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Marker or mediator? The effects of adolescent substance use on young adult educational attainment

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Abstract

Aims— We tested the effects of adolescent substance use on college attendance and completion by young adulthood in the context of the behavioral and familial risk factors that influence substance use.

Design, setting and participants— Longitudinal data were collected from a community sample of children of alcoholics (248) and matched controls (206) at three adolescent assessments ($\mu_{\text{age}} = 13\text{--}15$) and a long-term follow-up in young adulthood ($\mu_{\text{age}} = 25$).

Measurements— College attendance and degree completion by age 25 were self-reported in young adulthood. During adolescence, self-reports of alcohol and drug use were assessed with log-transformed quantity/frequency measures; substance use risk factors [e.g. parental monitoring, externalizing and internalizing symptoms and *Diagnostic and Statistical Manual* version III (DSM-III) diagnosis of parental alcoholism] were assessed by both self- and parent-report, and adolescent reading achievement was assessed using a standardized assessment of reading achievement (Wide Range Achievement Test).

Findings— Using growth curve modeling, we found that mean levels and growth in adolescent substance use mark, or identify, those adolescents who are at risk for reduced odds of attending and completing college. Moreover, adolescent substance use was not merely a marker of risk, in that growth in drug use (but not alcohol use) significantly mediated the effects of parental alcoholism and early externalizing behavior on later college completion, partially explaining the effects of these risk factors on college completion.

Conclusions— The current study provides evidence for both the marker and the mediator hypotheses, and identifies multiple pathways to higher educational attainment. The findings point to the importance of studying the effects of adolescent substance use in a broader developmental context of its correlated risk factors to specify more effectively the key pathways to later developmental outcomes.

Keywords

Adolescent development; education; outcomes of substance use; substance use

INTRODUCTION

Educational attainment is an important gateway to successful young adult outcomes, and is especially important for later occupational status and income [1]. However, adolescents who use alcohol and drugs may be at risk for lowered educational attainment [2–5]. Cross-sectional relations have been reported between adolescent substance use and lower educational

aspirations, lower educational expectations and an increased likelihood of dropping out of high school [6–11]. Taken together, these findings suggest that adolescent substance use may lead to lowered educational attainment in young adulthood.

A number of theories have been proposed to explain the mechanisms by which adolescent substance use negatively impacts young adult educational attainment. Alcohol and drug use during adolescence might exert ‘selection effects’ in which adolescent substance users are less likely to be selected into higher educational settings (e.g. because of lowered academic performance in high school) or are less likely to select higher education (e.g. because they make precocious transitions into adult roles such as marriage, parenthood and/or full-time employment) [12]. Even if substance-using adolescents choose to enter higher educational settings, they may have less academic success because their substance use both impedes the development of necessary social and behavioral competencies [13] and also increases activities, values and peer social norms that are incompatible with academic success.

Although adolescent substance use may affect educational attainment, it is also necessary to recognize that adolescent substance use itself is embedded in a broader matrix of behavioral and familial risk factors. In fact, many of the risk factors that have been shown to predict prospectively adolescent substance use are also related to educational attainment. For example, parental education is related to both adolescent substance use [7] and to educational outcomes [14]. Similarly, parental alcoholism has been related to both adolescent substance use [15] and to academic achievement [16]. Moreover, externalizing and internalizing symptoms have been related to both substance use [17,18] and to educational attainment [8,19,20]. Finally, poor parenting has been related to both adolescent substance use [21] and educational outcomes [22,23].

The fact that adolescent substance use and young adult educational attainment share common predictors raises questions about the specific role of adolescent substance use as an influence on educational attainment in young adulthood. Adolescent substance use may simply be a marker of a broader spectrum of risk factors which influence young adult educational attainment [24,25]; that is, adolescent substance use may identify which adolescents are at risk for poor educational outcomes but not play a direct role in producing those outcomes. If adolescent substance use is simply a marker of risk (rather than a causal influence) then intervention programs aimed at improving educational attainment should attempt to modify the true causal influences rather than reducing adolescent substance use. Fergusson & Horwood [8] explored this question among adolescents and found that adjusting for antecedents to early marijuana use reduced the effects of marijuana use on psychosocial adjustment to non-significance, but marijuana use retained a direct effect on leaving secondary school early. Their findings suggest that the effects of adolescent marijuana use on adolescent educational attainment are unique and not simply a reflection of a broader matrix of risk factors. Building on their findings, it is possible that adolescent substance use also may serve to mediate the effects of these more distal risk factors on educational attainment in young adults. For example, children who are externalizing and poorly regulated may have lower educational attainment because their substance use interferes with studying and with the behavioral and social competencies that are necessary for academic success. However, such mediational pathways have not been tested, and it is unclear whether substance use has persistent effects on educational outcomes beyond adolescence and into young adulthood.

