). Pubertal transitions, perceptions of being overweight, and adolescents' psychological maladjustment: Gender and ethnic differences. *Social Psychology Quarterly, 64*, 363-375. doi: 10.2307/3090160

Gee, G. C. (2002). A multilevel analysis of the relationship between institutional and individual racial discrimination and health status. *American Journal of Public Health, 92*, 615–623. Retreived from: http://login.ezproxy1.lib.asu.edu/login?url=http://search.proquest.com.ezproxy1.lib.asu.edu/docview/622145859?accountid=4485

Graber, J. A., Brooks-Gunn, J., &Warren, M. P. (1995). The antecedents of menarcheal age: Heredity, family environment, and stressful life events. *Child Development, 66,* 346–359. doi: 10.2307/1131582

Graber, J. A., Peterson, A. C., & Brooks-Gunn, J. (1996). Pubertal processes: Methods, measures, and models. In J. A. Graber, A. C. Peterson, & J. Brooks-Gunn (Eds.), *Transitions through adolescence: Interpersonal domains and context* (pp. 23–54). Mahwah, NJ: Erlbaum.

Grunbaum, J.A., Lowry, R., Kann, L. & Pateman, B. (2000). Prevalence of health risk behaviors among Asian American/Pacific Islander high school students. Journal of Adolescent Health, 27, 322-330. doi: 10.1016/S1054

Halpern, C.T., Kaestle, C.E., & Hallfors, D.D. (2007). Perceived physical maturity, age of romantic partner, and adolescent risk behavior. *Prevention Science, 8*, 1-10. doi: 10.1007/s11121-006-0046-1

Halpern, C.T., King, R.B., Oslak, S.G., & Udry, R.J. (2005). Body mass index, dieting, romance, and sexual activity in adolescent girls: Relationships over time. *Journal of Research on Adolescence, 15*, 535-559. doi: 10.1111/j.1532-7795.2005.00110.x

Hamilton, B.E., Sutton, P.D., & Ventura, S.J. (2003). *Revised birth and fertility rates for 1990s and new rates of Hispanic population, 2000 and 2001: United States. National Vital Statistics Report, 15, 12-13*. Hyattsville, MD: National Center for Health Statistics

Hayward, C., Killen, J.D., Wilson, D.M., Hammer, L.D., Litt, I.F., Kraemer, H.C. et al., (1997). Psychiatric risk associated with early puberty in adolescent girls. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 255-262. Retrieved from http://login.ezproxy1.lib.asu.edu/login?url=http://search.proquest.com.ezproxy1.lib.asu.edu/docview/619049340?accountid=4485

Herman-Giddens, M.E., Slora, E.J., Wasserman, R.C., Bourdony, C.J., Bhapkar, M.V., Koch, G.G. et al. (1997). Secondary sexual characteristics and menses in young girls seen in office practice: A study from the pediatric research in office setting network. *Pediatrics, 99*, 505-512. doi: 10.1542/peds.99.4.505

Herman-Giddens, M.E., Slora, E., Hasemeier, C., & Wasserman, R. (1993). The prevalence of secondary sexual characteristics of young girls seen in office practice. *Pediatrics, 88*, 147-455. Doi: http://login.ezproxy1.lib.asu.edu/login?url=http://search.proquest.com.ezproxy1.lib.asu.edu/docview/618310646?accountid=4485

Hetherington, E. M., & Clingempeel, W. G. (1992). Coping with marital transitions: A family systems perspective. *Monographs of the Society for Research in Child Development, 57*(2–3, Serial No. 227).

Hetherington, E. M., & Kelly, J. (2002). *For better or for worse: Divorce reconsidered*. New York: Norton.

Hin, H.S., Chen, J.Z., Su, J.Z., Zhu, F.C., Xing, W.H., & LiJ-Y, G.S. (1992). The menarcheal age of Chinese girls. *Annuals of Human Biology, 19*, 503-512. doi: 10.1080/03014469200002332

Hofferth, S.L. & Sandberg, J.F. (2001). How American children spend their time. *Journal of Marriage and Family, 63*, 295-308. doi: 10.1111/j.1741-3737

Hogan, D.P., Hao, L.X. & Parish, W.L. (1990). Race, kin networks, and assistance to mother headed families. *Social Forces, 68*, 797-812. doi: 10.1235/K1384-3001-23

James, J., Ellis, B.J., Schlomer, G.L., & Garber, J. (2012). Sex-specific pathways to early puberty, sexual debut, and sexual risk taking: Tests of an integrated evolutionary-developmental model. *Developmental Psychology, 48*, 687-702. doi: 10.1037/a0026427

Jencks, C.,&Mayer, S. E. (1990). Residential segregation, job proximity and Black job opportunities. In L. E. Lynn, Jr.&M.G.H. McGeary (Eds.), *Inner-city poverty in the United States* (pp. 187–222). Washington, DC: National Academy Press.

Johnson, D.J., Jaeger, E., Randolph, S.M., Cauce, A.M., Ward, J., & NICHD Early Child Care Research Network (2003). Studying the effects of early child care experiences on the development of children of color in the United States: Toward a more inclusive research agenda. *Child Development, 74*, 1227–1244. doi: 10.1111/1467-8624.00604

Jorm, A.F., Christensen, H., Rodgers, B. Jacomb, P.A. & Easteal, S. (2004). Association of adverse childhood experiences, age at menarche, and adult reproductive behavior: Does the androgen receptor gene play a role. *American Journal of Medical Genetics, 15*, 105-111. doi: 10.1002/ajmg.b.20114

Kaplowitz, P., Slora, E., Wasserman, R., Pedlow, S., & Herman-Giddens, M. (2001). Earlier onset of puberty in girls: Relation to increased body mass index and race. *Pediatrics, 108*, 347-353. doi: 10.1542/peds.108.2.347

Koplewicz, H.S., Gurian, A. & Williams, K. (2009). The era of affluence and its discontents. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*, 1053-1055. doi: 10.1097/CHI.0b013e3181b8be5c

Kim, K., & Smith, P. K. (1998). Retrospective survey of parental marital relations and child reproductive development. *International Journal of Behavioral Development, 22*, 729-751. doi: 10.1080/016502598384144

Liau, DiClemente, Wingood, Crosby et al., (2002). Associations between biologically confirmed marijuana use and laboratory-confirmed sexually transmitted diseases among African American adolescent females. *Sexually Transmitted Diseases, 29*, 387-390.

