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Adolescent Substance Use Outcomes in the Raising Healthy Children Project:

A Two-Part Latent Growth Curve Analysis

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Abstract

Raising Healthy Children (RHC) is a preventive intervention designed to promote positive youth development by targeting developmentally appropriate risk and protective factors. This study tested the efficacy of the RHC intervention on reducing alcohol, marijuana, and cigarette use during early to middle adolescence. Ten public schools, comprising 959 students, were matched and assigned randomly to either intervention or control conditions. A two-part latent growth modeling strategy was employed to examine change in both use-vs.-nonuse and frequency-of-use outcomes. Results indicated significant ($p < .05$) intervention effects in growth trajectories for frequency of alcohol and marijuana use but not for use vs. nonuse. For cigarette use, a trend ($p = .08$) was found for decreasing cigarette use (vs. nonuse) among intervention students compared to controls but not for frequency of cigarette use. These findings provide support for preventive interventions that take a social development perspective in targeting empirically supported risk and protective factors and demonstrate the utility of two-part models in adolescent substance use research.

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A Two-Part Latent Growth Analysis

Public health research suggests that reducing risks and enhancing protective factors is a promising strategy for the prevention of substance abuse and other related problems (Coie et al., 1993; Hawkins, Catalano, & Miller, 1992; Mrazek & Haggerty, 1994). Risk factors are conditions in the individual or environment that predict greater likelihood of developing a problem such as substance abuse. Research has shown that adolescent substance use is influenced by multiple risk factors in the individual, family, and environment. Recent examples of such risk factors include: early onset of substance use (Grant & Dawson, 1997; Hawkins et al., 1997; Pedersen & Skrondal, 1998), gender (Hops, Davis, & Lewin, 1999), antisocial behavior (Ellickson, Tucker, Klein, & McGuigan, 2001), low self-efficacy (Schulenberg, Wadsworth, O'Malley, Bachman, & Johnston, 1996), family history of substance abuse (Vitaro, Tremblay, & Zoccolillo, 1999), poor family bonding (Brook, Brook, Arencibia-Mireles, Richter, & Whiteman, 2001), low commitment to school (Williams, Ayers, Abbott, Hawkins, & Catalano, 1999), high perceived prevalence of substance use (Simons-Morton et al., 1999), and associating with substance-using peers (Griffin, Botvin, Scheier, & Nichols, 2002), among others (for a review see Hawkins et al., 1992). In addition to risk factors, researchers have identified protective factors that counter risk factors or increase resistance to them and, consequently, inhibit the development of substance use even in the face of risk exposure. Examples of protective factors include: self-esteem and affiliation with prosocial peers (Spath, Redmond, Hockaday, & Yoo, 1996), parental supervision and support (Marshall & Chassin, 2000; Vitaro et al., 1999), and psychosocial composite indices of protection (Felix-Ortiz & Newcomb, 1999; Griffin, Scheier, Botvin, & Diaz, 2000; Jessor, Van Den Bos, Vanderryn, Costa, & Turbin, 1995; Scheier, Botvin,

& Baker, 1997). Many risk and protective factors for substance abuse also are risk and protective factors for other problem behaviors including delinquency, school dropout, and teen pregnancy (Howell, Krisberg, Hawkins, & Wilson, 1995). Although some risk and protective factors may have a constant effect on outcomes, others appear to change in importance over the course of development (Hawkins et al., 1992). Evidence also has shown that exposure to more risk factors over time increases risk of substance use cumulatively (Bry, McKeon, & Pandina, 1982; Newcomb, Maddahian, & Bentler, 1986).

Only a few interventions that address multiple risk and protective factors at appropriate developmental periods have been tested. Most interventions have been brief (e.g., Bauman, Foshee, Ennett, Hicks, & Pemberton, 2001; Kellam, Rebok, Ialongo, & Mayer, 1994; Spoth, Redmond, & Shin, 2001), have addressed a narrow range of risk or protective factors (e.g., Botvin & Griffin, 2002; Eddy, Reid, & Fetrow, 2000; Griffin, Botvin, Nichols, & Doyle, 2003; Pentz, Trebow, Hansen, & MacKinnon, 1990), or have focused on a single social domain (e.g., Ellickson, Bell, & Harrison, 1993; Greenberg & Kusche, 1997). Two projects, the Fast Track project (Conduct Problems Prevention Research Group, 1992) and the Seattle Social Development Project (SSDP; Hawkins, Catalano, Kosterman, Abbott, & Hill, 1999) addressed a broad range of developmentally salient risk and protective factors in school, family, peer, and individual domains. These interventions targeted risk and protective factors in early childhood in order to prevent initiation and escalation of problem behaviors in adolescence. To date, these *social development* interventions have demonstrated positive effects in reducing substance use, violent behavior, conduct problems, and risky sexual behavior, as well as improving academic performance, commitment to school, and social cognitive skills (Catalano et al., 2003; Conduct

Problems Prevention Research Group, 2002; Hawkins et al., 1999; Lonczak, Abbott, Hawkins, Kosterman, & Catalano, 2002).

This study examines the efficacy of the Raising Healthy Children (RHC) project. Modeled after SSDP, RHC is a comprehensive, multicomponent preventive intervention designed to promote positive youth development by targeting developmentally appropriate risk and protective factors. However, unlike SSDP, the intervention extended beyond the elementary-school period to include universal and selective components in middle- and high-school years. As a theory-based intervention, RHC is guided by the social development model (SDM; Catalano & Hawkins, 1996; Farrington & Hawkins, 1991; Hawkins & Weis, 1985), which integrates empirically supported aspects of social control (Hirschi, 1969), social learning (Akers, 1985; Akers, Krohn, Lanza-Kaduce, & Radosevich, 1979; Bandura, 1973), and differential association theories (Burgess & Akers, 1966; Matsueda, 1982, 1988) into a framework for strengthening prosocial bonds and beliefs. Within this framework, the SDM emphasizes that prevention should (a) begin before the formation of antisocial beliefs and behaviors; (b) recognize the importance of individual and family characteristics as well as larger social contexts of community, school, and peer influences; and (c) identify and address the changing needs of its target population with regard to risk and protective factors that change in influence during the course of development. Specifically, the SDM organizes risk and protective factors into a causal model that explicates the mechanisms leading toward antisocial behavior. These mechanisms are specified as a sequence of mediated effects influenced by both prosocial and antisocial processes. Following the SDM, four distinct points of intervention were targeted by RHC: (a) opportunities for involvement with prosocial others (e.g., family, teachers, and nonsubstance-using peers); (b) students' academic, cognitive, and social skills; (c) positive reinforcements and rewards for

prosocial involvement; and (d) healthy beliefs and clear standards regarding substance use avoidance. According to theory underlying the SDM, increased opportunities for prosocial involvement, coupled with both positive reinforcements for that involvement and better skills on the part of the student, are theorized to lead to stronger bonds to prosocial others. Once strong bonds are established, individuals will tend to behave in a manner consistent with the norms and values of the individuals and groups with whom they associate. In turn, stronger prosocial bonds support positive belief formation against antisocial behaviors (e.g., adolescent substance use).

As the primary domains of social influence during elementary school years are theorized within the SDM to be the family and school, RHC intervention components during this period focused on these domains. Evaluation of early intervention effects found that teachers reported less disruptive and aggressive behavior and stronger effort on school work for intervention students compared to controls (Catalano et al., 2003). As students approach adolescence, peer influences become more important and bonds to family and school may become strained (Hawkins, Guo, Hill, Battin-Pearson, & Abbott, 2001). Preventive interventions that target norms and teach skills for resisting negative social influences during this period have been shown to be effective in reducing substance use (e.g., Ellickson & Bell, 1990; Griffin et al., 2002; Hansen & Graham, 1991). Thus, the focus of intervention components within RHC gradually shifted from early risk and protective factors in the social domains of school and family toward individual- and peer-related risk and protective factors that addressed antisocial associations and beliefs.

A social development perspective to intervention also suggests that the goals of the intervention need to be flexible, as well. Whereas preventive interventions for early-adolescent substance use often center around abstinence themes, once adolescents begin to use substances,

messages related to the prevention of escalating or problematic substance use become increasingly important. The importance of this shift has been noted by Ellickson and Bell (1990) who in their evaluation of Project ALERT (a social influence resistance and normative change intervention that centered on drug abstinence and refusal skills) found positive intervention effects for reduced cigarette use among baseline nonsmokers and experimental smokers. However, an iatrogenic effect was observed among regular cigarette smokers, thus highlighting the need for tailoring interventions to the specific developmental needs of its target population. Furthermore, recent data have shown that some degree of experimentation with substances is normative (e.g., Johnston, O'Malley, & Bachman, 2003). Noting this, an increasing number of researchers have suggested that a concomitant goal of prevention should be the reduction in the amount of use (quantity or frequency) among users (e.g. Maggs & Schulenberg, 1998; Marlatt, 1996; Marlatt & Witkiewitz, 2002; McBride, Midford, Farringdon, & Phillips, 2000; Schulenberg & Maggs, 2002). As the prevalence of substance use increases typically during adolescence, a corresponding increase in the frequency of use is likely. Thus, social development approaches to the prevention of substance use address risk and protective factors not only for initial and experimental use, but for heavy or problematic use as well.

The purpose of this study was to test the efficacy of the RHC intervention on rates of substance use during early to middle adolescence. As a social development intervention, RHC was designed to be flexible in addressing both the developmental needs and the particular goals of its target population of students and their families. Whereas a primary aim of RHC was to deter students from initiating substance use in earlier developmental periods, increasing emphasis also was placed on avoiding escalation of use. In light of this, this study addressed two related questions: First, has the intervention been efficacious in reducing students' likelihood to

use alcohol, marijuana, or cigarettes? And second, has the intervention been efficacious in altering the frequency at which students use alcohol, marijuana, or cigarettes?

