Published in final edited form as:

Drug Alcohol Depend. 2012 November 1; 126(1-2): 95–101. doi:10.1016/j.drugalcdep.2012.04.018.

Peer substance use as a mediator between early pubertal timing and adolescent substance use: Longitudinal associations and moderating effect of maltreatment

Sonya Negri^{ff} and Penelope K. Trickett

University of Southern California, School of Social Work

Abstract

Background—Early pubertal timing has received considerable empirical support as a risk for adolescent substance use. However few studies have examined the mediators linking these variables. Therefore, the aims of this study were 1) to examine peer substance use as a mediator between pubertal timing and adolescent substance use longitudinally and 2) to test gender and maltreatment experience as moderators of the mediational model.

Methods—Data were obtained from Time 1, 2, and 3 of a longitudinal study of maltreatment and development. At Time 1 the sample was comprised of 303 maltreated and 151 comparison children aged 9–13 years (213 females and 241 males). Longitudinal mediation was tested using structural equation modeling and moderating effects were tested using multiple group analysis.

Results—Peer substance use mediated the relationship between early pubertal timing and later adolescent substance use for the total sample. Moderation analyses indicated this significant indirect effect did not differ for males and females. However, it did differ for maltreated versus comparison adolescents with the mediational effect only remaining significant for the comparison group.

Conclusions—This is one of the first studies to examine peer substance use as a mediator of pubertal timing and adolescent substance use using a longitudinal design. Early maturing males are at equal risk to early maturing females for interacting with peers that may draw them into substance use. Additionally, the findings indicate that while peers are mediators for comparison adolescents a different mechanism may link early puberty to substance use for maltreated adolescents.

Keywords

puberty; substance use; peers; maltreatment

© 2012 Elsevier Ireland Ltd. All rights reserved.

Corresponding author: Sonya Negri^{ff}, University of Southern California, MRF MC 0411, Los Angeles, CA 90089, negri^{ff}@usc.edu; 213-821-2206.

Contributors

Author 1 developed the rationale and hypotheses, carried out the analyses, and wrote the manuscript. Author 2 designed the parent study and protocol, advised on the analyses, and contributed to revisions of the manuscript.

Conflict of Interest

All authors declare they have no conflicts of interest.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

1. Introduction

Substance use is a substantial problem in adolescence as indicated by recent findings from the Monitoring the Future study showing widespread substance use among adolescents in the United States (Johnston et al., 2011). Early initiation of substance use in adolescence is a risk factor for later substance abuse and has been linked to other negative events such as motor vehicle accidents, drug abuse, unwanted pregnancies, depression, and suicide (Grant, 1998; Grant and Dawson, 1997; 1998). The timing of puberty, particularly early timing, is consistently related to the initiation and prolonged use of substances (Bratberg et al., 2007; Downing and Bellis, 2009; Ge et al., 2006; Westling et al., 2008). Although most studies find that early pubertal timing puts an adolescent at increased risk for substance use there is sparse evidence of the mechanisms by which early timing confers risk for substance use.

Theory regarding the potential mechanisms points to the peer group as a key mediator between early maturation and substance use. Perhaps the most widely accepted hypothesis is that early maturation influences the composition of the peer network, resulting in increased exposure to substance use (Stattin and Magnusson, 1990). This hypothesis was first developed and applied to girls; early maturing girls attract older peers who then expose them to experimentation with substances. Whether a similar mechanism operates for males is largely unknown.

There are several existing studies that support peers as a risk for early maturers. Using the Add Health data, Drapela and colleagues (2006) found an indirect effect of early timing on smoking through peers' smoking for boys. Wichstrom (2001) found the association between early puberty and substance use was partially mediated by friends' problem behavior. Additionally, more recent research revealed a significant partial mediation effect of deviant peers on the association between pubertal timing and substance use for girls but not boys (Westling et al., 2008). Thus, despite a number of studies that have investigated the influence of peers on the substance use of early maturing adolescents, there is still uncertainty as to whether the mediational effect may be conditional on gender. Additionally, there is a need to examine the mediational processes between early timing and substance use across adolescence to determine if the effects are transient lead to persistent, long term substance use.

