Pubertal maturation and the development of alcohol use and abuse

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Abstract

Objective: To examine the impact of various aspects of puberty on risk of using alcohol and developing alcohol use disorder (AUD).

Methods: Data come from the Great Smoky Mountains Study, a longitudinal study of a representative sample of 1420 youth aged 9–13 at recruitment. Participants were interviewed annually to age 16. A parent was also interviewed. Information was obtained about use of a range of drugs including alcohol, drug abuse and dependence, other psychiatric disorders, life events, and a wide range of family characteristics. Pubertal hormones were assayed annually from blood samples, and morphological development was assessed using a pictorial measure of Tanner stage.

Results: Controlling for age, Tanner stage predicted alcohol use and AUD in both boys (OR 1.58, 95% CI 1.18–2.22) and girls (OR 1.62, 95% CI 1.17–2.23). The effect of morphological development was strongest in those who matured early. Early pubertal maturation predicted alcohol use in both sexes, and AUD in girls. The highest level of excess risk for alcohol use was seen in early maturing youth with conduct disorder and deviant peers. Lax supervision predicted alcohol use in early maturing girls, while poverty and family problems were predictive in early maturing boys.

Conclusions: Among the many biological, morphological, and social markers of increasing maturation, the visible signs of maturity are important triggers of alcohol use and AUD, especially when they occur early and in young people with conduct problems, deviant peers, problem families and inadequate parental supervision.

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1. Introduction

The transition from childhood to adolescence is a time of physiological changes that have important psychological and social consequences. The prevalence rates of many psychiatric disorders change across childhood and adolescence, some increasing over time and others decreasing (Costello et al., 2003). Among those that increase are alcohol use, and alcohol abuse and dependence (alcohol use disorder: AUD). This paper addresses questions about the relationship among puberty, alcohol use, and AUD. Does puberty cause any of the changes in alcohol use and AUD, or is it merely coincidental? If it plays a causal or mediational role, which of the many aspects of puberty are involved? Since individual development occurs in a complex biopsychosocial environment (Bronfenbrenner and Ceci, 1994), which aspects of this environment are protective against an increased risk of alcohol use and AUD across puberty, and which increase that risk?

The term puberty encompasses changes in multiple indices of adolescent development: several gonadal and steroidal hormones; height, weight, body fat, body hair, breasts and genitalia; powers of abstract thinking; family and peer expectations and behavior. It can also occur at the same time as major social changes such as moving to high school (Simmons and Blyth, 1992). We may be concerned with either linear effects: i.e., increased risk as development proceeds, or with non-linear effects of various kinds. For example, the impact of estrogen and testosterone on increased risk for depression in girls appears to operate only when average levels exceed a certain threshold (Angold et al., 1999, 2003). Another non-linear effect that has been widely studied is the impact of asynchrony between one’s own development and that of one’s peer group (Tschann et al., 1994). Of course, both the physiological and social impact of puberty may vary by gender, as has been found...
in the case of both depression (Angold et al., 1998) and conduct disorder (Maughan et al., 2000). Thus, the relationship between puberty and risk for alcohol use and AUD may vary widely depending on gender and which measures of puberty are used.

An aspect of the development of substance abuse that needs careful consideration is its relationship with the other kinds of rule-breaking behavior encompassed in the DSM-IV diagnosis of conduct disorder (CD). CD too has been linked to pubertal changes, especially in girls (Ge et al., 1996; Graber et al., 1997, 2004; Magnusson et al., 1985; Moffitt et al., 1992). CD is, of course, strongly associated with the onset and progress of substance use and abuse (Lynskey and Fergusson, 1995; Majumder et al., 1998; Myers et al., 1995; Riggs et al., 1999; True et al., 1999; Whitmore et al., 1997). One clinical study of substance abusing girls and a control group showed that there was no direct link between early menarche and AUD, but an indirect one via the greater propensity of early maturing girls to hang out with conduct disordered boys (Mezzich et al., 1999). A question for this paper is whether any observed links between pubertal development and alcohol use or AUD are direct, or are mediated by links with CD.

In the same way, the relationship between puberty and alcohol problems needs to take into account the fact that use and abuse of other drugs is highly comorbid with alcohol use and AUD (Kandel, 1975). This paper uses data from a longitudinal study of child and adolescent development to address the relationship between some aspects of puberty and two types of substance use: use of alcohol, and the transition from alcohol use to abuse and/or dependence (AUD). Alcohol was chosen because it is often the “gateway” substance; the first to be tried, and the one that often leads on to experimentation with other substances (Federman et al., 1997; Kandel, 1975). Also, alcohol use can begin around the time of puberty (Kandel, 1975), whereas other types of drug use (e.g., cannabis) often begins after puberty (Federman et al., 1997).