Method

Participants

Participants consisted of 1st- and 2nd-grade students originally enrolled in 1 of 10 public elementary schools in an urban school district in the State of Washington. Schools were selected on the basis of having student populations with high aggregate measures of risk (e.g., percentage receiving free/reduced lunch programs, percentage with low scores on standardized achievement tests). Schools were matched on various risk factors (i.e., low income status, ethnicity, single-parent families, low reading scores, high absenteeism, and mobility), and then one school from each matched pair was assigned randomly to either an intervention ($n = 5$) or control ($n = 5$) condition. Families of 1st- and 2nd-grade students from within these schools were recruited into the longitudinal study. To be included in the RHC sample, students had to remain in their school throughout the entire first year of their participation in the study and have a parent who spoke English, Spanish, Korean, or Vietnamese. In Year 1, 938 families from 1,239 eligible students provided written consent to participate in the study. In Year 2, the sample was augmented with an additional 102 students from a second eligible pool of 131 students yielding a total sample of 1,040 students. For the analysis sample, 77 students were excluded due to having missing data for all outcome substance use measures during Grades 6 through 10. Inspection of casewise patterns of self-reported substance use indicated questionable validity for an additional four students who reported maximal levels of substance use for almost all types of substances during all measurement occasions, prompting their exclusion from the analysis. These criteria resulted in a final sample of 959 students for analysis. Fifty-four percent of the analysis sample was male,

82% was European American, 7% was Asian/Pacific Islander, 4% was African American, 4% was Hispanic, and 3% was Native American. Mean age of students at the beginning of the study was 7.7 years ($SD = 0.6$), selected from both 1st- (52%) and 2nd-grade (48%) classrooms. Twenty-eight percent of the sample was from low-income households, defined as having received AFDC, TANF, food stamps, or free/reduced lunch programs during the first 2 years of the project.

Intervention Implementation, Fidelity, and Exposure

RHC consisted of prevention strategies that addressed risk and protective factors in four key domains (see Catalano et al., 2003; and Haggerty, Catalano, Harachi, & Abbott, 1998 for a detailed description of intervention components). First, *school intervention strategies* consisted of a series of teacher and staff development workshops that included training in instructional and proactive classroom management techniques. Workshops were administered to teachers in intervention schools while students were in elementary grades and in the first year of middle school. Additionally, one-on-one classroom-based coaching sessions with teachers were conducted periodically throughout the school year to enhance fidelity of school intervention strategies. These components were designed to enhance students' learning, interpersonal, and problem-solving skills, and increase their academic performance and bonding to school. Second, individual *student intervention strategies* consisted of student participation in after-school tutoring sessions and study clubs (during Grades 4 to 6), and individualized "booster" sessions and group-based workshops (during middle and high school years) designed to (a) improve academic achievement, (b) increase students' bonding to both family and school, (c) teach refusal skills, and (d) develop prosocial beliefs regarding healthy behaviors. Third, through annual summer-camps and social skills "booster" retreats in middle school, RHC provided

universal *peer intervention strategies* for students to learn and practice social and emotional skills in the classroom and other social situations. Fourth, *family intervention strategies* consisted of multiple-session parenting workshops (e.g., “Raising Healthy Children,” “How to Help Your Child Succeed in School,” “Preparing for the Drug Free Years,” and “Parenting for Respect and Responsibility”) and in-home services for selected families. Family intervention components were delivered to families in group and individual sessions during Grades 1 through 10. These were designed to (a) enhance parents' skills in child rearing and educational support, (b) decrease family management problems and conflict, and (c) identify and clarify family standards and rules regarding student behaviors (e.g., substance use, dating, and sex). These combined strategies were implemented to promote multiple positive youth outcomes (e.g., academic success and socio-emotional competency) and avoid or reduce antisocial outcomes (e.g., substance use).

Implementation of the intervention in each of the five intervention schools was coordinated by RHC-employed school-home coordinators (SHCs). SHCs were former elementary-school teachers or education specialists with experience in providing services to parents and families and were responsible for all aspects of coordinating and implementing the intervention, including hiring, supporting, and training teachers and parents to administer school and family intervention strategies; coordinating parent and student workshops; soliciting feedback from students and parents for intervention refinement; and periodic one-on-one follow-up visits with intervention students and their families. Teacher and staff development workshops were delivered by a Staff Development Coordinator who was an experienced educational trainer with a Ph.D. in curriculum and instruction. All intervention components were manualized with intervention training sessions monitored by the Project Director to ensure fidelity to curricula materials.

The number of sessions of each intervention component that were attended by teachers, school staff, families, and students were recorded to monitor exposure to the intervention. Over 94% ($N = 140$) of eligible teachers and staff in intervention schools attended development workshops with a mean attendance of 5.7 sessions ($SD = 3.1$). All intervention students and their families received at least one intervention component with overall means of 28.3 sessions ($SD = 44.5$) received by students and 12.6 sessions ($SD = 12.3$) received by their families. For student and peer intervention strategies, 27% of intervention students attended at least one study club, 40% attended at least one of the middle-school retreats or workshops, and 51% attended at least one summer camp. For family intervention strategies, 51% of intervention students' families attended at least one group workshop, 35% received individual contacts including at-home services, and 77% received at least one middle- or high-school period "booster" workshop.

Procedure

Student data collection in Years 6 through 8 (i.e., Grades 6 through 9) consisted of both group and one-on-one survey administration in students' schools during regular school hours. Trained interviewers read aloud survey questions to students who were instructed to confidentially record their responses on a response sheet and return it to the interviewer at the end of the interview. Students who were not at school at time of data collection (e.g., were absent, home-schooled, or had dropped out of school) were contacted at home and individually administered an in-person, telephone, or mail-in survey. In Year 9, (i.e., Grades 9 and 10), a one-on-one, computer-assisted personal interviewing (CAPI) mode of data collection was used in which interviewers read survey questions aloud to students and recorded their verbal responses directly into a laptop computer data collection program. Retention rates for student surveys during project Years 6 through 10 were all greater than 88%. In order to maintain confidentiality,

students' parents, teachers, and other school personnel were not present and did not participate in any student data collection activities. All students were informed that their responses would not be shared with their parents or other school personnel. A small yearly gift or monetary compensation was given to students for their participation in each wave of the study.

Measures

Substance use outcomes. Annual substance use measures were constructed from student self-reports of frequency of alcohol, marijuana, and cigarette use during both previous year and previous month time periods. Consistent with previous adolescent alcohol use research (Bryant, Schulenberg, O'Malley, Bachman, & Johnston, 2003; Li, Duncan, & Hops, 2001), a six-point scale was created for alcohol and marijuana use where 0 = *no use in the previous year*, 1 = *some use within the past year*, 2 = *once or twice within the past month*, 3 = *three to five times within the past month*, 4 = *6 to 19 times within the past month*, and 5 = *20 or more times within the past month*. The cigarette use measure was constructed using the following categorization: 0 = *no use in the previous year*, 1 = *less than one cigarette per day*, 2 = *one to five cigarettes per day*, 3 = *6 to 10 cigarettes per day*, 4 = *11 to 20 cigarettes per day*, 5 = *21 to 40 cigarettes per day*, and 5 = *more than 40 cigarettes per day*.

Intervention status and background variables. As an intent-to-treat analysis, intervention status was assigned using students' original school assignment; that is, students from the five program schools were coded "1" and students from the five control schools were coded "0." Background variables consisted of: students' grade-cohort status (coded "0" for students from the 1st-grade cohort with substance use data from Grades 6 through 9 and "1" for students from the 2nd-grade cohort with data from Grades 7 through 10) and gender (coded "0" for females and "1" for males). Although it was not possible to test for equivalency in pre-intervention rates of substance use (i.e., the intervention began before initiation of substance use for both intervention

and control groups), it was possible for the groups to be different in their latent propensity to use substances. Therefore, two additional measures theorized to be related to adolescent substance use were included as covariates. First, a measure of classroom antisocial behavior was constructed consisting of the average of 10 items taken from either the Teacher Report Form/4-18 (TRF; Achenbach, 1991) Aggressive syndrome behavior scale or the Teacher Observation of Classroom Adaptation-Revised (TOCA-R; Werthamer-Larsson, Kellam, & Wheeler, 1991), completed by teachers at baseline (i.e., students' first year of entry into the study). Response options for the items consisted of 1 = *rarely or never true*, 2 = *sometimes true*, and 3 = *often true*. Alpha reliability coefficient for the Year 1 antisocial behavior measure scale was .91 ($M = 1.24$, $SD = .38$). Second, a baseline measure of low income status was constructed to identify families that received AFDC, TANF, food stamps, or free-lunch school programs (coded "1" for receipt of service and "0" otherwise). Intervention status and all background variables were grand mean centered for analysis.

Data Analysis

Two-part latent growth model (LGM). To address the research questions posed in this study, we employed a two-part latent growth modeling strategy (Muthén, 2001; Olsen & Schafer, 2001). As a longitudinal adaptation to two-part (or two-equation) multiple regression models (e.g., Ellickson et al., 2001; Manning, Blumberg, & Moulton, 1995; also see Manning, 1997 for a description), this strategy decomposed the original distribution of substance use outcomes into two parts, each modeled by separate, but correlated, growth functions (see Figure 1). In Part 1 of the model, nonuse was separated from the rest of the distribution by creation of binary indicator variables distinguishing any positive level of use within the previous year (coded "1") from nonuse (coded "0"). Use-versus-nonuse outcome variables for each substance were analyzed as a

random-effects logistic growth model with the log-odds of use regressed on growth factors. Intervention status and background variables were included as covariates for examination of inter-individual differences in growth trajectories. Detailed specifications for this part of the model are described in B. O. Muthén (2001) and B. O. Muthén and Asparouhov (2002).