1.1 Maltreatment

The contextual amplification hypothesis proposes that experiencing early pubertal transition in a disadvantaged context increases the risk for psychopathology (Ge and Natsuaki, 2009). Early pubertal development in itself is presumed to be distressing for adolescents and is proposed to be more deleterious when coupled with other stressful experiences. This is supported by evidence that variables such as neighborhood disadvantage, stressful life events, and deviant peer affiliation moderate the relationship between puberty and various psychosocial difficulties (Ge et al., 2001; Lynne et al., 2007; Obeidallah et al., 2004). Of particular relevance to the present study are the findings regarding stressful life events (Ge et al., 2001). Maltreatment is an exceptionally stressful experience that by itself increases the risk for substance use (Rogosch et al., 2010; Tonmyr et al., 2010). It stands to reason that the effects of early pubertal development may be amplified for an adolescent with experience of maltreatment (Compas et al., 1993; Rutter, 1991; Simmons et al., 1987). Thus, in the context of maltreatment the effects of pubertal timing may be more detrimental.

The contextual amplification hypothesis suggests that the link between early timing and substance using friends may be stronger for maltreated youth. However, whether the moderating effect of maltreatment experience is relevant to the link between substance-using peers and adolescent substance use has been untested. Several studies have shown that

negative peer networks are detrimental for maltreated adolescents. For abused adolescents, peer delinquency was found to increase the risk for antisocial/delinquent behavior (Perkins and Jones, 2004). Similarly, in a sample of physically abused adolescents, abused youth with delinquent close friends were at highest risk for antisocial and delinquent behavior (Salzinger et al., 2007). Whether there are similar effects regarding substance use is unknown.

Other evidence suggests that for maltreated youth perhaps the influence of deviant or substance-using peers is less important than the adolescent's abuse experiences for the development of substance use or delinquency. For example, Salzinger and colleagues (2007) found that delinquent peers did not mediate the association between abuse and violent delinquency in adolescence. This demonstrates the need to delineate the specific conditional effects in the proposed mediational model for maltreated versus comparison adolescents.

Therefore, the aims of this study were 1) to examine peer substance use as a mediator between pubertal timing and later substance use and 2) to test gender and maltreatment as moderators of the mediational relationship. Understanding the mechanisms that underlie the association between pubertal timing and adolescent substance use is critical for informing prevention and intervention effort targeting substance use problems. Additionally, investigating the mechanisms for maltreated children, who are at higher risk for substance use problems, will provide significant information about vulnerable youth.

2. Research Design and Methods

2.1 Participants

The present study used data from the first three assessments (approximately 1 year apart) of an ongoing longitudinal study examining the effects of maltreatment on adolescent development. The sample was comprised of 454 adolescents (303 maltreated; 151 comparison) aged 9–13 years (Time 1 mean age=10.93, SD=1.16) with 241 males and 213 females. Sample characteristics (age, gender, ethnicity, caretaker) for Time 1 to Time 3 for the maltreatment and comparison groups can be found in Table 1. The maltreatment group ($n = 303$) were recruited from active cases in the Children and Family Services (CFS) of a large west coast city. The inclusion criteria were: (1) a new substantiated referral to CFS in the preceding month for any type of maltreatment (e.g. neglect, physical abuse, sexual abuse); (2) child age of 9–12 years; (3) child identified as Latino, African-American, or Caucasian (non-Latino); (4) child residing in one of 10 zip codes in a designated county at the time of referral to CFS. With the approval of CFS and the Institutional Review Board of the affiliated university, potential participants were contacted via postcard and asked to indicate their willingness to participate. Contact via mail was followed up by a phone call. Of the families referred by CFS, 77% agreed to participate.

The comparison group ($n=151$) was recruited using names from school lists of children aged 9–12 years residing in the same 10 zip codes as the maltreated sample to ensure the comparison group would have similar neighborhood experiences as the maltreatment group. Inclusion criteria included the child being within 9–12 years of age, identified as Latino, African-American, or Caucasian (non-Latino), and no involvement with CFS. Caretakers of potential participants were sent a postcard and asked to indicate their interest in participating which was followed up by a phone call. Approximately 50% of the comparison families contacted agreed to participate.

2.2 Group Comparisons

Upon enrollment in the study, the maltreatment and comparison groups were compared on a number of demographic variables. The two groups were similar on age, ($M = 10.93$ years,

$SD = 1.16$), gender (53% male), race (38% African American, 39% Latino, 12% Biracial, and 11% Caucasian), and neighborhood characteristics (based on Census block information; Trickett, Mennen, Negriiff, & Horn, 2011). However, they were different in terms of living arrangements. In the comparison group 93% lived with a biological parent, whereas this was the case for only 52% of the maltreatment group. The remainder of the maltreatment group was living in foster care, which is not unusual for those adolescents involved with social services. Of the maltreated group 27% were still living with the perpetrator of the referred abuse. Sample characteristic can be found in Table 1.

