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Perceived Pubertal Timing and Recent Substance Use among Adolescents: A Longitudinal Perspective

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Abstract

Aims—To determine the longitudinal associations between perceived pubertal timing and recent substance use between the ages of 11 and 17.

Design, setting, and participants—A school-based cohort sequential study of adolescents in rural North Carolina (N=6,892, 50% female) in the 6th to 8th grades at baseline and interviewed across five consecutive semesters.

Measurements—Self-administered questionnaires in a group setting measured perceived pubertal development using the Pubertal Development Scale and adolescents reported past three month use of cigarettes, alcohol, and marijuana. Latent class growth analysis determined the longitudinal relationships between perceived pubertal timing (early, on-time, and late) and use of the three substances.

Findings—A negative quadratic model was the best fitting model for all three substances. Higher proportions of early developers had used cigarettes and marijuana within the past three months at age 11 compared with on-time (p<.001 and p=.013) and late developers (p=.010 and p=.014) and a higher proportion of early developers had recently used alcohol at age 11 compared with on-time adolescents (p<.001). However, the proportion of recent cigarette and marijuana users increased more across adolescence for on-time adolescents compared with early developers (p=.020 and p=.037). Desistance in the proportion of substance users was similar for all adolescents (all p>.050).

Conclusions—Adolescents who believe they are more advanced in puberty than their peers are more likely to have recently used cigarettes, alcohol, and marijuana compared with adolescents

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Declaration of Interest

The authors have no conflicts of interest to declare.

who believe they are on-time or late developing; these findings are mainly due to differences in use at age 11.

Keywords

substance use; adolescence; puberty

Introduction

Puberty is a highly salient process for adolescents because of the cascade of associated physical, cognitive, emotional, and social changes (1–8). There is variation in the onset and tempo of puberty by gender and race/ethnicity, as well as individual differences within these groups (9–13). This variation has prompted researchers to explore how adolescent health is impacted by pubertal timing, defined as the comparative pubertal development of an adolescent in relation to peers. This study examines longitudinal relationships between perceived pubertal timing and recent cigarette, alcohol, and marijuana use among a school-based sample of adolescents aged 11 to 17.

Research has consistently shown that early developing adolescents are at highest risk for substance use compared with their peers (14–34). A limitation of prior research, however, is that many studies have collapsed on-time and late developers into the same category, thereby obscuring differences between these categories (15, 19, 22, 24, 30, 33, 34). Research demonstrating that both male and female late developers are more likely to engage in substance use than their on-time peers underscores the need to examine early, on-time, and late developers (26, 35–37).

Most studies examining associations between perceived pubertal timing and substance use have focused on girls (15, 22–25, 33, 35, 38). In particular, early maturing females have the highest risk of substance use compared to those who are on-time or late (15, 22–25, 33). Earlier research theorized that pubertal timing was not a risk factor for boys, and that early developing boys might be better adjusted and less likely to engage in risky behavior than their on-time peers (39–41). More recent research has found a relationship between perceived pubertal timing and substance use in males (19, 20, 27–31, 36), with most finding that early developing males are at highest risk (19, 27–31). There continues to be a need to examine the role of gender in the relationship between perceived pubertal timing and substance use.

Another limitation of previous research on perceived pubertal timing and substance use is that it has been cross-sectional (15, 18, 20, 24, 30, 31, 35, 38, 39) or based on only two time points (14, 17, 26–29, 33, 36). Both puberty and substance use are individually variable processes that develop over time. Research with both males and females suggests that the relationship between perceived pubertal timing and substance use also may be changing over time (21, 22, 25, 28, 29, 32–34, 42, 43). For example, the impact of perceived pubertal timing may be stronger in mid-adolescence compared with early adolescence (28). One longitudinal study found that early developing girls began substance use earlier and transitioned into more advanced patterns of substance use faster than other females (33). In contrast, other longitudinal studies with female (22, 25) and mixed gender samples (21, 29,

32, 34) found that the effects of early maturation decrease over time, suggesting that on-time and late developers begin to “catch up” with their early developing peers when substance use becomes more normative in adolescence. These “catch-up” findings, also suggested in a recent retrospective study of college women (44) support the need to examine the relationship between perceived pubertal timing and substance use across adolescence.

An issue in examining the longitudinal relationship between perceived pubertal timing and substance use is the measurement of pubertal timing. There is increasing evidence of a relationship between pubertal onset and pubertal tempo, using both clinical (11) and perceived measures of pubertal timing (12, 45, 46), such that adolescents who begin puberty earlier take longer to reach full pubertal development compared with their same-aged peers who begin puberty later. Furthermore, research has shown that adolescents’ perceptions of their pubertal timing are relatively unstable across adolescence (47–49). These findings indicate the need to measure pubertal timing perceptions as unfolding over time, and to incorporate longitudinal patterns of perceived pubertal timing into research on the effects on health risk behavior, rather than relying on an assessment at one or two points in time (47).

This study addresses limitations of prior research by including both males and females, modeling longitudinal relationships between patterns of perceived pubertal timing and substance use, and examining multiple substances. We examine longitudinal associations between perceived pubertal timing and recent cigarette, alcohol, and marijuana use from ages 11 to 17. Based on prior studies, we expect both male and female early developers to emerge as at higher risk for substance use, although prior studies do not support stating expectations for whether they will remain at risk across adolescence. Evidence is also insufficient for making hypotheses about the risk associated with late development. Similarly, we do not offer hypotheses about differential relationships across substances. Few studies have examined the differential associations between perceived pubertal timing and various substances, and findings are mixed as to whether no differences (25, 29) or differences (18, 27, 35) exist, with no clear patterns emerging in the latter studies.