Lucky, A.W., Biro, F. M., Simbartl, L. A., Morrison, J. A., & Sorg, N.W. (1997). Predictors of severity of acne vulgaris in young adolescent girls: Results of a five-year longitudinal study. *Journal of Pediatrics, 130*, 30–39. doi: 10.1016/S0022-3476(97)70307-X

Luthar, S.S. & Becker, B.E. (2002). Privileged but pressed? A study of affluent youth. *Child Development, 73*, 1593-1610. doi: 10.1111/1467-8624.00492

Manlove, J. (1997). Early motherhood in an intergenerational perspective: The experiences of a British cohort. *Journal of Marriage and the Family, 59*, 263-279. doi: 10.2307/353469

McLoyd, V.C. (1990). The impact of economic hardship on black families and children: Psychological distress, parenting, and socioemotional development. *Child Development, 65*, 562-589. doi: 10.2307/1131096

Milkie, M. A. (1999). Social comparisons, reflected appraisals, and mass media: The impact of pervasive beauty images on Black and White girls' self-concepts. *Social Psychology Quarterly, 62*, 190–210. doi: 10.2307/2695857

Miller, B. C., Norton, M. C., Curtis, T., Hill, E. J., Schvaneveldt, P., & Young, M. H. (1997). The timing of sexual intercourse among adolescents: Family, peer, and other antecedents. *Youth & Society, 29*, 54–84. doi: 10.1177/0044118X97029001003

Miyazaki, Y., & Raudenbush, S.W. (2000). Tests for linkage of multiple cohorts in an accelerated longitudinal design. *Psychological Methods, 5*, 44-63. doi: 10.1037/1082-989X.5.1.44

Moffitt, T. E., Caspi, A., Belsky, J., & Silva, P. A. (1992). Childhood experience and the onset of menarche: A test of a sociobiological model. *Child Development, 63,* 47–58. doi: 10.2307/1130900

Moore, K.A., Miller, B.C., Glei, D., & Morrison, D.R. (1995). *Adolescent sex, contraception, and child bearing: A review of recent research.* Washington, DC: Child Trends, Inc.

Muthen, L.K. (2006, December 5). Multiple group growth model. Message posted to http://www.statmodel.com/discussion/messages/14/1867.html

Muthén, L.K & Muthén, B.O. (1998-2004). *Mplus 3.0: User's guide*. Los Angeles: Author.

Natsuaki, M.N., Biehl, M.C., & Ge, X. (2009). Trajectories of depressed mood from early adolescence to young adulthood: The effects of pubertal timing and adolescent dating. *Journal of Research on Adolescence, 19,* 47-74. doi: 10.1111/j.1532-7795.2009.00581.x

Neistein, L.S., & Kaufman, F.R. (2002). Normal physical growth and development. In L. Neinstein (Ed.). *Adolescent Health Care: A Practical Guide, 4th ed*. Philadelphia: Lippincott Williams & Williams, pp. 3-51.

O'Sullivan, L.F., Meyer-Bahlburg, H.F.L., & Watkins, B.X. (2000). Social cognitions associated with pubertal development in a sample of urban, low-income, African-American and Latina girls and mothers. *Journal of Adolescent Health, 27,* 227-235. doi: 10.1016/S1054-139X(99)00111-1

Obeidallah, D., Brennan, R., Brooks-Gunn, J., Kindon, T., Earls, F. (2000). Socioeconomic status, race, and girls' pubertal maturation: Results from the project on human development in Chicago neighborhoods. *Journal of Research on Adolescence, 10*, 443-464. Retrieved from http://www.tandfonline.com/doi/abs/10.1207/SJRA1004_04#preview

Okazaki, S. (2002). Influences of culture on Asian Americans' sexuality. *Journal of Sex Research, 39*, 34-41. doi: 10.1080/00224490209552117

Peterson, A. C., Graber, J. A. & Sullivan, P. (1990). Pubertal timing and grade effects on adjustment. *Journal of Youth and Adolescence, 15*, 191-206. doi: 10.1007/BF02090318

Petit, G.S., Bates, J.E., & Dodge, K.A. (1997). Supportive parenting, ecological context, and children's adjustment: A seven year longitudinal study. *Child Development, 68*, 908-923. Retrieved from http://login.ezproxy1.lib.asu.edu/login?url=http://search.proquest.com.ezproxy1.lib.asu.edu/docview/619219769?accountid=4485

Phinny, V.G., Jensen, L.C., Olsen, J.A., & Cundick, B. (1990). The relationship between early development and psychosexual behaviors in adolescent females. *Adolescence, 25*, 321-332. Retrieved from http://login.ezproxy1.lib.asu.edu/login?url=http://search.proquest.com.ezproxy1.lib.asu.edu/docview/617782451?accountid=4485

Piakoff, R.L., Brooks-Gunn, J., & Warren, M.P. (1991). Effects of girls' hormonal status on depressive and aggressive symptoms over the course of one year. *Journal of Youth and Adolescence, 20*, 191-215. doi: 10.1007/BF01537608

Population Reference Bureau (2010). Data brief: U.S. children in single mother families. Retrieved August 11, 2010. from The Population Reference Bureau, Web site: www.wkkf.org/~/media/39F70A1BE2364C50A610B7E806CC4D02.ashx.

Prelow, H.M. & Guarnaccia, C.A. (1997). Ethnic and racial differences in life stress among high school adolescents. *Journal of Counseling and Development, 75*, 442-450. doi: 10.1002/j.5556-6676.1997.tb02360.x

Romans, S.E., Martin, M., Gendall, K., & Herbison, G.P. (2003). Age of menarche: The role of some psychosocial factors. *Psychological Medicine, 33*, 933-939. doi: 10.1017/S0033291703007530

Rowe, D.C. (2000). On genetic variation in menarche and age at first sexual intercourse: A critique of the Belsky-Draper hypothesis. *Evolution and Human Behavior, 23*, 365-372. doi: 10.1016/S1090-5138(02)00102-2

Shrier, L.A., Emans, S.J., Woods, E.R., & Durant, R.H. (1997). The association of sexual risk behaviors and problem drug behaviors in high school students. *Journal of Adolescent Health, 20*, 337-383. doi: 10.1016/S1054-139X(96)000180-2

Siegel, J.M., Aneshensel, C.S., Taub, B., Cantwell, D.P., & Driscoll, A.K. (1998). Adolescent depressed mood in a multiethnic sample. *Journal of Youth and Adolescence, 27*, 413-427. doi: 10.1023/A:1022873601030

Silbereisen, R. K., Petersen, A. C., Albrecht, H. T.,&Kracke, B. (1989). Maturational timing and the development of problem behavior: Longitudinal studies in adolescence. *Journal of Early Adolescence, 9,* 247–268. doi: 10.1177/0272431689093005