The current study attempts to clarify the unique roles of adolescent substance use and its antecedent behavioral and familial risk factors in the prediction of young adult educational attainment. We distinguish between two different outcomes, entry into post-high school education and successful degree completion. Entry into post-high school education reflects selection effects, such that a risk factor either impacts the adolescent’s ability to be selected

into higher education (perhaps by impairing high school performance) or increases the likelihood that the adolescent chooses to forgo higher education. Successful degree completion, on the other hand, reflects both selection effects and the social and behavioral competencies that are necessary for educational success.

In predicting entry into post-high school education and successful degree completion, we consider multiple pathways which are not mutually exclusive. First, adolescent substance use may directly and independently lower young adult educational attainment, reflecting other unidentified mediators such as lack of interest in higher education. Secondly, substance use may indirectly negatively affect later educational attainment by hampering academic achievement during adolescence. Thirdly, the effects of substance use on educational attainment may be a marker of a broader spectrum of risk. In this case, the relation between substance use and educational attainment should be eliminated when the effects of broader risk factors are considered. Finally, adolescent substance use may mediate the relation between the risk factors that produce it and later educational attainment. That is, familial alcoholism, poor parenting and adolescent conduct problems may lower educational attainment because they produce adolescent substance use which, in turn, lowers educational attainment.

In testing whether adolescent substance use is a marker or a mediator of the effects of behavioral and family risk factors on educational attainment, the current study contributes to the literature by testing the effects of both adolescent alcohol use and illegal drug use on educational attainment. Both alcohol and illegal drug use involve peer social activities and pharmacological effects that can be incompatible with educational attainment. However, because alcohol use is more prevalent than drug use and thus more normative in high school [26], there may be stronger effects of illegal drug use than of alcohol use, reflecting greater ‘deviance proneness’ of drug users [27]. In addition, the current study models growth over time in both drug and alcohol use to test their effects on young adults’ educational attainment. It may be a pattern of greater escalation in substance use involvement that affects educational attainment, rather than any static level of use [28].

In short, the current study extends the literature in several important ways. To our knowledge, it is the first to test whether adolescent substance use is a mediator or a marker of the effects of pre-existing risk factors on young adult educational attainment. Further, the longitudinal design allows us to model growth in adolescent substance use in relation to its behavioral and familial antecedents to predict prospectively young adult educational attainment. Finally, we distinguish between two different types of educational attainment: initiating the pursuit of education beyond high school and successfully obtaining a degree.

METHOD

Participants

Participants were from an ongoing longitudinal study of familial alcoholism [15,29–31]. At Time 1, there were 454 adolescents (mean age = 13.22 years, range = 10.5–15.5), 246 of whom had at least one biological, custodial alcoholic parent (COAs) and 208 demographically matched adolescents with no biological or custodial alcoholic parents (controls).

COA families were recruited using court records of driving under the influence (DUI) arrests, health maintenance organization wellness questionnaires and community telephone screening. Direct interview data from the computerized Diagnostic Interview Schedule (DIS version III [32]) confirmed that a biological and custodial parent met diagnostic criteria for life-time alcohol abuse or dependence per criteria listed in the *Diagnostic and Statistical Manual of Mental Disorders* version III (DSM-III [33]). Demographically matched control families were recruited using telephone interviews. When a COA participant was recruited, reverse

directories were used to locate families living in the same neighborhood. Families were screened to match the COA participant in ethnicity, family structure, target child's age (within 1 year) and socio-economic status (SES) (using the property value code from the reverse directory). Structured interviews were used to confirm that neither parent met life-time DSM-III criteria for alcohol abuse or dependence.

A complete description of sample recruitment and representativeness is reported elsewhere [15,34]. The sample was unbiased with respect to alcoholism indicators available in archival records (e.g. blood alcohol levels recorded at the time of the arrest, see Chassin *et al.* [34] for details). Moreover, the alcoholic sample had rates of other psychopathology similar to those reported for a community-dwelling alcoholic sample [35]. However, those who refused participation were more likely to be Hispanic, suggesting some caution in generalization.