Method

The Context Study

Data are from the Context of Adolescent Substance Use study (Context Study), a school-based cohort sequential study of adolescents from three rural North Carolina counties. Wave 1 began when adolescents were enrolled in the 6th (Cohort 1), 7th (Cohort 2), and 8th (Cohort 3) grades (Spring 2002); data collection occurred in April and October through Spring 2004 (Wave 5). All adolescents in the grades of interest in the sampled schools (eight middle schools, two K-8 schools, six high schools, and three alternative schools) were considered eligible for participation (eligible sample at Wave 1=5,906; Wave 2=6,226; Wave 3=6,251; Wave 4=6,342; and Wave 5=6,161). Response rates ranged from 88% at Wave 1 to 76% at Wave 5. The Context Study was approved by the Institutional Review Board at the sponsoring institution. The study received a waiver of written parental consent and written adolescent assent was obtained. Data were collected by the research team in a group setting using self-administered questionnaires. Completion time was approximately one hour and there was no monetary compensation.

Study Sample

Data are from adolescents who participated in at least one wave of data collection (N=6,892). Approximately 13% participated in one wave but the largest percentage of participants 42%, completed all five waves. The sample was limited to adolescents who were aged 11 to 17 at any wave to include only students who were within the typical age range for their grade (N=172 excluded) and those who provided information on sex and race/ethnicity (N=295 excluded). Excluded adolescents were less likely to be White, more likely to be male, and less likely to have participated in all five waves of data collection (all $p < .001$). The final sample included 6,425 adolescents (50% male, 53% White, 36% African-American, 4% Latino, and 7% indicating another racial/ethnic category, Wave 1 age M=13.1 (SD=.97).

Measures

Recent substance use—The three outcomes of interest were recent cigarette, alcohol, and marijuana use. Adolescents who responded affirmatively to lifetime use were asked on how many days in the past three months they had smoked at least one full cigarette (range 0 to 20 days or more), had one or more full drinks of alcohol, not including for religious purposes, (range 0 to 20 days or more), or used marijuana (range none to 10 times or more). Because of low response frequencies, particularly among younger age adolescents (see Table 1), three dichotomous measures were created (0=no recent use, 1=any recent use).

Perceived pubertal timing—*Perceived pubertal timing* was measured with the Pubertal Development Scale (PDS) (50). The PDS consists of five questions each for boys and girls assessing development of body hair growth, skin changes, and height for boys and girls, voice and facial hair growth for males and breast development for females (1=not yet started to 4=seems complete). Females are also asked if they started menstruating (1=no, 4=yes). Items were averaged to obtain a mean PDS score. We calculated the mean pubertal status by age, sex, and race/ethnicity and compared each adolescent's pubertal status to the mean for their demographic subgroup. Adolescents were classified as "early" (1=more than one standard deviation above the mean), "on-time" (0=from one standard deviation above the mean to one standard deviation below the mean), or "late" (-1=more than one standard deviation below the mean).

Previous research with this sample found that perceived pubertal timing was unstable (47). In other words, an adolescent classified as on-time at one age could be classified as early or late developing at another age. To capture individual patterns of perceived pubertal timing formed across the multiple assessments, we used latent class analysis (LCA). This person-centered analytic approach allows for individual variability over time in an outcome of interest (46). With LCA we were able to test and confirm that the instability in perceived pubertal timing was due to measurement error, such that an adolescent occasionally deviated from an underlying stable pattern of perceived pubertal timing. In the prior analyses a three-class solution was the best fitting model (Bayesian Information Criterion (BIC) = 31,153; entropy = .81). There was not support for a consistent pattern of change, as would be expected if there were significant pubertal tempo differences in our sample (i.e., we did not observe a latent class that shifted from early developing in early adolescence to on-time in

mid-adolescence). The current study uses the three stable latent classes of perceived pubertal timing (Class 1: always on-time; Class 2: always early; and Class 3: always late) as the predictors of recent substance use in order to take into account this measurement error.

Analytic Strategy

The analytic approach was based on an accelerated longitudinal design, which maximizes the advantages of the cohort sequential design of the Context Study (51, 52). As a result, we were able to collapse data across the three cohorts and use age as the unit of time instead of data collection wave. First, however, we tested the assumption that there are no cohort differences in any of the variables of interest (i.e., predictor variables, outcome variables, and covariates). We found only one difference: adolescents in the youngest cohort were more likely to be classified as late developers than as on-time compared with adolescents in the middle cohort ($B=.376, p=.001$). To account for this difference all of the analytic models included cohort one membership as a control variable.

To examine whether the development of recent substance use varied by the three latent classes of perceived pubertal timing, we used latent class growth analysis (LCGA). LCGA is a special case of growth mixture modeling, a person-centered approach to longitudinal data analysis extending from longitudinal growth modeling (53, 54). LCGA allows a test of whether growth model parameters (the fixed effects that explain the development of substance use across adolescence) vary by unobserved subpopulations (individuals in each pubertal timing latent class) (53, 54). Separate growth models are estimated for each perceived pubertal timing latent class and it is possible to test whether these models statistically differ. All analyses were conducted using MPlus 6.1 (55). The models were estimated using the maximum likelihood estimator with robust standard errors (MLR) (56). This estimator, based on Full Information Maximum Likelihood (FIML), addresses missing data by using all available data to maximize the information available for data analysis (57). By using MLR, all adolescents with at least one wave of data were retained in the analytic sample. MPlus can also account for multilevel data, allowing us to control for the nesting of individuals in schools.

The first analytic step was to determine the shape over time of the unconditional longitudinal growth models (the average development) for each substance of interest (cigarettes, alcohol, and marijuana). When modeling dichotomous data, MPlus uses thresholds as a corrective procedure. In order to have an identified model the thresholds were fixed to zero (57–59). The intercept is thus interpreted as the amount of deviation from 50% probability of the outcome. Because this is a difficult metric to substantively interpret, we used the probit regression parameters to transform the outcome into the proportion of adolescents who were recent substance users (ranging from 0 to 1) at each age. Standard fit statistics are not available for use with MLR estimator. Instead we used the likelihood ratio chi-square test to determine the best fit, where $p<.05$ indicated an improvement in fit compared with the previous model. Additionally, the best fitting model should have the lowest values for the BIC, sample-size adjusted BIC (aBIC), and Akaike information criterion (AIC), as these fit indices provide estimates for the relative difference in the likelihood function of a given model and the unknown true likelihood function of the data.