Part 2 of the model consisted of continuous indicator variables representing the frequency of substance use, given that some use had taken place. Here, each frequency-of-use outcome was modeled as a LGM with growth factors of nonzero substance use regressed on intervention status and background variables following traditional latent growth modeling techniques for normally distributed substance use measures (e.g., Curran, 2000; Duncan & Duncan, 1996; Duncan, Duncan, Strycker, Li, & Alpert, 1999; Taylor, Graham, Cumsille, & Hansen, 2000). However, in this part of the model, substance nonuse within each time period was treated as missing data for frequency of use, following standard assumptions of data missing at random (MAR; Little & Rubin, 1987).

The procedure for constructing the two-part LGMs consisted of first identifying the unconditional (i.e., without intervention status or background variables) functional form of each part of the model separately. Change in use-versus-nonuse and frequency-of-use outcomes was modeled as linear, quadratic, or piecewise growth. Loadings for linear and quadratic growth factors were specified as orthogonal polynomial contrasts with the intercept centered at the middle of the time points (Raudenbush & Liu, 2001). Loadings for piecewise growth functions were specified as segmented linear growth functions, again with the intercept centered at the mid-point. Although growth factors between model Parts 1 and 2 were allowed to be correlated, they were also free to follow different functional forms. Thus, when intervention status and background variables subsequently were added to the unconditional models, it was possible for

them to have differential effects on growth factors for each part of the model. All models were analyzed using Mplus 3.0 (Muthén & Muthén, 2004), which provided maximum-likelihood parameter estimates with robust standard errors under MAR via numerical integration.¹

Model fit for each part of the two-part LGMs was assessed using chi-square difference tests based on model log-likelihood values and by plotting observed rates against model-predicted values and visually inspecting for misfit. Additionally, standardized residuals (i.e., observed minus model-predicted values) were plotted for each time point and assessed for potential outliers (Olsen & Schafer, 2001; Wang, Brown, & Bandeen-Roche, 2002). For frequency-of-use outcomes, model fit also was assessed using the comparative fit index (CFI; Bentler, 1990, 1992), Tucker-Lewis fit index (TLI; Tucker & Lewis, 1973), and root mean square error of approximation (RMSEA; Browne & Cudeck, 1993; Steiger & Lind, 1980).

Units of analysis. Although random assignment of students to the intervention was conducted at the school level, the majority of intervention components theorized to have direct effects on substance use (e.g., “Preparing for the Drug Free Years”) were delivered via parent and family workshops, independent of the original school-level clustering. Examination of the effects of school clustering on alcohol, marijuana, and cigarette use trajectories (i.e., linear slopes) indicated that the proportions of variance among schools were relatively small (i.e., less than .01 for all three outcomes). This was not surprising given that over half the sample (51%) had transferred out of their original schools by sixth grade. To examine the potential influence of students’ original school-level clustering on intervention effects, three additional models were examined: (a) a two-part LGM incorporating an empirical sandwich estimator for standard errors, (b) a multilevel two-part LGM, and (c) a three-level hierarchical linear model (HLM; Raudenbush & Bryk, 2002).² The latter two models incorporated the nesting of students in their

original schools as the highest level in the hierarchy and tested the effects of the intervention as a school-level effect. Comparison of results from these models with results from the original two-part LGMs demonstrated no substantive difference in conclusions made regarding the effects of the intervention or background variables. However, certain limitations existed with each of these three approaches. For example, the HLM did not allow for correlated growth factors across model Parts 1 and 2 and use of the sandwich estimator or multilevel latent growth models with small numbers of clustering units may result in poorly estimated standard errors and variance components (Heck, 2001; Jo, Muthén, Ialongo, & Brown, 2002). Noting these caveats and the consistency of results across all models, we present results from the original two-part LGM testing intervention effects at the student level.

Missing data. To determine whether there was differential attrition among students excluded from the analysis because of missing outcome data ($n = 81$), proportions of missingness were examined for intervention status and background variables. Results indicated no significant difference in the proportion of students with missing outcome data for intervention versus control groups, 1st- versus 2nd-grade cohorts, low income status, or by level of student antisocial behavior ($ps > .05$). However, a significantly greater proportion of females had missing outcome data (9.8%) than males (6.0%), $\chi^2(1, N = 1,040) = 5.03, p < .05$; therefore, a follow-up logistic regression was conducted to examine the difference in proportions of missingness between intervention and control groups by gender. Results indicated no significant Intervention Status \times Gender interaction, Wald $\chi^2(1, N = 1,040) = 1.05, p > .05$. Given these results and the small degree of missing outcome data, we relied on full information maximum likelihood estimation under the assumption of data MAR.

Results

Prevalence and Frequency of Substance Use

Prevalence rates for alcohol, marijuana, and cigarette use for the measured time periods are presented in Table 1. For marijuana and cigarette use, extremely low prevalence rates in 6th grade precluded the use of this time point in the analysis and are not shown in the table.

Prevalence rates for all three substances increased generally during Grades 6 to 10. For example, 29% of all students in 6th grade had used alcohol at least once in the previous 12 months. By Grade 10, the percentage of students who had tried alcohol in the preceding 12 months had increased to 51%. The percentage of students who used marijuana increased from 8% in 7th grade to 31% in 10th grade. Additionally, prevalence of cigarette use doubled from 9% in 7th grade to 18% in 10th grade. Rates of substance use in the RHC sample during Grade 10 were similar to population-based rates for students in the State of Washington (Washington State Department of Health, 2003). As shown in Table 1, apparent differences in rates of alcohol and marijuana use between male and female students are notable. In sixth grade, females experienced lower rates of alcohol and marijuana use (24% and 5%, respectively) than males (34% and 11%, respectively). However, by ninth grade, rates of alcohol and marijuana use by females (50% and 27%, respectively) had reached or surpassed rates of use by males (44% and 27%, respectively).

Descriptive statistics for the frequency of alcohol, marijuana, and cigarette use, for those having some use, are presented in Table 2. Longitudinal patterns of growth in frequency of alcohol and marijuana use were different from patterns of growth in prevalence rates for these two substances. Whereas the prevalence of alcohol and marijuana use increased each year during Grades 6 through 10, mean frequency of alcohol and marijuana use peaked at eighth grade and declined thereafter. Mean frequency of cigarette use, however, increased during Grades 7 to 10. Excluding nonusers from the frequency-of-use distributions substantially improved the normality

of the outcomes. For example, skewness and kurtosis for frequency of alcohol use in seventh grade decreased from 1.94 and 3.59, respectively, for the entire sample to 1.31 and 1.37, respectively, for users only. For frequency of marijuana use in seventh grade, skewness and kurtosis decreased from 4.35 and 19.46 to 0.60 and -0.87, respectively. Similarly, skewness and kurtosis for frequency of cigarette use in seventh grade decreased from 4.91 and 30.35 to 1.99 and 3.45, respectively.

Two-part Latent Growth Model of Alcohol Use

Unconditional model. Comparison of intercept-only, linear, quadratic, and piecewise growth functions for Part 2 of the alcohol use model indicated that frequency of alcohol use was best modeled as a two-segment piecewise model consisting of separate linear growth functions for Grades 6 to 8 and Grades 8 to 10, $\chi^2(8, N = 628) = 13.52, p = .10, CFI = .944, TLI = .937,$ and $RMSEA = .033$.³ Although prevalence rates for alcohol use (versus nonuse) in Part 1 of the model appeared to increase linearly throughout Grades 6 through 10, previous studies have shown covariates to have potentially differential impact on middle and high school growth segments for adolescent substance use (Crawford, Pentz, Chou, Li, & Dwyer, 2003; Li et al., 2001), therefore, we modeled alcohol use in Part 1 of the model in a similar piecewise fashion.

Examination of growth factor variances and covariances indicated significant variation in intercept (i.e., Grade 8 status) growth factors for both Part-1 and Part-2 outcomes (Variances = 5.105 and .341, $SEs = .567$ and $.060, ps < .001$, respectively) and significant covariation between intercept growth factors ($r = .686, p < .001$). Growth factor variances for both outcomes were estimated at zero for linear growth during Grades 6 to 8 (and set to zero for subsequent analysis of intervention status and background variables) and freely estimated for linear growth during Grades 8 to 10 (Variances = .576 and .048, $SEs = .306$ and $.041, ps > .05$, for model Parts 1 and

2, respectively). The covariance between Grade 8 to 10 linear growth factors, however, was required to be fixed at zero in order to obtain stable parameter estimates.

Intervention status and background variables. Intervention status and background variables were added to both parts of the model and regressed on intercept and piecewise growth segments. Parameter coefficients (i.e., growth factor means) and standard errors for the final two-part LGM are shown in Table 3. Results of the alcohol use-versus-nonuse part of the model indicated a significant gender effect with females being more likely to use alcohol at Grade 8 and having a significantly greater rate of increase in their likelihood to use alcohol during Grades 6 through 8 relative to males. Higher baseline classroom antisocial behavior was associated with both a greater likelihood to use alcohol at Grade 8 and growth in the likelihood to use alcohol during Grades 8 to 10. Additionally, students from low socioeconomic status households were at greater likelihood of using alcohol at Grade 8 and had greater growth in use during Grades 6 to 8. No significant difference was found between students in the intervention group and controls in change in alcohol use versus nonuse ($p > .05$).

Results of the frequency-of-alcohol use part of the model indicated a significant intervention effect indicating a lower linear decline in the frequency of alcohol use during Grades 8 to 10 for the intervention group relative to controls (see Figure 2). The standardized effect size for the difference in mean trajectories was $\delta = .91$.⁴ In terms of an adjusted mean difference in frequency-of-use rates at Grade 10, the corresponding effect size was $d = .53$ (Cohen, 1992). Additionally, a significant grade-cohort effect was present for growth in frequency of alcohol use during Grades 8 through 10 with a greater decline for the 1st-grade cohort than the 2nd-grade cohort. To determine whether the intervention effect was consistent for both grade cohorts, an Intervention Status \times Grade Cohort interaction term was added to the

Grade-8-through-10 segment of the model. Results indicated that the interaction term had no significant effect on growth in frequency of alcohol use during this period ($\beta = -.089$, $SE = .139$, $p > .05$), indicating that grade-cohort status did not moderate the effects of the intervention on frequency of alcohol use.

