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Adult Substance Use as a Function of Growth in Peer Use Across Adolescence and Young Adulthood in the Context of ADHD: Findings from the MTA

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Abstract

Peer substance use strongly predicts adolescent and young adult substance use, but its role in ADHD-related risk for substance use, especially in adulthood, is unclear. In a sample with (n=516) and without (n=249) childhood ADHD from the Multimodal Treatment Study of ADHD, we compared associations between change over time in peer substance use and personal substance use (alcohol, cigarettes, marijuana, illicit drugs) from age 14–26 by ADHD status. Developmentally typical peer substance use trajectories across adolescence and young adulthood coincided with similar changes in personal use – but less so for those with ADHD histories. Concurrent

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associations between peer and personal use in adolescence and young adulthood were weaker for those with ADHD histories than without for commonly used substances (alcohol, marijuana). Prospectively, escalating peer use during adolescence forecasted adulthood declines for commonly used substances, yet persistently high substance use at age 25, regardless of ADHD history. In the reverse direction, growth in adolescent substance use predicted developmentally normative young adult declines in peer use – but for the ADHD group, adolescent heavy drinking predicted *increases* in young adult peer use. Findings suggest that individuals with ADHD may have difficulty emulating their peers' developmentally normative declines in substance use, highlighting the importance of social factors when treating young adults affected by ADHD and substance abuse.

Keywords

ADHD; substance use; adolescence; young adulthood; peers; longitudinal

Introduction

Attention-deficit/hyperactivity disorder (ADHD) confers risk for substance use in adolescence and adulthood (Lee, Humphreys, Flory, Liu, & Glass, 2011; Molina et al., 2018). Given long-term difficulties with social functioning, (peer rejection, deviant peer affiliation; Hoza, 2007; Molina et al., 2009), the social context of substance use may play an important role in this ADHD-substance use link (Molina & Pelham, 2014). Specifically, peer substance use a strongly influences substance use outcomes in adolescence and young adulthood (Chassin, Pitts, & Prost, 2002; Fergusson et al., 2008; Neighbors et al., 2007; White et al., 2014), when most use occurs with peers (Chassin, Colder, Hussong, & Sher, 2013; Creswell, Chung, Clark, & Martin, 2014). This robust link reflects both socialization (influence by substance-using peers) and selection (affiliation with substance-using peers; Curran, Stice, & Chassin, 1997).

Adolescents/young adults with ADHD may be especially susceptible to peer influences. For example, social rejection (Hoza, 2007) may increase their desire for acceptance from substance-using peers (Belendiuk et al., 2016). In the Pittsburgh ADHD Longitudinal Study, perceived peer use was cross-sectionally associated with heavy alcohol use, alcohol problems, and illicit drug use more strongly for adolescents with childhood ADHD (probands) than for typically developing controls (M_{age}=15.2; Marshal, Molina, & Pelham, 2003). In the same sample, prospective *growth* in peer alcohol use and adolescents' own alcohol use were more strongly correlated among probands than controls (Belendiuk et al., 2016). Thus, these processes *change together over time*, especially for youth with ADHD – suggesting *how* adolescents with ADHD reach atypically high levels of use. However, this study was limited to alcohol use. Given that individuals with ADHD disproportionately use other substances (Molina et al., 2018), a critical step is to examine associations between peer-personal substance use trajectories for several substance use outcomes.

Further, it is important to identify whether associations between peer and personal substance use continue into young adulthood for individuals with ADHD. One cross-sectional study

showed that peer substance use mediated the link between ADHD symptoms and substance use among college students (M_{age}=20.2; Van Eck, Markle, Dattilo, & Flory, 2014). The authors postulated that college students with ADHD may overestimate their peers' alcohol use due to misinterpreting social cues (Flory et al., 2006), leading them to increase their own drinking to gain peer acceptance. However, many young adults with ADHD histories do not attend college (Hechtman et al., 2016), necessitating study beyond the college population.

Understanding co-occurring changes in peer and personal substance use from adolescence into young adulthood requires a prospective, longitudinal design spanning these developmental periods. Substance use peaks in young adulthood (SAMHSA, 2014); however, as responsibilities increase and unstructured time decreases toward the mid-twenties, substance use may begin to detach from peer contexts.

Current Study

Using the Multimodal Treatment Study of ADHD (MTA) follow-up, in which substance use trajectories increased across adolescence then remained stable through young adulthood (Molina et al., 2018), we primarily aimed to compare co-occurring and prospective associations between change over time in peer and personal substance use for individuals with versus without childhood ADHD. Hypotheses are: (1) both peer and personal substance use will increase during adolescence and remain stable during young adulthood; (2) growth in peer use will predict growth in personal use, and vice versa, concurrently and prospectively; and, of primary interest, (3) these relations will be stronger for individuals with versus without ADHD histories.

