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# Does the Direction of Effects in the Association Between Depressive Symptoms and Health-Risk Behaviors Differ by Behavior? A Longitudinal Study Across the High School Years

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A B S T R A C T

**Purpose:** Adolescence is associated with the onset of depressive symptoms as well as significant increases in health-risk behaviors. Potential explanations for the direction of effects in the association between depressive symptoms and health-risk behaviors include the self-medication/acting out hypothesis (i.e., early depressive symptoms predict increases in risk behaviors over time) and the failure hypothesis (i.e., early participation in health-risk behaviors predicts increases in depressive symptoms over time). The purpose of the present longitudinal study was to assess these competing hypotheses across the high school years, and to examine whether the direction of effects (and therefore the self-medication/acting out and failure hypotheses) may differ depending on the *type* of risk behavior under consideration.
**Methods:** The sample consisted of 4,412 adolescents (49% female) who were followed up from grade nine to 12. Adolescents reported on their depressive symptoms and six health-risk behaviors (frequency of alcohol use, amount of alcohol consumed per drinking episode, cigarette smoking, marijuana use, hard drug use, and delinquency). Analyses were conducted with dual trajectory growth curve modeling.
**Results:** Adolescents who had higher depressive symptoms in grade nine reported faster increases than their peers in smoking, marijuana, and hard drug use across the high school years, supporting the self-medication hypothesis. The failure hypothesis was not supported.
**Conclusion:** The results are important because they suggest that by targeting depressive symptoms during early adolescence, treatment programs may prevent increases in the frequency of these risk behaviors later in adolescence.



Depression and health-risk behaviors are major public health problems, which have negative consequences for both individuals and society as a whole. Adolescence, in particular, is an age associated with the onset of depressive symptoms [1], as well as significant increases in health-risk behaviors, such as alcohol and marijuana use [2]. Moreover, positive correlations have been found between depressive symptoms and risk behaviors among adolescents [3,4]. As a result, researchers increasingly have attempted to uncover the direction of effects between these two behaviors, and have generated two hypotheses to potentially explain these effects [5].

First, the *self-medication/acting out hypothesis* states that early depressive symptoms predict increases in risk behaviors over time. For instance, negative emotions may provide adolescents with a strong motivation to self-medicate (e.g., through marijuana use) so as to minimize depressive symptoms [6,7]. Several longitudinal studies provide support for the self-medication hypothesis with regard to alcohol, cigarette, and marijuana use [2,8]. Early depressive symptoms

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may also predict delinquent behaviors, as researchers have suggested that youth may "act out" with delinquent behavior to mask underlying depressive symptoms [9–11].

In contrast, the *failure hypothesis* holds that early participation in health-risk behaviors predicts increases in depressive symptoms over time because engaging in risk behaviors may lead to negative evaluation from others, which in turn may lead to feelings of failure that contribute to depressive symptoms [12]. For example, cigarette smoking has been hypothesized to predict increased depressive symptoms over time because of the social stigma attached to smoking [13]. Furthermore, engaging in delinquent behaviors may elicit rejection from family members and teachers, leading to failures in social relationships as well as lower academic achievement, thus increasing adolescents' vulnerability to depressive symptoms [14].

There is no consensus about whether the self-medication/acting out hypothesis or the failure hypothesis is more accurate for adolescents, specifically across the high school years. Some studies have also found significant bidirectional relations between depressive symptoms and risk behaviors, suggesting support for both hypotheses [2,5,15]. However, other studies have reported that longitudinal relations do not exist between these two constructs [4,16], and, interestingly, some studies have even found that increases in one construct predict declines or less steep increases in the other [17,18].

These inconsistencies across studies may be because of several factors. First, many researchers have included only one type of health-risk behavior in their analyses [5,12,15], or conversely, have used composite variables encompassing several health-risk behaviors [2]. This represents a significant limitation because the direction of effects in the association between depressive symptoms and risk behaviors may differ depending on the *type* of behavior under consideration. For example, although substance use (e.g., smoking, marijuana) may affect brain chemistry, which in turn may affect emotion regulation and thereby depressive symptoms, this may not be true for delinquency. Second, many studies have not controlled for the typical co-occurrence found among health-risk behaviors [4,18]. For example, if depressive symptoms are associated with alcohol use, but alcohol use co-occurs with marijuana use [19,20], it is necessary to control for alcohol use to obtain the *unique* association between marijuana use and depressive symptoms. Third, some studies have focused only on select populations (e.g., clinical samples, girls) [2,3].

