

The Family Antecedents and the Subsequent Outcomes of Early Puberty

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Abstract The purpose of this study was to examine both the family antecedents and the outcomes of early puberty, with a particular focus on factors related to family socioeconomic status (SES). The study employed a comprehensive measurement of pubertal development and longitudinal data from the Canadian National Longitudinal Survey of Children and Youth. The sample ($N = 8,440$; 49% girls) included four cohorts of children who were followed biennially for 10 years, starting from age 4–11 to 14–21 years. Data were drawn at different years of age from these cohorts of children. Girls whose fathers were unemployed were more likely to experience early puberty than those whose fathers were employed. For boys, those living with fathers who had not finished secondary school were more likely to experience early puberty. Early maturing girls tended to engage in smoking and drinking at

an earlier age compared with their peers. These findings provide support for psychosocial acceleration theory and suggest that different aspects of low family SES may act as a psychosocial stress for early pubertal maturation in boys versus girls, which may lead to engagement in drinking and smoking at a younger age, at least for girls.

Keywords Puberty · Pubertal timing · Risk behaviors · Family background · Longitudinal

Puberty is one of the most profound transitions in the life span (Susman and Rogol 2004), characterized by rapid and simultaneous changes in the physical, cognitive, social, and emotional development of an individual. The impact of puberty on adolescent psychosocial development has received much attention in the literature. In particular, early pubertal maturation has been identified as a potential risk factor for adolescent mental health and behavioral problems (Arim and Shapka 2008; Ge et al. 2006; Graber et al. 1997; but see Dorn et al. 2003; Graber et al. 2004). Early pubertal maturation also has been examined as an outcome in its own right, in an attempt to understand the genetic and contextual factors that are of influence when an individual will go through puberty (e.g., Belsky et al. 1991, 2007; Ellis 2004; Ge and Natsuaki 2009). Thus, both the antecedents and the consequences of early maturation have been studied, although methodological limitations and mixed findings have limited conclusions that can be made from these studies.

Influences on the Early Onset of Puberty

It is generally accepted that pubertal development is influenced by an interaction of genetic, hormonal,

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nutritional, and environmental factors; however, research has focused on particular domains of factors that are hypothesized to influence the onset of puberty. Early onset of puberty has been linked with several psychosocial factors within the family. For example, for girls, the onset of puberty is more likely to occur earlier when there are stressful conditions in the family (Tremblay and Frigon 2005), a parent–child conflict (Graber et al. 1995), maternal psychopathology, and the presence of a stepfather (Ellis and Garber 2000). For boys, although research is scarce, greater emotional distance from the mother (Kim and Smith 1999), parental marital conflict, and father absence (Kim and Smith 1998) have all predicted the early onset of puberty. Higher socioeconomic status (SES) also has been associated with the early onset of puberty among girls (Ayatollahi et al. 1999; Qamra et al. 1991) and boys (Adegoke 1989), although a recent study found that “economic anxiety” (defined as adolescents’ worry about themselves or their parents for not having a job in the future or not having enough money to pay for things) predicted early maturation among boys, but not in girls (Meschke et al. 2003). Ellis and Essex (2007) found a corollary relationship—that higher SES predicted later pubertal development in girls. Similarly, the findings from a recent Canadian longitudinal study indicated that adolescents from higher SES families were less likely to have entered puberty by age 13, but were more likely to go through puberty at a faster rate (Arim et al. 2007). These findings suggest that various psychosocial factors within the family are associated with the early onset of puberty although mixed findings exist in the literature.

Several theories or hypotheses have emerged in the literature to describe the timing of puberty (see Belsky et al. 1991, 2007; Ellis 2004; Ge and Natsuaki 2009 for reviews). For example, the evolutionary theory of socialization emphasized that contextual stressors, such as inadequate resources in the family, contribute to early pubertal maturation (Belsky et al. 1991). Ellis (2004) reviewed five theories that provide an explanation for the environmental influences on pubertal timing. One of these theories, psychosocial acceleration theory, provides the theoretical basis for this study. Psychosocial acceleration theory posits that relatively early pubertal timing occurs under conditions of uncertainty and moderately high psychosocial stress. It is important to note that this theory was developed based on research with a sole focus on the pubertal development of girls. Therefore, one of the aims of the current study was to provide further insight into this theory by focusing on family SES and structure on pubertal timing for *both* girls and boys. In addition, most research that has focused on family SES used a composite score derived from several indicators of SES (e.g., Tremblay and Frigon 2005). To our knowledge, our study is the first to isolate the specific

aspects for SES, including family income, both mothers’ and fathers’ education and employment status that may be responsible for the association between SES and pubertal timing.

Effects of Early Pubertal Timing

Pubertal timing refers to “whether an individual’s overall pubertal development occurs *earlier*, *later*, or at about the same time (*on-time*) as most adolescents” (Graber et al. 1996, p. 27). Pubertal timing has been considered particularly salient for adolescent mental health problems and problem behaviors (see Ge et al. 2006 for a review). Two major perspectives on the effects of pubertal timing have dominated the literature: the *deviance hypothesis*, which states that any deviation from the norm (i.e., early or late versus on-time pubertal timing) is a risk factor for adolescent problem behaviors (Neugarten 1979); and the *stage termination hypothesis*, which proposes that it is early pubertal timing (i.e., early termination of childhood) that poses a greater risk for developing problem behaviors (Simmons and Blyth 1987). Although both perspectives have been supported in research (e.g., Caspi and Moffitt 1991; Williams and Dunlop 1999), most studies have focused on the latter hypothesis by examining early pubertal timing, particularly, in girls.