We use data from the Great Smoky Mountains Study (GSMS), a longitudinal population study of children and adolescents, to test the following hypotheses:

1. Among the various manifestations of puberty, alcohol use will be directly influenced by morphological development (measured by Tanner staging) more than by hormonal development or psychological response to puberty. The reason for this hypothesis is that for a large majority of children the major hormonal changes that drive morphological change have already occurred several years before the onset of alcohol use. The immediate effects of early hormonal development will likely have dissipated by the time most children begin to be at serious risk for alcohol use or AUD, while morphological development will be ongoing.

2. We predict that early morphological maturation will be associated with early onset of alcohol use and AUD in girls through the mechanism of increased risk of conduct disorder. Some (Caspi et al., 1993; Stattin et al., 1989) have argued that alcohol use and AUD are a greater risk for early maturing girls only if they associate with conduct disordered boys, or older boys for whom drinking is normative. However, it is worth noting that the Carolina Longitudinal Study (Cairns and Cairns, 1994) did not find any effect of early puberty on behavioral deviance in either girls or boys.

3. Early morphological maturation will be associated with early onset of alcohol use and AUD in girls through the mechanism of increased risk of conduct disorder. Some (Caspi et al., 1993; Stattin et al., 1989) have argued that alcohol use and AUD are a greater risk for early maturing girls only if they associate with conduct disordered boys, or older boys for whom drinking is normative. However, it is worth noting that the Carolina Longitudinal Study (Cairns and Cairns, 1994) did not find any effect of early puberty on behavioral deviance in either girls or boys.

4. Aspects of the family environment, notably supervision and parent–child relations, will interact with pubertal maturation, so that early maturation is a risk factor for poorly reared children, and protective factor for well-reared children.

5. Previous studies have advanced a special case of the family risk story: they have proposed that girls who have suffered early maltreatment are likely to reach menarche earlier than other girls, which leads in turn to association with deviant peers, conduct problems, and alcohol use (Belsky et al., 1991; Moffitt et al., 1992). We test the hypothesis that early maltreatment leads to early maturation and thence to alcohol use and AUD, in both boys and girls.

2. Methods

2.1. Sampling frame

A representative sample of 4500, 9, 11, and 13-year olds, recruited through the Student Information Management System (SIMS) of the public school systems of 11 counties in western North Carolina, was selected using a household equal probability design. As close as possible to the child’s birthday, a screening questionnaire was administered to a parent (usually the mother), by telephone or in person. This consisted of 55 questions from the Child Behavior Checklist (Achenbach, 1991) about the child’s behavior problems, together with extra questions about substance use, and some basic demographic and service use questions. All children scoring above a predetermined cutoff score of 20 (designed to include about 25% of the population) on the behavioral questions, plus a 1-in-10 random sample of those scoring below the cutoff, were recruited for the longitudinal study. In addition, all age-appropriate American Indian children living in the area, most of them on a federal reservation, were recruited irrespective of their screen scores. Eligible children and one of their parents were interviewed annually until the subjects were 16.

Informed consent was obtained from both children and parents. Because sexual development is a sensitive topic, we showed the Tanner stage pictorial assessment instrument (see below) to parents before giving it to the children, and specifically asked permission to use it. At each wave, between 6% and 13% of parents refused to have the measure administered to their children (average across waves 8%). Surprisingly, the refusal rate was twice as high for boys as it was for girls (11.5% versus 5.9%; p = 3 × 10−9). Thus girls were significantly over-represented for analyses involving Tanner staging. However, refusal to complete the Tanner stage assessment was not significantly related to age or pubertal status, so it seems unlikely that the missing data caused biased results.

2.2. Measures

2.2.1. Psychiatric and substance use symptoms and disorders. Children and parents were interviewed using the Child and Adolescent Psychiatric Assessment (CAPA) (Angold et al., 1995), which generates diagnoses based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association, 1994). The CAPA is an interviewer-based interview (Angold and Fisher, 1999). The goal of interviews using this format is to combine the advantages of clinical interviews with those of highly structured epidemiologic interview methods (Wing et al., 1974). While using a highly structured
format of questions and probes, the interviewer-based approach trains the interviewer to ensure that the parent or child being interviewed understands the construct under review, and provides enough detail and examples for a clear rating of the clinical severity of each symptom to be made. A detailed glossary provides the operational rules for identifying a clinically significant level of severity for each symptom. Two-week test–retest reliability (N = 78) ranged from $\kappa = 0.6$ for DSM-IV conduct disorder to $\kappa = 1.0$ for substance abuse (Angold and Costello, 1995).