Singer, J.D. & Willett, J.B. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. Oxford University Press, New York: NY

Smith, E., Udry, J., & Morris, N. (1985). Pubertal development and friends: A biosocial explanation of adolescent sexual behavior. *Journal of Health and Social Behavior, 26,* 183-192. doi: 10.2307/2136751

Spaights, E. & Whitaker, A. (1995). Black women in the workforce: A new look at an old problem. *Journal of Black Studies, 25*, 283-296. doi: 10.1177/002193479502500301

Stattin, H., & Magnusson, D. (1990). *Pubertal maturation in female development*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Steinberg, L. (2007). Risk taking in adolescence: New perspectives from brain and behavioral science, *Current Directions in Psychological Science, 16,* 55-59. doi: 10.1111/j.1467-8721.2007.00475.x

Steinberg, L., Lamborn, S.D., Darling, N., Mounts, N.S., & Dornbusch, S.M. (1994). Over-time changes in adjustment and competence among adolescents from authoritative, authoritarian, indulgent, and neglectful families. *Child Development, 65*, 754-770.

Steinberg, L., Mounts, N., Lamborn, S., & Dornbusch, S.M. (1991). Authoritative parenting and adolescent adjustment across varied ecological niches. *Journal of Research on Adolescence, 1*, 19-36.

Susman, E. J., Nottelmann, E. D., Dorn, L. D., Inoff-Germain, G., & Chrousos, G. P. (1988). Physiological and behavioral aspects of stress in adolescence. In G. P. Chrousos & D. L. Loriaux (Eds.), *Advances in experimental medicine and biology: Mechanisms of physical and emotional stress* (Vol. 245, pp. 341–352). New York: Plenum.

Tabachnick, B.G. & Fidell, L.S. (2007). *Using Multivariate Statistics (5th Ed.).* Boston, MA: Allyn and Bacon.

Taylor, P., Passel, J., Fry, R., Morin, R., Wang, W., Velasco, G. & Dockterman, D. (2010). The return of the multi-generational family household. Washing DC: Pew Research Center. http://pewsocialtrends.org /files/2010/10/752-multi-generational-families.pdf

Tither, J.M., & Ellis, B.J. (2008). Impact of fathers on daughters' age of menarche: A genetically and environmentally controlled sibling study. *Developmental Psychology, 44*, 1409-1420. doi: 10.1037/a0013065

U.S. Census Bureau (2001). *Overview of race and Hispanic origin*. Retrieved June 3, 2010, from The U.S. Census Bureau, Web site: http://www.census.gov/prod/2001pubs/c2kbr011.pdf.

Upchurch, D., Levy-Storms, L., Sucoff, C., & Aneschensel, C. (1998). Gender and ethnic differences in the timing of first sexual intercourse. *Family Planning Perspectives, 30*, 121–127. Retrieved from http://www.guttmacher.org.ezproxy1.lib.asu.edu/pubs/journals/3012198.html

Villarreal, S.F., Martorell, R.A., & Mendoza, F.S. (1989). Sexual maturation of Mexican-American adolescents. *American Journal of Human Biology, 1*, 87-95. doi: 10.1002/ajhb.1310010113

Wahl, A.G. & Eitle, T.M. (2010). Gender, acculturation and alcohol use among Latina adolescents: A multi-ethnic comparison. *Journal of Immigrant and Minority Health, 12*, 153-165. doi: 10.1007/s10903-008-9179

Waylen, A., & Wolke, D. (2004). Sex n drugs n rock n roll: The meaning and social consequences of pubertal timing. *European Journal of Endocrinology, 151*, 151-159. Retrieved from http://eje-online.org.ezproxy1.lib.asu.edu/content/151/Suppl_3/U151.long

Wierson, M., Long, P.J., & Forehand, R.L. (1993). Toward a new understanding of early menarche: The role of environmental stress in pubertal timing. *Adolescence, 28*, 913-924. Retrieved from http://login.ezproxy1.lib.asu.edu/login?url=http://search.proquest.com.ezproxy1.lib.asu.edu/docview/618440740?accountid=4485

Wilson,W. J. (1991). Public policy research and the truly disadvantaged. In C. Jencks&P. E. Peterson (Eds.), *The urban underclass* (pp. 460–482).Washington, DC: Brookings Institue.

Wilson,W. J. (1993). *The ghetto underclass: Social science perspectives.* NewburyPark, CA: Sage.

Wu, T., Mendola, P., & Buck, G.M. (2002). Ethnic differences in the presence of secondary sex characteristics and menarche among US girls: The third national health and nutrition examination survey. *Pediatrics, 110*, 752-757. doi: 10.1542/peds.110.4.752

Table 1

Participant Demographic Characteristics

Indicator	Full Sample M(SD)/%	European American M(SD)/%	African American M(SD)/%	Hispanic American M(SD)/%	Asian American M(SD)/%	*F* ratio; η^2/ χ^2; V_c
Puberty						
Early	30%	27%	36%	33%	22%	33.74; 09[***b]
On-Time/Late	70%	73%	64%	67%	78%	
Age	14.56(1.21)	14.52(1.20)	14.53(1.22)	14.82(1.19)	14.75(1.24)	8.08; .01[***c]
BMI	21.97(4.31)	21.44(4.01)	23.21(4.81)	22.98(4.01)	20.46(3.95)	2.06
Parent education	1.76(1.17)	1.84(1.12)	1.67(1.19)	1.00(1.12)	2.18(1.28)	59.34; .04[***d]
Income[a]	0-99	0-99	0-50	0-25	0-88	9.27; .01[***e]
Behavioral control	2.09(1.52)	2.03(1.47)	2.22(1.59)	2.36(1.60)	2.11(1.59)	7.57; .02[***f]
Mother warmth	4.41(0.67)	4.42(0.65)	4.43(0.68)	4.34(0.68)	4.30(0.73)	2.37
Father absence						
Father present	53%	60%	32%	53%	74%	369.68; .21[***g]
Not present	30%	30%	35%	30%	17%	
Never present	17%	10%	33%	17%	9%	

[a] Income is reported using the range in the 10,000's

[b] Higher proportion of African American and Hispanic Americans lower proportions of European and Asian Americans classified as early than would be expected by chance.

[c] Significant differences between Hispanic Americans and European and African Americans

[d] All comparisons were significantly different.