There were three annual assessments (Time 1–Time 3) of the adolescent participants and their parents, and two long-term follow-ups (Times 4 and 5). The follow-ups were conducted when participants were in emerging adulthood (Time 4, median age = 20) and in young adulthood (Time 5, median age = 25). Sample retention was excellent at both follow-ups (Time 4, $n = 407$, 90% of the total sample; Time 5, $n = 415$, 91% of the total sample). Retention was unbiased by gender and ethnicity, but slightly more COAs than controls were lost at Time 4, $\chi^2(1, n = 454) = 5.45, P < 0.05$, and at Time 5, $\chi^2(1, n = 454) = 4.12, P < 0.05$.

Data from the Times 1, 2, 3 and 5 assessment periods were used in the current analyses. Participants were included in the present study if they had valid data on all variables included in the analyses ($n = 374$). Those who were dropped from analyses due to missing data were more likely to be COAs than non-COAs, $\chi^2(1, n = 454) = 10.18, P < 0.05$. However, included and excluded participants were not different in terms of age, gender, ethnicity or parental income. Of the included participants, 46% were female and 51% were COAs. The ethnic composition of the sample was 70% non-Hispanic Caucasian and 27% Hispanic, with the remaining 3% representing other ethnicities. At Time 1, parents on average had completed high school, and reported an average mean income of approximately \$32 000. At Time 5, COAs and controls did not differ in age or gender. However, COAs were more likely than were non-COAs to be Hispanic and were less likely to be married at Time 5.

Procedure

Computer-assisted interviews were conducted at the family's residence or on campus at all waves. Interviewers read items aloud, and participants could either enter responses themselves or respond verbally to questions. In most cases, family members were interviewed simultaneously but in different rooms to avoid threats of contamination and to increase privacy. Interviewers were unaware of the family's group membership. To further encourage honest responding, confidentiality was reinforced with a US Department of Health and Human Services Certificate of Confidentiality. Interviews lasted 1–3 hours and participants were paid up to \$70 over the waves.

Measures

Parental alcoholism—At Time 1, life-time DSM-III diagnoses of parental alcoholism (abuse or dependence) were assessed by direct interview with the DIS [32]. For those parents who could not be interviewed (19.3% of fathers, 7.7% of mothers), life-time alcoholism diagnoses were established using the Family History Research Diagnostic Criteria (FH-RDC, Version 3 [36]) based on spousal report. Using the FH-RDC may underestimate the prevalence of alcoholism in the non-interviewed parents (13.5%) because the RDC has higher specificity (>90%) than sensitivity (50%, [37]). If so, this error would underestimate the effects of parental alcoholism. For the present analyses, diagnoses were dichotomous: either present (at least one

biological and custodial parent met life-time criteria) or absent (neither biological parent met life-time criteria).

Maternal monitoring—At Time 1, mothers self-reported their monitoring of participants' behaviors in the past 3 months with three items [e.g. I had a pretty good idea of (the adolescent's) plans for the day] rated on a scale from 1 (strongly agree) to 5 (strongly disagree). Coefficient alpha for this scale at Time 1 was 0.80. A confirmatory factor analysis suggested that a single latent factor best fitted these data, which was used as a latent indicator of maternal monitoring.

Externalizing and internalizing symptomatology—At Time 1, participants and their mothers reported on participants' past year externalizing (22 items; e.g. rebellious, stole things, mean or cruel to others, destroyed property, etc.) and internalizing (seven items; e.g. felt lonely, cried a lot, felt worthless, felt worried, felt fearful/ anxious, etc.) symptomatology using items from the Achenbach Childhood Behavior Checklist (CBCL [38]). Items were rated on a scale from 1 (almost always) to 5 (almost never), and were reverse-coded so that high scores indicated more symptomatology.

For externalizing, coefficient alphas = 0.89 for participant self-report and 0.81 for mother-report. Attempts to model externalizing symptoms as a single latent factor with two reporters (and separate single reporter factor models) were unsuccessful, due most probably to the symptom count nature of the items. Thus, we used the mean of participant and maternal report ($r = 0.42$) in order to reduce shared source variance. However, because participants' self-report and mothers' reports of internalizing symptoms were not significantly correlated, participants' Time 1 self-reports were selected as the more reliable indicator of their own internal state [39], coefficient alpha = 0.78.