Two-part Latent Growth Model of Marijuana Use

Unconditional model. Given the apparent nonlinear growth in marijuana use during Grades 7 through 10, a curvilinear growth model for the Part 1 use-versus-nonuse outcome containing intercept, linear, and quadratic growth factors was compared to an intercept-and-linear-only growth model (because only 4 time points were available to model marijuana use, the two-segment piecewise model was not considered). Results indicated better fit for the curvilinear model than the linear model, $\Delta\chi^2(1, N = 959) = 5.40$, $p < .01$. The unconditional curvilinear model for the Part 2 frequency of marijuana use exhibited marginally significant negative linear growth ($\beta = -.047$, $SE = .030$, $p = .058$) and nonsignificant quadratic growth ($\beta = -.057$, $SE = .047$, $p > .05$). However, fit of the intercept-only model was poor, $\chi^2(8, N = 340) = 15.75$, $p = .046$, $CFI = .718$, $TLI = .789$, and $RMSEA = .053$.⁵ Inclusion of a linear growth factor substantially improved model fit, $\chi^2(5, N = 340) = 8.46$, $p = .133$, $CFI = .890$, $TLI = .890$, and $RMSEA = .038$;⁵ therefore, the linear growth term was retained in the final unconditional model for frequency of marijuana use.

Significant variation existed in intercept (i.e., Grade 8.5 status) growth factors for both use-versus-nonuse and frequency-of-use outcomes (Variances = 9.113 and .691, $SEs = 1.251$ and .154, $ps < .001$, respectively). Growth factor intercepts between outcomes were significantly correlated ($r = .796$, $p < .001$). In Part 1 of the model, variances for linear and quadratic growth parameters both were estimated (and subsequently fixed) at zero. In Part 2 of the model, the

variance for the linear growth factor was freely estimated but was nonsignificant (Variance = .024, $SE = .017$, $p > .05$).

Intervention status and background variables. Results of the final two-part growth latent model for marijuana use, including intervention status and background variables, are shown in Table 4. Significant gender, grade cohort, baseline antisocial behavior, and income effects were found for the intercept growth factor in Part 1 of the model indicating that females, 2nd-grade-cohort students, students with high baseline antisocial behavior, and students from low socioeconomic status households had significantly higher rates of marijuana use (vs. nonuse) at Grade 8.5 than their respective counterparts. Additionally, during Grades 7 to 10, marijuana use increased more for (a) females than males and (b) students with higher baseline levels of antisocial behavior. No significant differences were found in marijuana use growth rates between intervention students and controls ($p > .05$). However, for frequency of marijuana use, results indicated a significant intervention effect with students in the intervention group exhibiting greater linear decline in the frequency of marijuana use than students in the control group (see Figure 3). Intervention effect sizes were $\delta = 1.44$ for the standardized difference in mean trajectories and $d = .53$ for the adjusted mean difference in frequency-of-use rates at Grade 10.

Two-part Latent Growth Model of Cigarette Use

Unconditional model. For the unconditional cigarette use-versus-nonuse outcome, results of the unconditional model indicated better fit with intercept, linear, and quadratic growth factors than the intercept-and-linear-only model, $\Delta\chi^2(1, N = 959) = 6.31$, $p < .01$. For the frequency-of-use outcome, a quadratic growth model similarly provided optimal fit to the data, $\chi^2(1, N = 239) = 6.93$, $p = .33$, CFI = .953, TLI = .953, and RMSEA = .026.⁶ Among growth factors for both parts of the model, significant variation existed only for use-versus-nonuse and frequency-of-use

intercepts (i.e., Grade 8.5 status; Variances = 10.342 and .865, *SEs* = 1.531 and .190, *ps* < .001, respectively). Again, growth factor intercepts between model Parts 1 and 2 were highly correlated ($r = .856$, $p < .001$). All other variances and covariances in the cigarette use two-part model were fixed at zero.

Intervention status and background variables. Results of the final two-part LGM of cigarette use, including intervention status and background variables, are shown in Table 5.⁷ Similar to marijuana use, significant effects for background variables indicated that females, 2nd-grade-cohort students, students with high baseline antisocial behavior, and students from low socioeconomic status households had higher rates of cigarette use (vs. nonuse) at Grade 8.5. Although a trend toward decreased cigarette use (vs. nonuse) was evident for intervention students relative to controls (see Figure 4), the effect of the intervention on the linear growth factor failed to achieve statistical significance ($p = .08$). The only significant effect for frequency of cigarette use was for baseline antisocial behavior with higher levels related significantly to more cigarette smoking at Grade 8.5. No other background variables were associated with change in either cigarette use-versus-nonuse or frequency-of-use outcomes.

Discussion

This study examined the efficacy of the Raising Healthy Children (RHC) intervention on trajectories of alcohol, marijuana, and cigarette use during early to middle adolescence. Using the social development model as a theoretical framework for the intervention, RHC targeted a broad set of empirically supported risk and protective factors through the multiple contexts of school, family, peers, and the individual student. As the aims of the intervention were designed to be both developmentally appropriate and consistent with the goals of its participating families,

we investigated students' substance use in terms of the likelihood to abstain from use as well as the frequency of use for those who did not abstain from use.

These related outcomes were analyzed using a two-part latent growth modeling (LGM) strategy. Similar to standard LGM techniques, this method allows for the examination of both intra- and inter-individual patterns of change in substance use trajectories. However, the two-part LGM decomposes the original semicontinuous outcome measures into dichotomous use-versus-nonuse and continuous frequency-of-use parts. In addition to providing a more detailed examination of the effects of the intervention, this approach substantially improved the normality of the frequency-of-use outcomes—a fundamental assumption underlying the appropriateness of LGMs in general. Consequently, we recommend this approach to other researchers faced with similarly distributed outcomes.

Results of this study provide evidence for the efficacy of the RHC intervention in reducing the frequency of alcohol and marijuana use. Between-group examination of alcohol and marijuana frequency-of-use trajectories shows greater decreases for intervention students relative to controls during middle to high-school periods. Standardized effect sizes associated with mean trajectory differences are substantial (.91 and 1.44, respectively), representing almost a full standard deviation unit difference in mean alcohol frequency-of-use trajectories and almost a 1½ standard deviation unit difference in mean marijuana frequency-of-use trajectories between intervention students and controls. In terms of adjusted mean differences in frequency-of-use rates at Grade 10, corresponding effects sizes represent medium to large intervention effects (.53 and .72, respectively). Although these findings support the intervention's goal of reducing frequent use, the lack of significant intervention effects on students' decision to engage in alcohol or marijuana use demonstrates a lack of support for the intervention's abstinence-

oriented goals regarding these two substances. However, for cigarette use, the finding of a marginally significant trend for intervention students demonstrates the potential of preventive interventions to affect different parts of the use distribution.

The differential impact of the RHC intervention on alcohol-, marijuana-, and cigarette-use outcomes is noteworthy. From a social development perspective, intervention students' bonding with those with prosocial beliefs and standards is keeping them from more frequent alcohol and marijuana use, which would disappoint those they are bonded to and threaten their investment in school or family relations if they were to do otherwise. On the other hand, experimentation with alcohol and marijuana, perhaps because of low risk of detection or acceptance as a rite of passage, may not pose as great a threat to bond disruption. Consequently, experimental use may not be as amenable to social development interventions. Findings by Ellickson et al. (2001) note the distinction between experimental and problematic use, suggesting that "prevention programs that target alcohol misuse may be more successful than those that advocate abstinence" (p. 773). In contrast, the highly addictive nature of cigarette smoking and knowledge of its deleterious health effects may account for its different relationship with the intervention. Experimental cigarette use is less likely to be considered normative, as evidenced by its low prevalence in our sample compared to alcohol or marijuana use. Furthermore, the greater potential for cigarette addiction may make escalating (i.e., more frequent) use less susceptible to social development intervention. More research is needed to disentangle the mediating processes regarding the effects of preventive interventions on students' decisions to use these substances.

Differences in the longitudinal patterns of substance between use-versus-nonuse and frequency-of-use outcomes within each type of substance are noteworthy, as well. Results of this

study showed that, whereas prevalence rates for alcohol and marijuana use increased during the middle- to early high-school period, frequency-of-use patterns for these substances were either nonlinear (for alcohol) or remained relatively unchanged (for marijuana). Conversely, although the prevalence of cigarette use changed very little during Grades 8 to 10, frequency of cigarette use increased steadily during the same period. Although different longitudinal patterns are apparent between use-versus-nonuse and frequency-of-use outcomes within each substance, we note that growth processes between outcomes are related, nonetheless. Large correlations ($.69 < rs < .86$) between growth factor intercepts in each model demonstrate the dependence of escalating substance use on the initial decision to use or not use. We caution researchers using two-part models to consider such dependencies in their analysis.

Results of this study also demonstrate that predictor variables can have differential effects on patterns of substance use depending on level of use. Gender, for example, was related to patterns of alcohol and marijuana use with female prevalence rates “catching up” to males’ rates by 10th grade. However, gender was not associated with patterns of frequency of alcohol or marijuana use. These findings are consistent with results from other studies that have found differential effects of risk factors on level-dependent substance use outcomes (Colder & Chassin, 1999; Gutierrez, Molof, & Ungerleider, 1994; Olsen & Schafer, 2001; Scheier & Newcomb, 1991). This suggests that the decision-making processes that adolescents go through with regard to substance use may not be uniform across all levels of use (e.g., Schulenberg & Maggs, 2002), that adolescents constitute heterogeneous populations characterized by distinct substance-use distributions (e.g., Colder, Campbell, Ruel, Richardson, & Flay, 2002; Li et al., 2001; Muthén & Muthén, 2000; Oxford et al., 2003), or both.

