Depressive Symptoms and Cigarette Smoking Among Middle Adolescents: Prospective Associations and Intrapersonal and Interpersonal Influences

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Using data from a 4-wave longitudinal study with a school-based sample of 1,218 middle adolescents, the authors investigated the directionality (e.g., unidirectionality and bidirectionality) of the prospective relationship between depressive symptoms and cigarette use within the context of potential confounding variables and common and unique intrapersonal and interpersonal predictors. Findings indicated that serious and persistent depressive symptoms were prospective predictors of increased cigarette use across time, after controlling for baseline levels of smoking. Similarly, heavy and persistent smoking prospectively predicted increases in depressive symptoms. Intrapersonal and interpersonal predictors of cross-temporal changes in depressive symptoms and cigarette use were more unique than common. Latent growth curve modeling indicated a quadratic trend in adolescent cigarette smoking across time with an initial acceleration followed by a deceleration, though there was substantial intraindividual variation in individual trajectories.

Recent national statistics from the Monitoring the Future Study (Johnston, O'Malley, & Bachman, 1998) indicated substantial rates of cigarette smoking among teens. For example, in 1997, the percentage of adolescents currently (i.e., within the last 30 days) using cigarettes was 19% for 8th graders, 30% for 10th graders, and 37% for 12th graders. Moreover, a considerable number of adolescents reported frequent use (i.e., daily use) of cigarettes, with approximately one in four high school seniors (24% of boys and 24% of girls) reporting daily cigarette smoking in the last 30 days. Among adolescents, short-term health complications associated with cigarette smoking include increased respiratory tract symptoms and infections, changes in pulmonary functioning, worsening of asthma, and declines in physical fitness (Pérez-Stable & Fuentes-Afflick, 1998).

Just as cigarette smoking among teens represents a health issue of great concern, so too does the occurrence of serious depressive symptoms and depressive disorders (e.g., Cicchetti & Toth, 1998; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Epidemiologic studies conducted in the United States and other countries have indicated high rates of depressive disorders–symptoms among community samples of adolescents and young adults. For example, among an older adolescent sample in Oregon, Lewinsohn et al. (1993) reported that 24% of the participants had experienced at least one lifetime major depressive episode. High rates of adolescent depressive disorders and symptoms are of concern, given that depression is a condition that may have an early onset, be of long duration, and may reoccur frequently (Cicchetti & Toth,

1998). Moreover, depression is a putative risk factor for suicide completions among youth (e.g., Brent et al., 1993).

Research findings with adults (e.g., Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Lerman et al., 1996) and with adolescents (e.g., Brown, Lewinsohn, Seeley, & Wagner, 1996; Choi, Patten, Gillin, Kaplan, & Pierce, 1997; Kandel & Davies, 1986) have consistently supported a significant association between depressive symptoms–disorders and cigarette smoking–nicotine dependence. Four alternative hypotheses have been promulgated regarding the nature (e.g., direction of effects) of the cigarette use–depressive symptoms relationship. One hypothesis suggests that cigarette use helps to "self-medicate" feelings of distress or negative mood, thereby positing that levels of depression causally influence subsequent levels of cigarette use (e.g., Breslau, Kilbey, & Andreski, 1991; Carmody, 1989; Lerman et al., 1996). A second hypothesis suggests that nicotine use alters neurochemical systems (e.g., neuroregulators such as acetylcholine, dopamine, and norepinephrine) that may, in turn, affect neural circuits in the brain, such as reward mechanisms associated with mood regulation (e.g., Pomerleau & Pomerleau, 1984; Pontieri, Tanda, Orzi, & Di Chiara, 1996).

A third hypothesis suggests that, rather than a unidirectional relationship, smoking and depression may reciprocally influence each other (Wang, Fitzhugh, Turner, Fu, & Westerfield, 1996). For example, some depressed smokers may smoke to alleviate their negative affect and, to the extent that nicotine has this desired effect, their smoking is positively reinforced (Lerman et al., 1996). However, upon smoking cessation, smokers with a history of major depression may be at increased risk to develop a new depressive episode and, thus, be more subject to smoking relapse (Covey, Glassman, & Stetner, 1998). Finally, a fourth hypothesis has been that, rather than there being a causal relationship between cigarette use and depression, a set of common, or highly correlated, variables (e.g., genetic and psychosocial factors) contribute to the expression of both these behaviors (Fergusson, Lynskey, & Horwood, 1996; Kendler et al., 1993). For example, Gilbert and

Gilbert (1995) postulated a biopsychosocial model in which genetic factors and biological structures influence the expression of certain personality phenotypes (e.g., neuroticism), with those phenotypes representing vulnerability factors that may interact with environmental circumstances and contribute to the development of both smoking and depression.

Which of these four alternative hypotheses most accurately accounts for the cigarette use–depression relationship is an important topic in the current literature (e.g., Breslau et al., 1998; Brown et al., 1996; Choi et al., 1997; Lerman et al., 1996), though this topic has been investigated more in the adult than in the adolescent literature. Nevertheless, with a 4-year longitudinal study of 1,901 adolescents, Killen et al. (1997) reported that higher levels of depressive symptoms at baseline prospectively predicted smoking onset. The findings of Kandel and Davies (1986) indicated that adolescent depressive symptoms were prospectively related to current and lifetime cigarette use in young adulthood. In terms of the direction of effects, these depression-to-cigarette-use findings have been countered by other adolescent studies supportive of a cigarette-use-to-depression pathway. For example, with a sample of 1,709 adolescents, Brown et al. (1996) reported that smoking prospectively predicted major depressive disorders, even when they controlled for other psychiatric disorders. Similarly, with a sample of 6,863 adolescents, the findings of Choi et al. (1997) indicated that smoking status prospectively predicted levels of depressive symptoms. Using a late childhood (8–9 years) and early adolescent (13–14 years) sample, Wu and Anthony (1999) reported that cigarette use prospectively predicted depressive symptoms but that depressive symptoms did not prospectively predict cigarette use. In a 4-year prospective study of 5,855 adolescents, Wang et al. (1996) reported significant cross-lagged correlations between depression and smoking, thereby providing support for a reciprocal influence model.