Parent and child were interviewed separately using different interviewers, who were trained to inter-rater reliability of $\kappa \geq 0.90$. A symptom was defined as present if reported by either or both respondents, or using the either/or rule common in clinical practice. The time frame of the CAPA for determining the presence of most psychiatric symptoms is the past 3 months. Diagnoses were based on DSM-IV criteria (American Psychiatric Association, 1994).

The interview also includes symptoms scales, measures of functional impairment, and of family structure, functioning, and child-rearing style, family burden, family psychiatric history, income, occupation, etc. (for details, see Costello et al., 1996). Computer algorithms which combined symptom information from parent and child were used to determine whether a symptom was present. When symptoms were found to be present during the interview, their dates of onset were also collected. If either parent or child reported a symptom as present in the past 3 months, it was counted toward the relevant scale score or diagnosis. This 3-month “primary period” was selected for current symptoms rather than, say, a 1-year or lifetime period, because shorter recall periods are associated with more accurate recall (see, e.g., Angold et al., 1996). Of course, onset dates might be earlier than the 3-month time frame of the interview. If a symptom was reported at multiple waves, the earliest reported date was used as age at onset.

Substance use was assessed in the CAPA using a screening interview that covered use of 18 types of substances (chewing tobacco or snuff, cigarettes, alcohol, cannabis, cocaine, crack cocaine, amphetamines, ice, inhalants, nitrite inhalants, heroin, other opioids, LSD, PCP, psilocybin, sedatives, steroids, and other). Tobacco use was counted if the child had smoked on average one cigarette a day, or used half a tin of chewing tobacco or snuff in a week, over the past 3 months. Alcohol use was counted if the child had had a drink of alcohol in the past 3 months without parental permission. All other substances were counted if they had ever been used in the past 3 months.

If there was substance use, subjects received a detailed interview covering age at first use, amount of use, source of the substance, intoxication, withdrawal, dealing, associated maladaptive functioning, etc., so that a diagnosis of substance abuse or dependence could be made. Because very few adolescents have yet suffered the physiological and social damage needed to meet DSM-IV criteria for dependence, we created a category for youth of Substance Use Disorder (SUD) for youth whose substance use is causing them to show significantly impaired functioning at home, at school, or with their peers. A similar category was created for alcohol use disorder (AUD).

### 2.2.2. Measures of development across puberty

The analyses take into account the following measures of maturation across the age range of 9–16: age, height, weight, development of secondary sex characteristics (morphologic status), age at menarche (girls), and self-perception of early, on-time, or late maturation. Levels of follicle stimulating hormone (FSH), luteinizing hormone (LH), testosterone (T), estradiol (E), and dihydroepiandrosterone sulphate (DHEAS) are available for the first three waves of the study.

### 2.2.3. Pubertal morphologic status

Ratings of pubertal morphologic status were based on the standard Tanner staging system (Tanner, 1962). Originally developed for clinical use, this involved a physical examination, which is impractical in non-clinical settings. Self-ratings performed with the aid of schematic drawings of secondary sexual characteristics (breasts and pubic hair in girls; genitalia and pubic hair in boys) have yielded good correlations with physical examination based on Tanner stages (Morris and Udry, 1980). Each child was provided with sex-appropriate schematic drawings and requested to rate her- or himself on each dimension. Unless otherwise noted, both self-ratings were averaged to yield a single individual score (ranging from I-pubertal, to V-adult level of development). Girls were also asked about their menstrual histories, and if post-menarcheal, questions about the date of onset of their menstrual periods.

### 2.2.4. Age at puberty

For data analyses, age at puberty was defined using the strategy developed for these data by Maughan et al. (2007). Self-reported Tanner stage was used for the youngest two cohorts, as the best approximation to morphological stage. Thus, the age reached at Tanner stage IV was used as a definition of ‘age at puberty’ for the youngest two cohorts. Given that the oldest cohort of children were first interviewed at age 13, which for many would be after reaching Tanner stage IV, reported age at menarche was used for the girls in the oldest cohort as an approximation for morphological development. The boys in the oldest cohort (N = 223) were excluded from analyses involving age at puberty because for those who had already reached Tanner stage IV at the entry to the study (44%), no adequate approximation of age at puberty is available in the data. The average age at reaching Tanner IV was 13.56 (S.D. = 1.55) for boys and 13.55 (S.D. = 1.58) for girls in Cohorts 1 and 2. For the girls in cohort 3, the average age at menarche was 12.64 (S.D. = 2.05). Thus, the estimated age at puberty was slightly, but not significantly, earlier for the oldest cohort of girls.