Alcohol and drug consumption—At each wave, participants reported their frequency of past-year consumption of beer/wine, hard liquor and eight illicit drugs (e.g. marijuana, amphetamines, cocaine, opiates, inhalants, etc.) with responses from (0) never to (7) every day. Quantity of alcohol consumption (two items) ranged from one to nine or more drinks per occasion. For beer/wine and for hard liquor, we computed quantity $\times$ frequency products and averaged them to index consumption. For drug use, we used the sum of the eight drug use frequency items. Because the modeling techniques are sensitive to non-normality, we used a log-transformation to reduce skewness and kurtosis, and multiplied the log-transformed variable by 10 to facilitate interpretation.

Alcohol and drug use were highly correlated across and within waves ($r_s = 0.35$ to 0.65 , $P_s < 0.001$). Moreover, only four drug users abstained completely from alcohol use during the adolescent waves, and those who reported drug use at any time-point reported higher alcohol use than those who used alcohol only, $t_{(103.03)} = 8.48$, $P < 0.001$. Thus, adolescent drug users were also heavier drinkers.

Academic achievement—At Time 3, reading achievement was measured with the Wide Range Achievement Test–Revised (WRAT–R [40]). In order to compare across different ages, we used standardized WRAT scores (mean = 102.82, SD = 15.17). Higher values reflected greater levels of academic competence.

Educational attainment—At Time 5, participants (median_{age} = 25) reported on their highest level of education. Responses were assigned to one of the following four categories to create an ordinal measure of educational attainment: 0 = did not graduate from high school ($n = 24$), 1 = received high school diploma or GED (general education diploma) ($n = 98$), 2 = received some college ($n = 127$) and 3 = earned an associate's degree or higher ($n = 125$).

Sixteen participants reported entering ($n = 4$) or completing ($n = 10$) a post-high school vocational program; for analytical purposes they were considered to have received 'some college'. Because this ordinal variable cannot be assumed to have equal intervals, we tested the models using two different dummy coded contrasts: one between those who had obtained some college education or better ($n = 252$) with those who had not ($n = 122$) and one comparing those who had completed an associate's degree or higher ($n = 125$) with those who had not ($n = 249$).

Analytical strategy

Substance use is consistently observed to increase with age during adolescence [26]. Longitudinal growth curve models can be used to capture this change in substance use as a function of time, describing both the average change in the population and the intra-individual variation in the rates of change (see Curran & Hussong [41] for an introduction to longitudinal growth curve models). This provides an advantage over traditional autoregressive models which confound change with mean levels and do not separate intra-and inter-individual variation in change. For the current study, we developed growth curve models using Meanstructure Analysis in MPlus 3.11 [42].

We tested mediational hypotheses by testing the significance of the indirect effects of familial and behavioral risk factors on educational outcomes through adolescent substance use [43]. The indirect effects are computed by multiplying the coefficient for the effects of the risk factors on substance use by the coefficient for the effects of adolescent substance use on educational attainment. Because the product of two coefficients are often skewed and highly kurtotic [44], we used confidence limits and critical values [45] obtained from the asymmetric distribution of the product of the coefficients. An indirect effect is considered significant if the interval between the upper confidence limit (UCL) and the lower confidence limit (LCL) does not contain zero. This approach has been shown to have higher power and lower Type I error rates than the Sobel [46] approach [44].

We included age, gender and ethnicity as covariates in all initial analyses. However, because gender and ethnicity had no significant effects across the models, we excluded them from the final models. We also tested for interactions by gender using multiple-group modeling methods. However, the two-group models did not fit the data significantly better than the one-group models, suggesting that there were no significant gender differences across models.

RESULTS

Correlations among variables

Bivariate correlations among the variables in our models are in Table 1. Alcohol and drug use during adolescence were correlated weakly with reading achievement in adolescence (average $r = -0.08$, $P < 0.05$). The point-biserial correlations between alcohol and drug use at any single time-point and college attendance and degree attainment were negative and significant, although small ($rs = -0.04$ to -0.18 , $Ps < 0.01$). Finally, drug and alcohol use were more strongly correlated with degree attainment than with college attendance.

Growth models of substance use

We developed separate growth models of alcohol and drug use during adolescence, and tested the effects of mean levels (intercepts) and changes (slope) in alcohol and drug use on educational attainment. We specified an intercept factor, representing participants' average level of substance use at Time 1 by fixing the factor loadings of Times 1, 2 and 3 substance use at 1.0. We also specified a linear slope factor, representing the average change in substance use, by fixing the factor loadings of Time 2 and Time 3 substance use to 1 and 2, respectively.

