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Pubertal maturation and sex steroids are related to alcohol use in adolescents

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ABSTRACT

Adolescents often show risk-taking behavior, including experimentation with alcohol. Previous studies have shown that advanced pubertal maturation is related to increased alcohol use in adolescents, even when controlling for age. Little is known about the underlying mechanisms of this relation between pubertal maturation and alcohol use. The goal of the present study was twofold. In Experiment 1, we investigated whether advanced pubertal maturation is associated with higher levels of alcohol use, when controlling for age. To this end, questionnaires on pubertal development and alcohol use were administered to a large sample of 797 Dutch adolescents (405 boys) aged 11–16 years. In Experiment 2, we explored whether sex steroids contribute to this relation between pubertal maturation and alcohol use by examining the association between salivary sex steroid levels and alcohol use in 168 adolescents (86 boys). It was found that, when controlling for age, advanced pubertal maturation is related to increased alcohol use in adolescent boys and girls. Controlling for age, higher testosterone and estradiol levels correlated with the onset of alcohol use in boys. In addition, higher estradiol levels were associated with a larger quantity of alcohol use in boys. Correlations between sex steroids and alcohol use were not significant in girls. These findings show that advanced pubertal maturation is related to advanced alcohol use, and that higher sex steroid levels could be one of the underlying mechanisms of this relation in boys. Sex steroids might promote alcohol use by stimulating brain regions implicated in reward processing.

Introduction

Alcohol is the most commonly used intoxicant among adolescents (Hibell et al., 2009; Johnston et al., 2009). For example, 43% of European adolescents have consumed alcohol at least once prior to the age of 13 (Hibell et al., 2012). Although experimentation with alcohol might be considered normative in European adolescents, early onset of use (i.e., <18 years) and high levels of alcohol use during adolescence are related to the development of alcohol abuse and dependence in adulthood (McCambridge et al., 2011). Investigating (biological) mechanisms underlying the onset and quantity of alcohol use in healthy adolescents might increase our understanding of the underpinnings of alcohol abuse and dependence in adulthood.

A growing body of literature exists on the relation between pubertal maturation and alcohol use in adolescents (Mendle et al., 2007). We will first explain the specific hormonal events of puberty and their specific effects on physical maturation. Puberty is a biologically determined period, typically ranging from 10 to 16 years, during which the (re)activation of the hypothalamic–pituitary–gonadal (HPG) axis causes children to change from a non-reproductive state into a reproductive state (Nussey and Whitehead, 2001). At the onset of puberty the hypothalamus starts to produce gonadotropin-releasing hormone (GnRH) in a pulsatile manner, which stimulates the pituitary to release luteinizing hormone (LH) and follicle stimulating hormone (FSH) into the bloodstream. FSH and LH stimulate the gonads to produce sex steroids, which contribute to the maturation of the reproductive organs and secondary sexual characteristics (Cameron, 2004; Dahl, 2004). In girls, the ovaries produce estrogens, such as estradiol (E2), which stimulate breast development and the onset of menarche. Growth of pubic and axillary hair in girls is initiated by testosterone (T) that is secreted by the adrenal glands (Grumbach and Styne, 2003). In boys, T is secreted by the testes, and it stimulates voice changes and the growth of body and facial hair. Within both sexes, part of T is converted into E2 via the enzyme aromatase. Other hormones also play an important role in the onset of puberty-related physical changes. The pubertal growth spurt is mediated by growth hormone (GH) and insulin growth factor-1 (IGF-1), in addition to estrogens, in boys and girls (Clark and Rogol, 1996). Skin changes (e.g., acne) and the growth of body hair are influenced by the hormone dehydroepiandrosterone (DHEA) and its sulfated product (DHEAS) (Dahl, 2004). Although exact estimates of puberty onset vary between countries, it is generally assumed that females show pubertal signs at an earlier age (i.e., 1–2 years earlier) than males (Herman-Giddens et al., 2012).