In this study we sought to advance the research literature in three ways. First, by using a short-term, intensive longitudinal design (four times of measurement at 6-month intervals), we attempted to better capture the change dynamics between adolescent cigarette use and depressive symptoms. Most prior studies had fewer measurement occasions, longer intervals, and/or did not repeatedly measure both cigarette use and depressive symptoms. Second, we included a relatively broad range of predictors of cigarette use and depressive symptoms to examine the simultaneous influence of these variables within the context of a more expansive multivariate model. Extant longitudinal studies of the adolescent smoking–depression relationship have included either a limited number of predictors or no predictors. The exclusion of predictor variables limits the inferences that can be made about the putative causal dynamics affecting the smoking–depression relationship. Third, given our research design and multiple predictors, we were able to advance the literature by specifying and evaluating statistical models with relevance to all four of the described hypothesized models (i.e., the two unidirectional models, the reciprocal influence model, and the common influence model).

The study had four specific objectives. First, we addressed the possible prospective, bidirectional relationship between depressive symptoms and cigarette smoking among adolescents within the context of other possible confounding influences (e.g., other problem behaviors, such as delinquency, alcohol use, and other substance use). That is, for example, smoking and depressive symp-

toms may be correlated with each other because of a common association with level of alcohol use.

Second, we tested hypothesized models of the smoking–depression relationship that included not only some potential confounding variables but also some key substantive predictors that might commonly or uniquely contribute to cross-temporal changes in both of these behaviors. Our selection of variables for these analyses, although not exhaustive, was based on research that has supported their predictive relationships with cigarette use and depression among teens. Predictors of depression included previous levels of depression (e.g., Kandel & Davies, 1986; Lewinsohn et al., 1994), temperament (e.g., Choi et al., 1997; Gjerde, 1995), gender (e.g., Angold, 1988), and levels of parental and family emotional support (e.g., Ge, Lorenz, Conger, Elder, & Simons, 1994). Predictors of cigarette use included prior smoking behavior (e.g., Choi et al., 1997; Patton et al., 1998); parents' smoking behavior (e.g., Chassin, Presson, Todd, Rose, & Sherman, 1998); friends' alcohol and other drug use, which are strong predictors of adolescents' initiation of and increases in cigarette use (e.g., Ennett & Bauman, 1994; Patton et al., 1998); temperament (e.g., Wills, DuHamel, & Vaccaro, 1995); and levels of family emotional support (e.g., Chassin et al., 1998; Melby, Conger, Conger, & Lorenz, 1993). The four temperament dimensions of general activity level, task orientation, positive mood, and flexibility have been supported in previous studies as significant risk factors for adolescent substance use (including cigarette use), and depression (e.g., Wills & Cleary, 1999; Wills et al., 1995; Windle, 1999).

Our third objective was to develop and use categorical variables for adolescent cigarette use and depressive symptoms as predictors that reflected both the severity and persistence of these phenotypes. We propose that any long-term relationships between cigarette use and depressive symptoms are likely to occur only among those participants for whom the reciprocal relationships and common intervening mechanisms are part of a relatively dominant, persistent, coregulatory system (e.g., Carmody, 1989). Hence, for example, single-occasion (statelike) low-to-moderate levels of depressive symptoms would not be hypothesized to prospectively predict higher levels of cigarette use, but persistently (multioccasion, traitlike) high levels of depressive symptoms would be hypothesized to prospectively predict higher levels of cigarette use. In essence, we argue that the prospective prediction of depressive symptoms on smoking and vice versa is more likely for traitlike than for statelike components because traitlike consistency reflects the establishment of stable neural and cognitive structures that underlie the coregulatory mechanisms (biological, cognitive, and social) that would maintain such a relationship across time. Such stable neural and cognitive structures to maintain the coregulatory activities of smoking and depressive symptoms appear improbable within a statelike model.

Our fourth objective was to evaluate the longitudinal relations between depressive symptoms and cigarette smoking with two alternative statistical models of change: (a) an autoregressive model that focused on individual differences in rank-order stability across a 1.5-year interval and (b) a latent growth curve model (LGC model) that focused on the variability of intraindividual growth trajectories. Because there are a range of definitions of change in behavior and a number of alternative statistical models to assess change (e.g., Bryant, Windle, & West, 1997; Collins & Horn, 1991), it is often valuable to evaluate hypotheses using more

than one statistical model. Using these two statistical models with a four-wave panel design (with 6-month intervals), we have attempted to capture features of adolescent changes (e.g., increases or decreases) in smoking and depressive symptoms and to identify influential variables and mechanisms that contribute to these changes.

Method

Sample

The data used in this study were collected as part of a larger, four-wave panel design that focused on vulnerability factors and adolescent substance use. We refer to the study by the acronym MAVS, which stands for Middle Adolescent Vulnerability Study. The principal objective of the MAVS was to assess the initiation, maintenance, and continuation (or termination) of alcohol and other substance use during the high school years in relation to a range of vulnerability, or risk, factors. These risk factors included temperament and family and peer functioning. Data were collected within the adolescents' high school setting. The initial wave of assessment occurred during the fall semester of the teens' 10th- and 11th-grade years. Waves 2–4 of data collection occurred every 6 months thereafter, with Wave 2 data being collected in the spring semester of Year 1, and Waves 3 and 4 being collected in the fall and spring semesters of Year 2, respectively, when adolescents were in 11th- and 12th-grades. Because of time constraints associated with testing in classroom settings and the desire to measure a broad range of risk and protective factors and health outcomes, not all measures were assessed at all four waves of measurement.