[e] Asian Americans were significantly different from all other groups

[f] European Americans were significantly different from African and Hispanic Americans

[g] Larger proportion of father absence for African and Hispanic American and lower proportion of European and Asian Americans than would be expected by chance

Table 2

Correlations among Study Variables for European American Below the Diagonal and African Americans Above the Diagonal

Indicator	1.	2.	3.	4.	5.	6.	7.	8.
1. Puberty	--	-.14***	.07*	.06*	.03	-.05	-.02	.06**
2. Age	.01	--	.11***	.01	.06	-.20***	-.14***	.01
3. BMI	.16***	.15**	--	.06*	-.04	-.03	-.07*	.12***
4. Parent education	-.06***	-.03	-.10***	--	.41***	-.06*	.04	-.22***
5. Income	-.04*	.02	-.07***	.27***	--	-.01	.03	-.34***
6. Behavioral control	-.03	-.30***	-.02	-.05*	-.06***	--	.04	.01
7. Warmth	-.02	-.14***	-.01	.02	.00	.06**	--	-.05
8. Father absence	.07**	.02	.04*	-.09***	-.17***	-.02	-.06***	--

Note. * $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 3

Correlations among Study Variables for Hispanic American Below the Diagonal and Asian Americans Above the Diagonal

Indicator	1.	2.	3.	4.	5.	6.	7.	8.
1. Puberty	--	-.14*	.15*	.04	.09	.08	-.03	.08
2.Age	-.05	--	.15*	.05	-.13	-.08	-.10	-.07
3. BMI	.08	.14**	--	-.02	-.14	.20**	.06	.01
4. Parent education	-.01	-.16**	-.07	--	.26***	.17*	.00	.18*
5. Income	.00	-.09	-.06	.37***	--	.02	-.17*	-.14
6. Behavioral control	.06	-.21***	-.05	-.04	-.04	--	-.21**	.10
7. Warmth	.02	-.12*	.04	.02	.12*	.03	--	.00
8. Father absence	.07	.06	-.10	.04	-.20***	-.02	-.05**	--

Note. * $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 4

Models Predicting Pubertal Development with European Americans as the Reference Group

Model	Parameter	*B*	Wald χ^2 Square	Odds Ratio	95% CI for Odds Ratio	Model χ^2	$\Delta \chi^2$
Model 1	Intercept	3.78	295.79***				
	BMI	.07	57.03***	1.07	1.05-1.09	52.30	
Model 2	Intercept	3.75	280.61***				
	BMI	.06	39.24***	1.06	1.04-1.07		
	African American	.56	28.58***	1.75	1.41-2.07		
	Hispanic American	.43	6.27**	1.52	1.08-2.04		
	Asian American	-.16	.29	.87	.86-1.17	83.30	31.00
Model 3	Intercept	3.22	60.23***				
	BMI	.06	33.90***	1.06	1.04-1.08		
	African American	.44	16.34***	1.56	1.26-1.85		
	Hispanic American	.35	4.07*	1.19	1.01-2.02		
	Asian American	-.09	.09	.91	.51-1.63		
	Parent Education	-.05	1.14	.95	.87-1.04		
	Income	-.01	2.34	.99	.99-1.00		
	Behavioral Control	-.05	2.93	.95	.89-1.00		
	Warmth	-.06	.83	.94	.82-1.08		
	Father Absence	.16	6.18**	1.18	1.04-1.24	102.26	49.96
Model 4	Intercept	3.35	35.87***				
	BMI	.07	29.68***	1.07	1.07-1.11		
	African American	.63	.52	1.87	1.87-2.75		
	Hispanic American	1.61	1.16	5.00	4.99-9.56		
	Asian American	-.17	.01	.84	.53-2.74		
	Parent Education	-.07	6.19**	.93	.92-.96		
	Income	-.01	1.24	1.00	.99-1.00		
	Behavioral Control	-.08	3.18	.92	.92-.94		
	Mother Warmth	-.09	.85	.91	.91-.95		
	Father Absence	.28	9.60***	1.32	1.27-1.36		
	BMI x Af. Am.	-.05	11.91***				
	P. Ed. x Af. Am.	.21	4.49**			125.85	73.55

Note. Comparisons are made to the European American group.
* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 5

Models Predicting Pubertal Development with African Americans as the Reference Group

Model	Parameter	*B*	Wald χ^2 Square	Odds Ratio	95% CI for Odds Ratio	Model χ^2	$\Delta \chi^2$
Model 1	Intercept	3.78	295.79***				
	BMI	.07	57.03***	1.07	1.05-1.09	52.30	
Model 2	Intercept	3.19	169.27***				
	BMI	.06	39.24***	1.06	1.04-1.07		
	European American	-.56	28.58***	.57	.56-.57		
	Hispanic American	-.12	.51	.89	.86-.87		
	Asian American	-.72	5.82**	.49	.48-.49	83.30	31.00
Model 3	Intercept	2.77	43.68***				
	BMI	.06	33.91***	1.06	1.04-1.08		
	European American	-.44	16.34***	.64	.51-.79		
	Hispanic American	-.09	.24	.91	.63-1.31		
	Asian American	-.54	3.16	.58	.32-1.06		
	Parent Ed.	-.05	1.15	.95	.87-1.04		
	Income	-.01	2.34	.99	.99-1.00		
	Behavioral Control	-.06	2.93	.95	.89-1.01		
	Warmth	-.06	.84	.94	.82-1.08		
	Father Absence	.16	6.18**	1.17	1.04-1.34	102.26	49.96
Model 4	Intercept	2.71	15.65***				
	BMI	.04	7.46**	1.04	1.04-1.05		
	European American	-.64	.52	.52	.36-.53		
	Hispanic American	.97	.39	2.63	2.18-4.17		
	Asian American	-.81	.14	.44	.19-.45		
	Parent Education	.12	5.42*	1.12	1.11-1.16		
	Income	.01	.77	1.01	.99-1.01		
	Behavioral Control	-.03	.27	.97	.97-.98		
	Mother Warmth	-.01	.00	.99	.95-1.00		
	Father Absence	.17	4.84*	1.18	1.16-1.22		
	BMI x Eur. Am.	.05	11.91***				
	P. Ed. x Eur. Am.	-.19	8.92**			125.85	73.55

Note. Comparisons are made to the African American group.
* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 6

Models Predicting Pubertal Development with Hispanic Americans as the Reference Group