Early pubertal timing has been associated with a spectrum of poor psychosocial functioning in adolescence. Early maturing girls tend to display both externalizing problems, such as substance abuse (Dick et al. 2000; Graber et al. 1997; Wichstrom 2001), disruptive behavior (Graber et al. 1997), aggression and delinquency (Celio et al. 2006), and early sexual intercourse (Flannery et al. 1993); and internalizing problems such as depressive feelings (Siegel et al. 1999), suicide attempts (Graber et al. 1997), lower body-image (Siegel et al. 1999), lower self-esteem (Graber et al. 1997; Williams and Currie 2000), and eating disorders (Graber et al. 1997). Early maturing girls are also more likely to report higher levels of school problems and poorer health (Aro and Taipale 1987; Graber et al. 1997).

Although there is less research on boys, it has been found that early maturing boys tend to be more satisfied with their physical appearance (Simmons and Blyth 1987; Tobin-Richards et al. 1983) but have higher rates of internalized distress (Ge et al. 2006) and physical illness (Graber et al. 1997), as well as externalizing problems such as expressing hostile feelings (Ge et al. 2006), substance abuse (Wichstrom 2001; Wiesner and Ittel 2002), and getting into trouble with the law (Duncan et al. 1985). These findings suggest that early pubertal timing in both girls and boys represents a significant risk factor for adolescent psychosocial development. In our study, we focused on a wide

range of developmental outcomes in academic achievement, psychosocial, and behavioral domains. Specifically, we examined whether early puberty was associated with low mathematics achievement and low self-esteem scores, as well as high smoking, drinking, and sexual activity behaviors as adolescents go through puberty. More importantly, though, we examined these outcomes from a developmental perspective; that is, we looked at longitudinal trajectories of these outcomes, as they changed over the course of adolescence (ages 10–16). This allowed us to better understand the longitudinal effects of early puberty and identify critical risk periods when the effects of early puberty are more pronounced over the course of adolescence.

Research Questions and Hypotheses

The purpose of this study was to examine the family antecedents and the subsequent developmental outcomes of early pubertal timing using longitudinal data. Two research questions were addressed in this study. First, what is the relationship between family SES factors and the early onset of puberty? Are these relationships the same for boys and girls? Second, what is the relationship between early puberty and adolescents' subsequent developmental outcomes over time? We first identified *within*-sex differences in pubertal timing to define early onset of puberty and examined separate models for boys and girls due to the well-known *between*-sex differences in the onset of puberty for boys and girls, namely, that girls enter puberty, on average, 18 months ahead of boys (Fechner 2003).

Regarding antecedents for the early onset of puberty, we focused on family SES variables to elucidate the associations between different aspects of family SES and the early onset of puberty. Consistent with recent studies in this area (Arim et al. 2007; Ellis and Essex 2007), we expected that aspects of low family SES, such as low family income and single parenting, would predict early pubertal timing. We also expected that family SES variables may be more predictive of girls' than boys' early puberty because of the view that girls' pubertal development is more sensitive to social experiences, such as family influences compared to boys' pubertal development (Belsky et al. 2007). However, as previous research showed that economic anxiety was associated with early maturation in boys, but not in girls (Meschke et al. 2003), we hypothesized that different aspects of low family SES may be associated with girls' and boys' early pubertal timing separately. For example, while single parenting may be associated with girl's early puberty, family income, fathers' education and/or employment status may be predictive of only boys' early puberty.

Regarding the relationships between early puberty and adolescent academic achievement, psychosocial, and

behavioral outcomes, given the mixed findings (e.g., Dorn et al. 2003; Ge et al. 2006), we considered the preponderance of evidence from longitudinal studies. For adolescents' mathematics scores, we did not expect a significant association between early puberty and low mathematics scores because longitudinal findings from an early study had indicated that early pubertal timing was not associated with mathematics achievement from 6th to 12th Grade (Dubas et al. 1991).

In the absence of longitudinal evidence, we turned to the findings from population-based studies with cross-sectional designs. For self-esteem, we found mixed findings in the literature. For example, although Williams and Currie (2000) found an association between early pubertal timing and low self-esteem in girls, Siegel et al. (1999) did not report a relationship between early pubertal timing and low self-esteem in girls nor in boys. Notably, the latter study (but not the former) controlled for the effects of household income in the analyses. Based on these results, we expected that early puberty would not be associated with low self-esteem after controlling for the effects of family SES variables. Finally, based on previous findings from longitudinal studies (Copeland et al. 2010; Crockett et al. 1996; Dick et al. 2000; Wichstrom 2001), we expected that adolescents' experience of early puberty would be associated with higher rates of smoking, drinking, and early sexual activity in both girls and boys.