The initial sample included two adolescent cohorts consisting of 975 high school sophomores (53%) and juniors (47%) recruited from two homogeneous suburban high school districts in western New York. Fifty-two percent ($n = 517$) of the sample were young women and 48% ($n = 458$) were young men. The average age of the respondents at the first occasion of measurement was 15.54 years ($SD = 0.66$), and 98% were white. Seventy percent of the sample was Catholic, 18% Protestant, and 12% Other. (Note that these were not Catholic high schools, but rather the enrollment reflected the religious composition of participants in this community.) Ninety-six percent of the fathers and 43% of the mothers were employed full time outside the home (37% of mothers were employed part time outside the home). Fathers completed an average of 13.79 years of education ($SD = 2.39$) and mothers completed an average of 13.55 years ($SD = 2.01$). The average number of children per family was three. The median family income was about \$40,000, with only 3% of the sample reporting family income less than \$12,000. Eighty-eight percent of the adolescents' primary caregivers were currently married, 12% were divorced, and 1% were widowed. Approximately 76% of the high school students eligible for the study participated (see *Procedure*). At the second occasion of measurement (6 months later) the retention rate was 93%. Sample retention across waves of measurement was uniformly high, in excess of 90%.

A supplemental sample of 243 adolescents participated at the second, but not the first, occasion of measurement and were eligible to participate at subsequent measurement occasions. These supplemental participants were added to the study to increase the sample size because of some relatively low base-rate variables (e.g., family history of alcoholism) of interest to the objectives of the larger federally funded project. Statistical comparisons on sociodemographic variables (e.g., parental education level and income) and adolescent problem behaviors (e.g., alcohol use and delinquent activity) indicated that the supplemental sample did not differ in any systematic way from those participants who participated initially at the first measurement occasion. Because active informed consent procedures were used in this study, we did not have information on nonparticipants; hence, it is unclear how representative the participating sample was relative to the total eligible sample except on major sociodemographic variables (e.g., gender and ethnic group composition), which did not reflect participant bias. It has been determined, however, that the drinking and substance-use practices and rates of suicidal ideation and attempts among adolescents in this sample were highly similar to findings in national survey studies (see Reifman & Windle, 1995; Windle, 1996).

Seventy-one percent of the participating adolescents' primary caregivers (92% of whom were women) completed and returned mail survey materials that contained questions about sociodemographic characteristics (e.g., income and parental educational attainment) and family history of alcoholism, mental health problems, and regular smoking. For those primary caregivers who did not return the mail survey, selected information (e.g., income and regular smoking) was collected through telephone calls. This resulted in complete response information for these parent variables for all but 26 of the families participating in the study.

Measures

Depressive symptoms. Depressive symptoms were assessed at all four waves of data collection with the Center for Epidemiologic Studies Depression Scale (CES–D; Radloff, 1977). The CES–D consists of 20 self-report items and provides a unitary measure of current depressive symptomatology, with an emphasis on the affective component, depressed mood. Adolescents were asked to indicate how many days during the past week they experienced the emotions or behaviors indicated in each of the items. Items included "I thought my life had been a failure," "I felt that everything I did was an effort," and "I felt lonely." Serious depressive symptomatology was indicated by a criterion score of ≥23 (Roberts, Andrews, Lewinsohn, & Hops, 1990). Although a score of 16 or more on the CES–D has been commonly used in adult samples as indicating serious depressive symptomatology, Roberts et al. (1990) reviewed several articles that indicated that this criterion yielded rates in excess of 50% for adolescents, compared with 16–20% for general adult population samples. Roberts et al. reported that a more stringent criterion of 23 or more on the CES–D with adolescents yielded a sensitivity estimate of 89.7 and specificity estimate of 74.8 in relation to a *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; American Psychiatric Association, 1987) diagnosis of major depressive disorder. The internal consistency estimate for the CES–D with this sample was ≥.90 at each wave, and these alpha levels are consistent with prior research (e.g., Radloff, 1977; Roberts et al., 1990).

In addition to continuous scores of CES–D depressive symptoms, data from all four waves of measurement were used to construct three depressive symptom groups that represented varying degrees of persistence of serious depressive symptoms. We formed these groups using the following procedure: At each of the four waves, adolescents were divided into low and high depressive symptoms groups, with CES–D cutoff scores of <23 constituting low depressive symptoms and being scored as 0, and scores of ≥23 constituting high depressive symptoms and being scored as 1. Time 1 through Time 4 scores were then summed, and three depressive symptoms groups were formed on the basis of these summated scores: Group 1 consisted of participants who (out of four measurement occasions) never had a CES–D score of ≥23; Group 2 consisted of participants who had a CES–D score of ≥23 1–2 times (i.e., lower persistence of serious depressive symptoms); Group 3 were those participants who had a CES–D score of ≥23 3–4 times (i.e., higher persistence of serious depressive symptoms). The number of adolescents in each group was as follows: Group 1 $n = 598$ (49.1%), Group 2 $n = 339$ (27.8%), and Group 3 $n = 281$ (23.1%).

Cigarette smoking. At each measurement occasion, adolescents were asked, "How many cigarettes or packs of cigarettes did you usually smoke per day in the last 6 months?" Response options were as follows: none, less than 1 cigarette per day, 1–5 cigarettes per day, about ½ pack per day, about 1 pack per day, about 1 and ½ packs per day, and about 2 packs or more per day. The validity of self-report measures of cigarette smoking among school-based samples of adolescents has been supported by signif-

icant associations between biochemical indicators of smoking (e.g., levels of carbon monoxide from expired air) and self-reports of smoking (see, e.g., Wills & Cleary, 1997).

In addition to continuous scores of cigarette smoking, data from Times 1–4 were used to construct three cigarette smoking groups that represented varying degrees of persistence of lighter versus heavier smoking levels. We formed these groups using the following procedure: At each of the four measurement occasions, smoking categories were formed based on the following rating scheme: 0 = *no cigarette smoking, last 6 months;* 1 = *less than ½ pack per day, last 6 months;* 2 = *greater than or equal to ½ pack per day, last 6 months.* Next, three smoking groups were formed using the Time 1 through Time 4 data: abstainers/light smokers[1] were adolescents who received a score of 0 on at least two of the four measurement occasions and never received a score of 2; moderate smokers were those teens who received a score of 1 on at least two of the four measurement occasions and received a score of 2 on no more than two occasions; heavy smokers received a score of 2 on at least three of the four assessments. The number of adolescents in each group was the following: Group 1 *n* = 635, (52.1%), Group 2 *n* = 435 (35.7%), and Group 3 *n* = 148 (12.2%).