By setting the loading of Time 1 substance use on the growth factor to 0, we effectively set it as the intercept. Finally, we regressed the slope factors on the intercept factors to test whether the rate of growth in substance use depended on the initial level of use.

Our growth models of alcohol use [$\chi^2(2, n = 374) = 2.05, P = 0.36$; RMSEA (root mean square error of approximation) = 0.01; CFI (comparative fit index) = 1.00; TLI (Tucker Lewis index) = 1.00] and drug use [$\chi^2(2, n = 374) = 6.07, P = 0.19$; RMSEA = 0.03; CFI = 0.99; TLI = 0.99] fitted the data well. On average, participants increased their alcohol and drug use across the three waves, but they also varied significantly in how quickly they increased their alcohol (variance = 10.27, SE = 1.42, $P < 0.05$) and drug use (variance = 5.43, SE = 0.36, $P < 0.05$). The intercepts of alcohol and drug use also negatively predicted the slopes, such that higher initial alcohol or drug use predicted less acceleration in alcohol or drug use over time.

The relation between adolescent substance use and young adult educational attainment

Next, we tested whether the growth processes of alcohol and drug use were directly related to college attendance and degree completion in young adulthood after controlling for parental education and adolescent reading achievement. In all models, higher parental education and higher adolescent reading scores significantly predicted college attendance and degree completion. Generally, substance use during adolescence reduced the probability of completing but not attending college. Specifically, higher mean levels and acceleration in alcohol or drug use during adolescence reduced the odds of completing a college degree by Time 5, but only higher drug use at Time 1 reduced the likelihood of college attendance.

The marker hypothesis: does adolescent substance use predict young adult educational attainment over and above the effects of antecedents of adolescent substance use?

Predictors of substance use in adolescence—We first examined the effects of the risk factors on the alcohol and drug use intercepts and slope factors across the four models. The relations between the risk factors and the intercepts and growth factors of alcohol and drug use were nearly identical across the models predicting college attendance and degree completion. Thus, Table 2 presents estimates and standard errors for these paths from the final model predicting degree completion only. Externalizing symptoms at Time 1 were related positively to growth in alcohol and drug use during adolescence, whereas internalizing symptoms at Time 1 were related negatively to growth in alcohol use but were unrelated to growth in drug use. Parental alcoholism was related to higher alcohol and drug use intercepts and greater acceleration in alcohol use across adolescence, but was unrelated to acceleration in drug use during adolescence. Finally, maternal monitoring was not significantly related to the intercepts or growth in substance use.

Predicting college attendance—We next explored the effects of adolescent substance use on educational attainment controlling for substance use risk factors, adolescent academic achievement and parental education. Table 3 provides the path estimates and standard errors of the effects of the intercepts and growth factors of alcohol and drug use and the risk factors for substance use on college attendance. Fit indices suggested that both models predicting college attendance from alcohol use [$\chi^2(19, n = 374) = 29.41, P = 0.06$; RMSEA = 0.04; CFI = 0.99; TLI = 0.99] and drug use fitted the data well [$\chi^2(21, n = 374) = 30.54, P = 0.08$; RMSEA = 0.04; CFI = 0.99; TLI = 0.99]. None of the precursors to substance use (parental alcoholism, externalizing symptoms, internalizing symptoms and maternal monitoring) were related significantly to college attendance. However, the addition of these precursors to substance use eliminated the previously significant effect of the intercept of drug use on college attendance (suggesting that the mean level of drug use in adolescence may be a marker of a broader matrix of risk factors for a lower likelihood of college attendance).

Predicting college degree completion—Table 3 provides the path estimates and standard errors of the effects of the intercepts and growth factors of alcohol and drug use and the risk factors for substance use as well as adolescent academic achievement and parental education on college degree completion. Fit indices suggested that both models predicting college degree completion from alcohol use [$\chi^2(20, n = 374) = 33.01, P = 0.04$; RMSEA = 0.04; CFI = 0.99; TLI = 0.98] and drug use fitted the data well [$\chi^2(22, n = 374) = 33.43, P = 0.06$; RMSEA = 0.04; CFI = 0.99; TLI = 0.99]. Of the precursors to substance use, only externalizing symptoms at Time 1 were related to degree completion: higher externalizing symptoms in early adolescence predicted a lower likelihood of completing a college degree by young adulthood. After the addition of the precursors to substance use and those of educational attainment the observed effects of the intercepts of alcohol and drug use and growth in alcohol use on degree completion disappeared, suggesting that they are a marker of risk rather than specific mechanisms. However, the effects of growth in drug use during adolescence were more robust. Growth in drug use remained related negatively to the likelihood of college degree completion even with the addition of the precursors to substance use. To provide an example of the current models, Fig. 1 illustrates the model predicting degree completion from adolescent drug use and related factors.