### 2.2.5. Early maturation

Because the social and psychological effects of early puberty have been argued to stem from others’ perceptions of youths as physically mature, we aimed to base assessments of pubertal timing on Tanner stage ratings so far as possible, using Tanner stage IV as a marker of mature physical appearance. In the sample excluding the oldest cohort, 9.7% of girls reached Tanner stage IV by age 11, a further 20.4% by age 12, and another 14.4% by age 13; 12.5% of boys reached Tanner stage IV by age 11, another 17.9% by age 12 and a further 15.0% by age 13. In these two cohorts, we classified the 30.1% of girls and 30.4% of boys who had reached Tanner stage IV by age 12 as early maturers. In the oldest cohort, girls were classified as early maturers if their reported age at menarche fell in the earliest 30.1% for the sample as a whole (11.96 years). Using this classification, early maturers reached Tanner stage IV at a mean age of 12.05 years (S.D. = 1.74) by contrast with 14.24 years (S.D. = 1.82) for on-time/late maturers. Mean age at menarche in the two groups was 11.07 years (S.D. = 1.45) and 12.39 years (S.D. = 2.06), respectively. The difference in age between the early maturing groups from cohorts 1 and 2 versus cohort 3 was not statistically significant. As discussed earlier, boys from the oldest cohort were omitted from the analyses, because it was not possible to estimate their age at puberty.

### 2.2.6. Hormone assays

Hormone samples were obtained at the beginning of the interview session, as follows: two finger-prick samples were collected at 20 min intervals, applied to specially prepared paper, immediately refrigerated upon drying, and express shipped (without refrigeration) to the laboratory within 2 weeks of collection. Samples were then stored at –23 °C until they were assayed. At each wave, some 20% of participants refused to give blood for hormone measurements (separate consent for the finger prick procedure used here was sought). Since the response rate at each wave averaged 80%, this resulted in completed assays on 2988 of a possible 4660 observations (64.2%).

Blood spot FSH and LH were measured using modifications of commercially available fluoroimmunoassays for assay of these hormones in serum or plasma (DELFLIA; Wallac Inc., Gaithersburg, MD). The blood spot testosterone (T) and estradiol (E2) assays are modifications of commercially available serum/plasma radioimmunoassay kits (Binax, South Portland, ME; Pantex, Santa Monica, CA; DSL, Webster, TX; and Pantex, respectively). Complete details of protocol, validation, performance, sample stability, and comparability to plasma or serum values for each blood spot assay are provided elsewhere (Worthman and Stallings, 1997). To minimize effects of pulsatility, hormone values for each observation were taken as the average of the two blood spot samples. Formulae for conversion of blood spot assay values to serum/plasma equivalents are given in Worthman and Stallings (1997). All values in the present report represent plasma/serum total T and E2 (i.e., bound plus unbound) equivalents and hence are comparable to the extant literature based on that medium.

### 2.2.7. Environmental risk factors

Adolescent substance use and abuse have been associated with a wide range of family and environmental factors (Breslau et al., 2003; Chassin et al., 1999; Guo et al., 2000; Hawkins et al., 1997, 1992; Heath et al., 2002b; Hill et al., 2000; Hoffman and Cerbone, 2002). In these analyses we test the hypothesis that puberty exacerbates their effects. For these
analyses we aggregated large numbers of risk factors included in the GSMS data set into three broad categories using item–response modeling and factor analysis (Costello et al., 1996). The group labeled “poverty and adversity” includes family income below the federal poverty line, parental lack of education, parental unemployment, single, step, and adopted family structure, and multiple moves of home and school. The group labeled “child-rearing style” includes harsh or inconsistent parenting, lax supervision, scape-goating, and overinvasive parenting. The group labeled “family problems” includes parental history of mental illness, crime, drug problems, and family violence. Other risk factors included in these analyses are child psychiatric disorders, life events, and association with deviant peers.

### 2.3. Statistical analysis

Simple bivariate analyses were used to show the relationship of each measure of puberty to alcohol use and AUD. We retained for further analyses only those aspects of puberty that showed a significant bivariate association, using a generous criterion of \( p < 0.05 \). Because the two sexes have different developmental trajectories, analyses were run separately by sex.