Maternal and paternal regular smoking. Included as part of a measure of family history of alcoholism and other mental health problems (Andreasen, Endicott, Spitzer, & Winokur, 1977) was an index on which primary caregivers responded "no" or "yes" to questions as to whether the biological mother or the biological father ever smoked regularly. This lifetime index of ever smoking regularly correlated .60 with measures of current levels of self-reported smoking by the primary caregiver.

Temperament dimensions. Four dimensions of temperament were assessed at Time 1 with the Revised Dimensions of Temperament Survey (DOTS–R; Windle & Lerner, 1986; Windle, 1992). The DOTS–R is a 54-item, factor analytically developed self-report instrument that measures 10 temperament attributes—the four used in this study were General Activity Level, Rigidity–Flexibility, Positive Mood Quality, and Task Orientation. Each item has a 4-point response format ranging from 1 (*usually false*) to 4 (*usually true*). Summary scores for each of the temperamental dimensions were formed by summing individual items (after recoding reversed items). Higher subscale scores indicate higher levels of that temperamental characteristic. Windle and Lerner (1986) reported that Cronbach's alphas for the four dimensions were .84, .78, .89, and .80, respectively, and test–retest stability coefficients across a 6-week interval were .75, .64, .71, and .59, respectively. The correlations between parent–adolescent (interrater agreement) ratings of adolescent temperament among a clinical sample of girls ranged from .66 to .79 (Luby & Steiner, 1993). Validity data on the DOTS–R have indicated correspondence with the Big Five personality factors (e.g., Angleitner & Ostendorf, 1994), and Windle (1999) summarized findings regarding the predictive relations between temperament and several health-related outcome variables (e.g., delinquency, alcohol and illicit drug use, and perceived competence). In addition, positive mood quality correlated significantly with positive, but not negative, affect as measured by the Positive and Negative Affect Scale (Watson, Clark, & Tellegen, 1988).

Perceived Social Support—Family. At Time 1, adolescents completed the Perceived Social Support—Family measure, which assesses the amount of perceived emotional support provided by the family (Procidano & Heller, 1983). The measure consists of 20 items with 4 response options ranging from *generally false* to *generally true.* Examples of survey items include "My family gives me the moral support I need," "Members of my family are good at helping me solve problems," and "My family is sensitive to my personal needs." The response format of this measure was modified to a four-response option format from the original dichotomous response format (plus a "Don't know" response option) proposed by Procidano and Heller (1983) in order to increase the discriminative utility of the measure (Windle & Miller-Tutzauer, 1992). The internal consistency estimate for this measure at Time 1 with this sample was .95.

Delinquent activity. This was measured with 16 items used in prior delinquency research (e.g., Elliott, Huizinga, & Menard, 1989). A 6-point Likert scale (1 = *never,* 2 = *once,* 3 = *2–3 times,* 4 = *4–5 times,* 5 = *6–9 times,* 6 = *10 or more times*) was used for each item in reference to the past 6 months. The alpha level for the 16 items at Time 1 was .75, and test–retest reliability was .70. Items varied in terms of severity of offense and included skipped school, hit teacher or parent, stole something that was valued at more than $20, beat up someone, destroyed public property, and was suspended from school.

Alcohol consumption. This was measured with a standard quantity-frequency index (QFI) that assessed beer, wine, and hard liquor consumption in the past 30 days (Armor & Polich, 1982). Respondents were asked how often they usually had each beverage in the last 30 days (responses ranged on a 7-point scale from 1 = *never* to 7 = *every day*) and, when they had the beverage, on average how much they usually drank (10-point scale from 1 = *none* to 10 = *more than 8* cans, bottles, or glasses, depending on the beverage). The QFI, by including all three beverages, provides a measure of the average number of ounces of ethanol consumed per day in the past month.

Other substance use. A measure of other substance use included the self-reported frequency of using marijuana, hashish, and nonprescribed hard drugs (e.g., cocaine, stimulants, barbiturates, and hallucinogens) during the past 6 months using 7-point Likert scales that ranged from 1 (*never used*) to 7 (*used every day*). The validity of self-reports of substance use has been supported in numerous research studies (e.g., Oetting & Beauvais, 1990; Winters, Stinchfield, Henly, & Schwartz, 1991).

Percentage of friends who use alcohol and other drugs. Adolescents were requested to indicate the number of adolescents that they considered friends. Over 99% of adolescents reported at least one friend, with most reporting five or more. Adolescents were then requested to indicate how many of these friends consumed alcohol. Percentage scores were calculated by dividing the number of alcohol-using friends by the total number of friends and multiplying the dividend by 100, with a possible range of 0–100%. A similar procedure was used to calculate the number of illicit (e.g., marijuana or cocaine) drug-using friends.

Procedure

Subsequent to receiving approval from school administrators to conduct the study, schools provided a mailing list of the addresses of 10th- and 11th-graders. A packet of materials, including a letter of introduction by the principal, a description of the study, and informed-consent forms, was mailed to adolescents and their parents. Those individuals willing to participate in the study were requested to sign the informed-consent form (both the adolescent and one parent) and to return it to the investigator in a self-addressed, stamped envelope. Confidentiality was also assured with a Department of Health and Human Services Certificate of Confidentiality. Teachers made announcements about the study in home classrooms. Adolescents completed survey materials in large groups (e.g., 40–50 students) in their high school setting. A trained survey research team administered the survey to adolescents, and neither teachers nor school administrators were in the room during the time the students completed the surveys. The survey took about 45–50 min to complete and participants received $10.00 for their participation. A make-up date for testing was arranged for par-

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[1] In some research applications it is beneficial and of substantive importance to distinguish abstainers and light smokers. However, in this study in which both higher levels of smoking and persistence of smoking are key features of substantive import, the abstainer–light smoker distinction as defined in this study is of less importance. Furthermore, given that this was the referent group for the dummy variable coding in one of the prospective regression models, it provided for conservative tests for parameters associated with moderate and heavy smoking.

ticipants who were absent or unable to participate on the regularly scheduled day of testing. A similar procedure was used at each wave of measurement. Survey testing occurred in October and April of successive years.