The second analytic step was to estimate the substance use growth parameters (fixed effects) for each perceived pubertal timing latent class (51). Sex and race/ethnicity were added as predictors of the perceived pubertal timing latent classes (in addition to cohort one membership). The differences in the growth parameters were tested using contrast statements in the MPlus Model Constraint command.

Finally, we tested whether adolescent sex moderated the relationships between perceived pubertal timing and the substance use growth parameters. This was done by regressing the fixed effects of the substance use growth curve on adolescent sex. This is analogous to testing a perceived pubertal timing class by sex interaction effect on the substance use growth parameters.

Results

To determine the underlying form of development of recent use of each substance, we compared three nested models: intercept-only (no growth); intercept and slope (linear growth); and intercept, slope, and quadratic (nonlinear growth). The best fitting unconditional growth model for all three substances was the quadratic model (Table 2). For all three substances the mean growth parameters (fixed effects) were statistically significant (Table 3), indicating that for each substance the proportion of recent users increased from early adolescence and this growth began to decelerate in later adolescence. All of the random effects were significant except for the cigarette quadratic term, indicating individual variability around the mean curve. This individual variability provided justification for determining whether the perceived pubertal timing latent classes explained some of this variability.

We compared the growth parameters for each substance across the three perceived pubertal timing latent classes (on-time, early, and late) (Table 4, Figures 1–3). For all three substances, a higher proportion of early developers reported recent use at age 11 compared with on-time adolescents (cigarettes and alcohol $p < .001$, marijuana $p = .013$). However, the proportion of cigarette and marijuana users across adolescence increased faster among on-time adolescents compared with early developers (cigarettes $p = .020$ and marijuana $p = .037$). A higher proportion of early developers recently used cigarettes and marijuana at age 11 compared with late developers (cigarettes $p = .010$ and marijuana $p = .014$). There were no significant differences in the quadratic terms in any of the models (all $p > .050$), indicating the deceleration in the growth of the proportion of recent substance users was similar for all adolescents. When testing whether adolescent sex was a moderator, we found no significant effects in any of the models, indicating that the relationship between the perceived pubertal timing latent classes and the substance use growth parameters did not vary by adolescent sex.

Discussion

The purpose of this study was to determine the longitudinal relationships between perceived pubertal timing and past three month use of cigarettes, alcohol, and marijuana in a school-based sample of adolescents aged 11 to 17. As hypothesized, early perceived pubertal timing

places adolescents at higher risk for substance use (14–34). The results add to prior research by utilizing latent class growth analysis to account for the measurement instability of perceived pubertal timing and by demonstrating the importance of examining the longitudinal associations between perceived pubertal timing and substance use.

An important contribution of this study is the inclusion of both females and males, given that much of the prior research included only females (15, 22–25, 33, 35, 38). We tested whether sex moderated the association between perceived pubertal timing and substance use and found no significant differences. Early developing females and males were at higher risk for recent use of all three substances compared with their on-time and later developing peers, which supports previous research (14, 18, 19, 26–29, 31).

Contrary to some prior research, we found little support for differential associations between perceived pubertal timing and the three substances (cigarettes, alcohol, and marijuana) (18, 27, 35). A higher proportion of early developing adolescents were using all three substances at age 11 compared with their on-time or later developing peers. While not possible to test for statistically significant differences, the patterns of recent cigarette and recent marijuana use appear more similar to one another than to the patterns of recent alcohol use. This could be due, in part, to alcohol use being more socially acceptable in adolescence compared with cigarette or marijuana use (60, 61). Additional research is needed before conclusions about differential relationships, or not, across substances can be made.

The study results only partially support the theory that on-time and late developers “catch up” to their early developing peers in regards to substance use (22, 25, 29, 32, 42, 44). Although there were greater increases in the proportion of recent cigarette and marijuana users among on-time developers compared with early developers between the ages of 11 and 17, on-time developers never fully caught up with their early developing peers. Similar results were reported in a recent study investigating associations between perceived pubertal timing and cigarette use among similar aged adolescents in London, UK (21). Thus while there was some evidence of a catch-up effect, overall it was not enough to surpass the impact of perceived pubertal timing on adolescent substance use in early adolescence.

A number of mechanisms have been proposed for why early pubertal timing could be a risk factor for substance use and other adverse outcomes (62, 63). One hypothesis is that early developers are the first group to experience the structural brain changes linked to the surge of hormones during puberty, which may be related to early engagement in substance use (64). Another proposed mechanism is that early developing adolescents are at heightened risk because they appear older in age and thus may be more likely to affiliate with older peers, who could then expose them to substance use at an earlier age than their on-time or late developing peers (14, 62, 65). However, both of these mechanisms imply that on-time and late developers would “catchup” to their early developing peers as they begin experiencing the same biological changes and as substance use becomes more normative. Another hypothesis, the “maturation disparity hypothesis,” is that early developing adolescents are ill-prepared for pubertal development, such that they have not had the opportunity to gain the cognitive and social competencies to cope with their physical changes (19, 63, 66). Yet another possible mechanism is that there may be psychological

consequences of early development that cause adolescents to be more likely to engage in substance use as a coping strategy, which is supported by literature that demonstrates an association between perceived pubertal timing and psychological distress (7, 8, 36, 67). Which of these mechanisms might account for early developers' heightened risk of substance use throughout mid-adolescence is an area for future research.

Among the study limitations, the sample was 11 to 17 years of age, which did not capture very early developers or the completion of the pubertal process for some (68). This age range may also have prevented the observation of pubertal tempo differences seen in other samples (11, 12, 45, 46). The measure of perceived pubertal timing was based on adolescent self-report, which has been shown to be biased compared with clinically assessed measures of pubertal development. It has been argued, however, that self-report is acceptable when approximation (such as the categorization of early, on-time, and late used in this study) is acceptable (69, 70). Because bias is greatest at the earliest and latest pubertal stages and we assessed perceived pubertal development across the ages of 11 to 17, bias may have been reduced. It would be beneficial for future research to replicate these analyses with clinical measures of pubertal development.