Next we included all the measures of pubertal maturation in a single model. We ran two models, one for the basic measures of development (including age in the model as a separate independent variable), and a second incorporating age directly into the measures of puberty by dividing subjects into early maturers and the rest. Again, we ran one set of models for alcohol use, and one for AUD, by sex. After generating the best fitting model for the effects of puberty, we next tested the hypothesis that puberty’s effects on alcohol use were mediated by puberty’s effects on conduct disorder. Finally, we examined what aspects of puberty that showed a significant bivariate association, using a generous criterion of \( p < 0.05 \). Because the two sexes have different developmental trajectories, analyses were run separately by sex.

### 3. Results

Fig. 1 shows the sampling design of the study and the numbers selected and recruited at each stage. Of those selected for recruitment into the main study 81% of American Indians and 80% of the rest of the population agreed to participate. Across the eight waves of the study used in these analyses between 75% and 94% completed the interviews each year, with an average of 83% (\( N = 6674 \) observations). The final sample consisted of 790 males and 630 females (weighted percentages 52% and 49%, respectively). In the unweighted sample 69.2% (\( N = 983 \)) were Anglo, 6.2% (\( N = 88 \)) were African American, and 24.6% (\( N = 349 \)) American Indian. When weighted back to population probability of selection the respective proportions were 89.5%, 6.9%, and 3.6%. Race/ethnicity was forced into all analyses but no significant differences were found.

### 3.1. Effects of pubertal measures on alcohol use and alcohol use disorders (AUD)

Table 3 shows the results of regressing alcohol use on each separate measure of puberty controlling for age, for each sex. Table 4 shows the same analyses for AUD. Testosterone was transformed into a log scale to stabilize unequal variation, and to improve the fit of the model.

Increased age of course predicted increased likelihood of alcohol use. After controlling for age, however, Tanner stage was significantly associated with alcohol use for boys (OR 1.58, \( p < 0.01 \)) and girls (OR 1.62, \( p < 0.01 \)). Increased levels of log testosterone (OR 1.63, \( p < 0.0001 \)) and LH (OR 1.19, \( p = 0.02 \)) were associated with risk of alcohol use in boys, controlling for age.

The picture of puberty-related predictors of AUD was different. Once again, age was a strong predictor, but in this case after controlling for age there was no longer an effect of Tanner stage for either sex. FSH was significantly associated with AUD for both boys (OR 1.17, \( p = 0.04 \)) and girls (OR 1.14, \( p = 0.03 \)).
Models including all the significant pubertal predictors and their interactions were run separately for girls and boys. For boys, the effect of log(testosterone) remained significant (OR 1.5, \( p < 0.01 \)), but the effect of LH on alcohol use became insignificant. The effects of both Tanner stage and the age-by-Tanner stage interaction were significant. The effect of Tanner stage varied by age (OR = 0.49, 0.63, 0.80, 1.02, 1.29, 1.65, 2.09, 2.66 for ages 9–16, respectively). Up to age 11, mature boys had rates of alcohol use that were as low as, or lower than, those of immature boys. From age 12 onward, mature boys had much higher rates of alcohol use than did immature boys (Fig. 2).

For girls, both age (OR 1.39, \( p < 0.01 \)) and Tanner stage (OR 1.62, \( p < 0.01 \)) independently predicted alcohol use. The interaction was not significant. Fig. 3 shows that while alcohol use increased with age, mature girls had higher risk of alcohol use than immature girls after age 12.