Method

Source of Data

The National Longitudinal Survey of Children and Youth (NLSCY) is a longitudinal survey that is designed to collect a wide range of information about factors influencing a child's social, emotional, and behavioral development over time. The survey, conducted jointly by Statistics Canada and Human Resources and Skills Development Canada (HRSDC), began in 1994 and follow-up surveys were administered biennially. The target population of the survey in the first cycle was children who were newborn to 11 years old. The sample design was based on national household demographics but excluded households located in the Yukon, Nunavut, and Northwest Territories, First Nations (Aboriginal) reserves, and children living in institutional settings. There were 13,439 households in the first cycle resulting in 22,831 children who participated in the survey (Statistics Canada, Human Resources Development Canada [HRDC] 1997). The response rate of this longitudinal sample in the second cycle was 76% (Statistics Canada, HRDC 1997).

Data were collected in two different contexts—the household and the school. The household-collected data

included information about the person most knowledgeable (PMK) about the child, the spouse/partner of the PMK, and the child. The PMK was the biological mother for 90% of responding children (Statistics Canada, HRDC 1995). The school collection included a teacher's questionnaire, a principal's questionnaire as well as a Math Computation and Reading Comprehension test (added in the second cycle) administered to the child. The current study used data from the questionnaires that were administered to the PMK and the child in the household as well as the Mathematics Computation test administered to the child by a teacher at the school.

Sample

The sample ($N = 8,440$), balanced by gender (4,127 girls and 4,313 boys), included four cohorts of children born between 1983 and 1990, who were followed biennially for 10 years (Cycle 1 through Cycle 6). These children were between 4–11 years old in Cycle 1 (1994–1995) and became 14–21 years old in Cycle 6 (2004–2005). Data were drawn at different time points (i.e., at different years of age) from these cohorts of children. Approximately 50% of the sample was retained in Cycle 6 of the survey. Previous research has shown that children who withdrew from the NLSCY at some point, on average, were more likely to live in families with lower income and report higher levels of problem behaviors compared to children who remained in the study (Arim et al. 2011), but the effect size of these differences were found to be trivial to small.

Measures

Early Pubertal Timing (Ages 10–15)

Three items derived from the boys' and girls' versions of the Pubertal Development Scale (PDS; Petersen et al. 1988) were asked of participants between the ages of 10 and 15 years to assess their pubertal maturation: for boys, body hair, facial hair growth, and voice change; for girls, body hair, breast development, and menarche. A sample item is "Would you say that your body hair has": (1) *not yet started growing*, (2) *barely started*, (3) *is definitely underway*, and (4) *seems complete*. All items were measured on a 4-point scale except the item on menstruation that has a yes/no response. The use of the PDS is likely to be less valid than a pediatrician's assessment of pubertal maturation; however, this method is feasible for a large scale study and preferred by adolescents (Bond et al. 2006).

To enhance the validity and reliability of the PDS, in line with other researchers (e.g., Eaves et al. 2004), we utilized longitudinal data and Item Response Theory (IRT; see Tramonte and Willms 2009 for details). Previous research

showed the use of longitudinal data and IRT modeling can improve the meaningful assessment of pubertal timing (Eaves et al. 2004; Huang et al. 2009). The procedure for classifying adolescents as early maturing was completed in two steps. First, the criteria for reaching puberty were established based on the age-prevalence curves for each response for each of the three items for boys and girls using IRT, which assigns scores to observations based on the probability that an adolescent report full development of puberty items at various ages. Girls were considered to have reached puberty if they had begun to menstruate, or had both breast development and body hair definitely underway. Boys were considered to have reached puberty if both body hair and voice change were definitely underway, or if facial hair had barely started growing and either body hair or voice change were definitely underway. Next, the median ages of puberty (age at which 50% of girls and boys reached puberty) were calculated. Early puberty, separately for boys and girls, was defined with a cut-point score that indicated a probability of reaching puberty for 25% of the sample. For girls the median age was 11.99 years, and the cut-point for early puberty was 11.22 years. For boys, the median age was 12.61 years, and the cut-point for early puberty was 11.36 years. There were 1,473 girls and 405 boys who were classified as early maturing adolescents using this approach.

Family Antecedents (Ages 4–11)

Six variables describing family SES and structure were examined as family antecedents of early pubertal timing and were also used as covariates when examining the relationship between early puberty and subsequent developmental outcomes: (1) *Low Family Income*. Families with annual incomes less than \$30,000 were considered to have a low family income, (2–3) *Mothers' and Fathers' Low Level of Education*. Parents who had not completed secondary school were considered to have a low level of education, (4–5) *Mothers' and Fathers' Employment Status*. Parents were classified as being employed or unemployed, and (6) *Single Parent*. Families with one parent living at home were compared with families with two parents living at home, including families with one or more step-parents. All family variables were drawn from Cycle 1. If a family variable had a missing value in Cycle 1, then the information was drawn from Cycle 2 to prevent loss of data.

Adolescent Developmental Outcomes

We focused on three different domains of development: academic achievement, psychosocial, and behavioral with five outcome variables assessed at each data collection time point. All outcomes variables were dichotomized due to skewed response patterns. It should also be noted that the

use of dichotomized data simplified both the interpretation and the presentation of the findings.

Mathematics Computation Skills (Ages 10–15)

The math computation test was a shortened version of the Mathematics Computation Test of the standardized Canadian Achievement Tests, Second Edition (CAT/2). CAT/2 is a series of tests designed to measure achievement in basic academic skills. Adolescents were considered to have a low mathematics scores if they were about 1.5 grade levels behind their same-age peers.