Material and Methods

Sample and Procedure

The MTA was a 14-month randomized trial of ADHD treatment that continued as a naturalistic longitudinal study through 16 years post-baseline. Participants included 579 children diagnosed with *DSM-IV* ADHD–Combined Type at baseline (per parent-report Diagnostic Interview Schedule for Children and teacher ratings) and 258 age- and sex-matched children without ADHD in the Local Normative Comparison Group (LNCG; 31 with ADHD excluded from analyses). ADHD probands ages 7.0–9.9 (M=8.5, SD=.80) were recruited from schools, pediatricians, mental health practitioners/clinics, family-based referrals, and advertisements at seven sites in the U.S. and Canada (Arnold et al, 1997; Hinshaw et al., 1997), and randomly assigned to receive medication management, behavior therapy, their combination, or community care. Assessments occurred during treatment and at 2, 3, 6, 8, 10, 12, 14, and 16 years post-baseline. LNCG participants, recruited 2 years after ADHD baseline, underwent the same assessment schedule. Details of the MTA recruitment strategy, inclusion/exclusion criteria, treatment, and sample are described elsewhere (Arnold et al.; MTA Cooperative Group, 1999). This study includes data from ages 14–26, spanning assessments 3 years (M_{age}=14.24, *Range*=14.00–15.19) to 16 years (M_{age}=24.83, *Range*=21.69–26.95) post-baseline.

Among ADHD probands, 89% were reassessed at least once between the 3- and 16-year assessments and were retained for analysis (Swanson et al., 2017 Table 1). Substance use data were provided at least once in young adulthood (12, 14, and/or 16 years post-baseline) by 81.3% of probands (n=471/579) and 93% of LNCG (n=240/258). Final sample sizes were *n*=516 ADHD and *n*=249 LNCG. Participants with versus without complete data did not significantly differ on most baseline sociodemographics. Non-completers were more often characterized by: younger biological mothers, male sex, racial minority, low income, less educated parents (ADHD and LNCG); fewer fathers with mental health histories (ADHD); and receipt of welfare (LNCG).

Measures

Substance Use.—On the Substance Use Questionnaire (Molina & Pelham, 2003), participants reported frequency of alcohol, marijuana, cigarette, and illicit/prescription drug (mis)use. At 2- through 10-year assessments, past 6-month frequency was scored from 1 (*never*) to 9 (*>2x/week*). Beginning with the 12-year assessment, when participants were >18, past-year frequency was scored from 1 (*not at all*) to 12 (*>several times/day*). The two response scales were harmonized.

As in previous reports (Molina et al., 2018), we defined substance use variables to be optimally developmentally relevant across the age range examined here based on expert internal review of the literature. *Heavy drinking* (alcohol) reflected the highest frequency between binge drinking (five or more drinks) and drunkenness; *marijuana use* was assessed with a single item. Responses to each substance were coded on a four-point scale: 0 (*none*), 1 (*<monthly*), 2 (*at least monthly, <weekly*), 3 (*weekly or more*). *Daily smoking* (nicotine) was a binary variable (0=no, 1=yes) coded 1 through the 10-year assessment if at least one cigarette/day on an average day during the past month, and at 12–16 years if participants smoked “once a day” or more in the past year, *or* if they responded affirmatively to, “Are you currently a daily cigarette smoker?” *Other illicit drug* use was maximum frequency of any illicit drug used (heroin, inhalants, hallucinogens, cocaine, other substances to get high) or prescription drug misused (stimulants, sedatives, opioids). Responses were coded on a three-point scale: 0 (*none*), 1 (*<once/month*), and 2 (*≥once/month*).

Peer Substance Use.—The Peer Substance Use Questionnaire (Johnston, O’Malley, & Bachman, 1988) assessed participants’ perceptions of their friends’ use of alcohol, cigarettes, marijuana, and other drugs at all assessments except 12-year (M_{age}=20.90, Range=17.65–25.04). The composite scale averaged 6 items at assessments before age 18, plus five additional items (totaling eleven) at age 18 and older. The stem was, “How many of your friends would you estimate…” Items before age 18 included “smoke marijuana or hashish occasionally” and “regularly”, “drink alcohol occasionally” and “regularly”, and “take drugs other than marijuana occasionally” and “regularly”. Additional items after age 18 included five or more drinks and getting drunk once or twice each weekend, three or more times a week, and smoking cigarettes regularly. Responses ranged from 1 (*none*) to 6 (*all*). Cronbach’s alphas=.89 (age 17) to .94 (age 20).

Covariates.—Baseline demographic covariates included: sex, race, parental income, research site, and household (dis)advantage represented by two weighted effects codes – one comparing children from two-parent households and at least one college-educated parent to the sample average (*household advantage*), and the other comparing children from single-parent households and no college-educated parent to the sample average (*household disadvantage*). Analyses controlled for the proportion of time between ages 14–26 that participants were in college/university and that they lived at home with parents/caregivers/relatives.