### Table 3

<table>
<thead>
<tr>
<th></th>
<th>Girls</th>
<th></th>
<th></th>
<th>Boys</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Effects</td>
<td>Age</td>
<td>Odds Ratio</td>
<td>95% CI</td>
<td>p-value</td>
<td>Effects</td>
</tr>
<tr>
<td>Tanner stage</td>
<td>1.62</td>
<td>(1.17–2.23, &lt;0.01)</td>
<td>1.39</td>
<td>(1.17–1.65, &lt;0.001)</td>
<td>1.58</td>
<td>(1.18–2.22, &lt;0.01)</td>
</tr>
<tr>
<td>Log testosterone</td>
<td>0.89</td>
<td>(0.64–1.23, 0.47)</td>
<td>1.92</td>
<td>(1.50–2.45, &lt;0.0001)</td>
<td>1.63</td>
<td>(1.29–2.11, &lt;0.0001)</td>
</tr>
<tr>
<td>Androstenedione</td>
<td>0.83</td>
<td>(0.56–1.22, 0.34)</td>
<td>1.95</td>
<td>(1.57–2.43, &lt;0.0001)</td>
<td>1.34</td>
<td>(0.90–1.98, 0.15)</td>
</tr>
<tr>
<td>Estradiol</td>
<td>1.00</td>
<td>(0.99–1.01, 0.31)</td>
<td>1.79</td>
<td>(1.41–2.26, &lt;0.0001)</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>FSH</td>
<td>0.93</td>
<td>(0.79–1.08, 0.33)</td>
<td>1.92</td>
<td>(1.57–2.35, &lt;0.0001)</td>
<td>1.17</td>
<td>(0.95–1.44, 0.14)</td>
</tr>
<tr>
<td>LH</td>
<td>1.00</td>
<td>(0.99–1.01, 0.65)</td>
<td>1.89</td>
<td>(1.51–2.35, &lt;0.0001)</td>
<td>1.19</td>
<td>(1.03–1.38, 0.02)</td>
</tr>
<tr>
<td>DHEAS</td>
<td>1.00</td>
<td>(0.99–1.01, 0.06)</td>
<td>1.70</td>
<td>(1.33–2.18, &lt;0.0001)</td>
<td>1.00</td>
<td>(0.99–1.01, 0.27)</td>
</tr>
</tbody>
</table>

Table 4
Estimated strength of association (odds ratio, 95% confidence interval, p-value) linking age, and measures of pubertal development adjusted for age, with alcohol use disorder in the past 3 months

<table>
<thead>
<tr>
<th></th>
<th>Girls</th>
<th>Boys</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Effects</td>
<td>Age</td>
</tr>
<tr>
<td>Tanner stage</td>
<td>1.60 (0.56–4.60, 0.38)</td>
<td>2.00 (1.34–2.99, &lt;0.001)</td>
</tr>
<tr>
<td>Log testosterone</td>
<td>2.95 (0.18–47.44, 0.44)</td>
<td>4.92 (1.94–12.48, &lt;0.001)</td>
</tr>
<tr>
<td>Androstenedione</td>
<td>1.17 (0.69–2.00, 0.55)</td>
<td>4.47 (2.08–9.62, &lt;0.0001)</td>
</tr>
<tr>
<td>Estradiol</td>
<td>1.00 (0.99–1.01, 0.13)</td>
<td>5.19 (2.16–12.45, &lt;0.001)</td>
</tr>
<tr>
<td>FSH</td>
<td>1.14 (1.01–1.29, 0.03)</td>
<td>5.98 (2.01–17.75, 0.001)</td>
</tr>
<tr>
<td>LH</td>
<td>1.00 (0.99–1.01, 0.85)</td>
<td>5.42 (2.42–12.14, &lt;0.0001)</td>
</tr>
<tr>
<td>DHEAS</td>
<td>1.00 (1.00–1.01, 0.01)</td>
<td>4.00 (1.35–11.88, 0.01)</td>
</tr>
</tbody>
</table>


3.2. Effects of early puberty on onset of alcohol use

We used Cox proportional hazard model to test the hypothesis that early puberty increased risk for alcohol use and AUD. The model was fit using the SAS PROC PHREG with COVSANDWICH option, which fits proportional hazards regression models with a robust covariance matrix. Table 5 shows the results. In boys, neither risk of alcohol use nor AUD was affected by early puberty. For girls, risk of alcohol use decreased with age at puberty for girls (HR 0.80, p < 0.01); that is, girls who were younger at puberty were at increased risk of alcohol use. Risk of AUD also decreased with age at puberty for girls, but the statistical significance was not attained (HR 0.81, p = 0.09).

These analyses demonstrate that while alcohol use and AUD increased with age, morphological stage also played a role; one which differed by sex. For boys of 12 or older, physical maturity was more important than age in predicting alcohol use. For girls, age and physical maturity both predicted use, but early maturing girls were at increased risk of use and AUD at every age. Analyses run using only the younger two cohorts of girls, and omitting the use of menarche as a marker of pubertal stage, showed similar results.

3.3. Impact of conduct disorder and other risk factors on early maturers

One of the themes of the literature on puberty and substance abuse is that early puberty increases girls’ exposure to alcohol and drugs through increased likelihood of conduct disorder (CD) and of association with deviant youth. The same model could be equally relevant to boys. We fit logistic regression models to assess the effect of CD and association with deviant peers, separately and together, on alcohol use and AUD for early maturing boys and girls compared with those on a normal pubertal trajectory.