Limitations

Although this study addresses several methodological deficits that often characterize prevention studies of adolescent substance use (e.g., nonexperimental design, lack of theoretical or empirical basis, lack of longitudinal data, differential attrition), the study is limited by relying solely on adolescent self-reported substance use (although this is not uncommon; see Campanelli, Dielman, & Shope, 1987; Needle, McCubbin, Lorence, & Hochhauser, 1983; and Smith, McCarthy, & Goldman, 1995 on the reliability and validity of self-reported substance use) and by the predominantly European American composition of the sample. Additionally, this study did not investigate the timing of substance use initiation (i.e., age onset of first use) nor did it examine whether the efficacy of the RHC intervention on frequency-of-use was due to the same students actually decreasing their frequencies of use or by new students, characterized by later age-onset and lower levels of use, entering into the frequency-of-use trajectories. Finally, this study did not exhaustively examine other explanatory variables (i.e., risk and protective factors) with regard to their potential prediction of substance use. As the focus of the study was to test the efficacy of the RHC intervention, covariates were limited to those variables that had well established predictive relationships with substance use (e.g., antisocial behavior and low socioeconomic status) and could statistically control for pretest differences between intervention and control students.

Conclusion

As a comprehensive, longitudinal preventive intervention with universal and selective components, the Raising Healthy Children project incorporates principles of effective prevention programs (Nation et al., 2003) to address empirically identified and developmentally appropriate risk and protective factors for adolescent substance use. Although the effects of the intervention presented in this study are limited, they support the efficacy of the intervention in reducing the

frequency of alcohol and marijuana use. It will be important to see if these effects demonstrated primarily in middle and early high school are maintained and are associated with outcomes related to heavy or problematic use as students reach the ages of peak use. Additional research into the etiology of patterns of different levels of substance use (e.g., using two-part latent growth models) may help clarify the causal mechanisms leading to successful prevention interventions.

References

- Achenbach, T. M. (1991). *Manual for the Teacher's Report Form and 1991 Profile*. Burlington: University of Vermont Department of Psychiatry.
- Akers, R. L. (1985). *Deviant behavior: A social learning approach* (3rd ed.). Belmont, CA: Wadsworth.
- Akers, R. L., Krohn, M., Lanza-Kaduce, L., & Radosevich, M. (1979). Social learning and deviant behavior: A specific test of a general theory. *American Sociological Review, 44*, 636-655.
- Bandura, A. (1973). *Aggression: A social learning analysis*. Englewood Cliffs, NJ: Prentice Hall.
- Bauman, K. E., Foshee, V. A., Ennett, S. T., Hicks, K., & Pemberton, M. (2001). Family matters: A family-directed program designed to prevent adolescent tobacco and alcohol use. *Health Promotion Practice, 2*, 81-96.
- Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107*, 238-246.
- Bentler, P. M. (1992). On the fit of models to covariances and methodology to the Bulletin. *Psychological Bulletin, 112*, 400-404.
- Botvin, G. J., & Griffin, K. W. (2002). Life skills training as a primary prevention approach for adolescent drug abuse and other problem behaviors. *International Journal of Emergency Mental Health, 4*, 41-48.
- Brook, J. S., Brook, D. W., Arencibia-Mireles, O., Richter, L., & Whiteman, M. (2001). Risk factors for adolescent marijuana use across cultures and across time. *Journal of Genetic Psychology, 162*, 357-374.

- Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136-162). Newbury Park, CA: Sage.
- Bry, B. H., McKeon, P., & Pandina, R. J. (1982). Extent of drug use as a function of number of risk factors. *Journal of Abnormal Psychology, 91*, 273-279.
- Bryant, A. L., Schulenberg, J. E., O'Malley, P. M., Bachman, J. G., & Johnston, L. D. (2003). How academic achievement, attitudes, and behaviors relate to the course of substance use during adolescence: A 6-year, multiwave national longitudinal study. *Journal of Research on Adolescence, 13*, 361-397.
- Burgess, R. L., & Akers, R. L. (1966). A differential association-reinforcement theory of criminal behavior. *Social Problems, 4*, 128-147.
- Campanelli, P. C., Dielman, T. E., & Shope, J. T. (1987). Validity of adolescents' self-reports of alcohol use and misuse using a bogus pipeline procedure. *Adolescence, 22*, 7-22.
- Catalano, R. F., & Hawkins, J. D. (1996). The social development model: A theory of antisocial behavior. In J. D. Hawkins (Ed.) *Delinquency and crime: Current theories* (pp. 149-197). New York: Cambridge University Press.
- Catalano, R. F., Mazza, J. J., Harachi, T. W., Abbott, R. D., Haggerty, K. P., & Fleming, C. B. (2003). Raising healthy children through enhancing social development in elementary school: Results after 1.5 years. *Journal of School Psychology, 41*, 143-164.
- Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*, 155-159.
- Coie, J. D., Watt, N. F., West, S. G., Hawkins, J. D., Asarnow, J. R., Markman, H. J., et al. (1993). The science of prevention. A conceptual framework and some directions for a national research program. *American Psychologist, 48*, 1013-1022.

Colder, C. R., Campbell, R. T., Ruel, E., Richardson, J. L., & Flay, B. R. (2002). A finite mixture model of growth trajectories of adolescent alcohol use: Predictors and consequences. *Journal of Consulting and Clinical Psychology, 70*, 976-985.

Colder, C. R., & Chassin, L. (1999). The psychosocial characteristics of alcohol users versus problem users: Data from a study of adolescents at risk. *Development and Psychopathology, 11*, 321-348.

Conduct Problems Prevention Research Group. (1992). A developmental and clinical model for the prevention of conduct disorders: The FAST Track Program. *Development and Psychopathology, 4*, 509-527.

Conduct Problems Prevention Research Group. (2002). Predictor variables associated with positive Fast Track outcomes at the end of third grade. *Journal of Abnormal Child Psychology, 30*, 37-52.

Crawford, A. M., Pentz, M. A., Chou, C., Li, C., & Dwyer, J. H. (2003). Parallel developmental trajectories of sensation seeking and regular substance use in adolescents. *Psychology of Addictive Behaviors, 17*, 179-192.

Curran, P. J. (2000). A latent curve framework for the study of developmental trajectories in adolescent substance use. In J. S. Rose, L. Chassin, C. C. Presson, & S. J. Sherman (Eds.), *Multivariate applications in substance use research: New methods for new questions* (pp. 1-42). Mahwah, NJ: Erlbaum.

Duncan, S. C., & Duncan, T. E. (1996). A multivariate latent growth curve analysis of adolescent substance use. *Structural Equation Modeling, 3*, 323-347.

- Duncan, T. E., Duncan, S. C., Strycker, L. A., Li, F., & Alpert, A. (1999). *An introduction to latent variable growth curve modeling: Concepts, issues, and applications*. Mahwah, NJ: Lawrence Erlbaum Associates
- Eddy, J., Reid, J. B., & Fetrow, R. A. (2000). An elementary school-based prevention program targeting modifiable antecedents of youth delinquency and violence: Linking the Interests of Families and Teachers (LIFT). *Journal of Emotional & Behavioral Disorders, 8*, 165-176.
- Ellickson, P. L., & Bell, R. M. (1990). Drug prevention in junior high: A multi-site longitudinal test. *Science, 247*, 1299-1305.
- Ellickson, P. L., Bell, R. M., & Harrison, E. R. (1993). Changing adolescent propensities to use drugs: Results from Project ALERT. *Health Education Quarterly, 20*, 227-242.
- Ellickson, P. L., Tucker, J. S., Klein, D. J., & McGuigan, K. A. (2001). Prospective risk factors for alcohol misuse in late adolescence. *Journal of Studies on Alcohol, 62*, 773-782.
- Farrington, D. P., & Hawkins, J. D. (1991). Predicting participation, early onset and later persistence in officially recorded offending. *Criminal Behaviour and Mental Health, 1*, 1-33.
- Felix-Ortiz, M., & Newcomb, M. D. (1999). Vulnerability for drug use among Latino adolescents. *Journal of Community Psychology, 27*, 257-280.
- Grant, B. F., & Dawson, D. A. (1997). Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Substance Abuse, 9*, 103-110.

- Greenberg, M. T., & Kusche, C. A. (1997, April). *Improving children's emotion regulation and social competence: The effects of the PATHS curriculum*. Paper presented at the Annual Meeting of the Society for Research in Child Development, Washington, DC.
- Griffin, K. W., Botvin, G. J., Nichols, T. R., & Doyle, M. M. (2003). Effectiveness of a universal drug abuse prevention approach for youth at high risk for substance use initiation. *Preventive Medicine, 36*, 1-7.
- Griffin, K. W., Botvin, G. J., Scheier, L. M., & Nichols, T. R. (2002). Factors associated with regular marijuana use among high school students: A long-term follow-up study. *Substance Use and Misuse, 37*, 225-238.
- Griffin, K. W., Scheier, L. M., Botvin, G. J., & Diaz, T. (2000). Ethnic and gender differences in psychosocial risk, protection, and adolescent alcohol use. *Prevention Science, 1*, 199-212.
- Gutierrez, S. E., Molof, M., & Ungerleider, S. (1994). Relationship of "risk" factors to teen substance use: A comparison of abstainers, infrequent users, and frequent users. *International Journal of the Addictions, 29*, 1559-1579.
- Haggerty, K. P., Catalano, R. F., Harachi, T. W., & Abbott, R. D. (1998). Description de l'implementation d'un programme de prévention des problèmes de comportement à l'adolescence. (Preventing adolescent problem behaviors: A comprehensive intervention description). *Criminologie, 31*, 25-47.
- Hansen, W. B., & Graham, J. W. (1991). Preventing alcohol, marijuana, and cigarette use among adolescents: Peer pressure resistance training versus establishing conservative norms. *Preventive Medicine, 20*, 414-430.