Data Analyses

All data analyses were conducted with the Mplus software program (L. K. Muthén & Muthén, 1998). Mplus estimates missing data with full information maximum likelihood estimation, under the assumption that the data are missing at random (Little & Rubin, 1987). Prior research with this data set has supported the reasonableness of this assumption (e.g., Tubman, Windle, & Windle, 1996). The sample size for all analyses was 1,218. Two different statistical models of change were used in the analyses. First, a standard longitudinal, autoregressive multiple regression model was used in which Time 1 scores were statistically controlled to evaluate the prospective predictive relations of other Time 1 predictors on rank-order changes in the dependent variable between Time 1 and Time 4. Second, latent growth-curve modeling (e.g., Duncan, Duncan, Strycker, Li, & Alpert, 1999; B. O. Muthén & Curran, 1997) was used to model individual differences in intraindividual change trajectories of cigarette use and depressive symptoms, as well as predictors of these change trajectories (for a more complete explication of differences between these and other models of change, see Windle, 1997).[2] For the LGC models, three fit indexes were used in addition to the chi-square test. These three fit indexes were the Tucker–Lewis Index (TLI), the comparative fit index (CFI), and the root-mean-square error of approximation (RMSEA).[3]

Results

Prospective Multiple Regression Analyses

Two multiple regression models were specified to investigate the prospective relationships between depressive symptoms and cigarette use after controlling for their earlier expression. Additionally, other potentially important confounding variables and predictor variables were included in the analyses to identify common and unique predictors of each. The prospective predictors of Time 4 cigarette use and depressive symptoms were primarily measured at Time 1. An exception was that the percentage of friends using alcohol and the percentage of friends using drugs (which were measured at Times 2 and 3, but not Time 1) were included as prospective predictors of Time 4 dependent variables. In these regression analyses, dummy variable coding was used for the (lower and higher) persistent groups for smoking and depressive symptoms when they were used as predictor variables.[4]

The results of the multiple regression models predicting Time 4 smoking and Time 4 depressive symptoms are presented in Tables 1 and 2, respectively. The results of analyses predicting Time 4 cigarette use indicated that smoking at Time 1 was the strongest predictor of smoking at Time 4. After controlling for Time 1 cigarette smoking, statistically significant predictors of rank-order changes in cigarette use were a higher percentage of alcohol and drug-using peers, higher levels of alcohol and illicit drug use, lower levels of positive mood, and, it is important to note, higher (but not lower) persistent depressive symptoms. These variables accounted for 31% of the variance of cigarette smoking at Time 4.

Time 1 depressive symptoms was the strongest predictor of Time 4 depressive symptoms. Controlling for Time 1 depressive symptoms, statistically significant predictors of rank-order changes in Time 4 depressive symptoms were a higher percentage of alcohol-using peers, lower marijuana use, temperamental inflex-

Table 1

Standardized Coefficients for Multiple Regression Model Prospectively Predicting Changes in Adolescent Cigarette Smoking

Predictor variable	Cigarette smoking (T4)
Cigarette smoking	.40***
Gender (1 = boys; 2 = girls)	−.03
Maternal smoking	.01
Paternal smoking	.03
Family social support	.04
Alcohol-using peers	.08**
Drug-using peers	.19***
Delinquent activity	−.04
Alcohol use	.16**
Marijuana use	−.01
Other illicit drug use	.06*
General activity level	−.01
Task orientation	−.02
Positive mood	−.17**
Flexibility	.03
Lower persistent depressive symptoms[a]	.06
Higher persistent depressive symptoms	.13*
Estimated R^2	.31

Note. $N = 1,218$. T4 = Time 4.
[a] Lower persistent depressive symptoms = Center for Epidemiologic Studies Depression Scale (CES–D) ≥23 on 1–2 measurement occasions; higher persistent depressive symptoms = CES–D ≥23 on 3–4 measurement occasions.
* $p < .05$. ** $p < .01$. *** $p < .001$.

ibility in adjusting to changes in the environment, and, it is important to note, heavy (but not moderate) persistent cigarette smoking. These variables accounted for 13.6% of the variance of depressive symptoms at Time 4.

LGC Model

Figure 1 depicts the LGC model that was used to evaluate hypotheses about the shape (e.g., linear or quadratic) of the latent growth curve for cigarette use across the four waves of measurement. The first factor (F1) represents the intercept and contains sample information about the mean and variance of the collection, or group, of individual intercepts that describe each person's growth curve. Each of the four manifest variables (cigarette use Time 1 to Time 4) has factor loadings fixed to 1.0 on F1 to

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[2] Simultaneous group LGC models were conducted for groups age 15 and 16 years, respectively, at Time 1 to address concerns about the potential biasing effects of pooling across age groups on estimated parameters (Mehta & West, 2000). The 15 and 16 year-old age groups constituted 92% of the sample. The simultaneous group models supported the invariance of estimated parameters across age groups; hence, the sample was pooled rather than treated as two separate cohorts.

[3] For the TLI and CFI, values greater than .90 indicate an acceptable fit. For the RMSEA, values between .05 and .09 reflect a well-fitting model (e.g., Browne & Cudeck, 1993).

[4] Cigarette use was nonnormally distributed, therefore all data analyses were conducted using both raw scores and log-transformed scores. The regression and LGC findings were unaltered by the log transformation; hence, the results are presented in the raw score metric.