The early maturing girls were more likely to have CD (OR 2.07, p = 0.015) but this was not true for boys (OR 0.78, p = 0.304). However, both early maturing boys (OR 3.46, p < 0.01) and girls (OR 2.28, p < 0.01) were more likely than others to report deviant peers. In early maturing youth with CD, the risk of deviant peers increased considerably for both boys (OR 2.98, p < 0.01) and girls (OR 34.92, p < 0.01). Thus, the expected associations between conduct disorder and deviant
peers, and early maturation and deviant peers, were seen for both sexes. However, the link between early maturation and conduct disorder was only seen in girls.

Table 6 shows how these risk factors affected alcohol use and AUD. Looking first at alcohol use, there was a main effect of early maturity for both boys (OR 2.46, \( p < 0.0001 \)) and girls (OR 3.05, \( p < 0.0001 \)). The interaction of early maturation and conduct disorder, and early maturation and deviant peers, greatly increased the risk of alcohol use in both sexes. The joint effect of CD and deviant peers was even more powerful in increasing risk in early maturing youth. There were no early maturing girls with CD who did not have deviant peers; 80% of those early maturing girls with CD and deviant peers were using alcohol. Three-quarters of early maturing boys with CD had deviant peers, and 83% of these were using alcohol. The increased risk of alcohol use associated with all three risk factors was significant.

In the case of AUD, numbers were smaller and for one analysis the model failed to converge. There was no effect of early maturity on AUD in boys, alone or in interaction with other risk factors. In girls, the main effect and the interaction with all three risk factors were significant.

The fourth hypothesis predicted an increase in likelihood of alcohol use and AUD associated with aspects of the family environment. Table 7 shows the results of testing for an interaction between early maturation and family problems. We concentrate on prediction of alcohol use, as cell sizes were too small for reliable tests of effects on AUD.

Poverty and adversity did not increase the risk of alcohol use in early maturing boys or girls. Interestingly, poverty marginally decreased the risk of alcohol use in early maturing boys. However, child rearing style (specifically, lack of adequate supervision) increased the risk of alcohol use for both. Family problems dramatically increased the risk of alcohol use in early maturing boys (OR 22.02, \( p = 0.002 \)), but the effects on girls, while in the same direction, were not significant (OR 1.40, \( p = 0.14 \)).

The fifth hypothesis concerned the impact of early maltreatment (neglect, physical abuse or sexual abuse) on age at maturation as defined above, and thence on alcohol use. By age 13, 14.3% of the sample had been maltreated according to their own or parental report for physical abuse (4.3%) or sexual abuse (10.0%) or interviewer report for neglect (2.7%). Girls who had been maltreated reached maturity on average 8 months earlier than non-maltreated girls (11.6 years, S.D. 1.0 versus 12.1 years, S.D. 1.3, OR 0.75, 95% CI 0.58–0.93, \( p = 0.011 \)). However, there was no effect of early maltreatment on alcohol use, alone or in interaction with age at maturation. Maltreated boys were no more likely to mature early than other boys, and there was no association between maltreatment and alcohol use.

### 4. Discussion

The aim of this paper is to deepen our understanding of the role played by pubertal development in the onset of alcohol use and the transition from use to abuse. We have extended the existing literature on the subject in three ways. First, the study includes multiple measures of pubertal development, encompassing hormonal changes as well as the morphological, psychological and sociological changes that they entrain. Second, the study includes both boys and girls. Third, it is able
to look at the interaction of puberty with a range of individual and environmental risk factors that have been associated with adolescent alcohol use and abuse in previous studies.

Tanner stage predicted alcohol use and AUD in both boys and girls, when age was included in the models. The effect of morphological development was strongest in those who matured early. Early pubertal maturation predicted alcohol use in both sexes, and AUD in girls. The highest level of excess risk for alcohol use was seen in early maturing youth with conduct disorder and deviant peers. Lax supervision predicted alcohol use in early maturing girls, while poverty and family problems were predictive in early maturing boys.

4.1. Different measures of puberty

Other analyses from this data set have shown that different aspects of the pubertal process are associated with different types of psychopathology, and that these associations can differ by sex. Depression in girls was associated with increasing levels of the steroid hormones estrogen and testosterone, controlling for all the other measures of hormonal and social-psychological development (Angold et al., 1999). In the case of conduct disorder in girls, testosterone levels proved to be a better predictor than Tanner stage or age. In boys, the link between rising testosterone levels and conduct problems was rather different: testosterone was related to non-aggressive CD symptoms in boys with deviant peers, and to leadership in boys with non-deviant peers (Rowe et al., 2004).