Analytic Plan

We first estimated growth trajectories separately for peer substance use ("peer use") and personal use ("substance use") of each substance (heavy drinking, daily smoking, marijuana, illicit drugs) between ages 14–26 using generalized linear multilevel modeling for ordered categorical outcomes. Age was centered at 25, the mean age of the sample at the 16-year assessment. Visual data inspection showed that both peer use and substance use increased steadily across adolescence before leveling off in the early twenties. Preliminary unconditional growth models using competing functional forms were compared (linear, quadratic, 2-piece and 3-piece linear with inflection points at varying ages). Based on model fit statistics and successful convergence, a piecewise linear function (Flora, 2008) with an inflection point at age 21 was selected for all outcomes, modeling rates of change through adolescence (age 14–21; *adolescent slope*) and young adulthood (age 21–26; *adult slope*). We retained factor scores representing the adolescent slope, adult slope, and age 25 intercept from each univariate growth model. Four bivariate path analyses assessed relations among peer use trajectory growth factors and each substance use growth factor, adjusting for demographic covariates[1] (Figure 1). Concurrent relations between peer use and substance use (i.e., adolescent growth, adult growth, and intercepts) were estimated as covariances; all other relations were regressed. Finally, we sequentially constrained each model parameter to be equal between ADHD and LNCG to test group differences. We constrained each growth factor mean, followed by variances, then each covariance, each regression estimate, and lastly, all covariates simultaneously. Model comparisons yielding significant chi-square difference tests reflect a decrement in model fit, suggesting the parameters constrained differ between ADHD and LNCG. Analyses were conducted in M*plus* (Version 8.1; Muthén & Muthén, 1998–2017) using full information maximum likelihood estimation with robust standard errors.

Results

Unconditional Growth Models

Peer substance use significantly increased through adolescence (14–21), as did personal use of three substances (heavy drinking, marijuana, illicit drugs; $B(SE)$ ranged from .17(.01) to 1.02(.05), all $p<.001$). The log odds of daily smoking marginally increased during adolescence ($B(SE)=.17(.09)$, $p=.063$). All adult (21–26) trajectories were nonsignificant

[1]We adopted this analytic approach after encountering substantial difficulty obtaining estimates of growth factor relations from a conventional parallel process latent growth model, due in part to skewness in the distribution of peer use, and low endorsement of some substance use ordinal categories during adolescence.

(*B*(*SE*) ranged from - .001(.03) to −.55(.37), *p* ranged from .130 to .984), indicating stable peer and personal use in young adulthood. Figure 2 illustrates the peer use trajectories, which closely parallel substance use trajectories for all substances (Molina et al., 2018).

Peer and Personal Substance Use Growth

Table 1 (diagonal entries) shows that, adjusting for covariates, peer substance use increased in tandem with concurrent personal use throughout adolescence, and both remained stable through early adulthood to age 25. Associations were stronger for LNCG than ADHD participants for adolescent heavy drinking, and for age-25 heavy drinking and marijuana. Table 1 (above diagonal) shows a general pattern of significant prospective prediction from adolescent peer use trajectories to adult substance use trajectories for both groups. Individuals whose number of substance-using peers increased more quickly during adolescence tended to *decrease* their own heavy drinking and marijuana use more quickly in young adulthood (conditional on adolescent substance use and covariates; Figure 3), and reported less daily smoking and illicit drug use at age 25. For the ADHD group only, however, steeper increases in adolescent peer use predicted *higher* age-25 heavy drinking. Additionally, individuals whose peer use declined more steeply during young adulthood endorsed greater use of all substances at age 25 (regardless of ADHD).

Table 1 (below diagonal) displays the reverse prospective relations– growth in substance use predicting later peer use for both groups. First, adolescents who escalated in heavy drinking more quickly across adolescence reported increasingly more substance-using peers across young adulthood(Figure 4). In contrast, adolescents who escalated more quickly in their odds of daily smoking (Figure 5) and illicit drug use *decreased* more quickly in peer use through young adulthood. Only growth in marijuana use (adolescent and adult) consistently predicted higher age-25 peer use.

ADHD/LNCG Group Differences

Of greatest interest, Table 1 highlights several associations described above that differed by ADHD diagnosis based on chi-square difference tests comparing nested models. The positive adolescent and age-25 concurrent relations between heavy drinking and peer use were stronger for LNCG than ADHD participants. Likewise, peer use was more strongly associated with age-25 marijuana use for LNCG than ADHD participants.

Three prospective relations also differed significantly by diagnostic group. The relation between adolescent peer use and age-25 heavy drinking was significant and positive for the ADHD group, but nonsignificant for the LNCG. Second, although adolescent peer use did not significantly predict age-25 marijuana use for either group, the relation was positive for ADHD but negative (and significantly different) for LNCG. Third, increases in adolescent heavy drinking more strongly predicted adult peer use increases in LNCG than ADHD participants.

Sensitivity Analyses

We considered that path coefficients predicting age-25 peer use might have been attenuated due to a high correlation with adolescent growth in peer use (r=.981). We conducted a series

of regression analyses predicting age-25 peer use from covariates, peer use adult slope (excluding peer use adolescent slope), and substance use adolescent and adult slopes and intercept. Table S1 shows that all four substances predicted age-25 peer use after excluding the collinear peer use adolescent slope. We therefore consider the path coefficients predicting age-25 peer use (Table 1) to be conservative estimates. We ran parallel regressions predicting each substance at age 25, excluding adolescent peer use growth but retaining the intercept, then excluding the peer use intercept but retaining adolescent growth. Effects were generally similar to the path analysis results (Table S2).[2]

Discussion

On the whole, changes in peer and personal substance use during adolescence and young adulthood tracked together, as expected, with some notable differences for individuals with versus without childhood ADHD. Several prospective, reciprocal associations between peer personal use survived stringent adjustments for prior growth, consistent with both selection and socialization processes. Findings suggest that peers play an important role in individuals' substance use trajectories into early adulthood for young people with and without childhood ADHD. However, several associations were unexpectedly weaker in the ADHD group compared to the LNCG, possibly reflecting ADHD participants' failure to meet normative developmental expectations of detaching their own substance use from their peers' in their mid-twenties.