2.3 Attrition

The attrition rate was 13.4% ($n=61$) from Time 1 to Time 2 and 31% ($n=141$) from Time 1 to Time 3. The results of attrition analyses indicated that the participants who were not seen at Time 2 were more likely to be in the maltreatment group ($OR=4.38$, $p<.01$; Nagelkerke's $R^2=.10$) and those not seen at Time 3 were more likely to be Latino ($OR=3.37$, $p<.01$) and in the maltreatment group ($OR=5.36$, $p<.01$; Nagelkerke's $R^2=.19$).

2.4 Procedures

Assessments were conducted at an urban research university. After assent and consent were obtained from the adolescent and caretaker, respectively, the adolescent was administered questionnaires and tasks during a four-hour protocol. The measures used in the following analyses represent a subset of the questionnaires administered during the protocol, which also included hormonal, cognitive, and behavioral measures. Both the child and caretaker were paid for their participation according to the guidelines of the National Institutes of Health Healthy Volunteer Program.

2.5 Measures

2.5.1 Pubertal Development

Tanner Stages: Pubertal stage was measured using the adolescent's self-report on his/her stage of pubertal development using Tanner criteria (Marshall and Tanner, 1969; 1970). Five stages of pubertal development are represented by sets of serial line drawings that depict the development of two different secondary sexual characteristics from prepubertal (stage = 1) to postpubertal (stage = 5; Morris and Udry, 1980). Female drawings are of breast development and pubic hair growth; male drawings are of genital development and pubic hair growth. Self-report on Tanner stages is highly correlated with physician assessment and sufficient when approximate estimation of pubertal stage is adequate (Dorn et al., 1990). Scores on each drawing (breast/genital and pubic hair) were used as separate indicators of pubertal development.

Pubertal Development Scale: The Pubertal Development Scale (PDS) is a measure of the physical changes associated with pubertal development. It was developed as an alternative to physician rating measures and has been shown to have adequate reliability and validity (Petersen et al., 1988). On a 4-point scale ranging from 1 (has not yet started) to 4 (has completed), each subject is asked to indicate the level of development on each of the physical changes. Five items were used for both males and females (height spurt, body hair, skin changes, breast growth/deepening of voice, menarche/facial hair; $\alpha = .61$ for males, $\alpha = .76$ for females). A coding system developed by Shirtcliff and colleagues (2009) was used to convert the PDS scores to a 5-point scale to parallel the Tanner stages. Other studies have found similar reliability coefficients for the PDS (Ge et al., 2003; Petersen et al., 1988; Stroud and Davila, 2008), although it should be noted that in this sample the reliability for males was lower than for females.

2.5.2 Pubertal Timing—When the degree of physical development is standardized within same-age peers, the resulting score can be used as an index of pubertal timing (Ge et al., 2001). For the present study the scores on each of the Time 1 Tanner stage ratings (breast/genital and pubic hair) and the PDS scores were standardized within each age cohort (e.g., 9, 10, 11) and gender. The resulting z-scores for each of the three measures had a mean of 0 and a standard deviation of 1 with higher scores indicating earlier maturation relative to peers. The three scores were retained separately to be used as indicators for a latent variable of pubertal timing.

2.5.3 Substance use—The adolescent reported on their own substance use within the past 12 months via two items from the Adolescent Delinquency Questionnaire (ADQ; adapted from Huizinga and Elliott, 1986). Computerized administration was used to ensure participants' confidentiality. The number of times the adolescent used alcohol (0 to five or more) and the number of times they used marijuana were used as indicators of their substance use at Time 1, 2, and 3. Smoking was obtained by this measure but not used for analyses because it was a different metric (yes/no) than the other two substances.

2.5.4 Peer Substance Use—The adolescent reported on the substance use of their peers within the past 12 months via two items from the ADQ. Similar to the adolescent self-report, they were asked "how many of your friends or people your age you know have used alcohol in the past 12 months." Answer options were 0=none, 1=some, 2=a lot. A second item asked about peers' marijuana use.