In the case of alcohol use and AUD, we found little independent effect of the hormonal precursors of the morphological and social-psychological changes of puberty. This is not surprising given that many of these changes preceded by months or years the average age at which adolescents begin to use alcohol (Angold et al., 1999). However, visible changes such as body hair and breast development were important precursors of alcohol use in both boys and girls. Even at age 15, boys were unlikely to drink unless they were also physically mature. Alcohol use increased with age in all girls, but in immature girls its course lagged a couple of years behind that of mature girls. It should be borne in mind, however, that the numbers in each group change over time; relatively few participants were in the mature groups at age 11, or the immature groups at age 15.

The most potent aspect of pubertal development in these analyses was early maturation in girls. This observation goes back to the 1980s (Magnusson et al., 1985) and has been repeatedly confirmed (Andersson and Magnusson, 1990; Prokopcakova, 1998; Tarter, 2002; Tschann et al., 1994; Wilson et al., 1994), sometimes as a main effect and sometimes through the high level of comorbidity between substance use onset and other deviant behaviors in the early teens (Graber et al., 2004).

4.2. Puberty, CD, and family risk

Earlier analyses of the GSMS data showed that, among the psychiatric disorders, CD was by far the strongest predictor of substance use and substance abuse or dependence (SUD) (Sung et al., 2004), with particularly strong effects up to age 14. This paper extends those findings by presenting evidence that it was the early matures with CD who were at highest risk of alcohol use, especially if, as others have noted, they had deviant peers (Andrews et al., 2002; Dick et al., 2001; Tarter, 2002).

There is a considerable literature on puberty and conduct problems in girls, some of which includes alcohol use and abuse as an indicator of deviance (Graber et al., 1997; Kaltiala-Heino et al., 2003; Nottelmann et al., 1990; Ge et al., 2002; Moffitt and Caspi, 2001; Dick et al., 2000; Graber et al., 1997; Laitinen-Krispijn et al., 1999; Magnusson, 1988). Others have argued that specific environmental risk factors, such as early maltreatment, may be linked to early puberty in girls and thence to adolescent deviance (Belsky et al., 1991; Moffitt et al., 1992). In the GSMS data early maltreatment predicted early maturation in girls, but not boys, and did not predict either conduct disorder or alcohol use.

When we tested the inter-relationship of early maturation with different aspects of familial risk in predicting alcohol use, only one family factor increased the risk for early maturing girls: a child-rearing style that included lax supervision. Risk also increased for boys, however, if they came from families with a history of drugs, crime, or psychiatric problems. This is consistent with a large literature (e.g., Heath et al., 2002a; Hoffman and Cerbone, 2002; Xian et al., 2000). Interestingly, in a multivariable model family poverty was a not significant predictor of alcohol use in early maturing girls, and in boys it was marginally protective. In studies where poverty appears to increase risk (Boardman et al., 2001; Marcenko et al., 2000), it may serve as a marker for other family problems.

Limitations of the study include its geographical specificity: participants live in a rural area of the southeastern USA, and findings may not apply elsewhere. Also, findings are limited to the white and American Indian youth who made up 69% and 25%, respectively, of the sample; there were too few African American or other race/ethnic groups for comparisons to be made. However, the findings in the two main groups did not differ. The measurements of alcohol use and of pubertal status are based on self and parent reports.

This pattern of risk found in this longitudinal study supports the argument that risk for alcohol use increases when young people show signs of maturation that can be read by others, especially by peers who are already involved in rule-breaking behavior. It reinforces the thesis that alcohol use is driven by social norms within adolescent groups (Jessor et al., 1995). The implications for prevention thus point to social interventions. For example, as Simmons pointed out 20 years ago, the structure of American schools, with a transition to high school that exposes young teenagers to an environment ruled by the norms of 16 and 17-year olds, might have been designed expressly to put intense strain on the early-developers, especially girls (Simmons and Blyth, 1987, 1992). School systems that concentrate the most deviant children in special schools or classes are also likely to increase the risk of alcohol use and abuse, as well as other forms of deviance (Dishion et al., 1999). In an earlier paper (Sung et al., 2004) we noted that the impact of behavioral deviance on substance use was marked in the early years of adolescence, but that by age 16 its effects had disappeared as substance use
became more “normal” (see also Hipwell et al., 2005, etc.). The findings from this study of pubertal effects are consistent with the earlier analyses, with the added implication that the earlier the onset of puberty the longer the period of risk.

References