Model	Parameter	*B*	Wald χ^2 Square	Odds Ratio	95% CI for Odds Ratio	Model χ^2	$\Delta \chi^2$
Model 1	Intercept	3.78	295.79***				
	BMI	.07	57.03***	1.07	1.05-1.09	52.30	
Model 2	Intercept	3.32	143.56***				
	BMI	.06	39.24***	1.06	1.04-1.08		
	European American	-.43	6.26*	.65	.46-.91		
	African American	.13	.51	1.14	.80-1.61		
	Asian American	-.59	3.26	.55	.29-1.05	83.30	31.00
Model 3	Intercept	2.86	44.93***				
	BMI	.06	33.91***	1.06	1.04-1.08		
	European American	-.36	4.08*	.70	.49-.98		
	African American	.09	.25	1.10	.77-1.57		
	Asian American	-.45	1.81	.64	.33-1.23		
	Parent Ed.	-.05	1.15	.95	.87-1.04		
	Income	-.01	2.34	.99	.99-1.00		
	Behavioral Control	-.06	2.93	.95	.89-1.01		
	Warmth	-.06	.83	.94	.82-1.08		
	Father Absence	.16	6.18**	1.17	1.04-1.34	102.26	49.96

Note. Comparisons are made to the Hispanic American group.
* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 7

Models Predicting Pubertal Development with Asian Americans as the Reference Group

Model	Parameter	*B*	Wald χ^2 Square	Odds Ratio	95% CI for Odds Ratio	Model χ^2	$\Delta \chi^2$
Model 1	Intercept	3.78	295.79***				
	BMI	.07	57.03***	1.07	1.05-1.09	52.30	
Model 2	Intercept	3.94	123.46***				
	BMI	.06	39.24***	1.06	1.04-1.08		
	European American	.16	.30	1.18	.69-2.10		
	African American	.72	5.82**	2.06	1.15-3.71		
	Hispanic American	.59	3.24	1.81	.95-3.46	83.30	31.00
Model 3	Intercept	3.31	46.80***				
	BMI	.06	33.91***	1.06	1.03-1.07		
	European American	.09	.10	1.10	.61-1.96		
	African American	.54	3.17	1.72	.95-3.21		
	Hispanic American	.45	1.81	1.57	.82-3.02		
	Parent Ed.	-.05	1.14	.95	.87-1.04		
	Income	-.01	2.34	.99	.99-1.00		
	Behavioral Control	-.06	2.93	.95	.89-1.01		
	Warmth	-.06	.84	.94	.82-1.08		
	Father Absence	.16	6.18	1.18	1.04-1.34	102.26	49.96

Note. Comparisons are made to the Asian American group.
* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 8

Composite Sexual Risk Score

Item	Category	Value	Percent of Sample		
			Wave I	Wave II	Wave III
Number of sexual partners	0	0	85%	87%	16%
	1	1	4%	5%	18%
	2-3	2	5%	5%	24%
	4+	3	6%	3%	42%
Frequency of condom use	All of the time	0	96%	95%	37%
	Most of the time	1	1%	2%	14%
	Less than half the time	2	1%	1%	7%
	None of the time	3	2%	2%	42%
Sexual intercourse under the influence of alcohol or drugs	Never	0	94%	94%	87%
	Once	1	4%	4%	8%
	Twice	2	1%	1%	3%
	Three or more times	3	1%	1%	2%

Table 9

The Number of Participants by Wave and Age

	Age												
	12	*13*	*14*	*15*	*16*	*17*	*18*	*19*	*20*	*21*	*22*	*23*	*Total N*
Wave I	202	756	890	1,047	1,116	--	--	--	--	--	--	--	4,011
Wave II	6	275	742	899	1,051	1,013	23	2	--	--	--	--	4,011
Wave III	--	--	--	--	--	--	70	621	840	1,001	1,069	410	4,011
Total *N*	208	1,031	1,632	1,946	2,167	1,013	93	623	840	1,001	837	781	12,033

Note. Dashes indicate not applicable.

Table 10

Correlations and Descriptive Statistics for Study Variables with European Americans Below the Diagonal and African Americans Above the Diagonal

Indicator	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Puberty	--	.04	.04	.01	.01	.01	.01	.01	.01	.02
2. W1 Risk	.08***	--	.29***	.22***	.25***	.16***	.09**	.34***	.25***	.20***
3. W2 Risk	.06**	.42***	--	.16***	.11***	.05	.06	.18***	.20***	.13***
4. W3 Risk	.01	.22***	.22***	--	.07*	.03	.22***	.15***	.12***	.35***
5. W1 STIs	.05*	.23***	.10***	.05*	--	.30***	.01	.23***	.13***	.07*
6. W2 STIs	.04*	.17***	.10***	.04	.07***	--	.01	.12***	.09**	.04
7. W3 STIs	.01	.10***	.06**	.15***	-.01	.01	--	.06	.01	.10***
8. W1 Pregnancy	.04*	.26***	.11***	.06**	.20***	.02	.01	--	.54***	.27***
9. W2 Pregnancy	.06**	.34***	.18***	.12***	.21***	.14***	.08***	.61***	--	.28***
10. W3 Pregnancy	.11**	.28***	.18***	.25***	.09***	.10***	.07***	.22***	.35***	--
M European Americans		.45	.43	3.79	.01	.03	.10	.02	.05	.42
SD		1.20	1.10	2.25	.15	.38	.42	.17	.25	.82
M African Americans		.53	.42	3.48	.05	.08	.27	.06	.11	.70
SD		1.10	1.00	2.05	.28	.46	.64	.27	.44	1.02

Note. * $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 11

Correlations and Descriptive Statistics for Study Variables with Hispanic Americans Below the Diagonal and Asian Americans Above the Diagonal

Indicator	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Puberty	--	.10	.09	.05	.04	.07	.17**	.12	.04	.05
2. W1 Risk	.02	--	.59***	.25***	.02	.37***	.01	.44***	.45***	.23***
3. W2 Risk	.00	.37***	--	.25***	.19**	.28***	.02	.26***	.49***	.35***
4. W3 Risk	.05	.20***	.20***	--	.03	.05	.15*	.09	.19**	.28***
5. W1 STIs	.08	.18**	.18**	.13*	--	.01	.01	.01	.21***	.15*
6. W2 STIs	.06	.17**	.35***	.12*	.27***	--	.03	.07	.04	.08
7. W3 STIs	.10	.03	.21***	.05	.02	.25***	--	.00	.07	.29***
8. W1 Pregnancy	.02	.32***	.40***	.18**	.41***	.23***	.04	--	.34***	.14*
9. W2 Pregnancy	.05	.12	.25***	.22***	.25***	.14*	.03	.57***	--	.47***
10. W3 Pregnancy	.05	.25***	.24***	.34***	.08	.23***	.12*	.34***	.39***	--
M Hispanic Americans		.46	.43	3.31	.03	.02	.13	.07	.09	.67
SD		1.18	1.13	2.11	.20	.19	.61	.28	.31	.92
M Asian Americans		.39	.34	2.93	.01	.02	.05	.09	.07	.35
SD		1.18	.97	2.23	.13	.21	.25	.44	.29	.74