3. Data Analysis

Structural equation models were used to test the hypotheses using Amos 18.0 (Arbuckle, 2007). Latent variables were constructed for pubertal timing (three manifest indicators: z-scores of Tanner breast/genital, Tanner pubic hair, and PDS), peer substance use (two manifest indicators: alcohol use and marijuana use), and adolescent substance use (two manifest indicators: alcohol use and marijuana use). Pubertal timing was only included at Time 1, peer and adolescent substance use variables were included at Time 1, 2, and 3. A cross-lagged model was tested which included direct effects from all Time 1 variables to all Time 2 variables and Time 2 to Time 3 variables. Additionally, the model included covariances between disturbance terms within time and autoregressive effects across time. Two models were tested, the first excluded the direct effect of Time 1 pubertal timing on Time 3 substance use and the second included this direct effect. The models were first tested with the total sample and then multiple group models were used to test gender and maltreatment experience as moderators. The one degree of freedom nested chi square difference test was used to determine if any parameters in the model were significantly different between groups. Covariates in all models were age at Time 1, gender, race (minority, non-minority), and maltreatment status (maltreated, comparison). Except in the case of the multiple-group models were either gender or maltreatment was the moderator and thus not included as a covariate.

Full Information Maximum Likelihood estimation method (FIML; Arbuckle, 1996) was employed to handle variable level missingness and longitudinal missingness across time. This procedure does not impute data but breaks down the likelihood function into components based on patterns of missing data, allowing estimation to proceed using all available data. Fit indices, such as the χ^2 (chi-square) *goodness-of-fit* statistic, the root mean square error of approximation (RMSEA) and comparative fit index (CFI), were used to evaluate the fit of the model to the data. Significant mediation effects were tested using MacKinnon et al.'s distribution of products (MacKinnon et al., 2002).

4. Results

4.1 Descriptive Statistics

Table 2 summarizes the means and standard deviations of the puberty, adolescent- and peer-substance use variables by maltreatment status and gender using the raw scores. Significant differences between groups were examined using independent samples t-test. Males had significantly higher scores than females on the Tanner stages and T1 alcohol use but females scored higher on the PDS and T3 peer alcohol use ($p < .01$). Maltreated adolescents scored significantly higher on T1 and T2 marijuana use and T1 peer marijuana use whereas comparison adolescents scored higher on T3 peer alcohol use ($p < .01$).

4.2 Measurement Models

Prior to examining the structural parameters, measurement models were examined for all latent variables using the total sample. All models were fully saturated and thus a perfect fit. Factor loadings for Time 1 pubertal timing were .44 for PDS, .77 for Tanner breast/genital, and .82 for Tanner pubic hair. For peer substance use, factor loadings for Time 1 were .84 and .68, for Time 2 were .78 and .73, and for Time 3 were .87 and .83 for alcohol and marijuana, respectively. For adolescent substance use, factor loadings for Time 1 were .65 and .76, for Time 2 were .71 and .83, and for Time 3 were .83 and .54 for alcohol and marijuana, respectively.

4.3 Mediation Models with the Total Sample

The first model fit the data well ($\chi^2 = 211.70$ (128), RMSEA=.038; CFI=.95). When compared to the model that included the direct effect of Time 1 pubertal timing on Time 3 substance use, there was not an improvement in model fit ($\chi^2 = 210.80$ (127), RMSEA=.038, CFI=.95) and there was not a significant direct effect of Time 1 pubertal timing on Time 3 substance use. Therefore, the results from the first model were selected. The results showed that peer substance use at Time 2 mediated the relationship between pubertal timing at Time 1 and substance use at Time 3 ($p < .01$). Time 1 pubertal timing predicted Time 2 peer-substance use ($\beta = .18$, $p < .01$) after accounting for the effects of Time 1 peer- and adolescent substance use (see Figure 1). In turn, Time 2 peer substance use predicted Time 3 adolescent substance use ($\beta = .25$, $p < .05$) after accounting for Time 2 peer- and adolescent substance use. There was no relationship between peer substance use at Time 1 and adolescent substance use at Time 2 and a trend relationship ($\beta = .16$, $p = .05$) between adolescent substance use at Time 1 and peer substance use at Time 2. The model explained 15.3% of the variance in substance use at Time 3. All factor correlations and autoregressive effects between the latent variables estimated in the model were statistically significant. Although not of primary interest for the aims, there was also a significant mediation effect from Time 1 substance use to Time 2 peers substance use to Time 3 adolescent substance use ($p < .01$).

4.4 Moderated Mediation

4.4.1 Gender as a Moderator—Using the same model as tested for the total sample, multiple-group analysis was used to examine moderating effect of gender. Measurement invariance was tested by imposing equality constraints on all the measurement parameters. Next, each mediational path was in turn fixed to be equal across gender and the model fit was compared to the freely estimated model. No significant differences were found when restricting the mediational paths to equivalence (T1puberty to T2 peer $\Delta\chi^2/\Delta df = 0/1$, ns; T2 peer to T3 substance use $\Delta\chi^2/\Delta df = .04/1$, ns), indicating that gender did not moderate the mediational relationships.