Researchers typically also have used traditional panel models (e.g., autoregressive cross-lagged [ACL] approaches) to explore the direction of effects [4,12]. Using ACL models, researchers have evaluated whether engagement in risk behaviors at one point in time predicts depressive symptoms at a later point in time, and vice versa, while accounting for rank-order stability in each behavior. These models, however, do not provide information about continuous developmental trajectories and individual-level change over time. For instance, the ACL approach may determine whether depressive symptoms at age 14 predict increases in health-risk behaviors at age 15 (and in models with >2 periods, whether depressive symptoms at age 15 predict increases in health-risk behaviors at age 16, etc.), but it cannot provide any indication of whether depressive symptoms at one point in time predict *individual-level change over many years* in health-risk behaviors [21]. Latent growth curve (LGC) models, in contrast, do provide this information. For instance, using LGC models, frequency of involvement in risk behaviors at one point of time can be used to predict *change* in depressive symptom scores across an extended period, and vice versa. Although much of the literature assessing the self-medication and failure hypotheses has focused on short-term, time-specific relations, the purpose of our study was to examine whether depressive symptoms experienced in grade nine set the stage for increasing involvement in risk behaviors over the course of high school (which would support the self-medication hypothesis), or, conversely, whether risky behavior in grade nine set the stage for increasing depressive symptoms over the course of high school (supporting the failure hypothesis).

Few studies have used an LGC approach to examine the self-medication/acting out and failure hypotheses. Studies also have not measured a broad range of health-risk behaviors within the same sample [5], or assessed change across each of the high school years [5,8,18]. This is an important limitation as the high school years involve significant changes in depressive symptoms and health-risk behaviors. The aim of the present study was to address these gaps by (a) modeling intraindividual trajectories of depressive symptoms and a wide range of health-risk behaviors (frequency of alcohol use, amount of alcohol consumed per drinking episode, cigarette smoking, marijuana use, hard drug use, and delinquency) in a large community sample of adolescents across all the high school grades (9 through 12), (b) simultaneously assessing both the *self-medication/acting out* and *failure* hypotheses for the associations between depressive symptoms and each health-risk behavior independently, by evaluating whether depressive symptoms in grade nine predict change across the high school years in health-risk behavior, and vice versa, and (c) examining whether the effects remain consistent when controlling for the level of engagement in other health-risk behaviors.

## Methods

### *Participants*

Students from eight high schools encompassing a school district in Ontario, Canada, were surveyed in each grade of high school. This cohort-sequential study was part of a larger project, and involved 4,412 participants (49% females). The overall participation rate across the longitudinal study ranged from 83% to 86%. Consistent with the broader Canadian population [22], 92.4% of the participants were born in Canada, and the most common ethnic backgrounds reported other than Canadian were Italian (31%) and French (18%). Data on socioeconomic status indicated mean levels of education for mothers and fathers falling between "some college, university or apprenticeship program" and "completed a college/apprenticeship/technical diploma" (25% of parents were university graduates).

Because of the study's cohort-sequential design, the sample included three cohorts of students who differed on the year they entered high school (i.e., the academic year 2002/2003, 2003/2004, or 2004/2005, for cohorts 1–3, respectively). An examination of mean differences on the study measures depending on cohort revealed significant differences among the cohorts in health-risk behaviors in grades 10 and 11, in which the second cohort reported significantly lower scores on the measures than the third cohort, with the exception of

**Table 1**
Study constructs and measures

| Domain | Variable | Number of items | Statement example | Scale |
|---|---|---|---|---|
| Gender | Gender | 1 | Are you male or female? | |
| Age | Age | 1 | How old are you? | |
| Parental education | Paternal education | 1 | What is the highest level of education your father/stepfather (male guardian) completed? | 1 *(did not finish high school) to* 6 *(professional degree)* |
| | Maternal education | 1 | What is the highest level of education your mother/stepmother (female guardian) completed? | 1 (*did not finish high school*) to 6 (*professional degree*) |
| Depressive symptoms | CES-D | 20 | During the past 2 weeks, how often have you felt or behaved this way? (e.g., I felt depressed) | 1 (*<1 day*) to 5 (*10–14 days*) |
| Health-risk behavior | Alcohol frequency | 1 | How often do you go drinking or have a drink (alcohol)? | 1 (*never*) to 8 (*every day*) |
| | Alcohol amount | 1 | On average, when you are drinking alcohol, about how many drinks do you have? | 1 (<1) to 6 (*over 10*) |
| | Smoking | 1 | How many cigarettes do you usually smoke each day? | 1 (*I no longer smoke*) to 8 (*more than a pack*) |
| | Marijuana | 1 | In the past 12 months, how often did you use marijuana? | 1 (*never*) to 6 (*every day*) |
| | Hard drugs | 6 | In the past 12 months, how often did you use the following substances? (e.g., cocaine) | 1 (*never*) to 6 (*every day*) |
| | Delinquency | 7 | In the past 12 months, how often have you done the following? (e.g., sneaking out at night) | 1 (*never*) to 4 (*>5 times*) |

CES-D = Centre for Epidemiological Studies Depression scale [27].