Recent substance use was relatively infrequent as would be expected in a general population sample, especially among the youngest adolescents, which precluded measuring substance use continuously. The dichotomous measures could have decreased the association between perceived pubertal timing and substance use because the substance use measures include a range of substance use, from adolescents who experimented once in the last three months to daily users. Furthermore, the "no recent substance use" category included adolescents who had never used the substance and those who had used but not in the past three months. While the substance use measures were self-report, research has supported the use of self-report measures in assessing adolescent risk behavior (71). Analyses did not include socioeconomic status because of the significant amount of missing data on the indicator of SES (parent education), but analyses including this measure did not change the results. Finally, the sample was from three rural counties in North Carolina so findings may not be generalizable to adolescents living in urban/suburban areas or those in other parts of the country. Despite these limitations, this study adds to the understanding of the relationship between perceived pubertal timing and adolescent substance use. The analyses were conducted using a longitudinal sample and advanced statistical methods that allowed for the control of the measurement instability associated with perceived pubertal timing classification. Because pubertal timing cannot be altered through psychosocial interventions, the implications for the prevention of substance use among early developers are less straightforward than with other risk factors. The findings do suggest a need for prevention programming at young ages, because by age 11 differences in use by perceived pubertal timing class are already present (72).

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References

1. Silbereisen, RK.; Kracke, B. Self-reported maturational timing and adaptation in adolescence. In: Schulenberg, JE.; Maggs, JL.; Hurrelmann, K., editors. *Health Risks and Developmental Transitions During Adolescence*. New York, NY: Cambridge University Press; 1997. p. 85-109.
2. Summers-Effler E. Little girls in women's bodies: Social interaction and the strategizing of early breast development. *Sex Roles*. 2004; 51(1–2):29–44.
3. Lee J. Never innocent: Breast experiences in women's bodily narratives of puberty. *Fem Psychol*. 1997; 7(4):453–74.
4. Beausang CC, Razor AG. Young western women's experiences of menarche and menstruation. *Health Care Women Int*. 2000; 21(6):517–28. [PubMed: 11235283]
5. Flaming D, Morse JM. Minimizing embarrassment: Boys' experiences of pubertal changes. *Issues Compr Pediatr Nurs*. 1991; 14(4):211–30. [PubMed: 1842786]
6. Forbes EE, Dahl RE. Pubertal development and behavior: Hormonal activation of social and motivational tendencies. *Brain Cogn*. 2010; 72(1):66–72. [PubMed: 19942334]
7. Weichold K, Buttig S, Silbereisen RK. Effects of pubertal timing on communication behaviors and stress reactivity in young women during conflict discussions with their mothers. *J Youth Adolesc*. 2008; 37(9):1123–33.
8. Sontag LM, Graber JA, Brooks-Gunn J, Warren MP. Coping with social stress: Implications for psychopathology in young adolescent girls. *J Abnorm Child Psychol*. 2008; 36(8):1159–74. [PubMed: 18465219]
9. Archibald, AB.; Graber, JA.; Brooks-Gunn, J. Pubertal processes and physiological growth in adolescence. In: Adams, GR.; Berzonsky, MD., editors. *Handbook of Adolescence*. Oxford, UK: Blackwell Publishers; 2003. p. 24–48.
10. Sun SS, Schubert CM, Chumlea WC, Roche AF, Kulin HE, Lee PA, et al. National estimates of the timing of sexual maturation and racial differences among US children. *Pediatrics*. 2002; 110(5):911–9. [PubMed: 12415029]
11. Marceau K, Ram N, Houts RM, Grimm KJ, Susman EJ. Individual differences in boys' and girls' timing and tempo of puberty: Modeling development with nonlinear growth models. *Dev Psychol*. 2011; 47(5):1389–409. [PubMed: 21639623]
12. Mendle J, Harden KP, Brooks-Gunn J, Graber JA. Development's tortoise and hare: Pubertal timing, pubertal tempo, and depressive symptoms in boys and girls. *Dev Psychol*. 2010; 46(5): 1341–53. [PubMed: 20822243]
13. Cance JD, Ennett ST. Demographic differences in self-report pubertal status among rural adolescents in the US. *Ann Hum Biol*. 2012; 39(1):84–7. [PubMed: 22092158]
14. Tschann JM, Adler NE, Irwin CE Jr, Millstein SG, Turner RA, Kegeles SM. Initiation of substance use in early adolescence: The roles of pubertal timing and emotional distress. *Health Psychol*. 1994; 13(4):326–33. [PubMed: 7957011]
15. Stattin H, Kerr M, Skoog T. Early pubertal timing and girls' problem behavior: Integrating two hypotheses. *J Youth Adolesc*. 2011; 40(10):1271–87. [PubMed: 21769611]
16. Negri S, Ji J, Trickett PK. Exposure to peer delinquency as a mediator between self-report pubertal timing and delinquency: A longitudinal study of mediation. *Dev Psychopathol*. 2011; 23(1):293–304. [PubMed: 21262055]
17. Ge X, Jin R, Natsuaki MN, Gibbons FX, Brody GH, Cutrona CE, et al. Pubertal maturation and early substance use risks among African American children. *Psychol Addict Behav*. 2006; 20(4): 404–14. [PubMed: 17176175]
18. Bratberg GH, Nilsen TI, Holmen TL, Turid L, Vatten LJ. Perceived pubertal timing, pubertal status and the prevalence of alcohol drinking and cigarette smoking in early and late adolescence: A population based study of 8950 Norwegian boys and girls. *Acta Paediatr*. 2007; 96(2):292–5. [PubMed: 17429923]
19. Westling E, Andrews JA, Peterson M. Gender differences in pubertal timing, social competence, and cigarette use: A test of the early maturation hypothesis. *J Adolesc Health*. 2012; 51(2):150–5. [PubMed: 22824445]