Table 2

Standardized Coefficients for Multiple Regression Model Prospectively Predicting Changes in Adolescent Depressive Symptoms

Predictor variable	Depressive symptoms (T4)
Depressive symptoms	.35***
Gender (1 = boys; 2 = girls)	.05
Maternal smoking	−.01
Paternal smoking	−.02
Family social support	−.07
Alcohol-using peers	.07*
Drug-using peers	−.03
Delinquent activity	−.01
Alcohol use	−.03
Marijuana use	−.10**
Other illicit drug use	−.01
General activity level	.04
Task orientation	.05
Positive mood	.01
Flexibility	−.16*
Moderate cigarette smoking[a]	.01
Heavy cigarette smoking	.08*
Estimated R^2	.14

Note. N = 1,218. T4 = Time 4.
[a] Moderate cigarette smoking = smoking < one half pack per day on at least 2 of the 4 measurement occasions and no more than 2 measurement occasions in which smoking ≥ one half pack per day occurred; heavy cigarette smoking = smoking ≥ one half pack per day on at least 3 of the 4 measurement occasions.
* $p < .05$.   ** $p < .01$.   *** $p < .001$.

constrain the intercept, or height of the reference curve. Factors 2 (F2) and 3 (F3) represent components of the shape of the growth trajectories for cigarette use across time. F2 represents the linear, accelerating growth component of the individual trajectories, and F3 represents the quadratic, decelerating component of the trajectories. Orthogonal polynomial contrast coefficients were used to scale the shape of the growth components. This was accomplished by fixing the factor loadings with vectors of orthogonal polynomial coefficients. This scaling of the shape of the growth components centers the estimated intercept across the four waves of measurement (i.e., between Waves 2–3).

A two-factor LGC model with an intercept and a linear shape (slope) component indicated a relatively poor fit, $\chi^2(5, N = 1,218) = 29.66$ for the observed data. The quadratic model portrayed in Figure 1 provided a better statistical fit, $\chi^2(1, N = 1,218) = 8.24$; RMSEA = .077; TLI = .98; CFI = .99, and all parameters (i.e., mean and variance estimates) of the intercept, linear, and quadratic factors were statistically significant at $p < .01$. The observed and model estimated means for this quadratic model are provided in Table 3 and indicate that cigarette use initially increases across time and then decreases between Time 3 and Time 4. The factor correlations in Figure 1 represent associations between initial individual differences in level of cigarette use (intercept) and intraindividual differences in the rate of growth or change in cigarette use. The estimated correlation of F1 and F2 was not statistically significant, but the correlation for F1 and F3 was statistically significant and the direction of the correlation
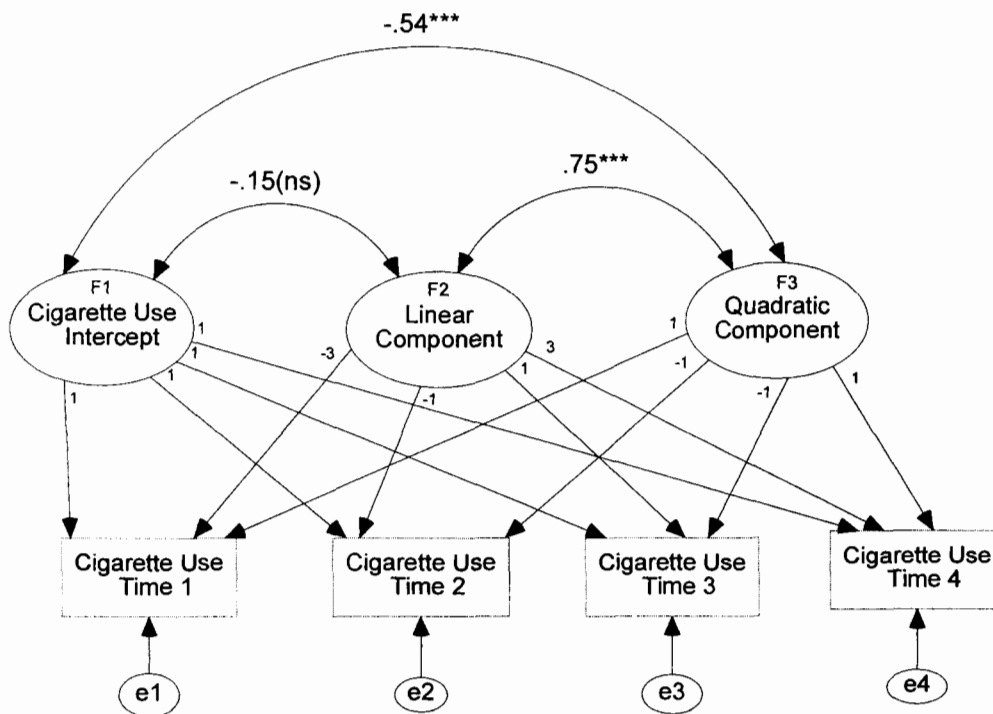

Figure 1. Latent growth model of cigarette use across four times of measurement, $\chi^2(1, N = 1,218) = 8.24$, $p < .001$; root-mean-square error of approximation = .07; Tucker–Lewis Index = .98; comparative fit index = .99. F = factor. The residual terms (e_1 to e_4) represent the residual variance of the four manifest indicators of cigarette use that are not accounted for by the growth curve model, and the double-arrows refer to correlations among the factors. ***$p < .001$.

Table 3

Observed and Estimated Means for Adolescent Cigarette Smoking

Means	Time 1	Time 2	Time 3	Time 4
Observed	2.48	2.62	3.14	2.79
Estimated	2.35	2.76	2.90	2.78

Note. $N = 1,218$.

indicated that higher levels of average cigarette use (at the intercept) were associated with a slower rate of deceleration. The F2 and F3 correlation was also statistically significant and positive, indicating that a more accelerated rate of linear change was associated with a higher rate of deceleration. Collectively, these factor intercorrelation findings suggest that those adolescents using cigarettes at higher average levels at the intercept are by and large maintaining their higher levels of use across time (i.e., they do not show a high rate of deceleration), whereas other adolescents appear to experiment with smoking during Times 2 and 3 but reduce or terminate their smoking between Time 3 and Time 4 (thereby accounting for the high positive correlation between F2 and F3).