depressive symptoms and frequency of alcohol use in grade 11 (Wilk's $\lambda < .001$). In addition, the first cohort reported significantly lower scores on all the measures than the third cohort in grade 11, with the exception of delinquency. However, magnitudes of the between-group differences were small (mean differences ranging from .08 to .50; individual $\eta^2$ values ranging from .006 to .029). As differences between cohorts were limited, all analyses combined students across cohorts into one sample. Cohort, however, was included as a covariate in all analyses. Participants who completed the survey at all time periods were not significantly different from participants missing one or more waves on any variables in grades 10, 11, or 12. However, participants with complete data did report significantly more positive scores in grade nine on the health-risk behaviors than participants missing one or more waves (Wilks $\lambda < .001$). Again, magnitudes of the between-group differences were small (mean differences ranging from .08 to .29; individual $\eta^2$ values ranging from .003 to .008). Finally, there were missing data because of (a) the longitudinal-sequential design, and (b) questions left blank within a survey (11% of data, consistent with other longitudinal survey studies [23]). Missing data were assumed to be missing at random [24], and were imputed using the multiple imputation procedure with 100 datasets [25,26].

### *Procedure*

Active informed assent was obtained from the adolescent participants. Parents were provided with written correspondence mailed to each student's home before the survey administration outlining the study; this letter indicated that parents could request that their adolescent not participate in the study. An automated telephone message about the study was also left at each student's home telephone number. This procedure was approved by the participating school board and the University Research Ethics Board. At all time periods, the questionnaire was administered to students in classrooms by trained research staff. Students were informed that their responses were completely confidential.

### *Measures*

Depressive symptoms, frequency of alcohol use, number of alcoholic drinks per drinking session, cigarette smoking, marijuana use, hard drug use, and delinquency were measured in each of the high school grades. Gender and parental education level were assessed in grade nine (or if the student was absent in grade nine, their first wave was used) and used as covariates in all analyses (Table 1).

## Results

Table 2 outlines the means and standard deviations, and Table 3 outlines the correlations among depressive symptoms and health-risk behaviors. All LGC analyses were conducted using MPlus 6.0 (Muthén & Muthén, Los Angeles, CA) [28]. Depressive symptoms, alcohol frequency, alcohol amount, and marijuana use exhibited acceptable skewness and kurtosis (i.e., skewness: <3, and kurtosis: <10 [29]), and were analyzed using maximum likelihood estimation. Cigarette smoking, hard drug use, and delinquency exhibited non-normality, and therefore were analyzed using maximum likelihood estimation with robust standard errors (MLR), a procedure that is robust to non-normality ([30]; e.g., with smoking). When using the multiple imputation procedure with MLR, an average MLR chi-square and standard deviation are calculated over imputations. These chi-square values were corrected using the scaling correction factor. In addition to using MLR estimation, we also reran all analyses for the

**Table 2**
Means and standard deviations of study measures across high school

| Domain | Variable | Grade nine M (SD) α | Grade 10 M (SD) α | Grade 11 M (SD) α | Grade 12 M (SD) α |
|---|---|---|---|---|---|
| Gender | Gender | 49% female | | | |
| Age | Age | 14 years (6 m) | 15 years (5 m) | 16 years (5 m) | 17 years (5 m) |
| Parental education | Paternal education | 3.26 (1.40) | | | |
| | Maternal education | 3.20 (1.41) | | | |
| Depressive symptoms | Depressive symptoms | 1.90 (.62) | 1.97 (.66) | 2.04 (.68) | 2.08 (.68) |
| Health-risk behaviors | Alcohol frequency | 1.84 (1.03) | 2.42 (1.38) | 2.80 (1.49) | 3.05 (1.57) |
| | Alcohol amount | 2.01 (1.19) | 2.74 (1.48) | 3.30 (1.51) | 3.59 (1.49) |
| | Smoking | 1.18 (.66) | 1.28 (.90) | 1.43 (1.17) | 1.52 (1.28) |
| | Marijuana | 1.55 (1.04) | 1.93 (1.40) | 2.34 (1.60) | 2.50 (1.68) |
| | Hard drugs | 1.07 (.26) | 1.15 (.57) | 1.26 (.79) | 1.34 (.97) |
| | Delinquency | 1.22 (.36) | 1.29 (.45) | 1.39 (.55) | 1.40 (.57) |

*Note*. Higher scores for variables indicate higher frequency of depressive symptoms and health-risk behaviors. Scores reflect raw means.

smoking, hard drug use, and delinquency behaviors using transformed data (i.e., inverse scores), with no difference in the pattern of results.

*Univariate growth trajectories*

Separate growth models were identified for depressive symptoms and each of the health-risk behaviors by first testing each variable on a linear growth model, in which the paths from the slope factor to the measured variables were fixed in a linear progression (i.e., 0 = grade nine; 1 = grade 10; 2 = grade 11; 3 = grade 12), followed by a model in which the slope factor was replaced with a shape factor so as to test nonlinearity [31], in which the loadings were set to zero at ninth grade, three at 12th grade, and freely estimated at the 10th and 11th grades. For each of the models, the intercept factor loading was set to 1 at all time points. Thus, the mean of the intercept represents the average score for the sample at the starting point (i.e., grade nine) of the trajectory, whereas the mean of the slope represents the average rate of change from grade nine to 12. A well-specified model fit was indicated by a comparative fit index (CFI) of $>.95$ and a root-mean-square error of approximation (RMSEA) of $<.06$ [32].

