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Elucidating the mechanisms linking early pubertal timing, sexual activity, and substance use for maltreated versus nonmaltreated adolescents

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Abstract

Purpose—To test models linking pubertal timing, peer substance use, sexual behavior, and substance use for maltreated versus comparison adolescents. Three theoretical mechanisms were tested: 1) peer influence links early pubertal timing to later sexual behavior and substance use, 2) early maturers engage in substance use on their own and then select substance-using friends, or 3) early maturers initiate sexual behaviors which leads them to substance-using peers.

Methods—The data came from a longitudinal study of the effects of child maltreatment on adolescent development (303 maltreated and 151 comparison adolescents; age: 9–13 years at initial wave). Multiple-group structural equation models tested the hypotheses across three timepoints including variables of pubertal timing, perception of peer substance use, sexual behavior, and self-reported substance use.

Results—Early pubertal timing was associated with substance-using peers only for maltreated adolescents, indicating the mediation path from early pubertal timing through substance-using peers to subsequent adolescent substance use and sexual behavior only holds for maltreated adolescents. Mediation via sexual behavior was significant for both maltreated and comparison

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Implications and Contribution

Early pubertal timing is associated with substance-using peers only for maltreated adolescents, whereas sexual behavior links early maturation with risky friends regardless of adverse life experiences. These findings are a step toward elucidating the pathways from early puberty to risk behavior and identifying early experiences that may alter mediation effects.

adolescents. This indicates that sexual behavior may be a more universal mechanism linking early maturation with risky friends regardless of adverse life experiences.

Conclusions—The findings are a step toward elucidating the developmental pathways from early puberty to risk behavior and identifying early experiences that may alter mediation effects.

Keywords

pubertal timing; substance use; sexual activity; maltreatment

Introduction

There is overwhelming evidence that early pubertal timing is a risk factor for a variety of psychological and behavioral problems, including early sexual activity and substance use [1-3]. Similarly, studies show that maltreated adolescents engage in substance use and risky sexual activity more than nonmaltreated adolescents [4, 5]. Early puberty can be stressful and confusing, and for youth with maltreatment experiences these stressors may be exacerbated, potentially strengthening associations between early puberty and problem behaviors. However, as of yet, there is incomplete evidence delineating the specific mediators linking early puberty to risk behavior for either maltreated or nonmaltreated adolescents. It is likely there are multiple mechanisms, operating singularly or in combination, that link early pubertal timing with risk behavior (e.g. sexual activity and substance use). Elucidating the pathways leading from puberty to sexual activity and substance use is integral for identifying strategic targets in the delivery of prevention programs [6].

The overarching theory guiding much of the work on the effects of pubertal timing has been termed the maturation disparity hypothesis [7]. This theory states that early maturers will encounter new stressors and environments before they are cognitively and emotionally prepared, thereby increasing vulnerability to psychopathology. Increases in risk behavior may be due to early maturers' exposure to deviant peers or as a coping response to the mismatch between physical and social maturity. The peer influence mechanism is supported by evidence showing that exposure to deviant peers, especially older or male peers, increases the delinquent behavior of early maturing girls [8]. Additionally, Drapela et al.[9] found that peers' smoking was a mediator between early timing and smoking in a two wave study of adolescent boys, whereas Wichstrom [10] found the association between early puberty and substance use to be mediated by peer problem behavior across two time points. More recent research revealed a longitudinal mediation effect of deviant peers on the association between pubertal timing and substance use for girls but not boys [11]. These mediation effects have only been investigated with normative samples, and the comparison of peer influence processes for early-maturing maltreated versus nonmaltreated adolescents is limited. Research shows strong links between maltreatment and risk behavior, but whether these associations are due to peer influence is not clear. For example, in a cross-sectional study Perkins and Jones [12] found that physically abused adolescents who perceived their peers to be engaged in negative behaviors (i.e. substance, trouble in school) were more likely to use alcohol, drugs, and engage in sexual activity. Conversely, other research

showed that exposure to delinquent peers was not as significant in the development of delinquency for maltreated adolescents versus comparison adolescents [13].