Weaker Associations Between Peer and Personal Substance Use for the ADHD Group

As expected, findings extend evidence that high levels of both peer and personal substance use persist from adolescence through young adulthood (SAMHSA, 2014; Van Eck et al., 2014), and further highlight that they *change together* across adolescence and young adulthood for individuals with and without ADHD. These results expand on prior cross-sectional findings to mean age 20 (DiGuiseppi et al., 2018; Marshal et al., 2003; Van Eck et al.) and correlated growth between adolescent peer and personal use (Belendiuk et al., 2016) by extending further into young adulthood — mean age 25 -- and generalizing to substances besides alcohol, the focus of most previous peer use research.

Unexpectedly, concurrent relations between peer use and personal use were weaker in the ADHD group than LNCG for heavy drinking in adolescence, and for heavy drinking and marijuana use at age 25. These weaker associations indicate that those with ADHD deviate from normative developmental patterns. In contrast, Belendiuk and colleagues (2016) showed *stronger* correlated growth between peer use and alcohol use among adolescents with ADHD histories. However, they measured frequency of any alcohol consumption, not exclusively binge drinking/drunkenness as in the present study, and only to age 17. For adolescents with ADHD, developmentally atypical levels of drinking likely stem from a broader set of risk factors beyond peer influences, such as impulsivity (Pedersen et al., 2016)

[2]To probe the potential confounding influence of ADHD persistence in adulthood, additional regression analyses tested each significant ADHD/LNCG group difference in the path analyses, controlling for main effects of ADHD persistence/desistence and its interaction with each of the growth factors. Results yielded the same significant ADHD/LNCG group differences as the path analyses, and none of the ADHD persistence/desistence main or interaction effects were significant, suggesting that symptom persistence did not significantly contribute to the observed finding beyond the effect of childhood ADHD diagnosis.

and younger age of drinking onset (Molina et al., 2018). Substance-using peers may thus play a proportionally more salient role in heavy drinking risk for adolescents without ADHD. Although seemingly protective, the weaker relations between peer use and personal use for *young adults* with ADHD actually translate to *higher* substance use at a time when they should cease experimentation and begin tapering down. Substance use may become less socially-mediated for young adults with ADHD as they age (Pedersen, Harty, Pelham, Gnagy, & Molina, 2014). Solitary alcohol use predicts worse alcohol problems in young adulthood (Christiansen et al., 2002; Creswell et al., 2014), suggesting a potentially riskier pathway to long-term substance abuse among individuals with ADHD.

Selection and Socialization

Prospective effects of adolescent peer use on young adult substance use, and vice versa, confirmed developmentally typical selection and influence processes for alcohol and marijuana, the most commonly used substances in adolescence/young adulthood (Hasin et al., 2015; Johnston et al., 2016; SAMHSA et al., 2014). Similarly, faster increases in adolescent peer use predicted lower rates of age-25 daily smoking and illicit drug use, likely reflecting normative experimentation among adolescents and their peers that subsides by young adulthood (Chassin et al., 2013).

An exception to this normative pattern again occurred for the ADHD group, for whom faster increases in adolescent peer use predicted *more frequent* heavy drinking at age 25. Even though their increasing peer use in adolescence predicts decreasing substance use across young adulthood, these adult declines do not outweigh disadvantage accumulating in adolescence, potentially posed by starting higher in adolescence (Molina et al., 2018), resulting in persistently high substance use by young adulthood. Relatedly, ADHD/LNCG differences were unaffected by young adult ADHD symptom persistence, suggesting that ADHD-related impairments (which may persist despite symptom reduction) may contribute to the unique risk for age-25 heavy drinking in the ADHD group. Counterintuitively, adolescents in both groups reporting heavy-using peers (a temporary, developmentally normative phenomenon) reported sharp declines in peer use through young adulthood, but continued to use all substances at relatively higher levels themselves at age 25. Thus, escalating peer use in adolescence appears to increase risk for substance use at age 25 despite declines in young adulthood. Risks for young adult substance use posed by atypically precocious use by peers may transcend common risks specific to ADHD. Future studies that extend beyond this young adult period will help elucidate whether this trajectory ultimately declines further, and whether adults with ADHD lag behind in achieving these normative declines.

Limitations and Future Directions

Findings rely on individuals' *perceptions* of their peers' behavior. Although potentially biased, perceptions strongly influence young peoples' substance use (D'Amico & McCarthy, 2006; Schulte, Monreal, Kia-Keating, & Brown, 2010). In fact, one effective intervention reduces substance use by correcting misperceptions about peer use norms (Dotson, Dunn, & Bowers, 2015; Schulte et al., 2010). In addition, our peer use measure averaged across substances. However, polysubstance use is the norm among young people (Moss, Chen, &

Yi, 2014), evident in the high internal consistency of our peer use scale and correlated peer use across substance classes.