The mediator hypothesis: does adolescent substance use mediate the relations between antecedents for adolescent substance use and young adult educational attainment?

We then examined the indirect effects of the risk factors (parental alcoholism, maternal monitoring and externalizing and internalizing symptoms) for substance use on educational attainment through intercepts and growth in adolescent alcohol and drug use. Of the substance use growth factors, we found that growth in drug use (but not alcohol use) significantly mediated the effects of externalizing symptoms at Time 1 (indirect effect = -0.09 , $SE = 0.04$, $UCL = -0.002$, $LCL = -0.165$), such that higher levels of externalizing symptoms at Time 1 predicted greater growth in drug use during adolescence which, in turn, reduced the likelihood of obtaining a degree.

The effects of parental education and reading achievement: direct and indirect relations

Although not a central focus of the current investigation, we obtained interesting findings illuminating the role of parental education and adolescent academic achievement in the prediction of young adult educational attainment. Table 2 shows that higher levels of parental education predicted adolescent academic achievement; however, academic achievement was unrelated to alcohol or drug use during adolescence. As shown in Table 3, higher academic achievement in adolescence increased the likelihood of college attendance and completion in all models. Finally, mediational analyses revealed reading achievement to be a mediator of the effects of parental education on college attendance and degree completion. Higher parental education was related to increased reading achievement in adolescence, which led in turn to a higher likelihood of college attendance and degree completion (for college attendance: indirect effect from the drug use model = 0.07 , $SE = 0.02$, $UCL = 0.11$, $LCL = 0.04$, indirect effect from the alcohol use model = 0.07 , $SE = 0.02$, $UCL = 0.11$, $LCL = 0.04$; for degree completion: the indirect effect from the drug use model = 0.06 , $SE = 0.02$, $UCL = 0.10$, $LCL = 0.03$, indirect effect from the alcohol use model = 0.06 , $SE = 0.02$, $UCL = 0.10$, $LCL = 0.03$).

DISCUSSION

The present study examined the relation between adolescent substance use and young adult educational attainment, testing whether adolescent substance use serves as a marker or a mediator of a broad matrix of risk factors for lowered educational attainment in young adulthood. The findings provide evidence for both the marker and the mediator hypotheses, and identify multiple pathways to higher educational attainment.

As expected, parental education predicted both the pursuit of higher education and successful degree completion, in part via adolescent reading achievement, reflecting the intergenerational transmission of educational attainment. Adolescents with more educated parents are likely to be in family contexts that value, encourage and model academic pursuits, have economic resources to provide educational opportunities [14], as well as have heritable individual differences in academic abilities. Thus, one basic pathway to young adult educational attainment reflects the intergenerational transmission of higher educational attainment through early adolescent academic achievement.

The question of central interest was how adolescent substance use fits into the larger matrix of behavioral, familial, cognitive and self-regulatory influences on higher educational attainment. Initial models showed that after adjusting for the effects of parental education and adolescent reading achievement, there were significant effects of mean levels and growth in adolescent substance use on both college attendance (for drug use) and degree completion (for alcohol and drug use). This is consistent with previous research [6,7]. However, the effects of adolescent substance use were largely eliminated when their antecedent risk factors were entered into the model. Thus, to some extent, adolescent substance use seems to serve as a marker for a larger constellation of risk factors, identifying those adolescents who are at risk but not playing a causal role. These findings are similar to those of Fergusson & Horwood [8], who found that the effects of substance use on adolescent psychosocial outcomes were largely eliminated when prior risk factors were accounted for in the model [24].

Although many of the effects of adolescent substance use on educational attainment were eliminated after considering antecedent risk factors, a significant relation remained between growth over time in adolescent drug use and successful degree completion. This finding suggests that acceleration in adolescent drug use is not simply a marker for other risk factors in terms of its effects on finishing a college degree. Thus differentiating between mean levels of use and change in use over time is important in understanding the effects of substance use during adolescence.

In fact, growth in adolescent drug use significantly mediated the effects of externalizing behaviors on degree completion. Baumrind & Moselle [13] theorized that adolescent substance use impedes the development of self-regulatory and coping skills. Externalizing adolescents, who are poorly regulated, are also more likely to escalate their drug use, which may further impede the development of the social and behavioral competencies that are necessary for success in higher education.