20. Foshee VA, Ennett ST, Bauman KE, Granger DA, Benefield T, Suchindran C, et al. A test of biosocial models of adolescent cigarette and alcohol involvement. *The Journal of Early Adolescence*. 2007; 27(1):4–39.
21. van Jaarsveld CHM, Fidler JA, Simon AE, Wardle J. Persistent impact of pubertal timing on trends in smoking, food choice, activity, and stress in adolescence. *Psychosom Med*. 2007; 69(8):798–806. [PubMed: 17942841]
22. Copeland W, Shanahan L, Miller S, Costello EJ, Angold A, Maughan B. Outcomes of early pubertal timing in young women: A prospective population-based study. *Am J Psychiatry*. 2010; 167(10):1218–25. eng. [PubMed: 20478880]
23. Tanner-Smith EE. Negotiating the early developing body: Pubertal timing, body weight, and adolescent girls' substance use. *J Youth Adolesc*. 2010; 39(12):1402–16. [PubMed: 19967397]
24. Stice E, Presnell K, Bearman S. Relation of early menarche to depression, eating disorders, substance abuse, and comorbid psychopathology among adolescent girls. *Dev Psychol*. 2001; 37(5):608–19. [PubMed: 11552757]
25. Dick DM, Rose RJ, Viken RJ, Kapiro J. Pubertal timing and substance use: Associations between and within families across late adolescence. *Dev Psychol*. 2000; 36(2):180–9. [PubMed: 10749075]
26. Bratberg GH, Nilsen TI, Holmen TL, Vatten LJ. Sexual maturation in early adolescence and alcohol drinking and cigarette smoking in late adolescence: A prospective study of 2,129 Norwegian girls and boys. *Eur J Pediatr*. 2005; 164(10):621–5. [PubMed: 16012856]
27. Wiesner M, Ittel A. Relations of pubertal timing and depressive symptoms to substance use in early adolescence. *J Early Adolesc*. 2002; 22(1):5–23.
28. Wichstrom L. The impact of pubertal timing on adolescents' alcohol use. *J Res Adolescence*. 2001; 11(2):131–50.
29. Kaltiala-Heino R, Koivisto A, Marttunen M, Frojd S. Pubertal timing and substance use in middle adolescence: A 2-year follow-up study. *J Youth Adolesc*. 2011; 40(10):1288–301. [PubMed: 21533658]
30. Downing J, Bellis M. Early pubertal onset and its relationship with sexual risk taking, substance use and anti-social behaviour: A preliminary cross-sectional study. *BMC Public Health*. 2009; 9:446. [PubMed: 19958543]
31. Patton GC, McMorris BJ, Toumbourou JW, Hemphill SA, Donath S, Catalano RF. Puberty and the onset of substance use and abuse. *Pediatrics*. 2004; 114(3):300–6.
32. Biehl MC, Natsuaki MN, Ge X. The influence of pubertal timing on alcohol use and heavy drinking trajectories. *J Youth Adolesc*. 2007; 36(2):153–67.
33. Lanza ST, Collins LM. Pubertal timing and the onset of substance use in females during early adolescence. *Prev Sci*. 2002; 3(1):69–82. [PubMed: 12002560]
34. Costello EJ, Sung M, Worthman CM, Angold A. Pubertal maturation and the development of alcohol use and abuse. *Drug Alcohol Depend*. 2007; 88S:S50–S9-S-S9. [PubMed: 17275214]
35. Marklein E, Negriff S, Dorn LD. Pubertal timing, friend smoking, and substance use in adolescent girls. *Prev Sci*. 2009; 10(2):141–50. [PubMed: 19067163]
36. Graber JA, Seeley JR, Brooks-Gunn J, Lewinsohn PM. Is pubertal timing associated with psychopathology in young adulthood? *J Am Acad Child Adolesc Psychiatry*. 2004; 43(6):718–26. [PubMed: 15167088]
37. Silbereisen, RK.; Kracke, B.; Crockett, L. Timing of maturation and adolescent substance use. Paper presented at the biennial meeting of the Society for Research on Adolescence; Atlanta, GA. 1990;
38. Al-Sahab B, Ardern C, Hamadeh M, Tamin H. Age at menarche and current substance use among Canadian adolescent girls: Results of a cross-sectional study. *BMC Public Health*. 2012; 12(1): 195. [PubMed: 22424106]
39. Duke P, Carlsmith J, Jennings D, Martin J, Dornbusch SM, Gross RT, et al. Educational correlates of early and late sexual maturation in adolescence. *J Pediatr*. 1982; 100(4):633–7. [PubMed: 7062217]