Given the complex and dynamic nature of ADHD treatment over time, adjustment for naturalistic, time-varying treatment was beyond the scope of this paper; it will be the focus of a subsequent paper. We previously failed to find effects of treatment on substance use in the MTA (Molina et al., 2007; 2013), a finding that others have replicated (e.g., Biederman et al., 2008), so it is unlikely that findings were strongly affected by excluding treatment from our models. Further, we have found no evidence that original MTA assigned treatment group predicts substance use in adolescence or young adulthood (REFERENCE BLINDED); however, there may be opportunities for future studies to more closely examine treatment-related effects on associations between peer and personal substance through and beyond adolescence.

We also note two methodological limitations. Peer use was not assessed at the 12-year point (M_{age}=20.9), reducing certainty about peer use growth during this period. Second, our path analysis of growth factor scores are approximations of latent variables and therefore subject to estimation error. However, the presence of baseline covariates in the growth models from which we drew our factor scores improves score quality (Curran et al., 2016) and reduces "shrinkage" or bias toward the marginal factor means for participants with fewer observations (Bauer & Hussong, 2009). Lastly, future research should examine whether weaker relations between peer use and substance use for individuals with ADHD generalize to newer methods of consumption (e.g., e-cigarettes, edibles).

Conclusions

Concurrent and prospective relations provide the first evidence of longitudinal co-occurrence between peer use and substance use from adolescence through young adulthood when substance use peaks. The peer context appears to be critical in substance use escalation and desistence (and vice versa) across this developmental period, suggesting both selection and socialization. Thus, interventions that alter perceived peer substance use through corrective normative feedback should be emphasized (Dotson et al., 2015) for both high-risk (ADHD) and typically developing young people. Given that some peer-personal associations were stronger for individuals without ADHD, forecasting developmentally normative declines in substance use, these interventions could be tailored for young adults *with* ADHD by adding a training component to help them emulate peers who have age-appropriately lowered their substance use. Peer-enhanced interventions, which integrate a peer into treatment and train them to facilitate prosocial alternatives to substance use, have shown promise for young adults, including in non-college, community settings (Smith, Davis, Ureche, & Dumas, 2016). If young adults with ADHD do not effectively emulate their peers' declines in substance use, they may uniquely benefit from peer-enhanced interventions to more directly influence their substance use behavior and hasten the maturing out process.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations:

ADHD	Attention-Deficit/Hyperactivity Disorder
LNCG	Local Normative Comparison Group
MTA	Multimodal Treatment Study of ADHD

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Highlights

- Peer and personal substance use change together in adolescence and young adulthood
- Change in peer use prospectively predicts young adult substance use, and vice versa
- Peer-personal substance use relations are weaker for those with childhood ADHD
- Young adults with ADHD may need unique supports to mature out of substance use

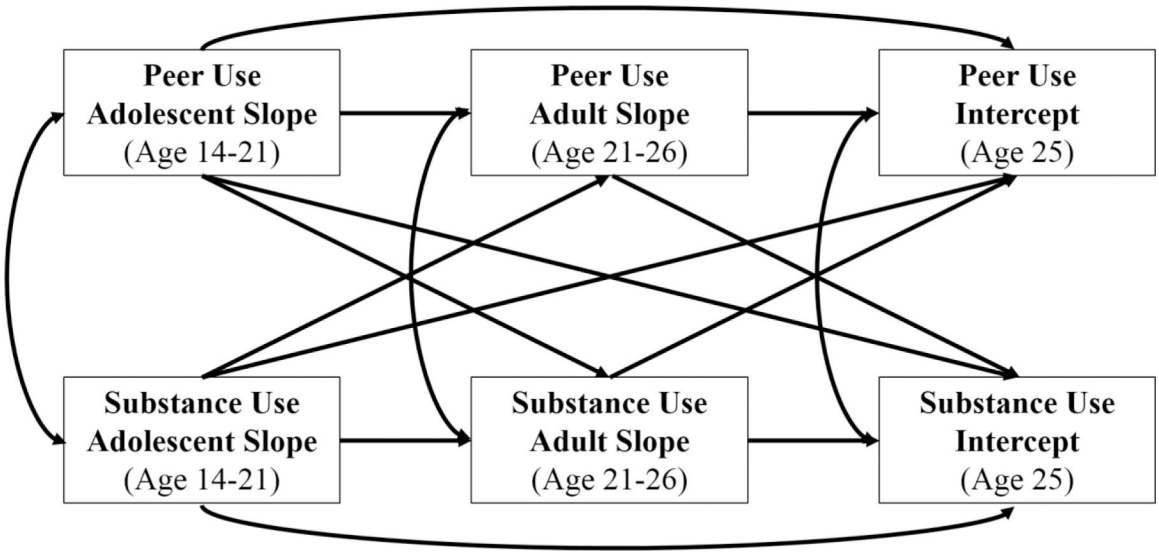

Figure 1.

Basic path model of expected relations among peer use and substance use growth factors. Cross-construct paths (i.e., Peer Use → Substance Use and Substance Use → Peer Use) are hypothesized to be stronger for ADHD than LNCG.