4.4.2 Maltreatment Experience as a Moderator—As in the previous analysis, measurement invariance was tested by imposing equality constraints. The mediation paths were then fixed to be equal across groups and compared to the model in which they were freely estimated. There was a significant difference between these models and subsequent tests restricting each structural parameter in turn indicated that maltreatment experience moderated the relationship between peer substance use at Time 2 and adolescent substance use at Time 3 ($\Delta\chi^2/\Delta df=5.86/1$, $p < .05$). The relationship was significant for the comparison group ($\beta=.45$, $p<.01$) but not significant for the maltreated group ($\beta=.04$, ns).

5. Discussion

The present study investigated the developmental mechanisms linking early pubertal timing to adolescent substance use. Although there is considerable evidence that demonstrates the risk of early pubertal maturation on substance use, few studies have investigated longitudinal relationships or mediators. This study sought to elucidate the role of peers in the development of substance use for early maturing adolescents while simultaneously testing moderating effects of gender. The current results supported a full mediation effect of peer substance use for the both boys and girls. Although this is consistent with some studies (Lynne et al., 2007; Wichstrom, 2001) it is counter to other studies that only find mediating effects for either males or females (Drapela et al., 2006; Westling et al., 2008).

Theoretical explanations for the influence of peers on early maturers centers on the proposal that early maturers attract older friends who are more likely to be using substances. In the present study the age of substance-using peers was not obtained and thus cannot be discounted as a possible mechanism. However, in a previous analysis with the current sample (Negriiff, Susman, & Trickett, 2011) having more older friends did not mediate between early timing and delinquency. Thus, it may not be the age of the peer group but the actual risk behavior of the peers that is integral for this mediating effect. Inclusion of both substance-using peers and older friends may aid in delineating this mechanism. However, based on the current findings, early maturers report more substance-using friends, which then increases the adolescent's subsequent substance use. The fact that this mediational relationship is not found to differ for males and females contributes to our knowledge about these developmental relationships. In the past, females have been the primary focus of the pubertal timing literature based on the assumption that they are at higher risk. However, the evidence is growing that males are similarly harmed by early maturation and should not be discounted.

These results also demonstrate longitudinal relationships from pubertal timing in early adolescence to substance use in mid/late adolescence. There was no direct effect of early timing on Time 2 adolescent substance use, which bolsters support for peer influence effects. Additionally, the indirect effect of early timing through peer substance use on substance use in later adolescence provides evidence of the longitudinal effects of early maturation. These findings are concomitant with several other studies that find long term effects of early timing on substance use (Biehl et al., 2007; Hayatbakhsh et al., 2009). However, this is the first study to demonstrate longitudinal mediation effects of early puberty on later substance use through peers' substance use.

Another important contribution of this study was the inclusion of maltreatment as a moderator. The findings indicated that the association between peer substance use and adolescent substance use was only significant for the comparison group, and in the expected direction (i.e. having more substance-using peers was related to more subsequent substance use). An implication of this finding is that for maltreated adolescents, their traumatic experience may be disrupting typical mediational processes. That is, for maltreated youth,

substance-using peers may not be integral to the development of substance use for early maturers. Although these results help us understand the mechanisms that link early timing to substance use for comparison adolescents, there clearly needs to be further investigation of the mechanisms for maltreated adolescents.

Several limitations of this study should be noted. First, the measures of substance use and peer substance use were self-report which could introduce bias. Perhaps adolescents who use substances are more likely to perceive that their peers are also using. However, due to the cross-lagged design we were able to account for previous substance use and the shared method variance when estimating the mediating effects. This contributes to our confidence that we are capturing the mechanistic processes. The substance use variables (both peer and adolescent) were skewed, with low means and variance. Despite the restricted variance we were still able to observe significant associations. Even transformation of variables that are highly skewed will not create a normal distribution; thus we accounted for this in our estimation algorithm. Lastly, there should be a note about the developmental appropriateness of the cross-lagged design by timepoint. There was a range of 9–13 years at Time 1 which may encompass different developmental stages. Creating the model by timepoint may not account for the possible developmental differences in these various ages. However it is our belief that for the purpose of the present analysis the cross-lagged design was best for investigating the moderated mediation hypotheses. In an age-cohort based design the number of parameters would have been unwieldy for testing moderating effects via multiple-group models. Age was included as a covariate and in doing so corrects for some possible age-related differences.