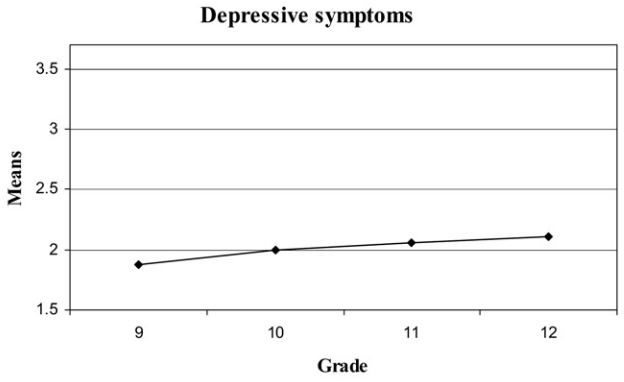
Chi-square difference tests were conducted to compare the model with the linear slope factor with the model with the shape factor for each behavior. The linear slope model was chosen for all behaviors unless $\chi^2$ difference tests between the linear slope and shape factor models indicated significant improvement for the shape model at $p < .01$. The model with the linear slope factor was the more parsimonious choice for

**Table 3**
Correlations between depressive symptoms and health-risk behaviors across the four high school years

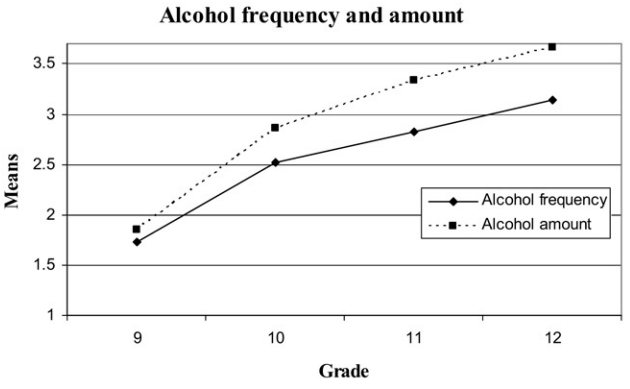
| Variable | Depressive symptoms grade nine | Depressive symptoms grade 10 | Depressive symptoms grade 11 | Depressive symptoms grade 12 |
|---|---|---|---|---|
| Alcohol frequency grade nine | .23* | .11* | .10* | .11* |
| Alcohol frequency grade 10 | .13* | .17* | .11* | .10* |
| Alcohol frequency grade 11 | .06** | .08*** | .11* | .09* |
| Alcohol frequency grade 12 | .03 | .05** | .04 | .12* |
| Alcohol amount grade nine | .22* | .10* | .07*** | .14* |
| Alcohol amount grade 10 | .14* | .16* | .12* | .11* |
| Alcohol amount grade 11 | .05** | .07*** | .11* | .08*** |
| Alcohol amount grade 12 | .00 | .02 | .05** | .04 |
| Cigarette smoking grade nine | .20* | .14* | .07*** | .15* |
| Cigarette smoking grade 10 | .13* | .19* | .10* | .11* |
| Cigarette smoking grade 11 | .12* | .13* | .17* | .09* |
| Cigarette smoking grade 12 | .14* | .13* | .14* | .20* |
| Marijuana grade nine | .21* | .10* | .08*** | .10* |
| Marijuana grade 10 | .18* | .20* | .13* | .14* |
| Marijuana grade 11 | .16* | .14* | .18* | .11* |
| Marijuana grade 12 | .11* | .10* | .12* | .17* |
| Hard drugs grade nine | .17* | .10* | .07*** | .12* |
| Hard drugs grade 10 | .11* | .21* | .09* | .12* |
| Hard drugs grade 11 | .07*** | .09* | .21* | .09* |
| Hard drugs grade 12 | .07*** | .09* | .10* | .30* |
| Delinquency grade nine | .24* | .15* | .15* | .16* |
| Delinquency grade 10 | .16* | .25* | .12* | .15* |
| Delinquency grade 11 | .12* | .13* | .27* | .15* |
| Delinquency grade 12 | .08*** | .12* | .10* | .28* |

*Note*. Higher scores for variables indicate higher depressive symptoms and frequency of health-risk behaviors. Full correlation table available by request from second author.