These results were found for escalation over time in drug use but not alcohol use, suggesting that there are important differences in the effects of growth in alcohol versus drug use on educational outcomes in young adulthood. For example, this finding may reflect a difference in the prevalence and relative deviance of adolescent alcohol use compared to illegal drug use [26]. Indeed, college students use alcohol at higher levels than do their same-age peers who do not attend college [47,48], but illicit drug use (particularly drugs other than marijuana) is more prevalent among non-college attending peers than among college students [49]. Therefore, adolescents who escalate illegal drug use over time may become part of a deviant peer network whose values and activities are less compatible with the academic demands and social climate of higher education. Indeed, the drug-using adolescents in our sample also were the heaviest drinkers, suggesting that their pattern of substance use was the least normative, particularly for college students, and that the effects of drug use in the current study may also include the comorbid effects of alcohol and drug use. Additionally, these differential findings for alcohol and drug use may reflect higher behavioral under-control or disinhibition among adolescents who use illegal drugs versus just alcohol [31,50].

The effects of growth in adolescent drug use were specific to degree completion, rather than to entry into post-high school education. Additionally, other significant predictors of degree completion (such as externalizing symptoms) were not related directly to college attendance, which was predicted only by parent education and adolescent academic achievement. In the current study, college attendance was defined with the highest level of education completed being 'some college or better'. This could reflect a spectrum of possibilities from enrolling full-time at a highly selective private university or taking a few courses part time at a community college. Thus, different findings might be obtained if the definition of 'college' is narrowed to include only 4-year colleges or full-time students. However, considering that only 59.6% of college students attend college full time [51], the data from our prospective community sample may provide a more realistic picture of the nature of college attendance. Our findings may also suggest that selection into college may be driven primarily by educational factors, but success in college may be influenced more strongly by behavioral factors. Many studies examine a single marker of educational attainment [3] or a continuous measure of educational attainment [7]. However, the current findings suggest that the effects of substance use may not be consistent across levels of educational attainment and that the barriers to performing well in and graduating from high school probably differ from barriers to being able to perform successfully in and graduate from college.

The current findings demonstrate the importance of considering a broader matrix of risk factors when examining the effects of adolescent substance use on later adult outcomes. The 'marker or mediator' hypothesis also has important implications for prevention research. For example, one long-term benefit of successful drug use prevention might be an increased likelihood of completing a college degree. However, our models also suggest that targeting externalizing behaviors in adolescence may have wider benefits on academic performance, both during and after high school. In a broader sense, the 'marker or mediator' hypothesis can be used to identify clearly potentially modifiable mediators to target for intervention by studying the outcomes of risk behaviors in light of the developmental processes that produce them, thus providing a means of identifying the most profitable targets for intervention.

In spite of the importance of the present findings, there are also limitations of our study that should be noted. First, we do not have data on the types of colleges our participants' attended or the reason for not completing a college degree by Time 5. This limits our ability to interpret the lack of relation between substance use and college attendance. Additionally, we examined consumption of alcohol and drug use, rather than substance use consequences or disorders, both of which may be more strongly related to high school academic achievement and college attendance. We examined only one indicator of early school success (reading achievement as measured by a standardized reading test) in our models. Future studies should include more comprehensive assessments of adolescent school performance, such as behavioral reports and grades.

In summary, we found that substance use during adolescence serves as a marker of a broader spectrum of problem behaviors which reduce academic achievement during adolescence and also impact later selection into and success in college. However, growth in drug use had specific direct effects on completing a college degree, accounting partially for the effects of externalizing symptoms on degree completion and suggesting that those adolescents whose drug use accelerates during adolescence experience additional risk for poor educational attainment due to their illicit use of drugs.

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Figure 1.