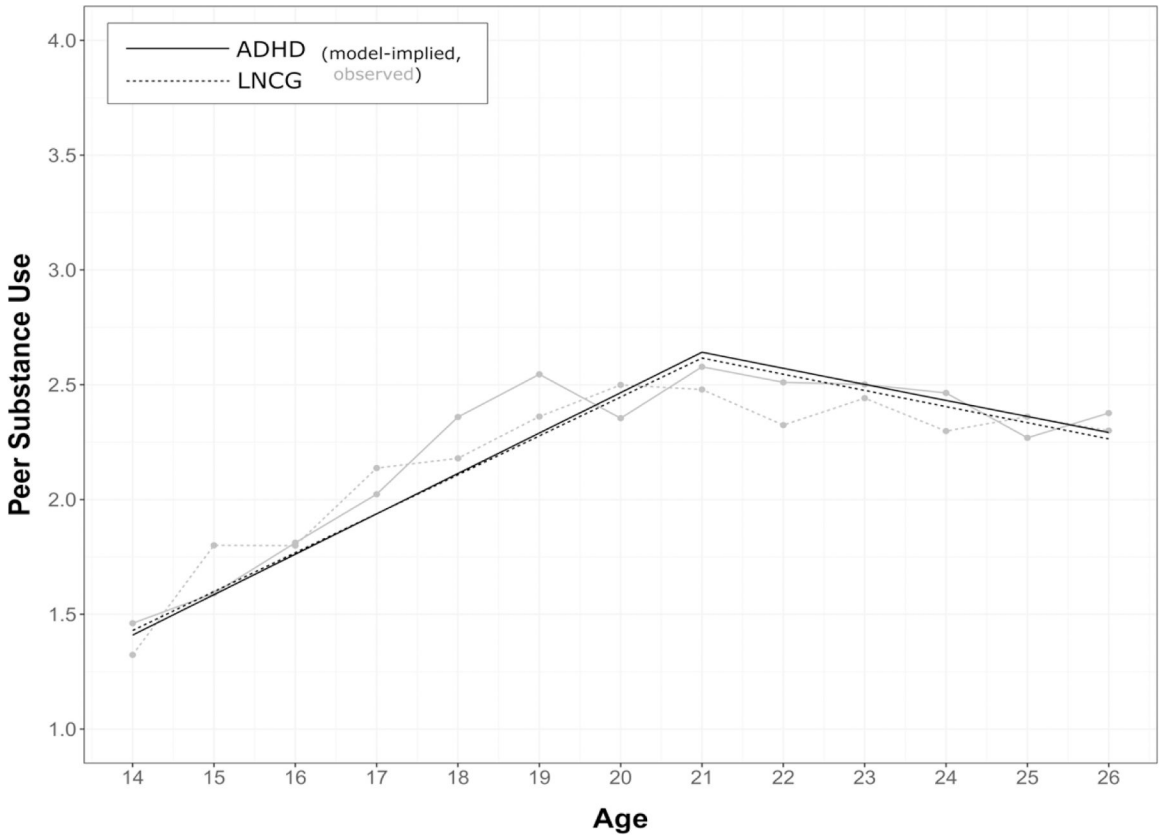

Figure 2.
Unconditional model-implied peer use growth trajectory.