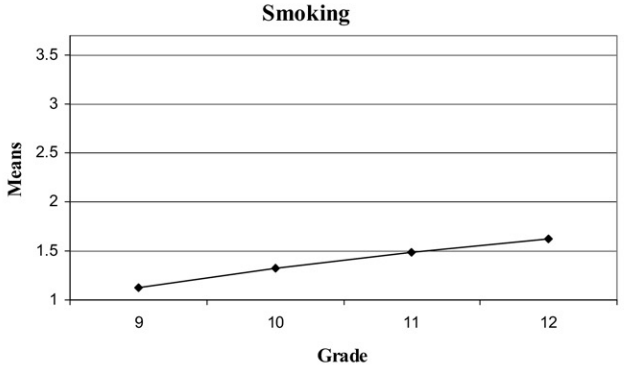
* $p < .05$.
** $p < .01$.
*** $p < .001$.



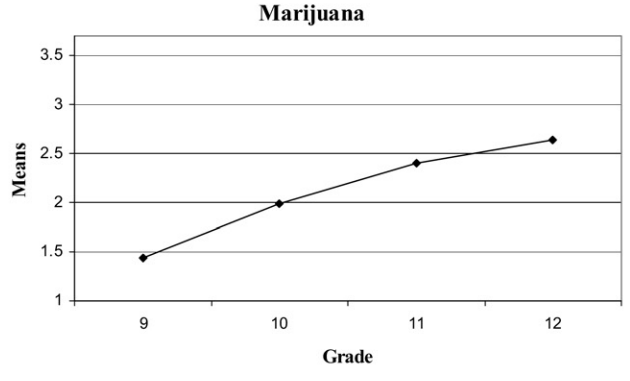

$\chi^2(11) = 28.36$; RMSEA = .019 (.010–.028); CFI = .99; TLI = .98.



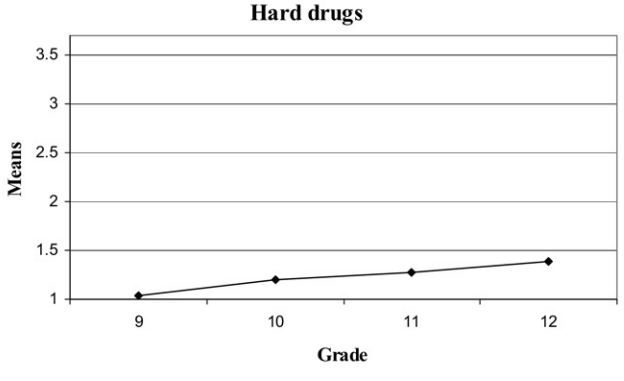

Alcohol frequency: $\chi^2(9) = 65.00$; RMSEA = .038 (.029-.046); CFI = .97; TLI = .93. Alcohol quantity: $\chi^2(9) = 103.19$; RMSEA = .049 (.041-.057); CFI = .96; TLI = .92.



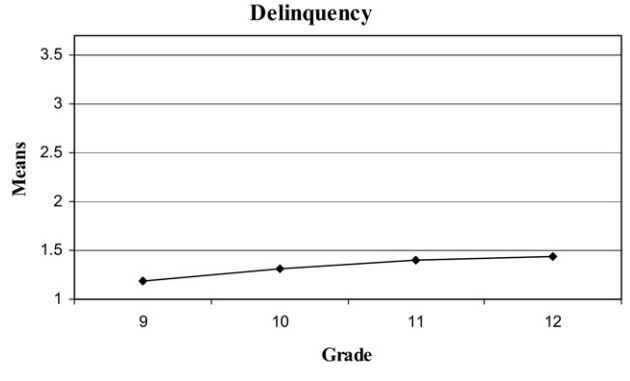

$\chi^2(9) = 114.07$; RMSEA = .051 (SD=.009); CFI = .96; TLI = .93.




$\chi^2(9) = 63.41$; RMSEA = .037 (.029-.046); CFI = .98; TLI = .96.




$\chi^2(11) = 76.13$; RMSEA = .036 (SD=.008); CFI = .94; TLI = .90.




$\chi^2(9) = 78.53$; RMSEA = .041 (SD=.006); CFI = .98; TLI = .96.

**Figure 1 .** Means for depressive symptoms, alcohol frequency and amount, smoking, marijuana, hard drugs, and delinquency across 4 years in high school. Scores are adjusted means.

depressive symptoms, $\chi^2 diff\ (2) = 6.25$, $p = .044$, and hard drug use, $\chi^2 diff\ (2) = 4.14$, $p = .126$. In contrast, the model with the shape factor indicated a better fit than the linear model for alcohol frequency, $\chi^2 diff\ (2) = 137.03$, $p < .001$, alcohol amount, $\chi^2 diff\ (2) = 248.64$, $p < .001$, cigarette smoking, $\chi^2 diff\ (2) = 14.33$, $p < .001$, marijuana use, $\chi^2 diff\ (2) = 98.32$, $p < .001$, and delinquency, $\chi^2 diff\ (2) = 92.51$, $p < .001$. For all behaviors, model fit was good, with CFIs >.94 and RMSEAs < .06 (Figure 1). These graphs indicate that depressive symptoms and health-risk behaviors all increase across adolescence, with alcohol use representing the largest growth over time.