40. Crockett, L.; Petersen, AC. Pubertal status and psychosocial development: Findings from the Early Adolescence Study. In: Lerner, R.; Foch, T., editors. *Biological-psychosocial interactions in early adolescence*. Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc; 1987. p. 173-88.
41. Jones M, Bayley N. Physical maturing among boys as related to behavior. *J Educ Psychol*. 1950; 41:129–48.
42. Graber, JA. Puberty in context. In: Hayward, C., editor. *Gender Differences at Puberty*. New York: Cambridge University Press; 2003.
43. Stattin, H.; Magnusson, D., editors. *Pubertal Maturation in Female Development*. Mahwah, New Jersey: Lawrence Erlbaum; 1990.
44. Richards M, Oinonen K. Age at menarche is associated with divergent alcohol use patterns in early adolescence and early adulthood. *J Adolesc*. 2011; 34:1065–76. [PubMed: 21115194]
45. Marti-Henneberg C, Vizmanos B. The duration of puberty in girls is related to the timing of its onset. *J Pediatr*. 1997; 131(4):618–21. [PubMed: 9386670]
46. Biro FM, Huang B, Crawford P, Lucky AW, Striegel-Moore R, Barton B, et al. Pubertal correlates in black and white girls. *J Pediatr*. 2006; 148(2):234–340. [PubMed: 16492435]
47. Cance JD, Ennett ST, Morgan-Lopez AA. The stability of perceived pubertal timing across adolescence. *J Youth Adolesc*. 2011; 41(6):764–75. [PubMed: 21983873]
48. Dubas JS, Graber JA, Petersen AC. The effects of pubertal development on achievement during adolescence. *Am J Educ*. 1991; 99(4):444–60.
49. Dorn LD, Sontag-Padilla L, Pabst SR, Tissot A, Susman EJ. Longitudinal reliability of self-reported age at menarche in adolescent girls: Variability across time and setting. *Dev Psychol*. 2012 Epub Aug 13, 2012.
50. Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. *J Youth Adolesc*. 1988; 17(2):117–32. [PubMed: 24277579]
51. Curran, PJ. A latent curve framework for the study of developmental trajectories in adolescent substance use. In: Rose, J.; Chassin, L.; Presson, C.; Sherman, J., editors. *Multivariate Applications in Substance Use Research*. Hillsdale, NJ: Erlbaum; 2000. p. 1-34.
52. Miyazaki Y, Raudenbush SW. Tests for linkage of multiple cohorts in an accelerated longitudinal design. *Psychol Methods*. 2000; 5(1):44–63. [PubMed: 10937322]
53. Jung T, Wickrama KAS. An introduction to latent class growth analysis and growth mixture modeling. *Soc Personal Psychol Compass*. 2008; 2(1):302–17.
54. Muthén, BO. Latent variable analysis: Growth mixture modeling and related techniques for longitudinal data. In: Kaplan, D., editor. *Handbook of Quantitative Methodology for the Social Sciences*. Newbury Park, CA: Sage Publications; 2004. p. 345-68.
55. Muthén, BO.; Muthén, LK. *MPlus User's Guide*. Los Angeles, CA: Authors; 2001.
56. Gold MS, Bentler PM, Kim KH. A comparison of maximum-likelihood and asymptotically distribution-free methods of treating incomplete nonnormal data. *Struct Equ Model*. 2003; 10(1): 47–79.
57. Bollen, KA.; Curran, PJ. *Latent Curve Models: A Structural Equation Perspective*. New York, NY: Wiley; 2006.
58. Skrondal, A.; Rabe-Hesketh, S. Entry for the *Encyclopedia of Statistics in Behavioral Sciences*. Wiley; 2005. p. 1-8.
59. Morgan-Lopez AA, Fals-Stewart W. Analytic methods for modeling longitudinal data from rolling therapy groups with membership turnover. *J Consult Clin Psychol*. 2007; 75(4):580–93. [PubMed: 17663612]
60. D'Amico E, McCarthy D. Escalation and initiation of younger adolescents' substance use: The impact of perceived peer use. *J Adolesc Health*. 2006; 39:481–7. [PubMed: 16982381]
61. Johnston, LD.; O'Malley, PM.; Bachman, JG.; Schulenberg, JE., editors. *Monitoring the Future national results on adolescent drug use: Overview of key findings, 2011*. Ann Arbor: Institute for Social Research, The University of Michigan; 2012.
62. Brooks-Gunn J, Petersen AC, Eichorn D. The study of maturational timing effects in adolescence. *J Youth Adolesc*. 1985; 14(3):149–61. [PubMed: 24301174]

63. Ge X, Natsuaki MN. In search of explanations for early pubertal timing effects on developmental psychopathology. *Curr Dir Psychol Sci*. 2009; 18(6):327–31.
64. Sisk CL, Foster DL. The neural basis of puberty and adolescence. *Nat Neurosci*. 2004; 7(10):1040–7. [PubMed: 15452575]
65. Caspi A, Moffitt TE. Individual differences are accentuated during periods of social change: The sample case of girls at puberty. *J Pers Soc Psychol*. 1991; 61(1):157–68. [PubMed: 1890586]
66. Dahl RE. Adolescent brain development: A period of vulnerabilities and opportunities. *Ann N Y Acad Sci*. 2004; 1021:1–22. [PubMed: 15251869]
67. Conley CS, Rudolph KD, Bryant F. Explaining the longitudinal association between puberty and depression: Sex differences in the mediating effects of peer stress. *Dev Psychopathol*. 2012; 24(2): 691–701. [PubMed: 22559140]
68. Tanner, JM. *Growth at Adolescence: With a General Consideration of Effects of Hereditary and Environmental Factors upon Growth and Maturation from Birth to Maturity*. Oxford, England: Blackwell Scientific Publications; 1962.
69. Dorn LD, Biro FM. Puberty and its measurement: A decade in review. *J Res Adolescence*. 2011; 21(1):180–95.
70. Dorn LD, Dahl RE, Woodward HR, Biro FM. Defining the boundaries of early adolescence: A user's guide to assessing pubertal status and pubertal timing in research with adolescents. *Appl Dev Sci*. 2006; 10(1):30–56.
71. Winters KC, Stinchfield RD, Henly GA, Schwartz RH. Validity of adolescent self-report of alcohol and other drug involvement. *Subst Use Misuse*. 1990; 25(11A):1379–95.
72. Pasch KEP, CL, Stigler MH, Komro KA. Sixth grade students who use alcohol: Do we need primary prevention programs for “tweens”? *Health Educ Behav*. 2009; 36(4):673–95. [PubMed: 18303109]