Alternatively, selection effects may perpetuate the risk behavior of early maturers. Previous theory about selection processes posits that risk behaviors are initiated in an effort to cope with the stress of early puberty, as well as to reconcile the disparity between social and physical maturity. This implicates the adolescent as an active participant in the selection of deviant peers [14]. However, it may be that the hormones linked with pubertal development, particularly androgenic hormones such as testosterone, may be the initial impetus for sexual interest, leading early maturers to seek out sexual partners, thereby exposing them to risky peers [15, 16]. Thus, the selection of risky peers may be initiated via hormonal drive rather than cognitions about reconciling their maturational disparity or as a coping mechanism. Evidence shows that early maturing females report more sexual motivation and exposure to sexual content in the media [17] while early maturing adolescent boys reported downloading and viewing pornography on the internet more than on-time and late maturers [18]. Hormonal changes are also responsible for the physical developments that may affect an adolescent's selection of peers or behavioral patterns [19]. Although maltreated youth are at higher risk for early sexual debut and sexual risk behavior [4], there is little evidence that they initiate normative sexual behaviors at an earlier age. Therefore, sexual behavior may link early puberty to deviant peers and/or substance use for both maltreated and nonmaltreated adolescents. There are still substantial gaps in the literature regarding the mechanisms linking early puberty with risk behavior; more importantly, there is little knowledge on how these mediation effects may be moderated by maltreatment.

The Current Study

The purpose of this study was to test mediation models linking pubertal timing, peer substance use, sexual behavior, and substance use for maltreated versus comparison adolescents. The peer influence model hypothesizes that early puberty leads directly to association with deviant peers and then to sexual behavior and substance use. On the other hand, the selection model has two possible pathways: a) early puberty leads to substance use and then association with deviant peers, or b) early puberty leads to increased sexual behavior and then to deviant peers and subsequent substance use. It is not hypothesized that these mediation models are mutually exclusive as it is possible that selection and peer influence are reciprocal processes. We hypothesize that selection effects will operate via increased sexual interest which will lead early maturers to sexual/romantic partners that may also be conduits for exposure to more advanced risk behavior.

Regarding maltreatment as a potential moderator, there is evidence that peers may influence both maltreated and nonmaltreated early maturers. Maltreatment is well-known to be associated with higher rates of risk behavior thus we expect that early-maturing maltreated youth may be more likely to engage in risk behavior on their own and then affiliate with other deviant peers (i.e., selection). Peer-influence effects may be more salient for comparison youth who are less likely to engage in risk behavior of their own volition. Sexual behavior may be a more universal mechanism whereby both maltreated and comparison youth become exposed to risky peers.

Research Design and Methods

Participants

Data were from the first three assessments of an ongoing longitudinal study examining the effects of maltreatment on adolescent development. At Time 1 (T1), the sample was composed of 454 adolescents aged 9–13 years (241 males and 213 females). Time 2 (T2) and Time 3 (T3) occurred approximately 1 and 2.5 years after baseline. The attrition rate between T1 and T2 was 13.8% ($n=63$) and between T1 and T3 was 29% ($n=132$). Participants not seen at T2 were more likely to be in the maltreatment group ($OR = 4.38, p < .01$), and those not seen at T3 were more likely to be Latino ($OR = 3.37, p < .01$) and in the maltreatment group ($OR = 5.36, p < .01$). Descriptives of the sample for all three timepoints can be found in Table 1.

Recruitment—The maltreatment group ($n = 303$) was 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, or emotional abuse); 2) child age of 9–12 years; 3) child identified as Latino, African-American, or Caucasian (non-Latino); and 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 (IRB) of the affiliated university, potential participants were contacted and asked their willingness 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. With IRB approval, caretakers of potential participants were contacted and asked to indicate their interest in participating. Comparison families were run through the CFS database to ensure they had no previous or ongoing experience with child welfare agencies. Unreported maltreatment could not be accounted for.

Procedures

Assessments were conducted at an urban research university and approved by the university IRB. After assent and consent were obtained from the youth and caretaker, respectively, questionnaires were administered in a standardized protocol. The measures used in this study represent a subset of the questionnaires in the full protocol. Both child and caretaker were compensated according to the National Institutes of Health standard rate for healthy volunteers.

Measures

Pubertal Development

Tanner stages—Five stages of pubertal development are represented by sets of serial line drawings that depict the development of two different secondary sexual characteristics (breast growth/pubic hair for females and genital development/pubic hair for males) from prepubertal (stage = 1) to postpubertal (stage = 5) [20]. Scores on each drawing (breast/genital and pubic hair) were used as separate indicators of pubertal development.