An LGC model was also specified for depressive symptoms and included both linear and quadratic specifications. However, parameter estimates corresponding to the variance of the shape factors (e.g., slope and quadratic component) were not statistically significant, though the parameter corresponding to the intercept was statistically significant ($p < .001$). The observed and estimated mean scores revealed a relatively flat profile. Given these findings and consistent with standard LGC modeling practices (e.g., Duncan et al., 1999), we restricted our predictor models to the intercept.

Predictors of LGC Models

With the exception of alcohol- and drug-using peers, all of the predictors used in the previous regression equations (see Tables 1 and 2) were used to predict the growth parameters identified in Figure 1. Because the measurement of alcohol- and drug-using peers occurred after the initial assessment of cigarette use (i.e., they were measured at Times 2 and 3), their entry into the model would have violated the principal of temporal ordering (i.e., independent variables should temporally precede dependent variables in terms of presumed causal ordering). Figure 2 represents the findings of the LGM parameters (i.e., initial status, linear growth, and quadratic growth) regressed on the predictors; to simplify the presentation, only statistically significant predictors are included in the model. With regard to the intercept, initial higher levels of cigarette use were predicted by female gender, inflexibility, higher general activity level, lower task orientation, and higher levels of
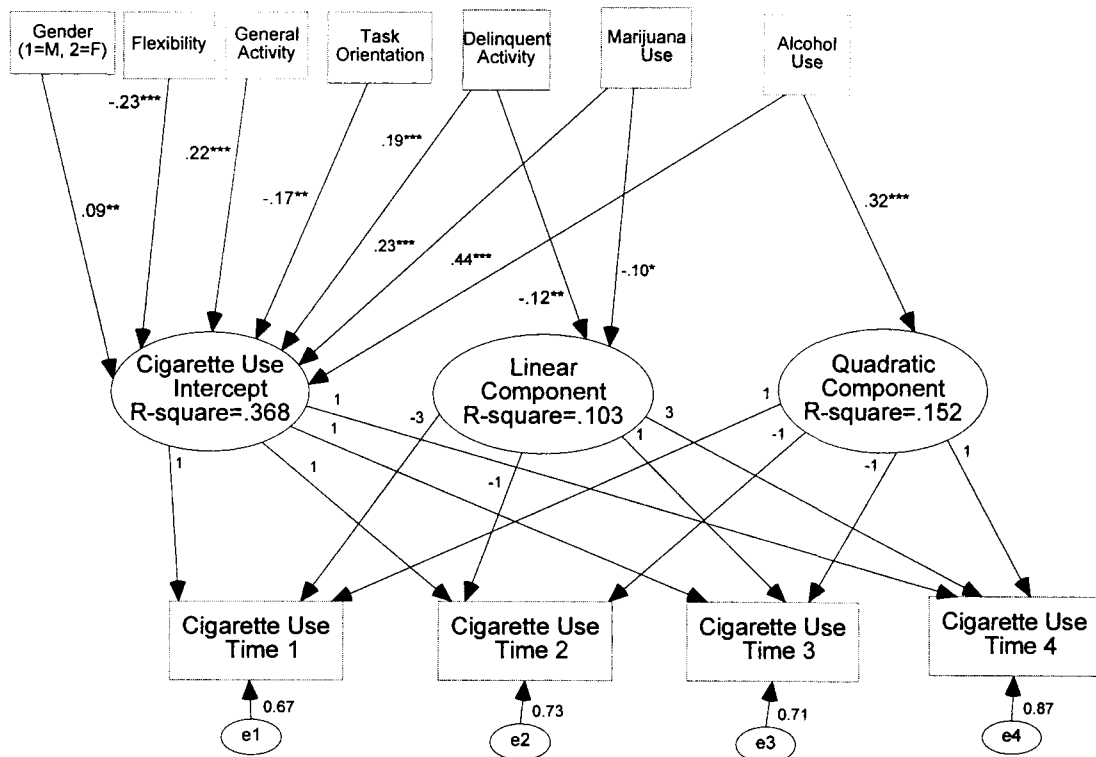

Figure 2. Time-invariant predictors of quadratic model of cigarette use across four times of measurement, $\chi^2(15, N = 1,218) = 54.41, p < .001$; root-mean-square error of approximation $= .06$; Tucker-Lewis Index $= .97$; comparative fit index $= .99$. F $=$ factor. The residual terms (e_1 to e_4) represent the residual variance of the four manifest indicators of cigarette use that are not accounted for by the growth curve model. $*p < .05$. $**p < .01$. $***p < .001$.

delinquent activity, alcohol use, and marijuana use; these variables account for 36.8% of the variance of the intercept.

The linear (accelerating) component is predicted by lower delinquent activity and marijuana use at Time 1. These findings for predictors of linear growth appear paradoxical but, nonetheless, reflect a common finding in studies of change across time (e.g., Kessler & Greenberg, 1981).[5] These predictors accounted for 10.3% of the variance of the linear component. The only significant predictor of the quadratic (decelerating) component was higher level of alcohol use at Time 1, and the predictors accounted for 15.2% of the variance of the quadratic component. This may reflect some sort of maturational process whereby earlier alcohol and other substance experimentation, including cigarette smoking, were at higher levels at Time 1 but across time covaried toward the deceleration trend indicated in Figure 2. Of importance for the LGC model, neither moderate nor high persistent depression significantly predicted the intercept or other growth parameters (neither did a continuously scored Time 1 CES–D variable specified in a separate model).

The prediction model for the intercept of depressive symptoms (i.e., Time 1 depressive symptoms) indicated that higher levels of depressive symptoms were predicted by female gender, heavier alcohol use, lower family support, higher general activity level, and lower positive mood quality. These predictors accounted for 43.3% of the variance of Time 1 depressive symptoms. Neither moderate nor heavy persistent smoking was a significant predictor in this regression model (nor was a continuously scored Time 1 cigarette-use variable specified in a separate model).