Self-esteem (Ages 10–16)

Eight items taken from the General-Self Scale and the Physical Appearance Scale of the Marsh Self Description Questionnaire (Marsh 1988) were used to assess the adolescent’s overall self-esteem and perceptions of physical appearance. Each item was rated on a 5-point scale ranging from (1) *false* to (5) *true*. These items were scaled using IRT techniques, and were transformed into a dichotomous variable with a 50% cut-off point to denote low self-esteem.

Smoking (Ages 10–16)

Participants were asked, “Have you ever tried cigarette smoking, even just a few puffs?” If they answered affirmatively, they were asked, “If you do smoke, how often do you smoke cigarettes?” Adolescents were considered to be smoking if they smoked “at least once or twice a month but not every day” through to “every day”.

Drinking (ages 10–16)

Participants were asked, “Have you ever drunk alcohol?” If they answered affirmatively, they were asked, “How often?” Adolescents were considered to be drinking if they drank alcohol “about once or twice a month” through to “every day”.

Sexual Activity (Ages 12–16)

Participants were asked whether they had ever had sexual inter-course? Their response was used to create a simple yes/no marker.

Data Analysis

All analyses were conducted using the Hierarchical Linear and Nonlinear Modeling (HLM) program (Raudenbush et al. 2005). Longitudinal sample weights were applied to

generate unbiased population estimates in all analyses. The first question examined family antecedents of early puberty. Adolescents’ pubertal status was assessed at each cycle. Using the criteria described above, a separate marker was constructed based on whether an adolescent reached puberty at an age below the early onset of puberty cut-off ages. The logistic analysis then regressed the early puberty marker on six variables describing family SES and structure.

The second question focused on the effects of early puberty on subsequent developmental outcomes over time. In these analyses, the marker of early puberty was used as a covariate, and we examined its effects on adolescents’ developmental trajectories. The analyses for this study entailed a multilevel logistic regression analysis for each outcome measure, separately for girls and boys. The first level of the unadjusted model described individuals’ growth trajectories:

$$Pr(Y_{it} = 1|x) = 1/(1 + e^{-\eta_{it}}) \quad \text{where} \quad (1)$$

$$\eta_{it} = \pi_{0i} + \pi_{1i}a_{it} + \pi_{2i}a_{it}^2 \quad \text{Intra - individual model}$$

where Y_{it} is the i th adolescent’s developmental outcome on occasion t , a_{it} is the adolescent’s age on occasion t and a_{it}^2 is the square of the adolescent’s age, which is included to capture any bends in the trajectory. The three regression coefficients, π_{0i} , π_{1i} , and π_{2i} , describe the growth trajectories for the adolescents in the sample.

At the second level, the between-child level, we have an indicator of whether the adolescent experienced early puberty, as well as the six markers of family antecedents. We first regressed the three parameters describing adolescents’ growth trajectories on the marker of early puberty

$$\pi_{0i} = \beta_{00i} + \beta_{01}\text{Early Puberty}_i + u_{0i} \quad \text{Model for initial status} \quad (2)$$

$$\pi_{1i} = \beta_{10i} + \beta_{11}\text{Early Puberty}_i + u_{1i} \quad \text{Model for linear growth} \quad (3)$$

$$\pi_{2i} = \beta_{20i} + \beta_{21}\text{Early Puberty}_i + u_{2i} \quad \text{Model for acceleration} \quad (4)$$

The models described by Eqs. 2, 3, and 4 are then extended to include the set of control variables pertaining to family SES and structure. The coefficients of these models are difficult to interpret not only because they stem from logistic regressions, but also because the effects of early puberty vary with age. Therefore, we chose to provide only the odds-ratios (OR) of the models and present the results graphically using the models that are unadjusted for family SES and structure. We should note that the age variable was centered on 15, which represented the most interesting timing with respect to differences in the outcome variables between adolescents who experienced early puberty and those who did not.

Results

Research Question 1 What is the relationship between family SES factors and early onset of puberty? Are these relationships the same for boys and girls?

Table 1 shows the OR associated with the six measures of family antecedents. The only factor that was a statistically significant predictor of early puberty for girls was father's employment status: the odds of girls experiencing puberty amongst those whose fathers were unemployed was 1.44 times that of girls whose fathers were employed. For boys, low father's education was a risk factor for early puberty, with an OR of 1.63. None of the other family antecedents was a statistically significant predictor of early puberty.

Research Question 2 What is the relationship between early puberty and adolescents' subsequent developmental outcomes over time?

The second set of analyses examined adolescents' developmental outcomes associated with early puberty over time. It should be noted that in these analyses early puberty was treated as a time-invariant (fixed) characteristic of the adolescent, even though the categorization as early versus not-early was based on the longitudinal records for each participant. Therefore, the longitudinal trajectories indicate the prevalence rates of outcome variables for adolescents who did and did not experience early maturation from age 10 to 15, even though at age 10 their status of early puberty was not yet determined.

Of the five developmental outcomes that we examined without controlling for the effects of family SES and structure, only two (smoking and drinking) were found to be statistically significantly associated with the probability of experiencing early puberty, and only for girls. The probability of having low mathematics scores, low self-esteem and early sexual activity was not found to be associated with the probability of experiencing early

puberty although the trajectories suggested a pattern where early puberty was associated with lower prevalence of low mathematics scores, higher prevalence of low self-esteem, and early sexual activity (see Table 2). As can be seen in Table 3, the probability of experiencing early puberty in girls was associated with the probability of smoking at the intercept, at age 15, and non-linear growth in the rate of change, but not the linear rate of change. Figure 1 (left panel) shows that girls who experienced early puberty were more likely to smoke than those who did not experience early puberty. The statistically significant differences for the intercept emerged after age 13.5. Our approach to detecting significant differences was based on testing the models with the age variable 'centered' at different ages. Specifically, we observed statistically significant results when we centered age at 13.5, 14, and 15 but not at age 13 and prior.