Pubertal Development Scale (PDS)—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 five physical changes associated with pubertal development (height spurt, body hair, skin changes, breast growth/deepening of voice, menarche/facial hair) [21]. The PDS scores were converted to a 5-point scale to parallel the Tanner stages [22].

Pubertal Timing

The scores on each of the T1 Tanner stage ratings (breast/genital and pubic hair) and the PDS scores were standardized within each age cohort (e.g., 9, 10, and 11) and gender to create an index of timing [23]. The resulting *z*-score for each measure had a mean of 0 and a *standard deviation* of 1, with higher scores indicating earlier maturation relative to peers.

Substance Use

The computerized Adolescent Delinquency Questionnaire (ADQ; adapted from [24]) assessed how many times the adolescent had used alcohol and marijuana (separately) in the past 12 months (0 times, 1, 2, 3, 4, 5 or more times). The separate scores on alcohol use and marijuana use were used as indicators of the adolescent's substance use at T1, T2, and T3.

Peer Substance Use

Participants reported on the perceived substance use of their peers within the past 12 months via two items from the ADQ. They were asked, "How many of your friends have used alcohol in the past 12 months?" Answer options were 0 = *none*, 1 = *some*, 2 = *a lot*. A second item asked about peer marijuana use.

Sexual Behavior

Sexual behavior was measured using the Sexual Activity Questionnaire [25]. This questionnaire asks about 11 sexual behaviors beginning with holding hands, then kissing or heavy petting, and lastly sexual intercourse (no = 0, yes = 1). The 11 items were summed to create a composite score of sexual behavior, with higher scores indicating more advanced sexual behavior.

Data Analysis

Structural equation models were used to test the hypotheses using Amos 18.0 [26]. Latent variables were constructed for Time 1 pubertal timing (three manifest indicators: *z*-score Tanner breast/genital, *z*-score Tanner pubic hair, and *z*-score PDS), T1, T2, T3 peer substance use, and T1, T2, T3 adolescent substance use (two manifest indicators each: alcohol and marijuana use). Sexual behavior was included as a manifest variable (T1, T2, T3). The cross-lagged model included direct effects from all T1 variables to all T2 variables and all T2 to T3 variables, as well as covariances among disturbance terms within time and autoregressive effects across time. Covariates in the model were age at T1, gender, and race (minority or non-minority). Full Information Maximum Likelihood (FIML) estimation method [27] was used to handle variable-level and longitudinal missingness. Due to skewness of the substance use variables, the model was also run using the Asymptotic Distribution Free (ADF) estimation method, which does not assume a normal distribution.

The pattern of significant parameter estimates was similar to the FIML and because ADF does not allow missing data, the results from the FIML are reported. The substance use variables were transformed using a square-root transformation for the FIML estimation to correct for the non-normality. Overall, an adequate model fit is indicated by a small χ^2 , RMSEA of .08 or smaller, and CFI above .90[28]. Significant mediation effects were tested using Mackinnon et al.'s [29] product of coefficients.

Multiple group models (MGSEM) were used to test maltreatment status as a moderator of the hypothesized mediation effects[30]. After factorial invariance between the maltreated and comparison groups was obtained for all latent variables, an unrestricted model (parameters free to vary between groups) was tested. Then sequential constraint of the individual direct effects relevant to the mediation hypotheses was imposed (requiring the parameter to be equal in both groups). The parameters tested were: 1) T1 pubertal timing to T2 peer substance use, 2) T1 pubertal timing to T2 substance use, 3) T1 pubertal timing to T2 sexual behavior, 4) T2 peer substance use to T3 substance use, 5) T2 peer substance use to T3 sexual behavior, 6) T2 sexual behavior to T3 peer substance use, 7) T2 sexual behavior to T3 substance use, 8) T2 substance use to T3 peer substance use. The one degree of freedom-nested χ^2 difference test was used to determine moderation effects. A significant decrement in model fit between the less-restricted versus more-restricted model indicated that specific parameter estimate was significantly different between groups.

Results

Descriptive Statistics

Mean differences by maltreatment status were tested for all variables in the model using an independent samples *t*-test and Mann-Whitney U test (Table 2). Maltreated adolescents reported more sexual behavior and peer marijuana use at T1 ($p < .05$) and more marijuana use at T2 ($p < .05$). Comparison adolescents reported more peer alcohol use at T3 ($p < .05$).