The results of this study provide evidence of a longitudinal mediation model from early pubertal timing to adolescent substance use through exposure to substance-using peers. It is important to note that although this mediational model did not vary for males and females, it did vary for maltreated adolescents. Future research should continue to consider traumatic experience such as maltreatment when developing models to understand the developmental pathways to adolescent substance use. Incorporating more specific information about the age, gender and behaviors of peers will allow us to determine the characteristics of peers that increase risk for substance use in early maturing adolescents. Overall, this study adds to our knowledge of the etiology of adolescent substance use. Understanding the specific risk factors for substance use as well as developmental mechanisms will facilitate more effective intervention efforts for at-risk adolescents.

Acknowledgments

Role of Funding Source

This research was supported by funding from a National Institutes of Health R01 grant HD39129, Penelope K. Trickett P.I. The NIH had no further role in study design; in the collection, analysis and interpretation of data; in the writing of the report; or in the decision to submit the paper for publication.

References

- Arbuckle, J.L. Full information estimation in the presence of incomplete data. In: Marcoulides, G.A.; Schumacker, R.E., editors. *Advanced Structural Equation Modeling*. Lawrence Erlbaum; Mahwah, NJ: 1996. p. 243-277.
- Arbuckle, J.L. Amos 18.0. Amos Development Corporation; Crawfordville, FL: 2007.
- Biehl MC, Natsuaki MN, Ge X. The influence of pubertal timing on alcohol use and heavy drinking trajectories. *J Youth Adolesc*. 2007; 36:153–167.
- Bratberg GH, Nilsen TI, Holmen TL, 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 Paediatrica*. 2007; 96:292–295. doi: 210.1111/j.1651-2227.2007.00102.x. [PubMed: 17429923]
- Compas BE, Ey S, Grant KE. Taxonomy, assessment, and diagnosis of depression during adolescence. *Psychol Bull*. 1993; 114:323–344. [PubMed: 8416035]
- Dorn LD, Susman EJ, Nottelmann ED, Inoff-Germain G, Chrousos GP. Perceptions of puberty: adolescent, parent, and health care personnel. *Dev Psychol*. 1990; 26:322–329.
- Downing J, Bellis MA. Early pubertal onset and its relationship with sexual risk taking, substance use and antisocial behaviour: a preliminary cross-sectional study. *BMC Public Health*. 2009; 9:446. [PubMed: 19958543]
- Drapela LA, Gebelt JL, McRee N. Pubertal development, choice of friends, and smoking initiation among adolescent males. *J Youth Adolesc*. 2006; 35:717–727.
- Ge X, Conger RD, Elder GH. Pubertal transition, stressful life events and the emergence of gender differences in adolescent depressive symptoms. *Dev Psychol*. 2001; 37:404–417. doi: 410.1037/0012-1649.1037.1033.1404. [PubMed: 11370915]
- Ge X, Jin R, Natsuaki MN, Gibbons FX, Brody GH, Cutrona CE, Simons RL. Pubertal maturation and early substance use risks among African American children. *Psychol Addict Behav*. 2006; 20:404–414. [PubMed: 17176175]
- Ge X, Kim IJ, Brody GH, Conger RD, Simons RL, Gibbons FX, Cutrona CE. It's about timing and change: pubertal transition effects on symptoms of major depression among African American youths. *Dev Psychol*. 2003; 39:430–439. doi:410.1037/0012-1649.1039.1033.1430. [PubMed: 12760513]
- Ge X, Natsuaki NM. In search of explanations for early pubertal timing effects on developmental psychopathology. *Curr Dir Psychol Sci*. 2009; 18:327–331. doi: 310.1111/j.1467-8721.2009.01661.x.
- Grant BF. Age at smoking onset and its association with alcohol consumption and DSM-IV alcohol abuse and dependence: results from the National Longitudinal Alcohol Epidemiologic Survey. *J Subst Abuse*. 1998; 10:59–73. [PubMed: 9720007]
- Grant BF, Dawson DA. Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: results from the National longitudinal alcohol epidemiologic survey. *J Subst Abuse*. 1997; 9:103–110. [PubMed: 9494942]
- Grant BF, Dawson DA. Age of onset of drug use and its association with DSM-IV drug abuse and dependence: results from the National Longitudinal Alcohol Epidemiologic Survey. *J Subst Abuse*. 1998; 10:163–173. [PubMed: 9854701]
- Hayatbakhsh MR, Najman JM, McGee TR, Bor W, O'Callaghan MJ. Early pubertal maturation in the prediction of early adult substance use: a prospective study. *Addiction*. 2009; 104:59–66. [PubMed: 18983626]
- Huizinga D, Elliott DS. Reassessing the reliability and validity of self-report delinquency measures. *J Quant Criminol*. 1986; 2:293–327.
- Johnston, LD.; O'Malley, PM.; Bachman, JG.; Schulenberg, JE. Secondary School Students. Vol. I. Institute for Social Research, The University of Michigan; Ann Arbor: 2011. Monitoring the Future National Survey Results on Drug Use, 1975–2010.
- Lynne SD, Graber JA, Nichols TR, Brooks-Gunn J, Botvin GJ. Links between pubertal timing, peer influences, and externalizing behaviors among urban students followed through middle school. *J Adolesc Health*. 2007; 40:181, e187–181.e113. doi:110.1016/j.jadohealth.2006.1009.1008. [PubMed: 17259062]
- MacKinnon DP, Lockwood CM, Hoffman JM, West SG, Sheets V. A comparison of methods to test mediation and other intervening variable effects. *Psychol Methods*. 2002; 7:83–104. [PubMed: 11928892]
- Marshall WA, Tanner JM. Variations in patterns of pubertal changes in girls. *Arch Dis Child*. 1969; 44:291–303. doi:210.1136/adc.1144.1235.1291. [PubMed: 5785179]
- Marshall WA, Tanner JM. Variations in patterns of pubertal changes in boys. *Arch Dis Child*. 1970; 45:13–23. doi:10.1136/adc.1145.1239.1113 [PubMed: 5440182]
- Morris NM, Udry RJ. Validation of a self-administered instrument to assess stage of adolescent development. *J Youth Adolesc*. 1980; 9:271–280.