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The focus of the current paper is on the contribution of puberty-related development to alcohol use. Pubertal development can be approximated by having teenagers report to what extent they are experiencing physical changes associated with going through puberty, such as body growth and the development of body hair. Based on the self-reported extent of these physical changes, it has been found that teenagers who report increased progression of physical changes (i.e., advanced pubertal maturation), show higher levels of alcohol use, even when controlling for age (Biehl et al., 2007; Bratberg et al., 2005; Westling et al., 2008; Wiesner and Ittel, 2002).

The first goal of the present study was to further test the hypothesis that advanced pubertal maturation is related to alcohol use in a large healthy adolescent sample. Moreover, very little is currently known about the underlying mechanisms of this relation. Several explanations have been proposed, including the gap between physical and psychosocial maturation in more mature adolescents, the affiliation with older and deviant peers, less parental monitoring, and sleep problems and evening-type tendencies in more mature adolescents (Engels, 2009; Ge and Natsuaki, 2009; Pieters et al., 2010). Another interesting hypothesis postulates that hormonal changes at puberty influence sensation-seeking behavior in adolescents, including alcohol use (Forbes and Dahl, 2010). Sex steroid receptors are found throughout limbic brain areas and the prefrontal cortex (Simerly et al., 1990) and pubertal hormones affect decision-making and brain development in adolescents (Peper et al., 2009, 2010, 2011; Sisk and Zehr, 2005). Consequently, these hormones might contribute to the relationship between pubertal maturation and alcohol use, for example by acting on receptors located in brain areas important for mediating reward responsiveness, risk-taking and cognitive control. Therefore, the second aim of our study was to test whether sex steroid levels are positively related to alcohol use in adolescents.

Only a handful of studies have examined this hypothesis in adolescents. Higher T-levels were found to be related to increased alcohol use in adolescent boys (Costello et al., 2007; Eriksson et al., 2005), and higher E2-levels were found to be associated with increased alcohol use in girls (Martin et al., 1999). Importantly, E2 is also present in pubertal boys, since the enzyme aromatase converts T into E2 in boys and girls (Lenz et al., 2011). Interestingly, higher levels of aromatase have been linked to addictive behaviors, including alcohol use (Lenz et al., 2011; Purohit, 2000). Moreover, higher E2 levels are correlated with greater sensation-seeking tendencies in adolescent boys (Vermeersch et al., 2009). Thus, E2 might also influence alcohol consumption in adolescent boys. However, to our knowledge, the potential contribution of E2 to elevated alcohol use in adolescent boys has not yet been addressed. Further, the contribution of T and E2 to both the onset and quantity of alcohol use in adolescent boys and girls has not been investigated in a single study. Thus, it remains unknown whether the effects of sex steroids on alcohol use in adolescent boys are specific for T. Moreover, it is currently unknown whether T and E2 are related to both the onset and quantity of alcohol use, or whether each of these hormones affects both aspects of alcohol use differently. The present study could therefore advance our knowledge on the detailed biological mechanisms underlying enhanced alcohol use in adolescents.

To address our two research aims, in Experiment 1, the relation between pubertal maturation and alcohol use was examined in a large normative sample of 797 adolescents, by administering questionnaires on pubertal development and alcohol use. In Experiment 2, we tested whether sex steroids could contribute to the relation between pubertal maturation and alcohol use. To this end, sex steroid levels were determined from saliva and correlated with the onset and quantity of alcohol use in a sample of 168 healthy adolescents.

Table 1
Demographic characteristics of the Experiment 1 sample (mean/*SD*).

	Boys ($n=405$)	Girls ($n=392$)
Age	14.10 (1.03)	14.18 (1.00) *ns*
PDS score[a]	2.35 (0.65)	2.71 (0.60)
Lifetime alcohol use[b]	1.13 (2.24)	1.03 (2.19) *ns*
Recent alcohol use[c]	0.68 (1.66)	0.52 (1.28) *ns*

Note. Table values represent means, with standard deviations in parentheses, unless otherwise reported.

[a] Ranging from 1 (=*no development*) to 4 (=*complete development*).

[b] Ranging from 0 (=*no alcohol use*) to 10 (=*more than 90 glasses of alcohol consumed*).