- Hawkins, J. D., Catalano, R. F., Kosterman, R., Abbott, R., & Hill, K. G. (1999). Preventing adolescent health-risk behaviors by strengthening protection during childhood. *Archives of Pediatrics and Adolescent Medicine, 153*, 226-234.
- Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance-abuse prevention. *Psychological Bulletin, 112*, 64-105.
- Hawkins, J. D., Graham, J. W., Maguin, E., Abbott, R. D., Hill, K. G., & Catalano, R. F. (1997). Exploring the effects of age of alcohol use initiation and psychosocial risk factors on subsequent alcohol misuse. *Journal of Studies on Alcohol, 58*, 280-290.
- Hawkins, J. D., Guo, J., Hill, K. G., Battin-Pearson, S., & Abbott, R. D. (2001). Long term effects of the Seattle Social Development intervention on school bonding trajectories. *Applied Developmental Science: Special issue: Prevention as altering the course of development, 5*, 225-236.
- Hawkins, J. D., & Weis, J. G. (1985). The social development model: An integrated approach to delinquency prevention. *Journal of Primary Prevention, 6*, 73-97.
- Heck, R. H. (2001). Multilevel modeling with SEM. In G. A. Marcoulides & R. E. Schumacker (Eds.), *New developments and techniques in structural equation modeling* (pp. 89-127). Mahwah, NJ: Lawrence Erlbaum.
- Hirschi, T. (1969). *Causes of delinquency*. Berkeley, CA: University of California Press.
- Hops, H., Davis, B., & Lewin, L. M. (1999). The development of alcohol and other substance use: A gender study of family and peer context. *Journal of Studies on Alcohol, Suppl. 13*, 22-31.

- Howell, J. C., Krisberg, B., Hawkins, J. D., & Wilson, J. J. (Eds.). (1995). *A sourcebook: Serious, violent, and chronic juvenile offenders*. Thousand Oaks, CA: Sage.
- Jessor, R., Van Den Bos, J., Vanderryn, J., Costa, F. M., & Turbin, M. S. (1995). Protective factors in adolescent problem behavior: Moderator effects and developmental change. *Developmental Psychology, 31*, 923-933.
- Jo, B., Muthén, B. O., Ialongo, N. S., & Brown, C. H. (2002). *Cluster randomized trials with nonadherence*. Manuscript submitted for publication.
- Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (2003). *Monitoring the Future national survey results on drug use, 1975-2002. Volume I: Secondary school students*. (NIH Publication No. 03-5375). Washington, DC: National Institute on Drug Abuse.
- Kellam, S. G., Rebok, G. W., Ialongo, N. S., & Mayer, L. S. (1994). The course and malleability of aggressive behavior from early first grade into middle school: Results of a developmental epidemiology-based preventive trial. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 35*, 259-281.
- Li, F., Duncan, T. E., & Hops, H. (2001). Examining developmental trajectories in adolescent alcohol use using piecewise growth mixture modeling analysis. *Journal of Studies on Alcohol, 62*, 199-210.
- Little, R. J. A., & Rubin, D. B. (1987). *Statistical analysis with missing data*. New York: John Wiley and Sons.
- Lonczak, H. S., Abbott, R. D., Hawkins, J. D., Kosterman, R., & Catalano, R. F. (2002). Effects of the Seattle Social Development Project on sexual behavior, pregnancy, birth, and sexually transmitted disease outcomes by age 21 years. *Archives of Pediatrics and Adolescent Medicine, 156*, 438-447.

- Maggs, J. L., & Schulenberg, J. (1998). Reasons to drink and not to drink: Altering trajectories of drinking through an alcohol misuse prevention program. *Applied Developmental Science, 2*, 48-60.
- Manning, W. (1997). Alternative econometric models of alcohol demand. In K. J. Bryant & M. Windle (Eds.), *The science of prevention: Methodological advances from alcohol and substance abuse research* (pp. 101-121). Washington, DC: American Psychological Association.
- Manning, W. G., Blumberg, L., & Moulton, L. H. (1995). The demand for alcohol: The differential response to price. *Journal of Health Economics, 14*, 123-148.
- Marlatt, G. A. (1996). Harm reduction: Come as you are. *Addictive Behaviors, 21*, 779-788.
- Marlatt, G. A., & Witkiewitz, K. (2002). Harm reduction approaches to alcohol use: Health promotion, prevention, and treatment. *Addictive Behaviors, 27*, 867-886.
- Marshall, M. P., & Chassin, L. (2000). Peer influence on adolescent alcohol use: The moderating role of parental support and discipline. *Applied Developmental Science, 4*, 80-88.
- Matsueda, R. L. (1982). Testing control theory and differential association: A causal modeling approach. *American Sociological Review, 47*, 489-504.
- Matsueda, R. L. (1988). The current state of differential association theory. *Crime and Delinquency, 34*, 277-306.
- McBride, N., Midford, R., Farrington, F., & Phillips, M. (2000). Early results from a school alcohol harm minimization study: The School Health and Alcohol Harm Reduction Project. *Addiction, 95*, 1021-1042.

- Mrazek, P. J., & Haggerty, R. J., (Eds.), Committee on Prevention of Mental Disorders, Institute of Medicine. (1994). *Reducing risks for mental disorders: Frontiers for prevention intervention research*. Washington, DC: National Academy Press.
- Muthén, B., & Asparouhov, T. (2002, December 9). Latent variable analysis with categorical outcomes: Multiple-group and growth modeling in Mplus. *Mplus Web Notes: No. 4, Version 5*. Retrieved January 27, 2004, from the World Wide Web: <http://www.statmodel.com/mplus/examples/webnote.html>.
- Muthén, B., & Muthén, L. K. (2000). Integrating person-centered and variable-centered analyses: Growth mixture modeling with latent trajectory classes. *Alcoholism: Clinical and Experimental Research, 24*, 882-891.
- Muthén, B. O. (2001). *Two-part growth mixture modeling*. Unpublished manuscript.
- Muthén, L. K., & Muthén, B. O. (2004). *Mplus user's guide* (3rd ed.). Los Angeles, CA: Muthén & Muthén.
- Nation, M., Crusto, C., Wandersman, A., Kumpfer, K. L., Seybolt, D., Morrissey-Kane, E., et al. (2003). What works in prevention: Principles of effective prevention programs. *American Psychologist, 58*, 449-456.
- Needle, R., McCubbin, H. I., Lorence, J., & Hochhauser, M. (1983). Reliability and validity of adolescent self-reported drug use in a family-based study: A methodological report. *International Journal of the Addictions, 18*, 901-912.
- Newcomb, M. D., Maddahian, E., & Bentler, P. M. (1986). Risk factors for drug use among adolescents: concurrent and longitudinal analyses. *American Journal of Public Health, 76*, 525-531.

- Olsen, M. K., & Schafer, J. L. (2001). A two-part random-effects model for semicontinuous longitudinal data. *Journal of the American Statistical Association, 96*, 730-745.
- Oxford, M. L., Gilchrist, L. D., Morrison, D. M., Gillmore, M. R., Lohr, M. J., & Lewis, S. M. (2003). Alcohol use among adolescent mothers: Heterogeneity in growth curves, predictors, and outcomes of alcohol use over time. *Prevention Science, 4*, 15-26.
- Pedersen, W., & Skrondal, A. (1998). Alcohol consumption debut: predictors and consequences. *Journal of Studies on Alcohol, 59*, 32-42.
- Pentz, M. A., Trebow, E. A., Hansen, W. B., & MacKinnon, D. P. (1990). Effects of program implementation on adolescent drug use behavior: The Midwestern Prevention Project (MPP). *Evaluation Review, 14*, 264-289.
- Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (2nd ed.). Newbury Park, CA: Sage.
- Raudenbush, S. W., & Liu, X. F. (2001). Effects of study duration, frequency of observation, and sample size on power in studies of group differences in polynomial change. *Psychological Methods, 6*, 387-401.
- Scheier, L. M., Botvin, G. J., & Baker, E. (1997). Risk and protective factors as predictors of adolescent alcohol involvement and transitions in alcohol use: A prospective analysis. *Journal of Studies on Alcohol, 58*, 652-667.
- Scheier, L. M., & Newcomb, M. D. (1991). Differentiation of early adolescent predictors of drug use versus abuse: A developmental risk-factor model. *Journal of Substance Abuse, 3*, 277-299.
- Schulenberg, J., Wadsworth, K. N., O'Malley, P. M., Bachman, J. G., & Johnston, L. D. (1996). Adolescent risk factors for binge drinking during the transition to young adulthood:

- Variable- and pattern-centered approaches to change. *Developmental Psychology*, 32, 659-674.
- Schulenberg, J. E., & Maggs, J. L. (2002). A developmental perspective on alcohol use and heavy drinking during adolescence and the transition to young adulthood. *Journal of Studies on Alcohol*, 14, 54-70.
- Simons-Morton, B., Haynie, D. L., Crump, A. D., Saylor, K. E., Eitel, P., & Yu, K. (1999). Expectancies and other psychosocial factors associated with alcohol use among early adolescent boys and girls. *Addictive Behaviors*, 24, 229-238.
- Smith, G. T., McCarthy, D. M., & Goldman, M. S. (1995). Self-reported drinking and alcohol-related problems among early adolescents: Dimensionality and validity over 24 months. *Journal of Studies on Alcohol*, 56, 383-394.
- Spoth, R. L., Redmond, C., Hockaday, C., & Yoo, S. (1996). Protective factors and young adolescent tendency to abstain from alcohol use: A model using two wave of intervention study data. *American Journal of Community Psychology*, 24, 749-770.
- Spoth, R. L., Redmond, C., & Shin, C. (2001). Randomized trial of brief family interventions for general populations: Adolescent substance use outcomes 4 years following baseline. *Journal of Consulting and Clinical Psychology*, 69, 627-642.
- Steiger, J. H., & Lind, J. C. (1980). *Statistically based tests for the number of common factors*. Paper presented at the annual meeting of the Psychometric Society, Iowa City, IA.
- Taylor, B. J., Graham, J. W., Cumsille, P., & Hansen, W. B. (2000). Modeling prevention program effects on growth in substance use: Analysis of five years of data from the Adolescent Alcohol Prevention Trial. *Prevention Science*, 1, 183-197.