- Negriff S, Susman EJ, Trickett PK. The developmental pathway from pubertal timing to delinquency and sexual activity from early to late adolescence. *Journal of Youth and Adolescence*. 2011; 40(10):1343–1356. [PubMed: 21191640]
- Obeidallah D, Brennan RT, Brooks-Gunn J, Earls F. Links between pubertal timing and neighborhood contexts: implications for girls' violent behavior. *J Am Acad Child Adolesc Psychiatry*. 2004; 43:1460–1468. doi: 1410.1097/1401.chi.0000142667.0000152062.0000142661e. [PubMed: 15564815]
- Perkins DF, Jones KR. Risk behaviors and resiliency within physically abused adolescents. *Child Abuse Neglect*. 2004; 28:547–563. [PubMed: 15159069]
- Petersen AC, Crockett LJ, Richards M, Boxer A. A self-report measure of pubertal status: reliability, validity, and initial norms. *J Youth Adolesc*. 1988; 17:117–133.
- Rogosch FA, Oshri A, Cicchetti D. From child maltreatment to adolescent cannabis abuse and dependence: a developmental cascade model. *Devel Psychopathol*. 2010; 22:883–897. [PubMed: 20883588]
- Rutter, M. Age changes in depressive disorders: some developmental considerations. In: Rutter, M.; Izard, CE.; Read, PB., editors. *Depression in Young People: Developmental and Clinical Perspectives*. Guilford Press; New York: 1991.
- Salzinger S, Rosario M, Feldman RS. Physical child abuse and adolescent violent delinquency: the mediating and moderating roles of personal relationships. *Child Maltreat*. 2007; 12:208–219. [PubMed: 17631621]
- Shirtcliff EA, Dahl RE, Pollak SD. Pubertal development: correspondence between hormonal and physical development. *Child Dev*. 2009; 80:327–337. [PubMed: 19466995]
- Simmons RG, Burgeson R, Carlton-Ford S, Blythe DA. The impact of cumulative change in early adolescence. *Child Dev*. 1987; 58:1220–1234. [PubMed: 3665641]
- Stattin, H.; Magnusson, D. *Pubertal Maturation in Female Development*. Vol. 2. Lawrence Erlbaum Associates; Hillsdale, NJ: 1990. *Paths Through life*.
- Stroud CB, Davila J. Pubertal timing and depressive symptoms in early adolescents: The roles of romantic competence and romantic experiences. *J Youth Adolesc*. 2008; 37:953–966. doi: 910.1007/s10964-10008-19292-10969.
- Tonmyr L, Thornton T, Draca J, Wekerle C. A review of childhood maltreatment and adolescent substance use relationship. *Curr Psychiatry Rev*. 2010; 6:223–234.
- Trickett, PK.; Mennen, FE.; Negriff, S.; Horn, JL. *The USC Young Adolescent Project: Sampling strategy, sample description and comparisons of neighborhood characteristics for maltreated versus comparison participants (Technical Report)*. Los Angeles, CA: University of Southern California, School of Social Work; 2011.
- Westling E, Andrews JA, Hampson SE, Peterson M. Pubertal timing and substance use: the effects of gender, parental monitoring and deviant peers. *J Adolesc Health*. 2008; 42:555–563. [PubMed: 18486864]
- Wichstrom L. The impact of pubertal timing on adolescents' alcohol use. *J Res Adolesc*. 2001; 11:131–150.