Substantive Analyses

Direct effects—The full model fit the data adequately ($\chi^2 = 285.64$ [166]; CFI = .94; RMSEA = .04). All significant parameter estimates are shown in Table 3. The MGSEM showed that maltreatment moderated only one parameter in the model. The direct effect of T1 pubertal timing on T2 peer substance use ($\chi^2/df = 3.84/1$, $p = .05$) was only significant for the maltreated group ($\beta = .18$, $p < .05$). Unmoderated significant direct effects can be found in Table 3.

Mediation effects—Mediation effects are provided in Figure 1. Due to the moderation effect of the parameter between T1 pubertal timing and T2 peer substance use, there emerged two moderated mediation effects. First, there was a mediation effect from T1 pubertal timing to T2 peer substance use to T3 sexual behavior ($z = 4.57$, $p < .01$). The second moderated mediation effect was from T1 pubertal timing to T2 peer substance use to T3 substance use ($z = 4.49$, $p < .01$). Both of these mediation effects were only significant for the maltreated group. There were also several mediation effects that were significant for both groups. First, there was a mediation effect from T1 pubertal timing to T2 sexual behavior to

T3 peer substance use ($z = 4.83, p < .01$). There were also significant mediation effects from T1 substance use to T2 peer substance use to T3 sexual behavior ($z = 4.60, p < .01$), T1 substance use to T2 peer substance use to T3 adolescent substance use ($z = 4.72, p < .01$), and T1 sexual behavior to T2 adolescent substance use to T3 peer substance use ($z = 5.17, p < .01$).

Discussion

The present study sought to advance our understanding of the mechanisms that lead from early puberty to risk behavior for maltreated versus nonmaltreated adolescents by testing an integrated biopsychosocial model. Specifically, three theoretical mechanisms were tested: 1) peer influence links early pubertal timing to later sexual activity and substance use, 2) early maturers engage in substance use on their own and then select substance-using friends, or 3) early maturers initiate sexual behaviors which leads them to substance-using peers. Although several significant mediation paths were identified within the total sample, moderation analyses uncovered that maltreatment alters some mediation effects.

The indirect effect from early pubertal timing through substance-using peers to subsequent adolescent substance use and sexual behavior only holds for maltreated adolescents, a finding that needs replication and should be interpreted cautiously. This is a unique finding because few studies have examined mediators between early puberty and problem behaviors for adolescents with a history of maltreatment. The literature shows that maltreated youth have difficulty forming healthy peer relationships, are likely to be habitually rejected, and form more antisocial peer ties [31]. Thus, the association between early puberty and substance-using peers is not unexpected, albeit somewhat contrary to previous research that found mediation effects via substance-using peers in normative samples [9-11]. Based on the existing evidence, the assumption was that pathways involving peer-influence effects would also emerge for comparison adolescents. However, perhaps because substance-using peers may be considered antisocial, maltreated youth are more likely to affiliate with them. It should be noted that there was no evidence for the first hypothesized selection mechanism; early pubertal timing was not directly related to substance use. Although the substance use literature has numerous examples of selection effects, there is scant evidence that selection is a relevant developmental mechanism for early maturers, and the present findings are congruent with the lack of empirical support for this theory. Instead, the findings show that selection may operate via sexual interest which is a more introductory avenue toward advanced risk behavior.

Although early maturation was a risk for maltreated adolescents being exposed to substance using peers, sexual behavior was a significant mediator for both maltreated and comparison adolescents. This indicates that sexual behavior may be a more universal mechanism linking early maturation with risky friends regardless of adverse life experiences. Early maturers may initiate sexual behaviors at a younger age, which then seems to be a risk for exposure to substance-using peers. However, the current analysis cannot determine whether this effect may continue from substance-using peers to subsequent substance use. The sequence of sexual behavior preceding exposure to peer substance use and adolescent substance use provides support for the possible hormonal initiation of the engagement in and exposure to

risk behavior. Although several studies have found early pubertal maturation linked with increased sexual interest and motivation [17, 18], they did not posit it as a mechanism by which early maturers are selecting friends that may increase exposure to substance use. The results of the current study are not able to definitively link the hormones of puberty with sexual behavior, but the findings do provide some initial support for this possible mechanism.