- Tucker, L. R., & Lewis, C. (1973). A reliability coefficient for maximum likelihood factor analysis. *Psychometrika*, 38, 1-10.
- Vitaro, F., Tremblay, R. E., & Zoccolillo, M. (1999). Paternal alcoholism, drug use in adolescence and protective factors. *Canadian Journal of Psychiatry - Revue Canadienne de Psychiatrie*, 44, 901-908.
- Wang, C. P., Brown, C. H., & Bandeen-Roche, K. (2002). *Residual diagnostics for growth mixture models*. Manuscript submitted for publication.
- Washington State Department of Health. (2003). *2002 Healthy Youth Survey*. Retrieved September 15, 2003, from the World Wide Web: <http://www3.doh.wa.gov/HYS>.
- Werthamer-Larsson, L., Kellam, S. G., & Wheeler, L. (1991). Effect of first-grade classroom environment on shy behavior, aggressive behavior, and concentration problems. *American Journal of Community Psychology*, 19, 585-602.
- Williams, J. H., Ayers, C. D., Abbott, R. D., Hawkins, J. D., & Catalano, R. F. (1999). Racial differences in risk factors for delinquency and substance use among adolescents. *Social Work Review*, 23, 241-256.

Footnotes

¹Mplus input scripts used in the analyses are available from the first author upon request.

²Substance use-versus-nonuse was analyzed using a three-level hierarchical generalized linear model (HGLM) with a logit-link function for the binary outcome.

³Fit indices based on $n = 628$ students with nonzero frequency of alcohol use.

⁴ δ is defined as the group difference in a growth factor divided by the population standard deviation of that growth factor (see Raudenbush & Liu, 2001, Equation 13).

⁵Fit indices based on $n = 340$ students with nonzero frequency of marijuana use.

⁶Fit indices based on $n = 239$ students with nonzero frequency of cigarette use.

⁷Given the high degree of skewness and kurtosis for Grade 7 frequency of cigarette use, parallel analyses were conducted with log transformed outcome data. Results indicated no substantive differences between analyses with log transformed and untransformed outcomes; therefore, for consistency, we report results from analysis of cigarette use in the original metric.

Table 1

Annual Substance Use Prevalence Rates by Intervention Status and Gender

Grade	Intervention	Controls	Females	Males	Total Sample
Alcohol					
6 ^a	.29	.30	.24	.34	.29
7	.33	.29	.29	.33	.31
8	.37	.40	.43	.34	.38
9	.46	.48	.50	.44	.47
10 ^b	.52	.50	.52	.50	.51
Marijuana					
7	.08	.09	.05	.11	.08
8	.16	.18	.16	.18	.17
9	.25	.28	.27	.27	.27
10 ^b	.30	.31	.27	.33	.31
Cigarettes					
7	.09	.08	.10	.08	.09
8	.14	.13	.17	.11	.14
9	.16	.17	.18	.15	.16
10 ^b	.16	.20	.20	.16	.18

Note. Prevalence rates denote the proportion of students having used each substance within the previous 12 months.

^aRepresents 1st-grade cohort only. ^bRepresents 2nd-grade cohort only.

Table 2

Descriptive Statistics for Frequency of Substance Use

Grade	N	Mean	Standard Deviation	Skewness	Kurtosis
Alcohol					
6 ^a	143	1.57	0.88	1.97	4.32
7	297	1.85	0.99	1.31	1.37
8	361	2.05	1.07	0.91	0.03
9	430	1.98	1.08	1.02	0.24
10 ^b	227	1.81	1.06	1.34	1.04
Marijuana					
7	79	2.41	1.33	0.60	-0.87
8	158	2.58	1.44	0.53	-1.10
9	245	2.29	1.31	0.72	-0.77
10 ^b	136	2.21	1.39	0.77	-0.87
Cigarettes					
7	82	1.68	0.95	1.99	3.45
8	128	2.02	1.29	1.32	1.30
9	149	2.03	1.16	1.25	1.49
10 ^b	80	2.09	1.06	0.88	0.91

Note. Scale ranges from 1 (some use within the past year) to 5 (20 or more times within the past month).

^aRepresents 1st-grade cohort only. ^bRepresents 2nd-grade cohort only.

Table 3

Means and Standard Errors for Alcohol Use Growth Factors and Covariates

Variable	Grade 8 status		Linear growth Grades 6 to 8 ^a		Linear growth Grades 8 to 10	
	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>
Part 1: Use versus nonuse						
Growth factor mean	.821***	.117	.440***	.106	.452***	.099
Intervention group	.013	.229	-.005	.174	.047	.167
Gender (male)	-.687**	.232	-.815***	.178	.092	.171
Grade cohort (older)	.398	.230	.274	.215	.069	.196
Antisocial behavior	.838**	.312	.191	.235	.567*	.264
Low income	.674**	.238	.495**	.179	-.131	.180
Part 2: Frequency of use						
Growth factor mean	1.774***	.061	.297***	.050	-.207***	.046
Intervention group	-.031	.098	-.029	.073	-.199*	.080
Gender (male)	.076	.098	-.081	.071	-.045	.077
Grade cohort (older)	.054	.099	.075	.100	.242**	.093
Antisocial behavior	.287*	.123	.005	.078	-.056	.105
Low income	.056	.102	.028	.078	.072	.087

Note. ^aGrowth factor variance and associated covariances set to zero in model Parts 1 and 2.

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

Table 4

Means and Standard Errors for Marijuana Use Growth Factors and Covariates

Variable	Grade 8.5 status		Linear growth		Quadratic growth ^a	
	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>
Part 1: Use versus nonuse						
Growth factor mean	3.233***	.212	.475***	.065	-.463***	.112
Intervention group	-.178	.268	.055	.081	-.008	.143
Gender (male)	.388	.274	-.170*	.088	.514***	.146
Grade cohort (older)	.888**	.311	.071	.120	.175	.217
Antisocial behavior	1.306***	.351	.203	.106	-.133	.214
Low income	.878*	.283	.030	.083	-.138	.149
Part 2: Frequency of use						
Growth factor mean	1.511***	.139	-.005	.037	na	na
Intervention group	.103	.126	-.223***	.052	na	na
Gender (male)	.100	.128	.088	.053	na	na
Grade cohort (older)	.160	.134	.001	.068	na	na
Antisocial behavior	.221	.148	.003	.082	na	na
Low income	.006	.121	.005	.053	na	na

Note. na = not applicable (i.e., quadratic growth factor not included in Part 2 model).

^aQuadratic growth factor variance and associated covariances in model Part 1 set to zero.

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

Table 5

Means and Standard Errors for Cigarette Use Growth Factors and Covariates

Variable	Grade 8.5 status		Linear growth ^a		Quadratic growth ^a	
	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>
Part 1: Use versus nonuse						
Growth factor mean	4.245***	.280	.146*	.074	-.395**	.133
Intervention group	.164	.305	-.153	.089	-.123	.155
Gender (male)	-.795**	.306	.051	.089	.149	.161
Grade cohort (older)	1.118**	.374	.183	.143	.249	.255
Antisocial behavior	1.030**	.374	-.105	.101	-.074	.206
Low income	.916**	.318	.094	.090	.029	.156
Part 2: Frequency of use						
Growth factor mean	.833***	.187	.133**	.044	-.048	.094
Intervention group	.017	.125	-.008	.040	-.033	.090
Gender (male)	-.112	.132	-.066	.048	-.055	.108
Grade cohort (older)	.022	.193	-.057	.086	-.271	.167
Antisocial behavior	.431*	.172	.000	.061	.205	.118
Low income	.108	.132	-.013	.040	-.010	.090

Note. ^aGrowth factor variance and associated covariances set to zero in model Parts 1 and 2.