Note. * $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 12

The Unconditional Models for European Americans by Outcome

Model	Means			Variances			Δ-2LL
	Intercept	Slope	Quadratic	Intercept	Slope	Quadratic	
Sexual Risk							
Model 1	.39(.02)***						
Model 2	.79(.04)***			1.12(.09)***			73.42***
Model 3	.90(.07)***	.51(.01)***		1.30(.25)***	.04(.00)***		49.70***
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	3.72
STIs							
Model 1	.27(.10)***						
Model 2	.51(.24)***			1.10(.51)***			130.58***
Model 3	.83(.44)***	.31(.06)***		1.79(1.10)***	.00(.00)		55.02***
Model 4	.97(1.29)***	.60(.50)***	-.23(.05)***	.66(.40)***	.31(.20)	.00(.00)	36.12***
Pregnancy							
Model 1	.79(.04)***						
Model 2	.97(.07)***			.47(.14)***			66.12***
Model 3	.80(.19)***	.47(.02)***		.99(.42)***	.00(.00)		112.08***
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	.96

Note. Model 1 estimates just the intercepts. Model 2 estimates the intercept mean and variance. Model 3 estimates the linear slope mean and variance. Model 4 estimates the quadratic mean and variance.

[a] The parameter was not included in the model because it did not improve model fit..

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 13

The Unconditional Models for African Americans by Outcome

	Means			Variances			Δ-2LL
Model	Intercept	Slope	Quadratic	Intercept	Slope	Quadratic	
Sexual Risk							
Model 1	.37(.03)***						
Model 2	.57(.06)***			.30(.11)***			223.48***
Model 3	.69(.11)***	.45(.01)***		.36(.31)***	.03(.00)***		173.26***
Model 4	.98(.41)***	1.63(.12)***	-.08(.00)***	1.39(2.77)***	.41(.29)***	.00(.00)	19.48***
STIs							
Model 1	.09(.09)***						
Model 2	.21(.19)***			.53(.36)***			88.38***
Model 3	.52(.37)***	.33(.05)***		.87(.80)***	.00(.00)		63.12***
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	4.22
Pregnancy							
Model 1	.28(.05)***						
Model 2	.10(.08)***			.82(.14)***			33.80***
Model 3	.90(.20)***	.37(.02)***		.87(.31)**	.00(.00)		60.20***
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	1.92

Note. Model 1 estimates just the intercepts. Model 2 estimates the intercept mean and variance. Model 3 estimates the linear slope mean and variance. Model 4 estimates the quadratic mean and variance.

[a] The parameter was not included in the model because it did not improve model fit..

* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 14

The Unconditional Models for Hispanic Americans by Outcome

	Means			Variances			Δ-2LL
Model	Intercept	Slope	Quadratic	Intercept	Slope	Quadratic	
Sexual Risk							
Model 1	.24(.06)***						
Model 2	.91(.13)***			1.98(.26)***			67.40***
Model 3	.81(.26)***	.51(.02)***		1.23(.83)***	.04(.00)***		40.86***
Model 4	--a	--[a]	--[a]	--[a]	--[a]	--[a]	.68
STIs							
Model 1	.43(.30)***						
Model 2	.59(.81)***			1.54(.99)***			36.08***
Model 3	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	5.46
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	
Pregnancy							
Model 1	.19(.08)***						
Model 2	.28(.13)***			.30(.21)***			7.36***
Model 3	.89(.24)***	.30(.04)***		.44(.30)	.00(.00)		14.74***
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	5.64

Note. Model 1 estimates just the intercepts. Model 2 estimates the intercept mean and variance. Model 3 estimates the linear slope mean and variance. Model 4 estimates the quadratic mean and variance.

[a] The parameter was not included in the model because it did not improve model fit..

* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 15

The Unconditional Models for Asian Americans by Outcome

	Means			Variances			Δ-2LL
Model	Intercept	Slope	Quadratic	Intercept	Slope	Quadratic	
Sexual Risk							
Model 1	.19(.07)***						
Model 2	.29(.16)***			1.34(.33)***			56.38***
Model 3	.50(.38)***	.41(.04)***		1.68(1.47)***	.07(.02)***		30.30***
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	1.74
STIs							
Model 1	.64(.38)***						
Model 2	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	1.30
Model 3	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	
Pregnancy							
Model 1	.10(.12)***						
Model 2	.67(.23)***			.30(.43)			6.64***
Model 3	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	.64
Model 4	--[a]	--[a]	--[a]	--[a]	--[a]	--[a]	

Note. Model 1 estimates just the intercepts. Model 2 estimates the intercept mean and variance. Model 3 estimates the linear slope mean and variance. Model 4 estimates the quadratic mean and variance.

[a] The parameter was not included in the model because it did not improve model fit..

* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 16

Models with Puberty Predicting Growth Trajectories by Ethnicity

Model	Intercept	Slope
European American		
Sexual Risk	.63(.12)***	-.07(.01)***
STIs	1.53(.80)**	--[a]
Pregnancy	.69(.20)***	--[a]
African American		
Sexual Risk	.53(.16)***	-.05(.19)***
STIs	.13(.04)	--[a]
Pregnancy	.22(.12)**	--[a]
Hispanic American		
Sexual Risk	.16(.38)	-.01(.05)
STIs	.15(.74)	--[a]
Pregnancy	.38(.35)	--[a]
Asian American		
Sexual Risk	.10(.55)	.02(.06)
STIs	--[a]	--[a]
Pregnancy	--[a]	--[a]

Note. * $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

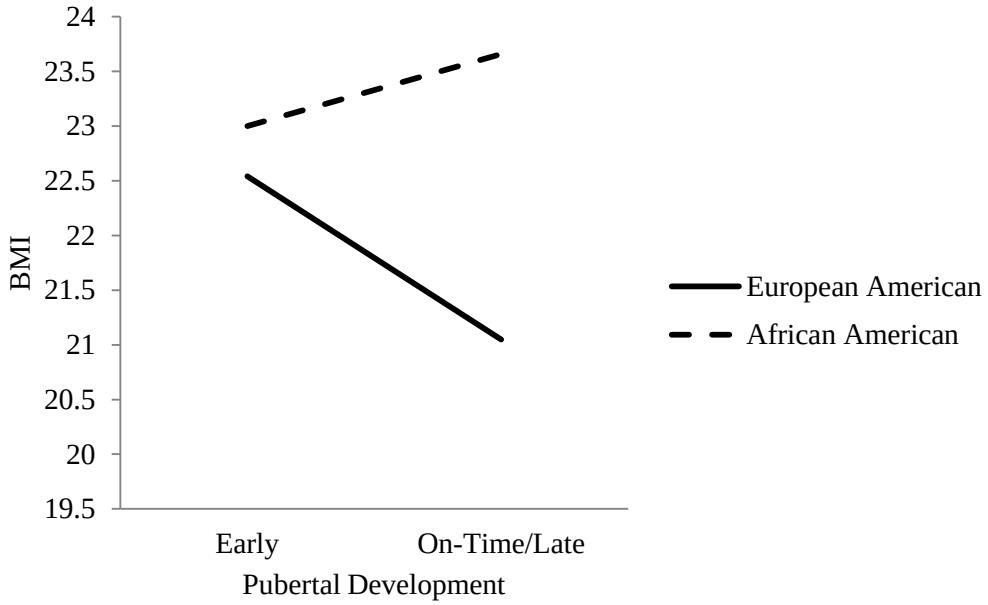

Figure 1. Interaction among BMI and ethnicity.