$\chi^2 = 211.70$ (128); RMSEA = .04; CFI = .95
 ** $p < .01$, * $p < .05$, † $p < .10$

Figure 1.

Longitudinal relationships between pubertal timing, peer substance use, and adolescent substance use.

All parameter estimates are standardized. Bold paths show significant mediation effects at $p < .05$. Dashed paths were included in the final model but were not significant.

Table 1

Sample Characteristics For Time 1, 2, and 3

Demographic Variable	Group					
	Maltreated			Comparison		
	Time 1	Time 2	Time 3	Time 1	Time 2	Time 3
N	303	250	191	151	142	128
Age (std deviation)	10.84 (1.15)	12.02 (1.21)	13.85 (1.48)	11.11 (1.15)	12.28 (1.26)	13.57 (1.38)
Gender (%)						
Male	50	48	46	60	60	57
Female	50	52	54	40	40	43
Ethnicity (%)						
African American	40	40	47	32	32	34
Latino	35	36	29	47	45	43
White	12	11	8	10	11	11
Mixed Biracial	13	13	16	11	12	12
Living Arrangement (%)						
With Parent	52	63	62	93	94	95
Foster Care or Extended Family						

Table 2

Means and standard deviations for study variables

	<u>Comparison</u>		<u>Maltreated</u>		<i>p</i>	<u>Boys</u>		<u>Girls</u>		<i>p</i>
	Mean	SD	Mean	SD		Mean	SD	Mean	SD	
<i>T1 Puberty</i>	n=151		n=301			n=240		n=212		
Tanner breast/genital	2.09	0.84	2.06	0.89	ns	2.26	0.92	1.85	0.76	<i>p</i> <.01
Tanner pubic hair	2.15	0.92	2.12	1.01	ns	2.26	0.98	1.99	0.96	<i>p</i> <.01
PDS	1.97	0.61	1.94	0.68	ns	1.85	0.57	2.07	0.72	<i>p</i> <.01
<i>T1 Substance use</i>	n=149		n=290			n=237		n=209		
Alcohol	.09	.52	.14	.65	ns	.11	.58	.14	.65	ns
Marijuana	.01	.08	.09	.59	<i>p</i> <.01	.11	.66	.00	.07	<i>p</i> <.01
<i>T1 Peer-substance use</i>	n=149		n=290			n=237		n=209		
Alcohol	.39	.61	.45	.71	ns	.41	.66	.46	.69	ns
Marijuana	.16	.45	.35	.64	<i>p</i> <.01	.32	.63	.25	.54	ns
<i>T2 Substance use</i>	n=142		n=246			n=203		n=185		
Alcohol	.13	.54	.21	.77	ns	.17	.71	.19	.67	
Marijuana	.01	.08	.11	.60	<i>p</i> <.01	.07	.53	.07	.44	ns
<i>T2 Peer-substance use</i>	n=142		n=246			n=203		n=185		
Alcohol	.54	.75	.43	.70	ns	.44	.70	.50	.74	
Marijuana	.37	.65	.40	.68	ns	.38	.65	.41	.69	ns
<i>T3 Substance use</i>	n=119		n=176			n=150		n=146		
Alcohol	.65	1.49	.40	1.10	ns	.40	1.11	.60	1.42	ns
Marijuana	.38	1.20	.40	1.29	ns	.37	1.23	.41	1.28	ns
<i>T3 Peer-substance use</i>	n=119		n=176			n=150		n=146		
Alcohol	.89	.83	.70	.78	<i>p</i> <.01	.66	.78	.90	.81	<i>p</i> <.01
Marijuana	.67	.82	.63	.81	ns	.59	.82	.70	.81	ns

Note: significant mean differences between groups tested using t-test