[c] Ranging from 0 (=*no alcohol use*) to 9 (=*more than 50 glasses of alcohol consumed*).

Method

Experiment 1: pubertal maturation and alcohol use

Participants

A total of 871 participants, recruited from local high schools in the Leiden area, The Netherlands, were included in Experiment 1 (Table 1) The first 48 questions (sets A–D) of the Raven's Standard Progressive Matrices (SPM) test (Raven et al., 1998) were administered, with a 10-minute time limit, to screen for general intelligence. Participants scoring more than 2 *SD* below the mean of the current sample (calculated for boys and girls separately) were excluded from the analyses (4.3% of boys, and 4.6% of girls). Further, participants with incomplete assessments of age, IQ and pubertal development were excluded from data analyses (7.9% of boys, and 3.4% of girls). After the exclusion of these participants, 405 boys (age range = 11.88–16.00 years, $M=14.10$ years, $SD=1.03$) and 392 girls (age range = 11.90–15.99 years, $M=14.18$ years, $SD=1.00$) were included in our analyses. Parents and teachers of the children gave informed consent. All procedures were approved by the Internal Review Board at the Leiden University Institute of Psychology, and were in agreement with the Declaration of Helsinki (Seoul amendments, 2008).

Measures

Pubertal development. The Pubertal Development Scale (PDS) was used to quantify pubertal development (Petersen et al., 1988). Adolescents indicated their physical development on a 4-point scale (ranging from 1 = *no development or change* to 4 = *complete development*). Boys were asked to indicate their body growth, body hair (pubic and axillary hair), facial hair, skin changes and voice changes, whereas girls indicated their body growth, body hair, breast development, skin changes and whether they had reached menarche.

Alcohol use. Boys and girls reported (a) lifetime alcohol use and (b) recent alcohol use. *Lifetime alcohol use* was measured as the number of glasses of alcohol adolescents had consumed in their lifetime on an 11-point scale (0, 1–10, 11–20, 21–30, 31–40, 41–50, 51–60, 61–70, 71–80, 81–90, and >90). Bottles and cans of alcohol had to be counted as 1.5 glasses. *Recent alcohol use* was assessed by asking the participants to indicate the number of glasses of alcohol they had consumed over the past 30 days on a 10-point scale (0, 1–2, 3–4, 5–6, 7–10, 11–15, 16–20, 21–30, 31–50, and >50) (Scholte et al., 2001). Self-report measures of alcohol use have been found to show high test–retest reliability in adolescents (*r*'s up to .86) (Brener et al., 2002). In order to ensure confidentiality, the instructions specifically stated that the adolescents' answers for both measures of alcohol use would not be disclosed to their parents, teachers or classmates.

Procedure

Online versions of the PDS, alcohol use questionnaires, and Raven's SPM were used in the current study. All measures were administered in computer rooms at the schools of the adolescents. One class, with on average 30 students, was tested at a time. Participants could not view

each other's computer screens and a team of research assistants ensured that participants did not discuss their answers with each other.

Participants first completed a demographic questionnaire, followed by the alcohol questionnaire and the PDS. Finally, participants performed a computerized version of the Raven SPM.

Statistical analysis

Since the PDS items differ by gender, boys and girls were analyzed separately. To control for the confounding effects of age, hierarchical multiple regression analyses were conducted with age and PDS score (mean score of all PDS items) as predictors and lifetime and recent alcohol use as dependent variables. To examine gender differences in pubertal maturation, we computed Puberty Category Scores (Crockett, 1988; see: http://www.sleepforscience.org/contentmgr/showdetails.php/id/91), which provide pubertal development scores on the same scale for boys and girls (i.e., 1 = *prepubertal* to 5 = *postpubertal*).