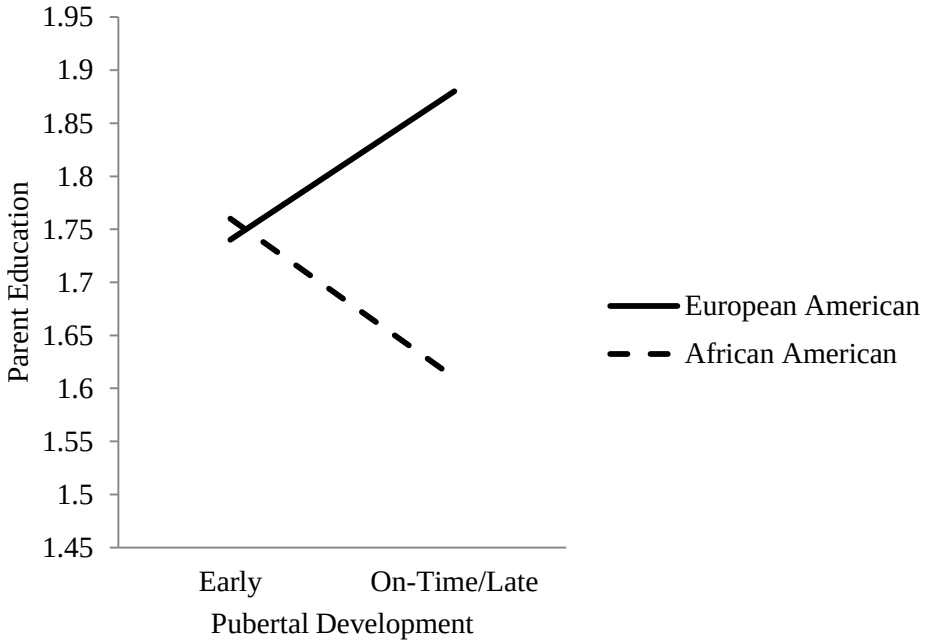

Figure 2. Interaction among parent education and ethnicity.

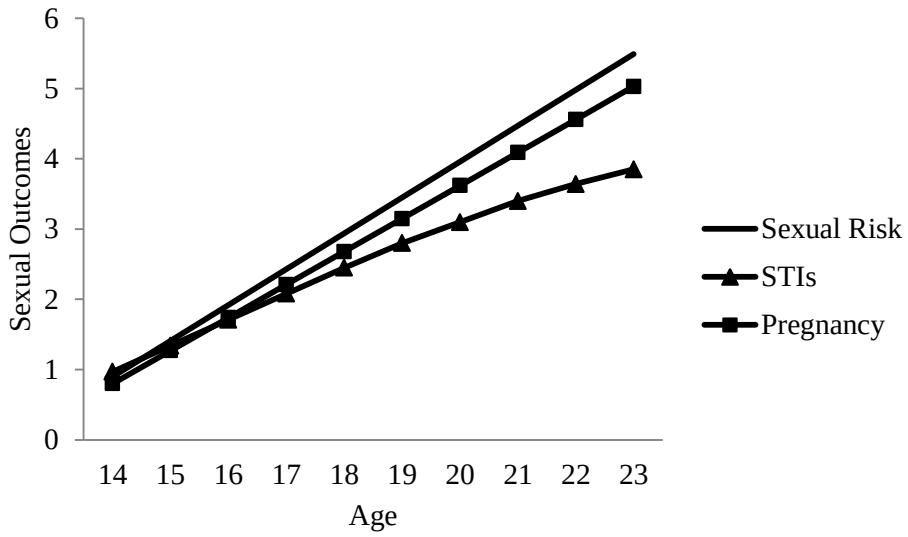

Figure 3. Growth trajectories for European Americans by outcome.

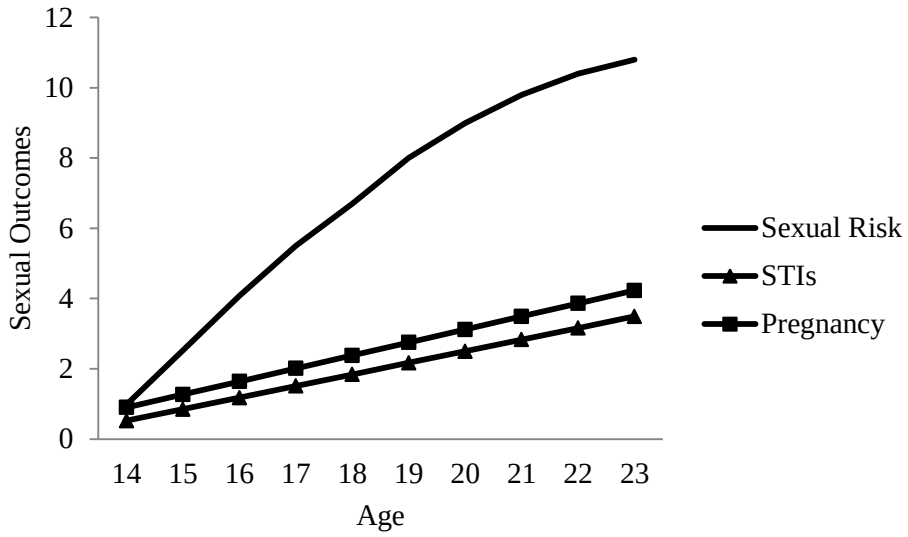

Figure 4. Growth trajectories for African Americans by outcome.