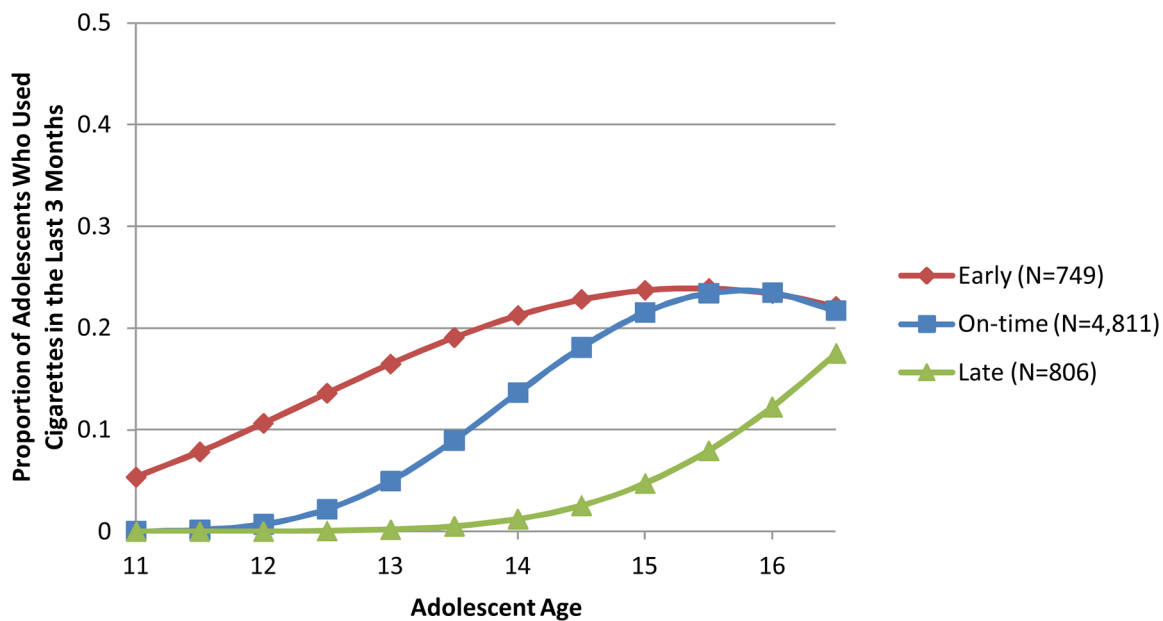


Figure 1. Proportion of adolescents (male and female) within each perceived pubertal timing latent class who endorsed using cigarettes in the past 3 months, measured across ages 11 to 17 (N=6,366)

Note: The sample sizes in each latent class are based on the most likely class membership.

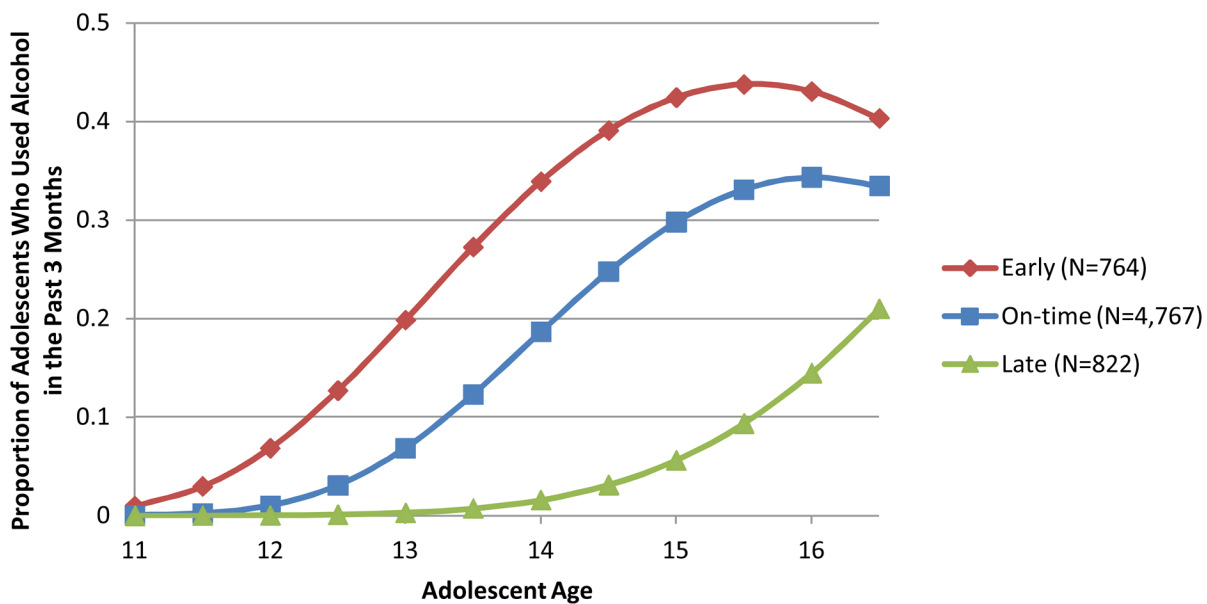


Figure 2.

Proportion of adolescents (male and female) within each perceived pubertal timing latent class who endorsed using alcohol in the past 3 months, measured across ages 11 to 17 (N=6,353)

Note: The sample sizes in each latent class are based on the most likely class membership.

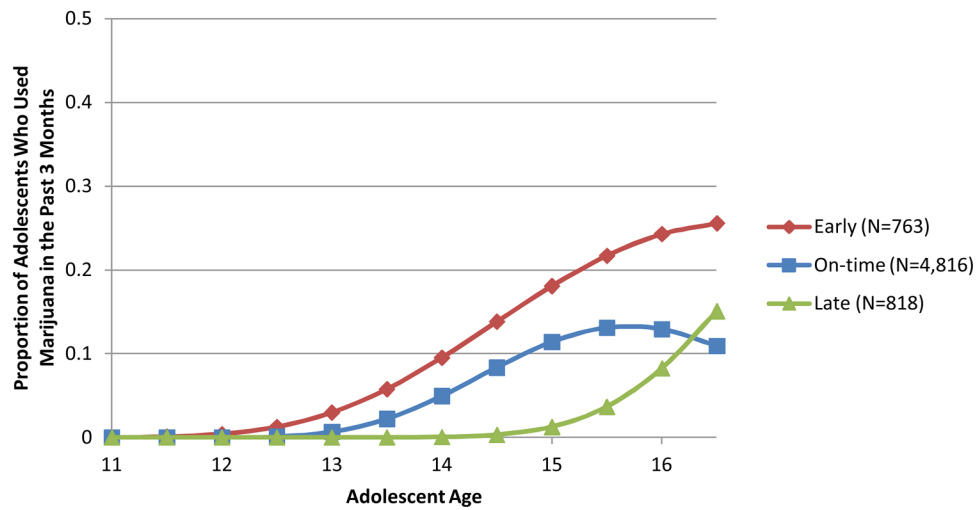


Figure 3.

Proportion of adolescents (male and female) within each perceived pubertal timing latent class who endorsed using marijuana in the past 3 months, measured across ages 11 to 17 (N=6,397)

Note: The sample sizes in each latent class are based on the most likely class membership.