Experiment 2: sex steroids and alcohol use

Participants

86 boys ($M = 14.66$ years, $SD = 1.64$) and 82 girls ($M = 14.66$ years, $SD = 1.81$) aged 12–17 years participated in the second experiment. Participants completed the WISC intelligence subscales Block Design and Similarities (Wechsler, 1991) to estimate their IQ (range = 80–135, $M = 108.4$, $SD = 10.4$). T-levels could not be reliably assessed in 5.9% of participants, and E2 could not be reliably assessed in 3.6% of participants. Participants who failed to provide a reliable saliva sample did not differ from those who could provide a reliable sample in terms of IQ, and lifetime and recent alcohol use (all F's<1, all p's>.33).

Participants were recruited with the help of local schools. Parents of the participants gave informed consent, and the adolescents gave informed assent. Participants were free from any history of psychiatric, endocrinological, or neurological illnesses, screened by an a priori interview with their parents. All procedures of this study were approved by the Medical Ethical Committee of the Leiden University Medical Centre (LUMC), and were in agreement with the Declaration of Helsinki (Seoul amendments, 2008).

Measures

Alcohol use. Participants filled out the same online questionnaires on lifetime and recent alcohol use that were used in Experiment 1. The questionnaires were e-mailed to the participants, and the instructions explicitly stated that their answers would not be disclosed to their parents.

Pubertal development. Participants completed an online version of the PDS, which was e-mailed to them.

Sex steroids. Boys and girls collected a saliva sample by passive drool at home, directly after waking up. To control for hormonal fluctuations across the menstrual cycle, postmenarcheal girls ($n = 61$, 74.4%) collected the saliva sample on the same day within the early follicular phase of the menstrual cycle (day 7), when hormone levels (e.g., progesterone) are relatively low (Dorn et al., 2006; Mihm et al., 2011; Peper et al., 2012). Similarly, girls using oral contraceptives ($n = 6$) collected a saliva sample on the last day within their stopping period (day 7). Girls using contraceptives without a stopping period, such as hormonal intrauterine devices (e.g. Mirena), were excluded from participating in this study.

The saliva samples of boys and girls were assayed for T- and E2-levels at the Department of Clinical Chemistry of the Free University Medical Centre (VUMC). The lower limit of detection was 4 pmol/L for T, and 0.1 pg/ml for E2. Salivary T was determined by isotope dilution–online solid phase extraction liquid chromatography–tandem mass spectrometry (ID–XLC–MS/MS; De Ronde et al., 2011; Peper et al., 2012). Intra-assay coefficient of variation (CV) was 11% and 4%, at 10 and 140 pmol/L, respectively and inter-assay CV was 8% and 5%, at 31 and 195 pmol/L, respectively.

Salivary E2 was determined using an enzyme linked immunosorbent assay (ELISA; DRG Instruments, Marburg, Germany). Inter-assay CV was 8% and 15% at 10 and 40 pg/L, respectively.

Statistical analyses

Due to the gender-specific nature of sex steroids, all analyses were carried out separately for boys and girls. Since T was highly skewed in boys and girls, T-levels were log transformed before they were entered as predictors in the analyses.

To test whether sex steroid levels are related to having ever consumed alcohol, logistic regression analyses were performed with T- and E2-levels as predictors and lifetime alcohol use (coded as 0 = *never used,* and 1 = *1 or more glasses consumed*) as dependent variable. Age was also included as predictor in these regression analyses, to control for the potentially confounding effects of age. Alcohol use and sex steroid levels were not normally distributed in boys and girls (Kolmogorov–Smirnov test; all p's<.05). Therefore, associations between quantity of alcohol use and sex steroid levels were examined by computing age-corrected Spearman correlations (Kendall and Gibbons, 1990; see http://imaging.mrc-cbu.cam.ac.uk/statswiki/FAQ/partsp) between lifetime and recent alcohol use and T- and E2-levels. Similar to Experiment 1, Puberty Category Scores (Crockett, 1988) were computed to test for gender differences in pubertal maturation.

Results

Experiment 1: pubertal maturation and alcohol use

Demographic characteristics

Boys and girls were the same age ($t(795) = -1.04$, $p = .30$). Lifetime ($t(795) = 0.65$, $p = .51$) and recent ($t(795) = 1.58$, $p = .11$) alcohol use did not differ between the sexes. Based on the Puberty Category Scores, girls showed more advanced pubertal maturation than boys ($p < .001$).