*Testing the self-medication/acting out and failure hypotheses*

To test the self-medication/acting out and failure hypotheses, we specified a path from the intercept of depressive symptoms to the slope of each health-risk behavior (i.e., self-medication/acting out), and a path from the intercept of each health-risk behavior to the slope of depressive symptoms (i.e., failure). In addition, paths from gender, cohort, and parental education to each intercept and slope were included as covariates. Covariances were estimated between the (a) intercepts, (b) error terms for the slopes, (c) intercept and slope within each behavior, and (d) depressive symptoms and health-risk behavior within each grade. For all behaviors, model fit was excellent, with CFIs $>.95$ and RMSEAs $<.06$ (Figure 2). The intercepts for depressive symptoms and each health-risk behavior were significantly correlated. The path from depressive symptoms to health-risk behavior was significant for cigarette smoking, $\beta = .26$, $p < .001$, marijuana, $\beta = .18$, $p < .01$, and hard drug use, $\beta = .25$, $p < .001$, indicating that adolescents who had higher depressive symptoms in grade nine reported faster increases in smoking, marijuana, and hard drug use across the high school years, in comparison with adolescents who had lower depressive symptoms in grade nine. Therefore, the self-medication hypothesis was supported for these behaviors. In contrast, the self-medication/acting out hypothesis was not supported for alcohol use (both frequency and amount) and delinquency. Furthermore, the failure hypothesis received no support in any of the models.

*Controlling for other health-risk behavior involvement*

To control for other health-risk behavior involvement, analyses for each dual trajectory model were rerun with all other health-risk behaviors. For each dual trajectory model (e.g., depressive symptoms and smoking), we incorporated initial (i.e., grade nine) levels of all other behaviors (e.g., alcohol frequency and amount, marijuana, hard drugs, and delinquency) as covariates. This was done by specifying paths from all other risk behaviors in grade nine to the intercept and slope of depressive symptoms and the specific health-risk behavior in question. Thus, results for these models represent the *unique* associations between depressive symptoms and each separate risk behavior. In all cases, adding these variables did not change the pattern of the results—information about these results can be obtained from the second author.

## Discussion

The present study specifically examined the self-medication/acting out versus failure hypotheses as possible explanations for long-term associations between depressive symptoms and health-risk behaviors, and found support for the self-medication hypothesis only. Higher levels of depressive symptoms in grade nine seem to set the stage for faster increases in cigarette smoking, marijuana use, and hard drug use across the high school years in comparison with adolescents with lower levels of depressive symptoms in grade nine. These types of substances may be perceived as providing mood-enhancing functions, which may explain the increased frequency of their use over time [6].

The self-medication hypothesis, however, was not supported for the relation between depressive symptoms and alcohol use. This finding may be related to the fact that most adolescents increase their frequency and amount of alcohol use over the high school years, regardless of their levels of depressive symptoms in grade nine. Increases in alcohol use are normative across high school and drinking often occurs during social activities such as parties [18]. As such, drinking alcohol for some youth may be associated with enhanced social networks and feelings of belongingness. Of course, some youth may engage in alcohol use for self-medication reasons, but depressive symptoms also are often associated with social withdrawal; thus, some adolescents with higher depressive symptoms may be less likely than their peers to participate in the social situations that co-occur with alcohol use [17]. The self-medication hypothesis with regard to alcohol use may be more likely to be supported in samples that differ from the current study, such as younger [9] or older [16] populations, where risk-taking is less normative, or for at-risk youth [33]. In contrast, in comparison with alcohol use, cigarette smoking, marijuana use, and hard drug use are less normative and accepted among adolescents [20], and therefore, adolescents with elevated depressive symptoms in grade nine who specifically want to self-medicate may be more likely than their peers to increase their use of these substances over an extended period.

We also did not find support for the acting out hypothesis with delinquency. However, similar to alcohol use, delinquent behavior in adolescence typically occurs in the presence of friends. Thus, delinquent behaviors may result from heightened arousal because of the presence of peers [34], as opposed to acting out because of depression. Similar to alcohol use, adolescents with elevated depressive symptoms also may be less interested than their peers in participating in the social context associated with delinquency [4]. Importantly, researchers who have found support for the acting out hypothesis often have used a broader category of antisocial behavior, including conduct problems, aggression, and delinquency [2].

In general, there was no support for the failure hypothesis in the present study. The failure hypothesis assumes that depressive symptoms would result from risk behaviors because of an internalization of social rejection [12]. Participating in substance-related risks, however, may not lead to social rejection from peers for adolescents. In fact, socially-accepted substance use, such as alcohol use, often occurs in social contexts and thus may help alleviate feelings of rejection and low self-esteem [35,36]. Even though cigarette and marijuana use are less normative and accepted than alcohol use, in the present study, enough adolescents increased in these behaviors over time that, for the majority of students, social rejection may not have been significantly related to involvement in these behaviors. Again, these results may differ for other age groups where risk-taking is not normative.