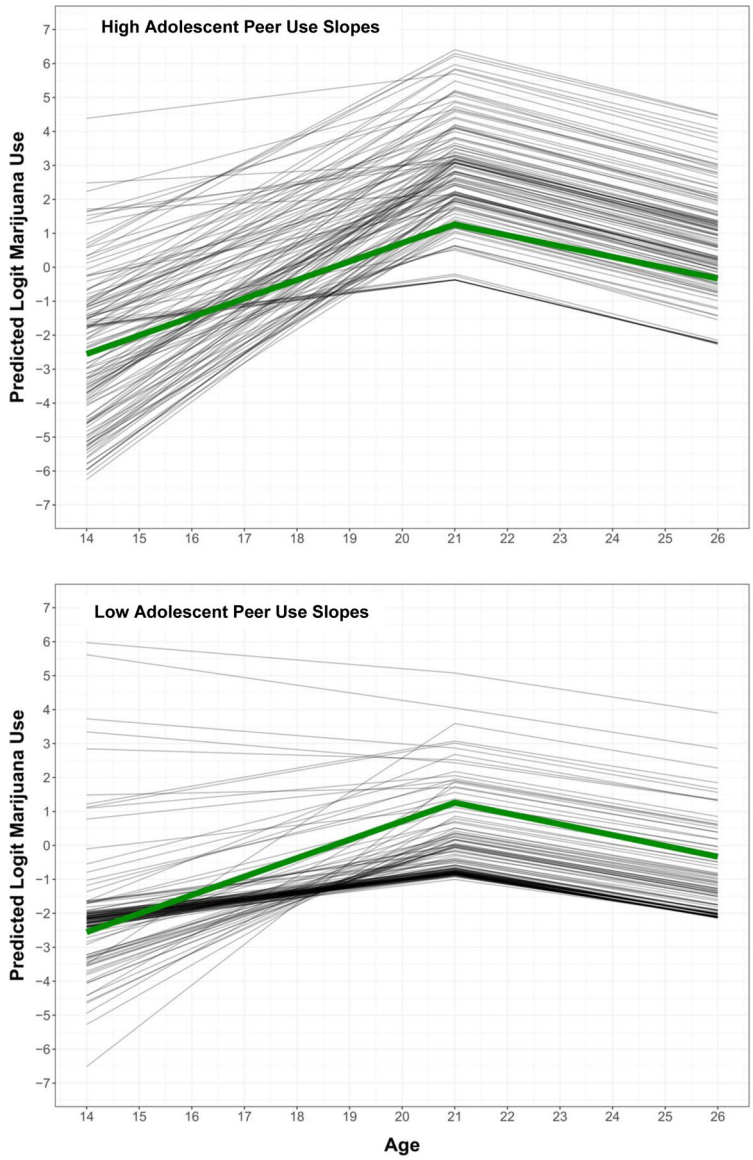

Figure 3.

Marijuana use trajectories predicted by adolescent peer use trajectories for individuals with high (top panel) versus low (bottom panel) adolescent peer use slopes (+/− 1 SD from average adolescent peer use slopes).

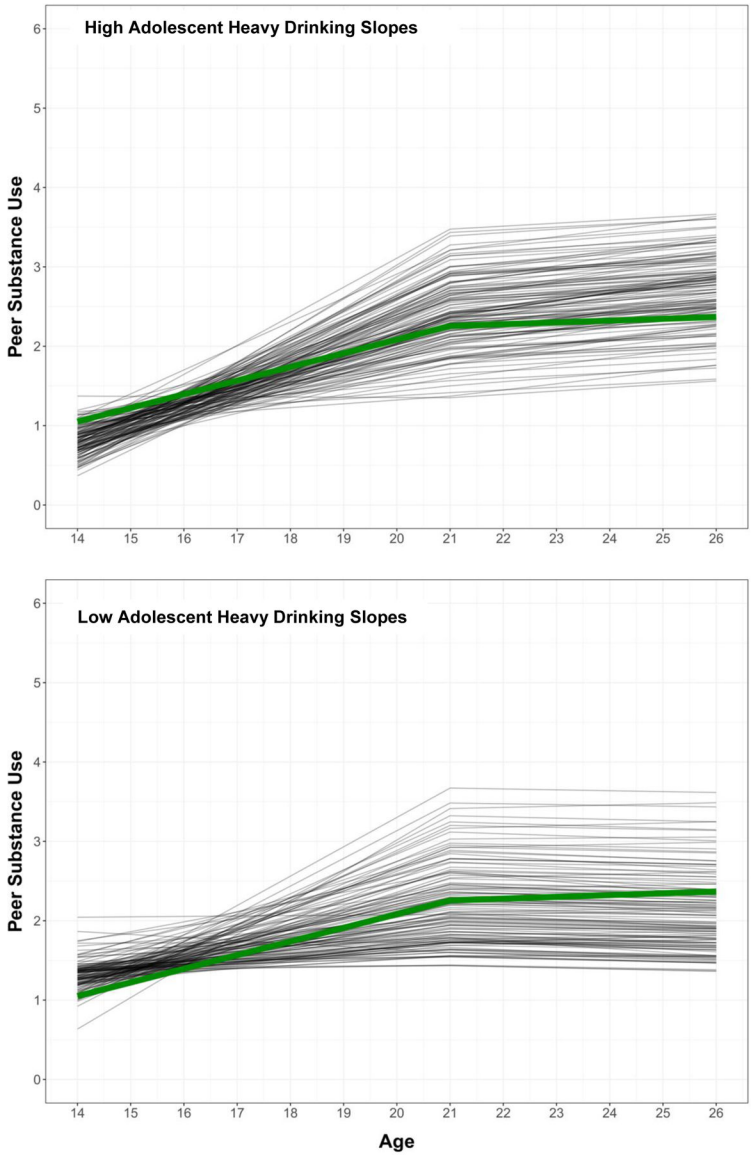

Figure 4.

Peer use trajectories predicted by adolescent heavy drinking trajectories for individuals with high (top panel) versus low (bottom panel) adolescent heavy drinking slopes (+/− 1 SD from average adolescent heavy drinking slopes).