Boys

Age explained a significant amount of the variance in lifetime alcohol use ($R = .36$, $F(1,403) = 60.70$, $p < .001$). However, when PDS score was added to the regression, a significant contribution was made to explaining the variance in lifetime alcohol use ($R = .40$, $F(1,402) = 13.73$, $p < .001$). In line with our expectations, PDS score was positively related to lifetime alcohol use in the model including both age and PDS score as predictors ($B = .66$, $p < .001$), indicating that advanced pubertal maturation is associated with increased lifetime alcohol use, when controlling for age.

Consistent with lifetime use, age explained a significant amount of variance in recent alcohol use ($R = .34$, $F(1,403) = 54.17$, $p < .001$), and adding PDS score to the regression contributed significantly to the explained variance in recent alcohol use ($R = .37$, $F(1,402) = 7.95$, $p < .01$). Consistent with our hypotheses, PDS score was positively correlated with recent alcohol use ($B = .38$, $p < .01$) in the model containing age and PDS as predictors. Multicollinearity was not an issue in these analyses, since the variance inflation factor (VIF) was relatively low (<1.27).

Girls

Age explained a significant amount of variance in lifetime alcohol use ($R = .39$, $F(1,390) = 71.55$, $p < .001$), and adding PDS score to the regression contributed significantly to this explained variance ($R = .41$, $F(1,389) = 7.18$, $p < .01$). As predicted, PDS score was positively associated with lifetime alcohol use ($B = .60$, $p < .01$) in the model including both age

and PDS, indicating that more advanced pubertal maturation is associated with greater lifetime alcohol use, after controlling for age.

The findings for recent alcohol use mirrored those for lifetime use, in that age explained a significant amount of variance in recent alcohol use ($R = .35$, $F(1,390) = 54.77$, $p < .001$), and adding PDS score to the regression contributed significantly to this explained variance ($R = .36$, $F(1,389) = 4.13$, $p = .04$). As expected, PDS score was positively related to recent alcohol use ($B = .27$, $p = .04$) in the model with age and PDS as predictors. Again, multicollinearity was not a problem in these analyses (VIF < 1.09).

Experiment 2: sex steroids and alcohol use

Demographic characteristics

Based on the Puberty Category Scores, girls showed more advanced pubertal maturation than boys ($p = .013$). As expected, boys had higher T-levels than girls ($t(153) = 13.51$, $p < .001$). No gender differences were found for age, E2-levels, and lifetime and recent alcohol use (all p's > .17) (Table 2). PDS scores were positively related to T ($rho = .55$, $p < .001$) and E2 ($rho = .38$, $p < .01$) in boys, and to T in girls ($rho = .33$, $p < .05$), indicating that salivary sex steroid levels are a relatively sensitive index of (self-reported) pubertal development. In boys, age was positively correlated with alcohol use, sex steroid levels, and total PDS score (Table 3), while T was positively associated with E2. In girls, age was positively correlated with alcohol use, but not related to sex steroid levels (Table 3). T was not significantly related to E2 in girls.

Boys: sex steroids and alcohol use

First, we tested whether sex steroid levels contributed to the onset of alcohol use in boys. Consistent with our predictions, after controlling for age, higher T- and E2-levels were associated with a greater probability of alcohol use (Table 4).

Second, the relation between quantity of alcohol use and sex steroid levels was studied in boys. In line with our hypotheses, higher E2-levels were significantly correlated with higher levels of lifetime alcohol use ($rho = .30$, $p < .001$). In contrast, T-levels were not related to quantity of lifetime alcohol use (all p's > .25). Similarly, sex steroid levels were not associated with recent alcohol use (all p's > .19).

Girls: sex steroids and alcohol use

Contrary to our findings for boys, sex steroid levels were neither significantly related to the onset of alcohol use (Table 4), nor to the quantity of lifetime and recent alcohol use in girls (all p's > .16). The heterogeneity of our sample, which included both pre- and postmenarcheal girls, might have affected the associations between sex steroids and alcohol use. Therefore, we repeated the analyses in postmenarcheal girls ($n = 61$) only. Again, no significant associations were found between sex steroid levels and alcohol use (all p's > .30).