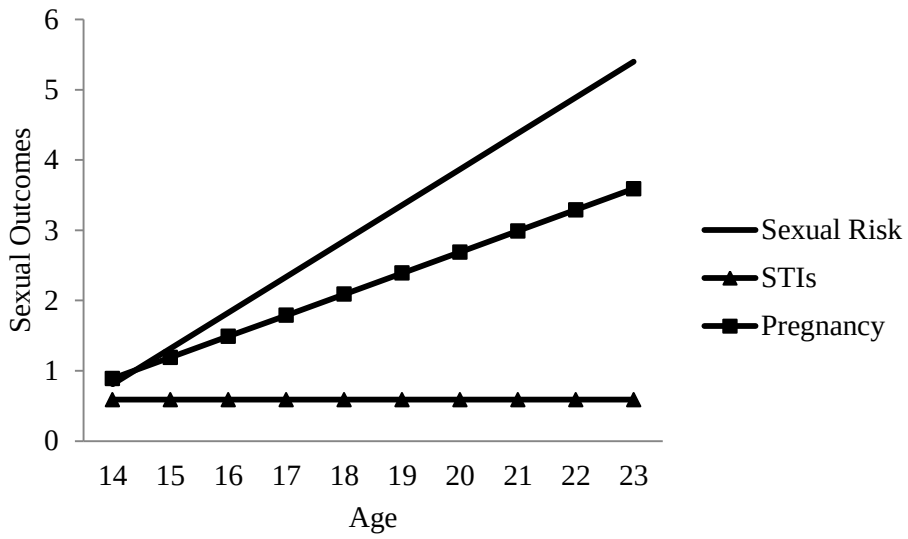

Figure 5. Growth trajectories for Hispanic Americans by outcome.

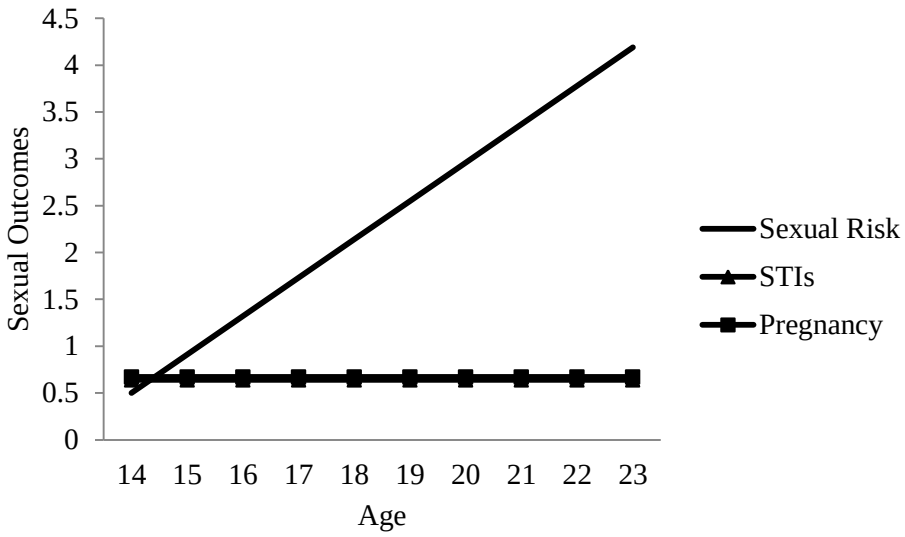

Figure 6. Growth trajectories for Asian Americans by outcome.

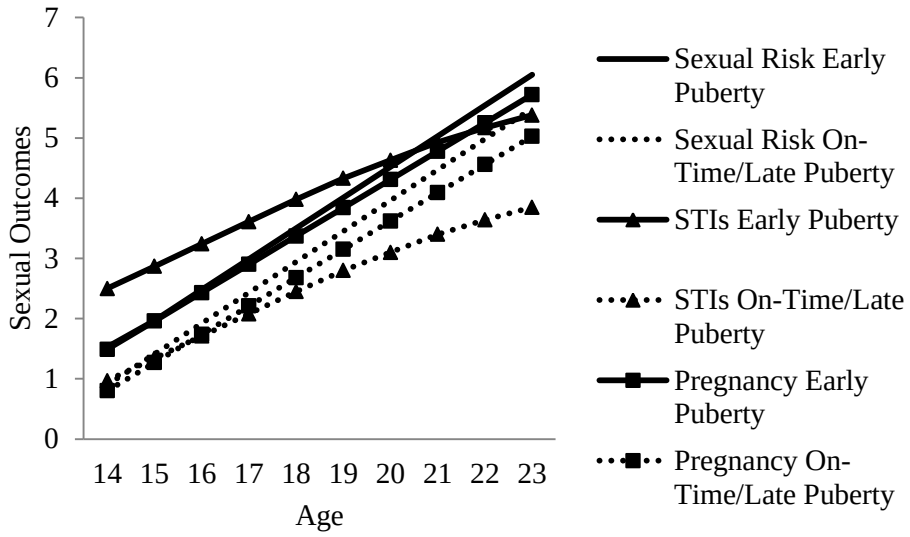

Figure 7. Growth trajectories for European Americans by puberty and outcome.

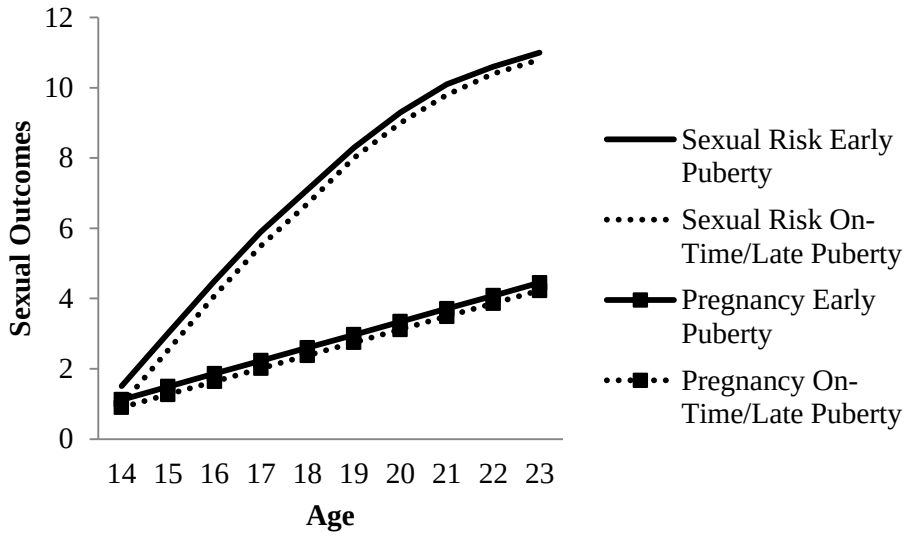

Figure 8. Growth trajectories for African Americans by puberty and outcome.