Prediction of college degree completion from adolescent academic achievement, the drug use intercept and growth factors and antecedents to adolescent drug use. Non-significant paths, latent factor loadings, estimated means, residual variances and covariances among predictors are not shown for ease of presentation. Bold type paths indicate significant mediated effects. Mon1 = Time 1 parental monitoring; Mon2 = Time 2 parental monitoring; Mon3 = Time 3 parental monitoring

Table 1

Correlations among main predictors and outcomes.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Parental education	—	0.12*	0.09	0.03	-0.12*	-0.09	0.28***	0.02	-0.01	-0.03	0.00	0.02	-0.05	0.36***	0.36***
2. T1 monitoring 1		—	0.55***	0.53***	-0.28***	-0.02	0.06	-0.12*	-0.17***	-0.14**	-0.21***	-0.16**	-0.13*	0.11*	0.21
3. T1 monitoring 2			—	0.68***	-0.25***	-0.04	0.12*	-0.21***	-0.18***	-0.17***	-0.23***	-0.20***	-0.12*	0.08	0.15
4. T1 monitoring 3				—	-0.25***	-0.01	0.10	-0.23***	-0.19***	-0.20***	-0.27***	-0.20***	-0.17**	0.08	0.12
5. Externalizing T1					—	0.43***	-0.23***	0.37***	0.41***	0.40***	0.33***	0.35***	0.39***	-0.20***	-0.26***
6. Internalizing T1						—	-0.13*	0.16**	0.16**	0.10	0.10	0.10	0.16**	-0.11	-0.08
7. T3 reading achievement							—	-0.06	-0.04	-0.12*	-0.09	-0.07	-0.05	0.34***	0.32
8. Time 1 alcohol use								—	0.72***	0.52***	0.67***	0.57***	0.43***	-0.04	-0.12
9. Time 2 alcohol use									—	0.67***	0.57***	0.66***	0.64***	-0.07	-0.18***
10. Time 3 alcohol use										0.67***	0.57***	0.47***	0.57***	-0.11*	-0.15**
11. Time 1 drug use										—	0.41***	0.60***	0.46***	-0.10	-0.11*
12. Time 2 drug use											—	0.60***	0.74***	-0.13*	-0.17***
13. Time 3 drug use												—	—	-0.14**	-0.18***
14. College attendance														—	0.49***
15. Degree attainment															—

* Correlation is significant, $P < 0.05$;

** correlation is significant, $P < 0.01$;

*** correlation is significant, $P < 0.001$.

Table 2
Prediction of the intercept and growth factors of adolescent substance use growth and adolescent reading achievement across adolescence.

	Alcohol use growth		Drug use growth		Reading achievement	
	Estimate	SE	Estimate	SE	Estimate	SE
Adolescent alcohol use intercept	-0.309***	0.035				
Adolescent drug use intercept			-0.239***	0.036		
Maternal monitoring	0.013	0.228	0.107	0.138	0.075*	0.067
Time 1 externalizing symptoms	1.943***	0.350	0.823***	0.195	-0.167*	0.068
Time 1 internalizing symptoms	-0.879**	0.338	-0.070	0.215	-0.085	0.072
Parental alcoholism	1.532***	0.448	0.612	0.361	-0.019	0.101
Time 1 age	0.979***	0.196	0.198	0.126		
Parental education					0.198***	0.036

	Alcohol use intercept		Drug use intercept	
	Estimate	SE	Estimate	SE
Time 1 age	1.929	0.242		
Parental alcoholism	2.759	0.771	0.854***	0.166
			1.304*	0.654

Estimates are presented from final models predicting college degree completion; estimates from models predicting college attendance were nearly identical.

* $P < 0.05$;
** $P < 0.01$;
*** $P < 0.001$.

Table 3

Predictors of educational attainment by Time 5.

	College attendance		College degree completion	
	Estimate	SE	Estimate	SE
Alcohol use slope	-0.02	0.02	-0.021	0.025
Alcohol use intercept	0.00	0.01	-0.01	0.013
Maternal monitoring Time 1	0.01	0.08	0.136	0.092
Externalizing Time 1	-0.15	0.09	-0.365***	0.104
Internalizing Time 1	-0.02	0.11	0.152	0.118
Parental education	0.29***	0.06	0.325***	0.06
Academic achievement Time 3	0.34***	0.06	0.294***	0.073
Parental alcoholism	-0.15	0.15	0.069	0.145
Drug use slope	-0.03	0.03	-0.11*	0.05
Drug use intercept	-0.03	0.02	-0.04	0.03
Maternal monitoring Time 1	-0.01	0.09	0.14	0.10
Externalizing Time 1	-0.13	0.08	-0.31**	0.10
Internalizing Time 1	0.00	0.11	0.16	0.12
Parental education	0.29***	0.06	0.32***	0.06
Academic achievement Time 3	0.34***	0.06	0.30***	0.07
Parental alcoholism	-0.12	0.14	0.11	0.15

* Coefficients are significant $P < 0.05$;

** coefficients are significant $P < 0.01$;

*** coefficients are significant $P < 0.001$.