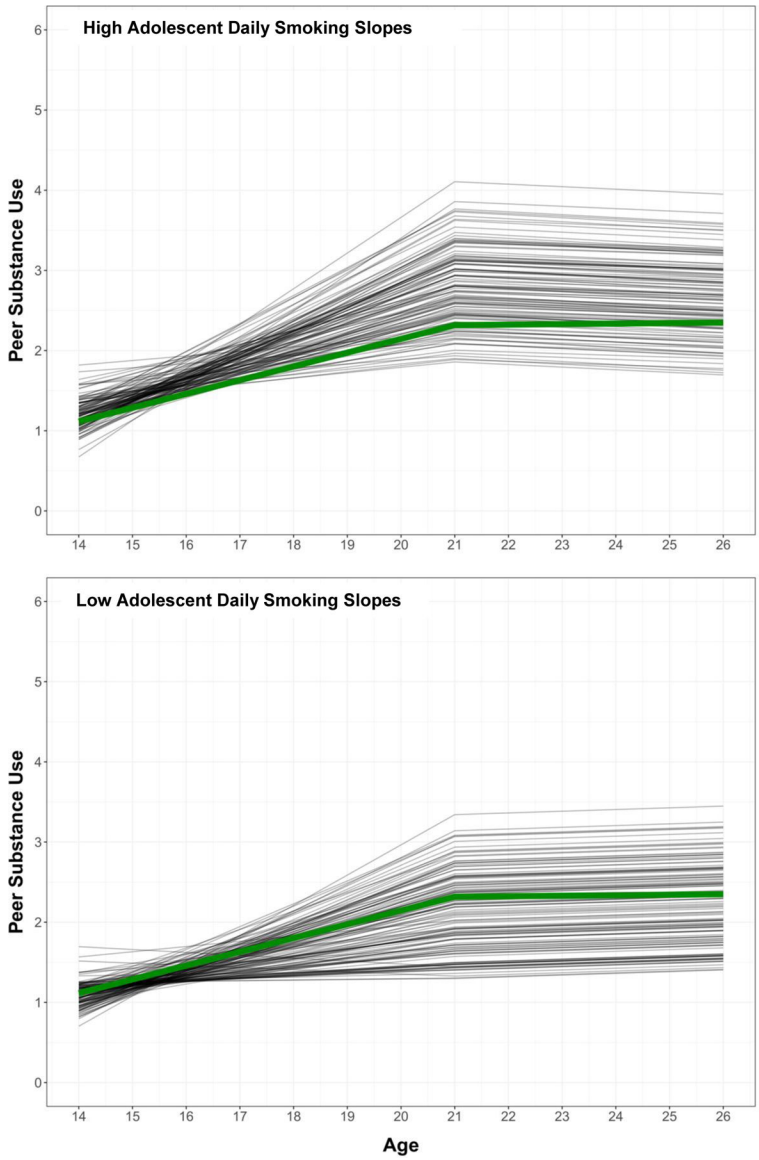

Figure 5.

Peer use trajectories predicted by adolescent daily smoking trajectories for individuals with high (top panel) versus low (bottom panel) adolescent daily smoking slopes (+/− 1 SD from average adolescent daily smoking slope).

Table 1

Predicted values of prospective substance use growth from peer use growth (above diagonal), prospective peer use growth from substance use growth (below diagonal), and concurrent associations (diagonal).

	Substance Use					
	Adolescent Growth		Adult Growth		Age 25 Intercept	
Peer Use	**ß**	**B (SE)**	**ß**	**B (SE)**	**ß**	**B (SE)**
ADHD						
Adolescent Growth						
Alcohol	**.32**A^{***}**/.48**L^{***}	**.01***A, L* **(.001***A, L***)**	−.48***	−1.32 (.11)	**.06**A^{*}**/.03***L*	**.42***A* **/ .22***L* **(.19** *A* **/.21***L***)**
Smoking	.32***	.01 (.001)	.02	.13 (.18)	−.11***	−.85 (.23)
Marijuana	.50***	.01 (.001)	−.22***	−.50 (.08)	**.02***A***/ −.01***L*	**.29***A***/−.24***L* **(.66***A***/.69***L***)**
Illicit	.42***	.01 (.001)	.01	.01 (.07)	−.08*	−.84 (.36)
Adult Growth						
Alcohol	**.19**A^{***}**/.27**L^{***}	**.06***A***/.09***L* **(.01** *A, L***)**	.48***	.01 (.001)	−.09**	−.57 (.21)
Smoking	−.21***	−.05 (.01)	.16**	.004 (.001)	−.16***	−1.15 (.26)
Marijuana	.01	.004 (.01)	.44***	.005 (0)	−.30***	−5.13 (.69)
Illicit	−.14***	−.06 (.02)	.34***	.003 (.001)	−.20***	−2.17 (.44)
Age 25 Intercept						
Alcohol	−.01	−.01 (.01)	−.01	−.01 (.01)	**.18**A^{***}**/.40**L^{***}	**.003** *A***/.01***L***(.001** *A, L***)**
Smoking	.003	.004 (.01)	.01	.004 (.01)	.13**	.003 (.001)
Marijuana	−.01*	−.02 (.01)	.02**	.04 (.02)	**.18**A^{***}**/.36**L^{***}	**.01***A* **/.02***L* **(.002***A* **/.004***L***)**
Illicit	−.01	−.01 (.01)	.01	.03 (.02)	.14**	.004 (.001)

Note. Coefficients from path model of growth factors. All paths are regressed, except for concurrent relations (diagonal), which are covaried. Within-construct relations (e.g., adolescent peer alcohol use predicting adult peer alcohol use) are also included in the model but are not displayed here for simplicity. Covariates include sex, race, income, proportion of time during study period living at home, proportion of time during study period enrolled in college, household (dis)advantage, and site. Adolescent growth spans age 14 to 21, and adult growth spans age 21 to 26. Parameters for the ADHD group are displayed with equivalent unstandardized coefficients (B) and standard errors (SE B) between ADHD and LNCG, except where significant ADHD/LNCG group differences are highlighted in boldface type.

* p<.05;

** p<.01;

*** p<.001

A Parameter for ADHD;

L Parameter for LNCG