As can be seen in Fig. 2(left panel), the pattern was similar for drinking, with a higher prevalence of drinking among girls who experienced early puberty. The results from the statistical models indicated that the probability of experiencing early puberty in girls was associated with the probability of drinking only at the intercept, at age 15. Early puberty was not associated with the linear rate of change of the probability of drinking and the non-linear growth in the rate of change (see Table 3). As can be seen in the right panel of Figs. 1 and 2, the trajectories suggested a similar pattern for boys; however the observed differences were not statistically significant for boys.

The observed relationships did not change substantially after controlling for family SES and structure. The findings were statistically significant for only two outcomes (smoking and drinking), and only for girls. As can be seen in Table 3, for girls, before controlling for the effects of family SES and structure, the OR for smoking and drinking were 1.34 and 1.33, respectively at age 15, and these were virtually unchanged when family SES and structure were added to the model.

Table 1 Odds-ratios indicating the relationship between early puberty and family antecedent variables

	Girls (n=4127)	Boys (n=4313)
Low family income	1.00	0.85
Low mother's education	0.90	0.90
Low father's education	1.01	1.63*
Mother is unemployed	0.95	0.80
Father is unemployed	1.44*	0.88
Single-parent family	0.97	0.97
Nagelkerke R ²	.002	.007

* $p < .05$

Discussion

Puberty has often been considered as an important factor that influences adolescent development and behavior (Graber et al. 2010). Numerous studies have linked early pubertal timing with poor developmental outcomes during adolescence, although mixed findings exist (see Susman and Rogol 2004 for a review). Relatively fewer studies focused on the factors that influence early pubertal timing. Despite a wealth of studies in the field, much uncertainty remains over the role of early pubertal timing in adolescent outcomes over time and significant gaps exist in our

Table 2 Odds-ratios associated with early puberty, with and without adjustment for family SES and structure

Variables	Low mathematics				Low self-esteem				Sexual activity			
	Boys (n=3,922)		Girls (n=3,873)		Boys (n=4,183)		Girls (n=4,015)		Boys (n=3,700)		Girls (n=3,564)	
	Unadj.	Adj.	Unadj.	Adj.	Unadj.	Adj.	Unadj.	Adj.	Unadj.	Adj.	Unadj.	Adj.
<i>Fixed effects</i>												
Initial status at age 15												
Intercept	.28***	.27***	.27***	.27***	.08***	.08***	.15***	.15***	.12***	.10***	.11***	.10
Early puberty	.97	.94	.95	.91	1.02	1.02	1.10	1.12	1.13	1.12	1.34	1.36
Low family income		2.49**		1.33		1.01		1.31		1.42		1.73
Low mother's education		1.53*		1.75**		.88		1.60*		1.52*		1.56
Low father's education		1.56*		1.61**		1.64*		1.20		1.64**		1.62
Mother is unemployed		1.35		.81		1.58		1.45		1.09		.80
Father is unemployed		.66		1.76		.59		.97		.79		.83
Single-parent family		1.20		1.27		1.17		1.41		1.92*		1.82
Linear slope (Age)												
Intercept	1.40***	1.42***	1.32***	1.35***	.98	.98***	1.00	1.01***	2.22***	2.07***	2.48***	2.49***
Early puberty	1.03	.98	1.17	1.17	.85	.84	1.00	1.00	1.29	1.53	1.02	1.01
Low family income		.97		.80		.89		1.04		1.09		.75
Low mother's education		1.07		1.32		1.05		.95		1.24*		.97
Low father's education		1.06		.86		1.05		1.02		1.16		1.02
Mother is unemployed		1.19		1.05		.95		1.04		.96		1.13
Father is unemployed		1.05		.77		.93		.90		.79		1.13
Single-parent family		.87		1.08		.97		1.03		1.13		1.57
Curvilinear slope (Age ²)												
Intercept	1.01	1.01	1.01	1.01	.97**	.97**	.93***	.93***	.83***	.86***	.82***	.82
Early puberty	1.00	.99	1.03	1.03	.99		.99	.99	.96	.90	.98	.98
Low family income		.98		.96		.97		.92*		.96		1.06
Low mother's education		1.01		1.04		1.00		.94**		.95		1.04
Low father's education		1.02		.96		.97		1.02		.95		.97
Mother is unemployed		1.03		1.02		1.01		1.00		1.01		.98
Father is unemployed		1.02		.93		.99		1.07		1.15		.98
Single-parent family		.98		1.02		1.03		1.05		.98		.90
<i>Random effects</i>												
Initial status at age 15	1.12	1.08	1.01	.96	1.47	1.46	1.29	1.29	1.11	1.06	1.82	1.77
Linear slope	.00	.00	.00	.00	.01	.01	.01	.01	.06	.07	.01	.01
Reliability—initial status	.14	.13	.13	.12	.17	.16	.22	.22	.12	.10	.17	.16
Reliability—slope	.00	.00	.00	.00	.01	.01	.01	.01	.03	.03	.01	.01
Tau ^a (correlation)	.32	.49	.18	.11	.47	.39	.21	.18	.09	.15	.80	.79