Furthermore, although some researchers have suggested that delinquency may lead to social rejection, thus increasing vulnerability to depressive symptoms, other researchers have found that the quality of friendships among delinquent adolescents does not differ from those of nondelinquent adolescents [37]. As depressive symptoms are highly correlated with interpersonal problems and feelings of loneliness [38], strong friendship networks among delinquent adolescents may protect these youth from increases in depressive symptoms. In contrast, other health-risk behaviors that were not assessed in the present study, such as conduct problems and aggression, may provide stronger support for the failure hypothesis because these types of behaviors may be associated with lower levels of interpersonal social skills when compared with substance use and delin-

**Figure 2.** Dual trajectory latent growth model of depressive symptoms and health-risk behaviors across four times of measurement. All values are standardized coefficients, and factor loadings for slopes were fixed to their estimates as determined by the univariate models. Not all residuals are shown. Covariances between depressive symptoms and each health-risk behavior within each grade are also not shown (e.g., depressive symptoms in grade nine with alcohol frequency in grade nine). Double-arrows refer to covariances among the factors. Although not shown, paths from the gender, cohort, and parental education covariates were added to each intercept and slope (these results can be obtained from the second author). Results reported do not partial out other risk behavior covariates. The results from these models also can be obtained from the second author. $^{\dagger}p < .07$, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$.

quency, thereby increasing social rejection and vulnerability to depressive symptoms [2,12,14].

Moreover, the failure hypothesis may not have been supported in the present study because our initial wave of assessment occurred in grade nine, when the mean frequency of involvement in health-risk behaviors generally was low. The failure hypothesis may be more likely to be supported in studies using LGC modeling when the initial wave of assessment (i.e., the

intercept) occurs at an age when frequency of risk taking is higher (e.g., in late high school as opposed to the grade nine starting point used in the present study).

This study is not without limitations. First, this study cannot infer causality. It is possible that other third variables, such as genetic and/or environmental influences [39], may better explain the prospective relations between depressive symptoms and various health-risk behaviors. Second, a dynamic relation may exist between depressive symptoms and risk behaviors such that the direction of effects between these behaviors changes across development [40], supporting the need for further research that examines change in these behaviors across different age periods.

Despite these limitations, this study was the first to test both the self-medication/acting out and failure hypotheses using LGC analyses over a long-term period that shows significant growth in these behaviors (i.e., adolescents in high school), for a wide range of health-risk behaviors, and while controlling for the co-occurrence of other health-risk behaviors. The results provide support for the self-medication hypothesis for cigarette, marijuana, and hard drug use, and are important because they suggest that by targeting depressive symptoms during early adolescence, treatment programs may prevent increases in the frequency of health-risk behaviors over the course of high school.