Limitations

It should be noted that the measures of sexual behavior, 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, studies show that perceptions of peer use are better predictors of adolescent use than the peers' actual substance use [32, 33]. The substance use variables (both peer and adolescent) had low means and variance. However, despite the restricted variance and increased risk of type 2 errors, we observed significant associations. Most variables were limited to less than three indicators which may have implications for the estimation of structural parameters. We were also unable to determine if the sexual partners are substance users or if exposure to substance-using peers is occurring through another process. Although there is evidence that maltreatment, particularly sexual abuse, is a risk for early pubertal timing [34], in the current study, there was no significant association between the two variables. Therefore, although a model from maltreatment to early puberty to sexual behavior and substance use might be expected based on theory and extant evidence, this is not supported by the current data. This may be due to the low rates of sexual abuse in our sample (13%) or to convoluted associations due to high rates of co-occurring maltreatment types (70%). We also could not account for unreported maltreatment that may have occurred in the comparison group. An additional limitation of this study is that we could not directly tie higher levels of sexual behavior to increased androgenic hormones or sexual motivation. However, there is substantial evidence that links adrenarche and the related hormones to the initiation of sexual attraction [35]. Finally, it may be an important distinction to examine these models by gender and maltreatment simultaneously. However, because of sample size limitations and the complexity of the model, there was not adequate power to perform a four-group MGSEM.

Conclusions

In summary, the results support a model in which both sexual behavior and substance-using peers may initiate a risk trajectory for early maturing youth. Additionally, the moderating effects of maltreatment demonstrate that there may be multiple pathways to risk behavior depending on early environmental experiences. The accumulating evidence demonstrating the importance of peers in the development of substance use has significant implications for prevention efforts. In identifying early maturers at higher risk for sexual behavior and exposure to deviant peers, prevention programs might intervene in preadolescence to curtail a deleterious trajectory. Previous research shows that early sexual debut is a risk for substance use, as well as long-term risky sexual behavior [36, 37]. The findings from the present study demonstrate that early puberty increases the risk of early sexual behavior (although, not necessarily intercourse); thus, prevention programs should take pubertal

development into account to be most effective. Early sex education (before sexual initiation) has been found to reduce risky sexual behavior [38]; therefore, sex education programs should be focused on children close to the initiation of puberty to effectively reduce the associated risk behaviors and problematic trajectories from early to late adolescence and possibly into adulthood. Lastly, this study identified pathways of risk for early-maturing maltreated youth. There is evidence that the same genes that influence the timing of puberty also predict risk behavior and that environmental experience may modify the expression of these genes[39]. Particular attention should be paid to this vulnerable population with efforts to increase the positive socialization of these youth.

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Figure 1.

Mediation effects in full cross-lagged model. All parameter estimates are standardized. * $p < .05$; ** $p < .01$; † $p < .08$. Covariates (not shown) are T1 age, race, gender. Bold paths show significant mediation effects at $p < .05$. Dotted path shows moderated mediation effects. Grayed-dotted paths show significant covariances and autoregressive effects.

Table 1

Sample characteristics for Times 1, 2, and 3

Demographic variable	Group					
	Maltreated			Comparison		
	Time 1	Time 2	Time 3	Time 1	Time 2	Time 3
<i>N</i>	303	249	193	151	142	129
Age (STD deviation)	10.84 (1.15)	12.01 (1.15)	13.59 (1.36)	11.11 (1.15)	12.28 (1.26)	13.59 (1.36)
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	48	37	38	7	6	5