Table 3
Correlations (*rho*) between PDS, sex steroid levels, age and alcohol use by gender.

	1.	2.	3.	4.	5.	6.
1. PDS		.55***	.38**	.67***	.39***	.27*
2. T (log-transformed)	.33**		.44***	.62***	.50***	.33**
3. E2	.13	.04		.40***	.39***	.19
4. Age (years)	.63***	.19	.12		.66***	.57***
5. Lifetime alcohol use	.46***	.05	−.01	.74***		.82***
6. Recent alcohol use	.42***	.07	.01	.65***	.90***	

Note. Correlations in boys are presented above the diagonal, girls below the diagonal.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

Discussion

The goal of the present study was to investigate whether advanced pubertal maturation and sex steroid levels are associated with alcohol use in healthy adolescents. In Experiment 1, we found that, after controlling for age, advanced pubertal maturation is associated with higher levels of lifetime and recent alcohol use in boys and girls.

The observed relationship between pubertal maturation and alcohol use is consistent with previous studies (Biehl et al., 2007; Bratberg et al., 2005; Westling et al., 2008; Wiesner and Ittel, 2002). More mature adolescents might engage in higher levels of alcohol use because of a gap between physical and psychosocial maturation, and because they affiliate with older and deviant peers (Engels, 2009; Ge and Natsuaki, 2009; Westling et al., 2008). Another explanation is that heightened sex steroid levels in more mature adolescents promote sensation-seeking tendencies, which in turn lead to experimentation with alcohol. In Experiment 2, we studied this hypothesis by examining the relation between sex steroid levels and alcohol use in adolescents.

In line with our predictions and previous research (Costello et al., 2007; Eriksson et al, 2005), we showed that higher T-levels are associated with a greater likelihood of lifetime alcohol use in boys. T might promote adolescent alcohol use through several means. First, it has been shown that higher T levels are related to increased activation in a reward-related brain region (i.e., the ventral striatum) during the anticipation and processing of monetary rewards in adolescents (Forbes et al., 2010; Op de Macks et al., 2011). This enhanced reward sensitivity is hypothesized to stimulate sensation-seeking behavior, including alcohol use, in adolescents (Steinberg, 2008). Furthermore, it has been suggested that T increases one's motivation to show status seeking behavior (Eisenegger et al., 2011), and that high levels of alcohol use are related to high status in the peer group in adolescents (Mayeux et al., 2008).

Consistent with our hypotheses, we also found that greater E2-levels are related to both the onset and quantity of alcohol use in boys. To the best of our knowledge, this is the first study that has demonstrated this relation between E2-levels and alcohol use in boys. E2 might directly contribute to alcohol use by stimulating

Table 2
Mean (*SD*) sex steroid levels, age and alcohol use of the Experiment 2 sample.

	Boys ($n = 86$)	Girls ($n = 82$)
T (pmol/L)	225.93 (149.09)	22.85 (14.02)***
E2 (pmol/L)	0.97 (0.41)	1.06 (0.43)
Age (years)	14.66 (1.64)	14.66 (1.81)
Lifetime alcohol use[a]	2.03 (3.55)	1.95 (3.13)
Recent alcohol use[b]	1.08 (2.22)	1.04 (1.97)

Note. Values represent means with standard deviations in parentheses.
[a] Ranging from 0 = *no alcohol use* to 10 = *>90 glasses consumed.*
[b] Ranging from 0 = *no alcohol use* to 9 = *>50 glasses consumed.*
*** $p < .001$.

Table 4
Age-corrected relations between sex steroid levels and lifetime alcohol use in boys and girls.