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## References

[1] Kandel DB, Davies M. Epidemiology of depressive mood in adolescents: An empirical study. Arch Gen Psychiatry 1982;39:1205–12.
[2] Measelle JR, Stice E, Hogansen JM. Developmental trajectories of co-occurring depressive, eating, antisocial, and substance abuse problems in female adolescents. J Abnorm Psychol 2006;115:524–38.
[3] Fergusson DM, Goodwin RD, Horwood LJ. Major depression and cigarette smoking: Results of a 21-year longitudinal study. Psychol Med 2003;33:1357–67.
[4] Vieno A, Kiesner J, Pastore M, Santinello M. Antisocial behavior and depressive symptoms: Longitudinal and concurrent relations. Adolescence 2008;43:649–60.
[5] Windle M, Windle RC. Depressive symptoms and cigarette smoking among middle adolescents: Prospective associations and intrapersonal and interpersonal influences. J Consult Clin Psychol 2001;69:215–26.
[6] Cooper ML, Frone MR, Russell M, Mudar P. Drinking to regulate positive and negative emotions: A motivational model of alcohol use. J Pers Soc Psychol 1995;69:990–1005.
[7] Khantzian EJ. The self-medication hypothesis of addictive disorders: Focus on heroin and cocaine dependence. Am J Psychiatry 1985;142:1259–64.
[8] Wills TA, Sandy JM, Yaeger AM. Stress and smoking in adolescence: A test of directional hypotheses. Health Psychol 2002;21:122–30.
[9] Carlson GA, Cantwell DP. Unmasking masked depression in children and adolescents. Am J Psychiatry 1980;137:445–9.
[10] Gold M, Mattlin J, Osgood DW. Background characteristics and responses to treatment of two types of institutionalized delinquent boys. Crim Justice Behav 1989;16:5–33.
[11] Rice F, Lifford KJ, Thomas HV, Thapar A. Mental health and functional outcomes of maternal and adolescent reports of adolescent depressive symptoms. J Am Acad Child Adolesc Psychiatry 2007;46:1162–70.
[12] Capaldi DM. Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: II. A 2-year follow-up at grade 8. Dev Psychopathol 1992;4:125–44.
[13] Martini S, Wagner FA, Anthony JC. The association of tobacco smoking and depression in adolescence: Evidence from the United States. Subst Use Misuse 2002;37:1853–67.
[14] Patterson GR, Reid J, Dishion T. A social interactional approach: Antisocial boys, Vol 4. Eugene, OR: Castalia Publishing Company, 1992.
[15] Locke TF, Newcomb MD. Alcohol involvement and dysphoria: A longitudinal examination of gender differences from late adolescence to adulthood. Psychol Add Behav 2001;15:227–36.
[16] Overbeek G, Vollebergh W, Meeus W, Engels R, Luijpers E. Course, co-occurrence, and longitudinal associations of emotional disturbance and delinquency from adolescence to young adulthood: A six-year three-wave study. J Youth Adolesc 2001;30:401–26.
[17] Fleming CB, Mason WA, Mazza JJ, Abbott RD, Catalano RF. Latent growth modeling of the relationship between depressive symptoms and substance use during adolescence. Psychol Add Behav 2008;22:186–97.
[18] Needham BL. Gender differences in trajectories of depressive symptomatology and substance use during the transition from adolescence to young adulthood. Soc Sci Med 2007;65:1166–79.
[19] Jessor R, Jessor SL. Problem behavior and psychosocial development: A longitudinal study of youth. New York, NY: Academic, 1977.
[20] Willoughby T, Chalmers H, Busseri MA. Where is the syndrome? Examining co-occurrence among multiple problem behaviors in adolescence. J Consult Clin Psychol 2004;72:1022–37.
[21] Bollen KA, Curran PJ. Autoregressive latent trajectory (ALT) models: A synthesis of two traditions. Sociol Methods Res 2004;32:336–83.
[22] Statistics Canada. Population by ethnic origin [Online]. Available at: http://www12.statcan.ca. Accessed April 6, 2003.
[23] Feldman BJ, Masyn KE, Conger RD. New approaches to studying problem behaviors: A comparison of methods for modeling longitudinal, categorical adolescent drinking data. Dev Psychol 2009;45:652–76.
[24] Little RJ, Rubin DB.Statistical analysis with missing data, 2nd edition. New York, NY: Wiley, 2002.
[25] Little TD, Card NA, Preacher KJ, McConnell E. Modeling longitudinal data from research on adolescence. In: Lerner RM, Steinberg L, eds. Handbook of Adolescent Psychology, Individual Bases of Adolescent Development, 3rd edition. Vol 1Hoboken, NJ: John Wiley & Sons Inc, 2009:15–54.
[26] Schafer JL, Graham JW. Missing data: Our view of the state of the art. Psychol Methods 2002;7:147–77.
[27] Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. Appl Psych Meas 1977;1:385–401.
[28] Muthén LK, Muthén BO. Mplus User's Guide, 6th edition. Los Angeles, CA: Muthén & Muthén, 1998-2010.
[29] Kline RB. Principles and practice of structural equation modeling, 3rd edition. New York, NY: Guilford Press, 2011.
[30] Audrain-McGovern J, Rodriguez D, Kassel JD. Adolescent smoking and depression: Evidence for self-medication and peer smoking mediation. Addiction 2009;104:1743–56.
[31] Duncan TE, Duncan SC, Strycker LA, Li F, Alpert A. An introduction to latent variable growth curve modeling: Concepts, issues, and applications, 2nd edition. Mahwah, NJ: Lawrence Erlbaum Associates Publishers, 2006.
[32] Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. Struct Equ Modeling 1999;6:1–55.
[33] Repetto PB, Zimmerman MA, Caldwell CH. A longitudinal study of the relationship between depressive symptoms and alcohol use in a sample of inner-city black youth. J Stud Alcohol 2004;65:169–78.
[34] Gardner M, Steinberg L. Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. Dev Psychol 2005;41:625–35.
[35] Wills TA, Cleary SD. Peer and adolescent substance use among 6th–9th graders: Latent growth analyses of influence versus selection mechanisms. Health Psychol 1999;18:453–63.
[36] Kaplan HB. Deviant behavior and self-enhancement in adolescence. J Youth Adolesc 1978;7:253–77.
[37] Giordano PC, Cernkovich SA, Pugh MD. Friendships and delinquency. Am J Sociol 1986;91:1170–202.
[38] Hartup WW, Stevens N. Friendships and adaptation across the life span. Curr Dir Psychol Sci 1999;8:76–9.
[39] Tambs K, Harris JR, Magnus P. Genetic and environmental contributions to the correlation between alcohol consumption and symptoms of anxiety and depression: Results from a bivariate analysis of Norwegian twin data. Behav Genet 1997;27:241–50.
[40] Orlando M, Ellickson PL, Jinnett K. The temporal relationship between emotional distress and cigarette smoking during adolescence and young adulthood. J Consult Clin Psychol 2001;69:959–70.