Table 2

Means and standard deviations for study variables

	<u>Comparison</u>		<u>Maltreated</u>		
	Mean	SD	Mean	SD	<i>p</i>
<i>T1 Puberty</i>					
Tanner breast/genital	2.09	0.84	2.06	0.89	ns
Tanner pubic hair	2.15	0.92	2.12	1.01	ns
PDS	1.97	0.61	1.94	0.68	ns
<i>T1 Sexual Behavior</i>	0.55	1.06	1.01	1.59	<i>p</i> <.01
<i>T1 Substance use</i>					
Alcohol	0.09	0.52	0.14	0.65	ns
Marijuana	0.01	0.08	0.09	0.59	<i>p</i> <.01
<i>T1 Peer-substance use</i>					
Alcohol	0.39	0.61	0.45	0.71	ns
Marijuana	0.16	0.45	0.35	0.64	<i>p</i> <.01
<i>T2 Sexual Behavior</i>	1.13	1.82	1.50	2.27	ns
<i>T2 Substance use</i>					
Alcohol	0.13	0.54	0.21	0.77	ns
Marijuana	0.01	0.08	0.11	0.60	<i>p</i> <.01
<i>T2 Peer- substance use</i>					
Alcohol	0.54	0.75	0.43	0.70	ns
Marijuana	0.37	0.65	0.40	0.68	ns
<i>T3 Sexual Behavior</i>	3.45	2.91	3.95	3.13	ns
<i>T3 Substance use</i>					
Alcohol	0.65	1.49	0.40	1.1	ns
Marijuana	0.38	1.20	0.40	1.29	ns
<i>T3 Peer- substance use</i>					
Alcohol	0.89	0.83	0.70	0.78	<i>p</i> <.01
Marijuana	0.67	0.82	0.63	0.81	ns

Note: significant mean differences between groups tested using t-test; puberty scores are stage not timing scores; substance use means reflect raw data (before transformation)

Table 3
Significant parameter estimates for the full cross-lagged model

<i>Correlations</i>		
Time 1	<i>r</i>	<i>p</i>
Pubertal timing ↔ Peer substance use	0.13	*
Substance use ↔ Peer substance use	0.37	**
Substance use ↔ Sexual behavior	0.26	**
Peer substance use ↔ Sexual behavior	0.24	**
Time 2		
Substance use ↔ Peer substance use	0.51	**
Substance use ↔ Sexual behavior	0.3	**
Peer substance use ↔ Sexual behavior	0.35	**
Time 3		
Substance use ↔ Peer substance use	0.63	**
Substance use ↔ Sexual behavior	0.32	**
Peer substance use ↔ Sexual behavior	0.4	**
<i>Auto-regressive effects</i>		
	β	<i>p</i>
T1 Substance use → T2 Substance use	0.18	**
T2 Substance use → T3 Substance use	0.17	†
T1 Peer substance use → T2 Peer substance use	0.45	**
T2 Peer substance use → T3 Peer substance use	0.5	**
T1 Sexual Behavior → T2 Sexual behavior	0.44	**
T2 Sexual Behavior → T3 Sexual behavior	0.51	**
<i>Direct effects (unmoderated)</i>		
T1 Pubertal timing → T2 Sexual behavior	0.14	**
T1 Substance use → T2 Peer substance use	0.18	*
T1 Sexual behavior → T2 Substance use	0.15	**
T2 Substance use → T3 Peer substance use	-0.22	*
T2 Peer substance use → T3 Substance use	0.17	**
T2 Peer substance use → T3 Sexual activity	0.22	*
T2 Sexual behavior → T3 Peer substance use	0.12	†
<i>Squared multiple correlations</i>		
	R^2	
T2 Substance use	0.06	
T2 Peer substance use	0.22	
T2 Sexual behavior	0.28	
T3 Substance use	0.17	
T3 Peer substance use	0.34	
T3 Sexual behavior	0.37	
<i>Moderated direct effects</i>		
	Maltreatment	Comparison
T1 Pubertal timing → T2 Peer Substance use	.18*	ns

<i>Mediation effects (unmoderated)</i>	<i>z</i>	<i>p</i>
T1 Pubertal timing → T2 Sexual behavior → T3 Peer substance use	4.83	**
T1 Substance use → T2 Peer substance use → T3 Substance use	4.6	**
T1 Substance use → T2 Peer substance use → T3 Sexual behavior	4.72	**
T1 Sexual behavior → T2 Substance use → T3 Peer substance use	5.17	**
<i>Moderated mediation effects</i>	<u>Maltreatment group only</u>	
	<i>z</i>	<i>p</i>
T1 Pubertal timing → T2 Peer substance use → T3 Sexual behavior	4.57	**
T1 Pubertal timing → T2 Peer substance use → T3 Substance use	4.49	**

*
 $p < .05$

**
 $p < .01$

†
 $p < .08$

^a Nonsignificant effects not included. Covariates were Time 1 age, gender, race (minority/nonminority); T1=Time 1, T2=Time 2, T3=Time 3.