Table 1

Means (M), standard deviations (SD), and proportions (%) of recent (past three month) cigarette, alcohol, and marijuana use, by age group, among male and female adolescents aged 11 to 17

| Age | Recent Cigarette Use | | | Recent Alcohol Use | | | Recent Marijuana Use | | |
|-------------|----------------------|---------------------|-------|--------------------|---------------------|-------|----------------------|---------------------|-------|
| | N | M (SD) ^a | % Any | N | M (SD) ^a | % Any | N | M (SD) ^b | % Any |
| 11 | 164 | .09 (.48) | 4.88 | 163 | .03 (.17) | 3.07 | 171 | .02 (.19) | 1.75 |
| 11.5 | 821 | .12 (.54) | 6.94 | 806 | .11 (.48) | 8.06 | 849 | .03 (.27) | 2.00 |
| 12 | 1535 | .19 (.73) | 9.51 | 1517 | .13 (.51) | 8.83 | 1571 | .05 (.33) | 2.99 |
| 12.5 | 2323 | .27 (.89) | 12.87 | 2318 | .22 (.70) | 13.76 | 2382 | .11 (.56) | 5.16 |
| 13 | 3107 | .40 (1.12) | 16.12 | 3082 | .30 (.87) | 16.61 | 3164 | .20 (.74) | 9.26 |
| 13.5 | 3532 | .47 (1.23) | 18.54 | 3504 | .38 (.94) | 20.46 | 3601 | .28 (.84) | 12.80 |
| 14 | 3531 | .62 (1.38) | 23.05 | 3505 | .49 (1.07) | 26.08 | 3593 | .39 (1.01) | 16.67 |
| 14.5 | 2880 | .80 (1.61) | 25.59 | 2869 | .63 (1.24) | 29.56 | 2952 | .48 (1.13) | 19.55 |
| 15 | 2199 | .89 (1.67) | 28.97 | 2199 | .70 (1.28) | 32.29 | 2268 | .58 (1.24) | 22.66 |
| 15.5 | 1468 | .96 (1.77) | 29.50 | 1465 | .79 (1.37) | 35.36 | 1503 | .60 (1.25) | 23.75 |
| 16 | 775 | 1.08 (1.89) | 30.84 | 779 | .86 (1.43) | 36.33 | 797 | .68 (1.31) | 26.85 |
| 16.5 | 183 | 1.36 (2.09) | 34.97 | 181 | .82 (1.48) | 33.15 | 189 | .74 (1.44) | 23.81 |

Note: The number of participants is greater than the total N due to the overlap in age groups in the three cohorts.

^a Range is 0=0 days to 5=20 or more days

^b Range is 0=none to 4=10 times or more

Table 2
Unconditional latent growth model fit statistics, by recent substance use outcome among male and female adolescents aged 11 to 17

| | Intercept only model | | | | | | Quadratic model | | | | | | |
|----------------------------|----------------------|-------|-------|--------|-------|-------|-----------------|--------|-------|-------|-------|--------|--------|
| | AIC | BIC | aBIC | LL | AIC | BIC | aBIC | LL | AIC | BIC | aBIC | LL | aLRT |
| Cigarettes (n=6366) | 18973 | 18986 | 18980 | -9484 | 18282 | 18316 | 18300 | -9136 | 18139 | 18200 | 18171 | -9060 | 167*** |
| Alcohol (n=6353) | 21022 | 21036 | 21029 | -10509 | 20176 | 20210 | 20194 | -10083 | 20051 | 20112 | 20083 | -10016 | 183*** |
| Marijuana (n= 6397) | 16039 | 16053 | 16046 | -8018 | 14971 | 15005 | 14989 | -7481 | 14791 | 14852 | 14824 | -7387 | 259*** |

p<.001

Note: AIC=Akaike Information Criteria, BIC=Bayesian Information Criteria, aBIC=adjusted Bayesian Information Criteria, LL=log-likelihood, aLRT=adjusted log-likelihood ratio test

Table 3

Fixed and random effects for the unconditional latent growth models, by recent substance use outcome among male and female adolescents aged 11 to 17

| | Cigarettes (n=6,366) | Alcohol (n=6,353) | Marijuana (n=6,397) |
|----------------|----------------------|-------------------|---------------------|
| Fixed effects | | | |
| Intercept | -5.987*** | -5.351*** | -9.193*** |
| Slope | 1.586*** | 1.516*** | 2.665*** |
| Quadratic | -0.166*** | -0.138*** | -0.268*** |
| Random effects | | | |
| Intercept | 10.631*** | 9.354*** | 14.584*** |
| Slope | 4.059*** | 4.075*** | 7.477*** |
| Quadratic | 0.104 | 0.080*** | 0.163** |

*
 $p < .05$,

**
 $p < .01$,

 $p < .001$

Note: When modeling dichotomous or ordinal data, MPLus uses thresholds as a corrective procedure. In order to have an identified model the thresholds were fixed to zero. The intercept is thus interpreted as the amount of deviation from 50 percent probability of the outcome.

Parameter estimates of recent substance use by substance use outcome and pubertal timing latent class among male and female adolescents aged 11 to 17

Table 4

| | Cigarettes (N=6,366) | | | Alcohol (N=6,353) | | | Marijuana (N=6,397) | | |
|----------------|------------------------|-----------------------|-----------------------|------------------------|-----------------------|-----------------------|-------------------------|-----------------------|-----------------------|
| | Intercept | Slope | Quadratic | Intercept | Slope | Quadratic | Intercept | Slope | Quadratic |
| Early | -1.611 ^{***o} | 0.412 ^o | -0.047 | -2.335 ^{***o} | 0.952 ^{***o} | -0.104 ^{**} | -3.577 ^{***ol} | 1.026 ^{**o} | -0.090 |
| On-time | -3.487 ^{***e} | 1.163 ^{***e} | -0.122 ^{***} | -3.378 ^{***e} | 1.180 ^{***e} | -0.117 ^{***} | -5.190 ^{***e} | 1.732 ^{***e} | -0.184 ^{***} |
| Late | -4.408 ^{***e} | 0.824 | -0.035 | -4.019 ^{***} | 0.672 | -0.016 | -7.504 ^{***e} | 1.688 | -0.093 |

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

eo Different superscripts indicate significant differences. e=different than early, o=different than on-time, l=different than late