Unadj. = Model unadjusted for family SES and structure, Adj. = Model adjusted for family SES and structure

* $p < .05$; ** $p < .01$; *** $p < .001$

^a Correlation between initial status and linear slope

Table 3 Odds-ratios associated with early puberty, with and without adjustment for family SES and structure

Variables	Smoking				Drinking			
	Boys (n=4,177)		Girls (n=4,012)		Boys (n=4,095)		Girls (n=3,935)	
	Unadj.	Adj.	Unadj.	Adj.	Unadj.	Adj.	Unadj.	Adj.
<i>Fixed effects</i>								
Initial status at age 15								
Intercept	.09***	.08***	.13***	.12***	.62***	.61***	.64***	.62***
Early puberty	1.47	1.52	1.34*	1.35*	1.48	1.45	1.33**	1.34**
Low family income		1.34		1.34		.93		1.12
Low mother's education		1.26		1.80**		.74		1.47*
Low father's education		1.91**		1.74**		1.38*		1.18
Mother is unemployed		1.18		1.19		.80		.51*
Father is unemployed		.94		1.14		.68		.82
Single-parent family		2.15*		2.38**		.99		1.35
Linear slope (Age)								
Intercept	1.77***	1.87***	1.66***	1.71***	1.93***	1.95***	2.04***	2.07***
Early puberty	.99	.99	1.10	1.10	.94	.92	1.03	1.03
Low family income		1.33*		.87		1.06		1.04
Low mother's education		.93		1.04		.98		.92
Low father's education		1.05		.93		1.11*		.90
Mother is unemployed		.93*		1.07		.98		.97
Father is unemployed		.63		.99		.82		1.04
Single-parent family		.81*		1.00		.94		.89
Curvilinear slope (Age ²)								
Intercept	.93***	.92***	.92***	.92***	.96***	.96***	.93***	.93***
Early puberty	1.00	1.00	.95*	.95*	1.00	.99	.98	.98
Low family income		.92*		1.07		.96		.98
Low mother's education		1.08**		1.00		1.02		.99
Low father's education		.96*		.97		.98		1.00
Mother is unemployed		1.00		.97		.98		1.02
Father is unemployed		1.05		.95		1.01		.99
Single-parent family		1.03		.98		1.03		1.00
<i>Random effects</i>								
Initial status at age 15	2.02	1.97	2.07	2.01	1.05***	1.04***	1.09***	1.09***
Linear slope	.01	.01	.01	.01	.03	.03	.02	.02
Reliability—initial status	.17	.16	.23	.21	.26	.26	.27	.27
Reliability—slope	.01	.00	.00	.00	.05	.05	.05	.04
Tau ^a (correlation)	.36	.25	-.06	.05	.30	.29	-.17	-.11

Unadj. = model unadjusted for family SES and structure, Adj. = model adjusted for family SES and structure

^a Correlation between initial status and linear slope

* $p < .05$; ** $p < .01$; *** $p < .001$

understanding of the factors that influence early onset of puberty (Graber et al. 2010). In this study, we attempted to elucidate the role of early pubertal timing in adolescent developmental outcomes and rectify the above-mentioned shortcomings.

There were several strengths of this study. First, this study focused on the family antecedents of early puberty and examined the effects of early puberty on a set of academic achievement, psychosocial, and behavioral outcomes that covered a period from age 10 to 16, the period

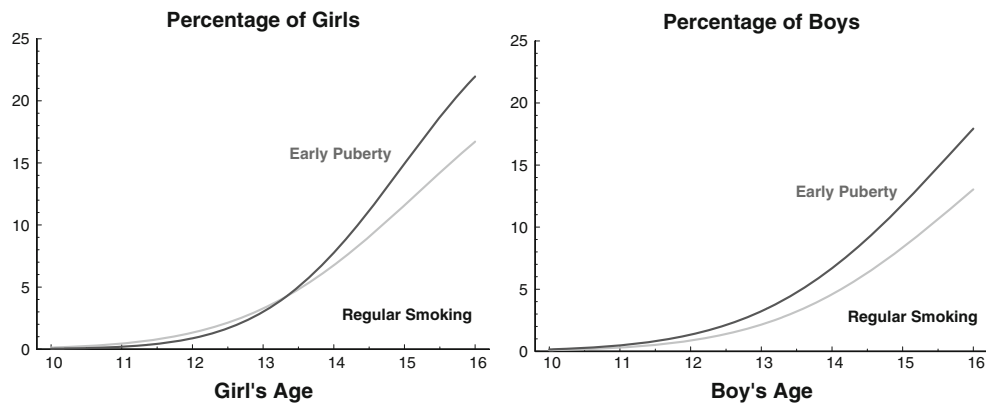


Fig. 1 Prevalence of youth who smoke regularly by age and pubertal timing. The *grey line* represents the average puberty curve at each age for adolescents who did not experience early puberty (the reference group)