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

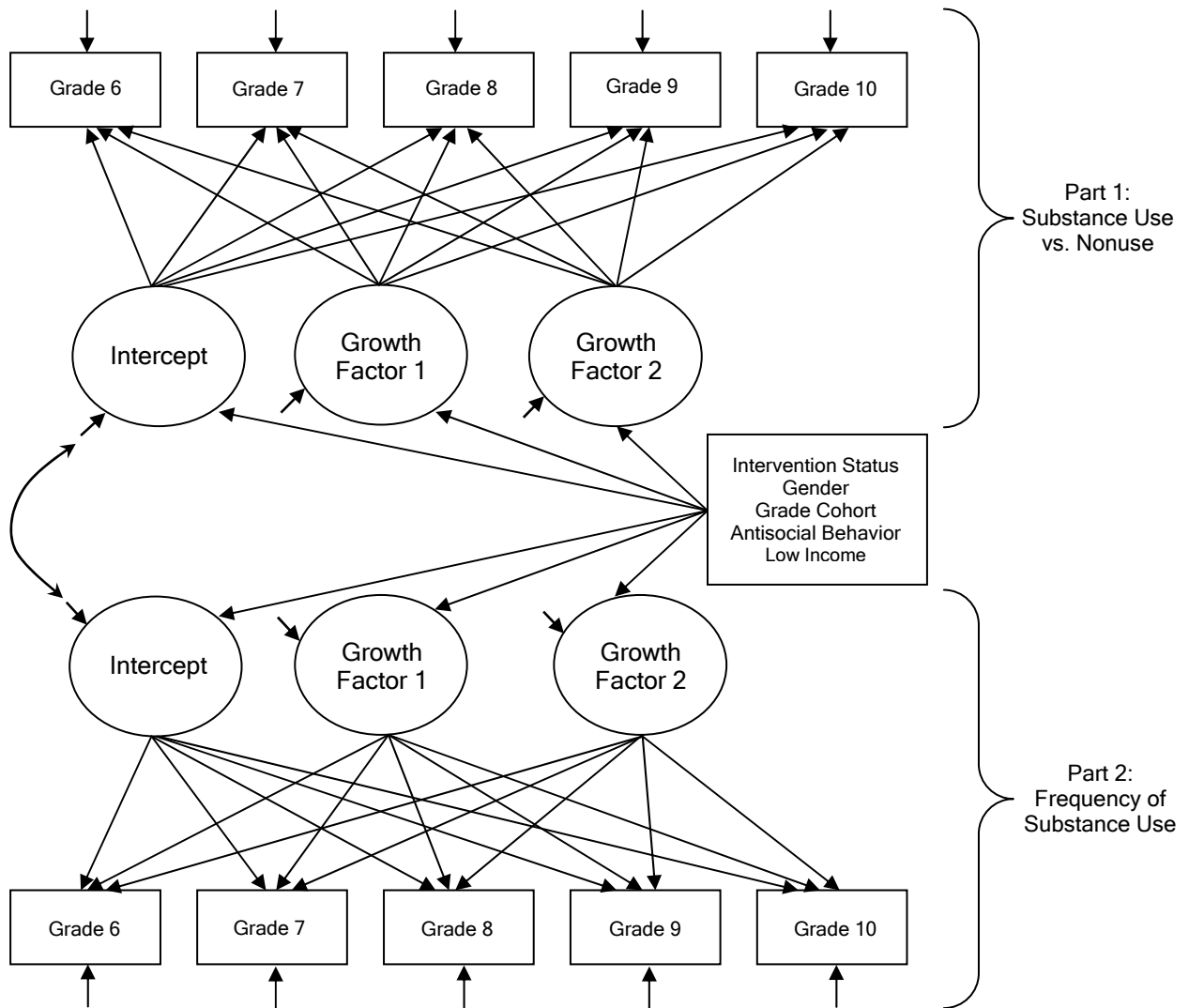
Figure Captions

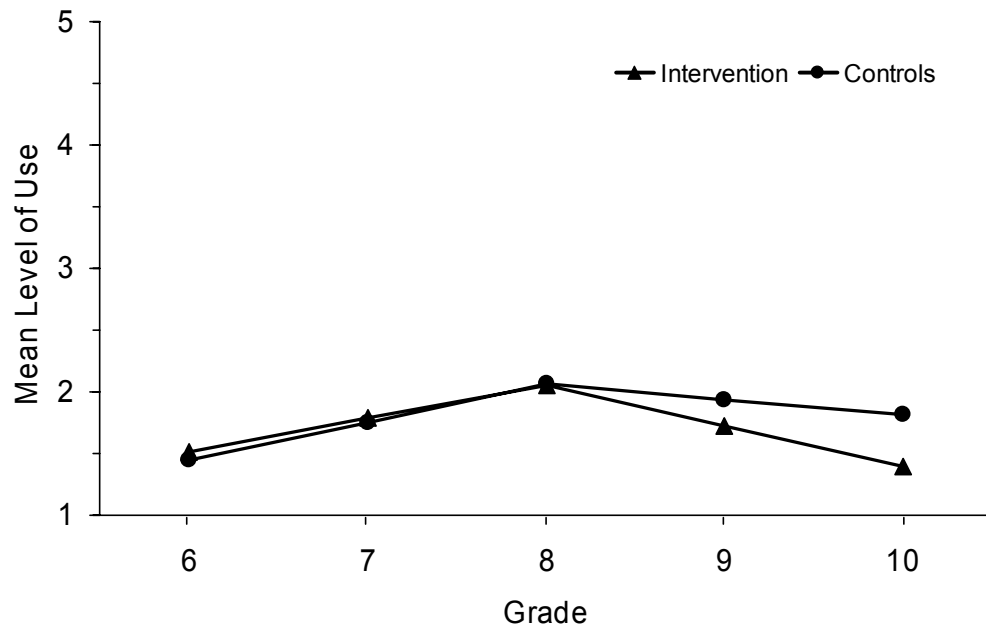
Figure 1. Path diagram for two-part latent growth model. Top portion of diagram depicts Part 1 of the model (i.e., substance use vs. nonuse); bottom portion depicts Part 2 of the model (i.e., frequency of substance use). Growth Factors 1 and 2 correspond to piecewise or linear and quadratic growth factors (correlations between growth factors within each model part omitted for clarity).

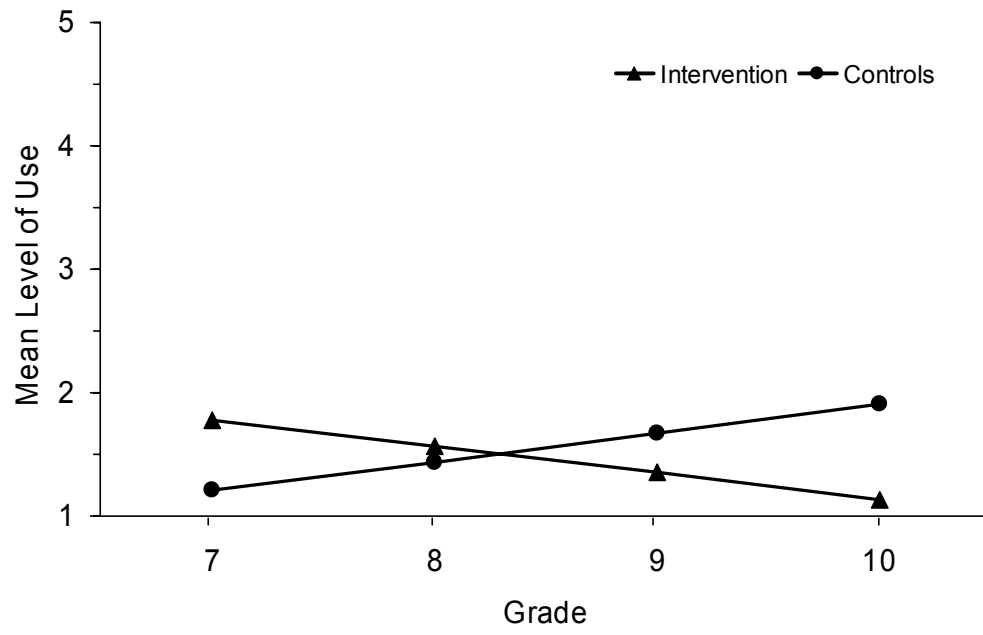
Figure 2. Adjusted means for frequency of alcohol use (excluding nonuse) during Grades 6 through 10 by intervention status. Scale ranges from 1 (*some use within the past year*) to 5 (*20 or more times within the past month*).

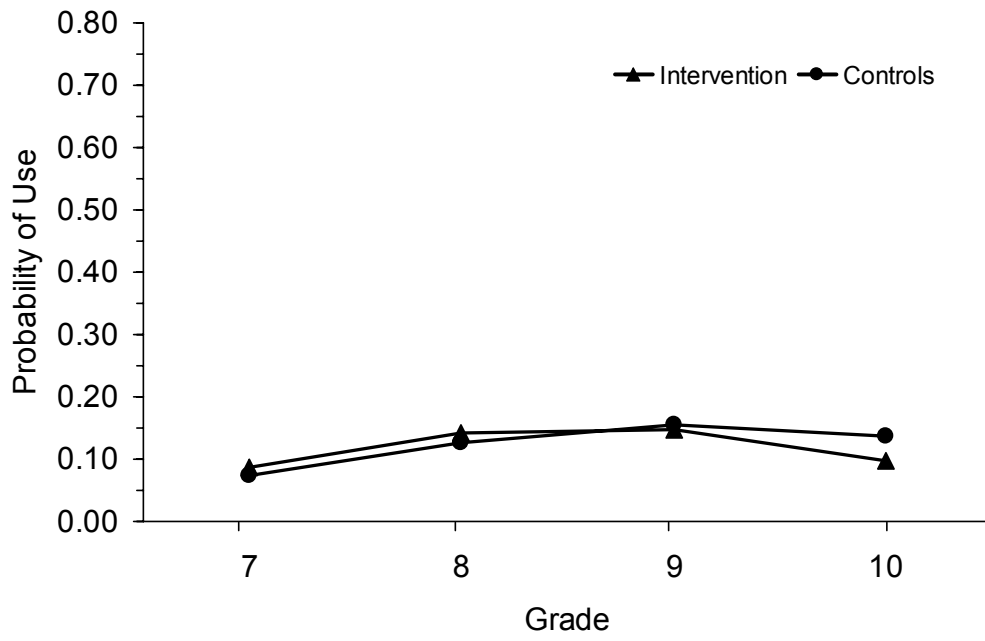
Figure 3. Adjusted means for frequency of marijuana use (excluding nonuse) during Grades 7 through 10 by intervention status. Scale ranges from 1 (*some use within the past year*) to 5 (*20 or more times within the past month*).

Figure 4. Predicted probabilities of cigarette use by intervention status.









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² Substance use-versus-nonuse was analyzed using a three-level hierarchical generalized linear model (HGLM) with a logit-link function for the binary outcome.

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⁴ δ is defined as the group difference in a growth factor divided by the population standard deviation of that growth factor (see Raudenbush & Liu, 2001, Equation 13).

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⁶ Fit indices based on $n = 239$ students with nonzero frequency of cigarette use.