Predictors	Boys ($n = 86$)			Girls ($n = 82$)		
	Lifetime alcohol use			Lifetime alcohol use		
	B	OR	CI	*B*	OR	CI
T (log-transformed)	2.23**	9.31	2.23–38.90	1.05	2.85	0.70–11.64
E2	1.48**	4.40	1.57–12.31	0.66	1.93	0.72–5.16

Note. Lifetime alcohol use was coded as 0 = *never used*, and 1 = *one or more glasses consumed*. OR = odds ratio, CI = 95% confidence interval.
** $p < .01$.

sensation-seeking behaviors (Vermeersch et al., 2009). Alternatively, the enzyme aromatase converts T into E2 and this enzyme has been implicated in addictive behaviors (Lenz et al., 2011; Purohit, 2000). Therefore, the heightened E2-levels in boys who report higher alcohol use might (indirectly) reflect increased activity of the enzyme aromatase.

Contrary to our predictions, E2 was not related to alcohol use in girls. These findings are in keeping with the findings of Costello et al. (2007), who also found no relation between E2 and alcohol use and abuse in adolescent girls. Nevertheless, Martin et al. (1999) did observe a positive correlation between E2-levels and alcohol consumption in late adolescent girls. These discrepant findings might be attributed to age differences between participants across studies, since the girls in the Martin et al. (1999) study were older (i.e., late adolescents) than the girls in the present study and in the Costello et al. (2007) study. Moreover, the girls in our sample had relatively low E2-levels, since they collected their saliva sample during the early follicular phase of their menstrual cycle, when hormone levels are low (Mihm et al., 2011). These low E2-levels could have obscured potential alcohol effects.

Inconsistent findings across studies might also be due to the complex association between estradiol and brain mechanisms contributing to risk-taking behavior in females. In female rodents and nonhuman primates, estradiol increases dopamine (DA) neurons and DA activity in brain areas implicated in reward processing (Kuhn et al., 2010). However, in human females, the relation between estradiol and (neuro)cognitive functioning is more complex, in that it differs by genotype (Jacobs and D'Esposito, 2011). Further, in adolescent girls, estradiol levels were found to be positively correlated with activation in brain areas associated with cognitive control (Op de Macks et al., 2011). Instead, it might also be hypothesized that the social impact of early and dramatic pubertal changes contributes to a greater risk for alcohol use in girls (through peer effects), whereas sex steroid hormones affect sensation-seeking and motivational tendencies in boys more directly. This hypothesis needs to be tested in future research.

Due to the cross-sectional design of the present study, the direction of the relationship between pubertal maturation, sex steroids and alcohol use remains unspecified. It might be hypothesized that alcohol use affects pubertal development, instead of the other way around (Pierucci-Lagha et al., 2006). Several longitudinal studies have partially dispelled this hypothesis by showing that advanced pubertal maturation in early adolescence is related to increased alcohol use several years later (Bratberg et al., 2005; Dick et al., 2001; Kaltiala-Heino et al., 2011).

While the focus of the current paper is on pubertal development and hormones, it should be noted that there is likely a myriad of factors contributing to alcohol use in adolescence. We focused on the contribution of biological factors to alcohol use, because the physical changes that are the hallmark of puberty are driven entirely by biological (i.e., hormonal) factors. Nonetheless, social, cognitive and emotional factors that are developing during adolescence (Blakemore and Choudhoury, 2006; Crone, 2009) also contribute to individual variation in adolescents' alcohol use (Weinberg et al., 1998), and future research should examine interactions between these factors and biological factors in the prediction of adolescent alcohol use (cf. Costello et al., 2007).

In conclusion, our study was able to replicate the positive association between advanced pubertal maturation and increased intake of alcohol in adolescents. Moreover, the results of the current study indicate a particular role for sex steroids in this association for boys. Specifically, we are the first to demonstrate that E2 contributes to alcohol use in adolescent boys, and that E2 and T are differentially associated with the onset and quantity of alcohol use. These findings increase our understanding of the underlying mechanisms of the onset and quantity of alcohol use across adolescence. In addition, our findings raise the intriguing hypothesis that the social impact of early and dramatic pubertal changes might contribute to alcohol use in girls (through peer effects), whereas sex steroids might contribute to alcohol use in boys more directly, by stimulation sensation-seeking and motivational tendencies. This hypothesis needs to be tested in future research.

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