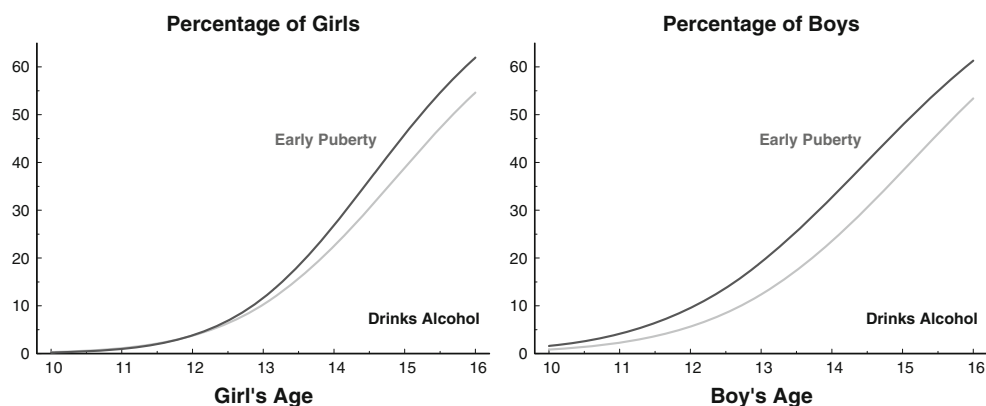


Fig. 2 Prevalence of youth who drink alcohol by age and pubertal timing. The *grey line* represents the average puberty curve at each age for adolescents who did not experience early puberty (the reference group)

when most children reach puberty. Moreover, this study used longitudinal data and Item Response Theory (IRT) modeling to combat some of the methodological concerns that plague this field (e.g., the reliance on cross-sectional data; Coleman and Coleman 2002; Dorn et al. 2003, 2006; Susman and Rogol 2004). Finally, this large-scale study involved both boys and girls, which helped narrow the gap in our understanding of boys' maturation processes.

Two key findings emerged from the study. First, pubertal timing showed a relatively weak relationship with family SES variables, and different aspects of family SES were associated with girls' and boys' early pubertal timing. Second, girls who experienced early puberty were more likely to engage in smoking and drinking at an earlier age compared with their girl peers, although these effects were also weak. The implications of these findings are discussed below in relationship to family antecedents and subsequent developmental outcomes of early puberty.

Regarding family antecedents, this study focused on family SES and structure variables, which can act as family context stressors. For girls, father's unemployment status

was a risk factor for early puberty, whereas for boys, it was low father's education (less than secondary school). It may be that having an unemployed father is related to economic anxiety that can be a risk factor for early puberty for girls and not just for boys, as previously thought (Meschke et al. 2003). For boys, it appears that low father's education can form the basis for economic anxiety in relation to early puberty. These findings are consistent with the tenets of the evolutionary theory of socialization (Belsky et al. 1991) suggesting that early rearing environment, especially family context, influences children's pubertal development such that a low SES family environment may be predictive of earlier pubertal maturation. In addition, our findings suggest that different aspects of low family SES are associated with girls' versus boys' early puberty. Furthermore, our findings also lend support to the psychosocial acceleration theory, which suggests that earlier pubertal timing may occur under conditions of moderately high environmental stress (Ellis 2004). In fact, it appears that both girls and boys are affected by stressors in the family context and enter puberty earlier than their peers. We should again note

that the observed effects were small in size but, nevertheless, different indicators of environmental stressors (father's unemployment for girls, father's low education for boys) were found to be predictive of early puberty in boys and girls.

Ellis (2004) distinguished among three types of environmental stressors: physical stressors (e.g., malnutrition), socioemotional stressors (e.g., lack of parental involvement), and father absence. It is possible that, in this sample, both father's unemployment and father's low education may be representative of prolonged familial distress (i.e., socioemotional stressor) rather than poverty (i.e., physical stressor). In line with this assertion, previous research indicated that girls who lived in dysfunctional families (e.g., parental alcohol abuse) had earlier menarche than girls who lived in families free of stressful events despite the lower SES (Hulanicka et al. 2001). Given these findings, it would be worthwhile to examine whether psychosocial aspects of family environment (e.g., parental involvement, parent-child relationships) can buffer the effects of family physical stressors (e.g., SES, family structure) on early puberty. It is possible that the former may play a moderating or a mediating role in the relationship between family physical stressors and pubertal timing.

Contrary to our expectations, neither family low income nor single parenting was significantly related to pubertal timing. While these findings may seem contradictory to previous research (e.g., Ellis and Essex 2007; Ellis et al. 1999), it should be noted that other researchers also have failed to find a relationship between family structure and pubertal development (Boothroyd and Perrett 2006). From a methodological perspective, it is possible that in the presence of multiple SES indicators, family income and single parenting lost their importance to predict significantly pubertal timing. Similarly, mothers' education and mothers' unemployment were not predictive of early onset of puberty in the presence of all other family SES indicators. Based on our findings, we conclude that aspects of low family SES, such as fathers' unemployment and fathers' low education can be a risk factor for early puberty, but the relationships are not strong. Future research should continue to disentangle the relationships between different aspects of family SES and the early onset of puberty separately for girls and boys.

Regarding subsequent developmental outcomes, the results suggest that early puberty may be a risk factor for smoking and drinking. Our findings indicated that girls who experienced early puberty were more likely to engage in smoking and drinking at an earlier age, which is consistent with previous research (Dick et al. 2000; Lynne-Landsman et al. 2010; Stattin and Magnusson 1990; Wilson et al. 1994). Similar patterns were also apparent for

boys' engaging in smoking and drinking in line with previous research (Andersson and Magnusson 1990; Dick et al. 2001; Lynne-Landsman, et al. 2010), but these effects were not statistically significant. The lack of statistically significant findings may be due to the relatively small sample size of boys who experienced early puberty ($n = 405$) in comparison with boys who did not experience early puberty ($n = 3,908$). It also should be noted that the age-prevalence curves for each of the three pubertal development items were less well-defined for boys than for girls. Thus, we recommend replication studies with a population sample of boys before we can draw solid conclusions about the effects of early puberty on smoking and drinking for boys.

Early puberty was not associated with low mathematics or low self-esteem scores. Although these results were anticipated, future replication studies are needed to elucidate these findings. It is possible that the use of cut-off scores for these outcomes variables limited our power to detect statistically significant differences (see Clarke and McKenzie 1994 for critics of using cut-off points). Alternatively, consistent with some previous research (e.g., Dubas et al. 1991; Siegel et al. 1999), it is possible that, in this sample, early puberty did not provide a meaningful differentiation for adolescents with low mathematics or low self-esteem from their peers. Finally, contrary to our expectations and previous findings (e.g., Flannery et al. 1993), early puberty was not associated with early sexual activity. A potential factor that may account for these contradictory findings may be the composition of our comparison group. Specifically, in this study, we compared adolescents who experienced early puberty to adolescents who did not experience early puberty, whereas other studies (Flannery et al. 1993; Lam et al. 2002) that suggested an association between early puberty and sexual activity made a comparison between early versus late maturing adolescents. In summary, although mixed findings exist in the literature, it seems that pubertal timing can be an important influence in adolescent psychosocial development.

Setting statistical significance aside, if one considers the magnitude of the observed effects of early puberty, the results suggest that for adolescent girls who do not go through puberty at an early age, their propensity to engage in smoking and drinking follows the same pattern; it is simply delayed by about 4–8 months. For example, the prevalence of smoking at age 15 among early maturing girls is approximately 15%, and this prevalence is not reached by not-early maturing girls until age 15 years 8 months. Similarly, for drinking, the prevalence of early puberty girls is 46% at age 15, while it reaches that level for not-early maturing girls at about age 15 years 5 months.

Technically, we could claim that early puberty is a risk factor, at least for an early initiation in smoking and drinking for girls at age 15. From a methodological perspective, a risk factor is a factor that precedes an outcome and is correlated with it (Kraemer et al. 1997). However, conceptually we tend to think of risk factors as having an ongoing effect, such as the effects of poverty on children's health outcomes. From this perspective, early puberty is not a risk factor; it simply starts the clock for adolescents' exposure to risk behaviors (i.e., it has an influence on the intercept at age 15, but on the rate of change). On the other hand, there is evidence that the earlier a risk behavior starts, the more entrenched it seems to become (Dekovic et al. 2004). Thus, earlier risk behaviors may be "riskier" due to greater immaturity and the possibility of forming friendships with older, risk-taking peers. Indeed, previous research has shown that affiliation with older peers (Stattin and Magnusson 1990) and deviant peers (Lynne et al. 2007) predicted adolescents' engagement in drinking and other delinquent behaviors. Consequently, from a practice perspective, these findings highlight the importance of early targeted prevention programs for adolescents who enter puberty early to prevent their engagement in risk behaviors.

Neither pubertal timing nor family SES or structure can be direct targets of prevention. However, existing research findings can help to inform the development of more comprehensive prevention and intervention programs (see Graber et al. 2010 for strategies for prevention). For example, our findings on family antecedents and early puberty highlight the need to promote better coping skills for stress among adolescents and increase their resiliency against economic and social challenges within the family. A family-based approach likely would be more powerful as research has consistently shown that quality of family processes are strongly associated with early pubertal timing (Belsky et al. 2007; Ellis et al. 1999; Graber et al. 1995).

Our findings should be interpreted in light of several limitations. First, although previous research has shown that adolescents can accurately identify their level of pubertal maturation (Duke et al. 1980), it still remains that self-reported pubertal development reflects self-perceptions, social comparisons, personal aspirations, and other attributes in addition to actual physical development (Silbereisen and Kracke 1993). Second, recent research has indicated that studies that use a cut-off score to categorize adolescents' pubertal development (as pre- or post-pubertal) rather than as a continuous process may be problematic when identifying early or late maturing adolescents (Shirtcliff et al. 2009). We should note that our procedure based on IRT modeling was somewhat different than using an arbitrary cut-off score. Specifically, our method used the

best discrimination slopes for each indicator of pubertal development using longitudinal data separately for boys and girls, and then assigned criteria to estimate a puberty curve representing the average age of puberty among boys and girls (Tramonte and Willms 2009). Furthermore, these average ages representing the probability of reaching puberty (11.99 for girls and 12.61 for boys) are in line with other research that has examined the sequence of pubertal development (see Fechner 2003; Susman et al. 2010). Hence, we are confident that our estimates of early puberty are meaningful.

In summary, the results from this longitudinal study suggest that family stressors can be associated with the early onset of puberty in both boys and girls. Early pubertal timing can be a significant risk factor in adolescents' early engagement in risk behaviors. Future research should continue to examine the antecedents and the consequences of early pubertal timing with longitudinal data using a broader focus on different social contexts and a wide range of adolescent developmental outcomes.

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