Discussion

Guided by the findings of the extant literature (e.g., Brown et al., 1996; Choi et al., 1997; Fergusson et al., 1996; Wang et al., 1996), we addressed four major objectives related to the prospective association between cigarette use and depressive symptoms among adolescents. First, our findings indicated that even within the context of some (but not all) potential confounding variables, such as alcohol and other substance use and delinquent activity, heavy and persistent levels of cigarette smoking significantly predicted increases in depressive symptoms across a 1.5-year interval. Similarly, within this context of potential confounding variables, persistently high levels of depressive symptoms prospectively predicted increases in smoking across a 1.5-year interval. To our knowledge, these mutual influence findings within the context of a prospective research design that included potentially confounding variables, substantive predictor variables, and Time 1 control of the dependent variables are unique to the adolescent literature.

Second, in addition to potential confounding variables associated with other problem behaviors, heavy and persistent levels of smoking and persistently high levels of depressive symptoms remained statistically significant predictors in their respective regression equations within the context of a broad set of potential "third variable" causes whose common influences may have eliminated the significant associations between smoking and depressive symptoms (e.g., Fergusson et al., 1996). Third, the derived categorical variables for heavy and persistent levels of smoking and persistently high levels of depressive symptoms appeared to capture joint information about severity and persistence that was valuable in the prospective regression analyses and indicated

stronger predictive relationships than single occasion, continuous measures. Fourth, each of the two statistical models (i.e., autoregressive regression model and LGC model) provided unique information on modeling changes in adolescent smoking behaviors and depressive symptoms and in the identification of predictors of these changes. Issues pertinent to the findings for each of these four objectives are provided below.

With regard to the four hypothesized models of the cigarette use–depressive symptoms relationship (i.e., unidirectional cigarette-use-to-depression, unidirectional depression-to-cigarette-use, bidirectional, and third variable), strongest support was provided for the bidirectional or reciprocal influence model. The strength of these bidirectional influence findings was bolstered by the inclusion of other possible confounding variables (e.g., alcohol and other substance use, delinquent activity) that may have been related to both smoking and depression and, thereby, may have contributed to a spurious correlation. Brown et al. (1996) reported that a statistically significant relationship still existed between smoking and depressive disorder when they controlled for other psychiatric disorders. Wang et al. (1996) did not include potentially confounding variables in their study of adolescent depression and smoking, and hence our study provides further support for the bidirectional-influence model within the context of a relatively rigorous research design.

Support for the bidirectional-influence model does not negate the theoretical and empirical research that has been completed using unidirectional models; it simply suggests that such models may not be comprehensive in relation to the dynamics of change involving smoking and depression. Accordingly, that persistently high levels of depressive symptoms significantly predicted cross-temporal increases in cigarette use is consistent with the self-medication hypothesis of smoking (e.g., Lerman et al., 1996). Similarly, heavy and persistent smoking may influence vulnerability to depression because of alterations in brain biochemistry. For example, nicotine positively affects levels of dopamine in the brain, both through stimulation of dopamine release and through inhibition of dopamine reuptake (Lerman et al., 1999), and it is through these actions on the dopaminergic system that nicotine may mediate the relationship between depressed affect and smoking in adolescents as well as adults. Some researchers have focused on affect-regulation models (e.g., Carmody, 1989; Parrott, 1995) that attempt to account for the regulatory dynamics underlying the reciprocal relationship between nicotine and negative mood, whereas others (e.g., Gilbert & Gilbert, 1995) have sought to identify genetic and neurobiological substrates that may be common across smoking, temperament–personality, and psychopathology. Although findings from our study obviously did not assess hypothesized intervening genetic or neurobiological mechanisms, they nevertheless suggest the utility of future research that ex-

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[5] Those adolescents who were smoking at higher levels at Time 1 were also probably engaging in higher levels of delinquency and marijuana use; therefore, they exhibited very little (if any) increase in smoking across time (and therefore Time 1 delinquency and marijuana use did not covary with changes in smoking). However, those adolescents who smoked less at Time 1 but increased their smoking across time also probably had lower levels of delinquent activity and marijuana use at Time 1; hence, their levels of delinquency and marijuana use at Time 1 significantly covaried with linear changes in smoking across time.

plores potential biological mechanisms linking depression and smoking among adolescents. The early identification of common (and unique) vulnerability genes for smoking and depression could provide enormous guidance for prevention and treatment with children and adolescents.

In addition to testing the directional relationship between depressive symptoms and cigarette use, an additional focus was to identify common and unique predictors of cross-temporal changes in both smoking and depression, after controlling for their baseline levels. Our findings suggest that the predictors of such change are both common and unique. In accordance with the findings of previous research, the strongest predictor of continued smoking was previous smoking (e.g., Choi et al., 1997), and, similarly, baseline levels of depressive symptoms at Time 1 were the strongest predictor of symptoms at Time 4 (e.g., Kandel & Davies, 1986; Lewinsohn et al., 1994).

It is not surprising that some of the most potent predictors of individual differences in rank-order stability in smoking and depressive symptoms across time were other substance use, especially alcohol use, and peer alcohol- and illicit-drug use. An increase in cigarette, alcohol, and other drug use commonly occurs during adolescence within the peer context (e.g., Ennett & Bauman, 1994; Kandel, 1980), and such substance-using behaviors are often associated with depressive symptoms (e.g., Windle & Windle, 1996). Given the strengths and interrelatedness of these substance-using behaviors and substance-related peer context indicators (i.e., percentage of alcohol- and drug-using peers), the challenge for other predictors to be statistically significant in our prospective analyses was substantial. Nevertheless, temperamental inflexibility prospectively predicted increases in depressive symptoms. Temperamental inflexibility refers to difficulties in adjusting to changes in the environment and has been associated with indicators of stress and anxiety (e.g., Windle, 1999). Given that temperamental factors are typically about 50% heritable, it is plausible that temperamental inflexibility may function as a vulnerability factor for negative mood–nicotine escalation across time. The temperamental factor of low positive mood quality was a prospective predictor of increases in cigarette use, further supporting the linkage between smoking and mood regulation (e.g., Parrott, 1995). Although existing literature indicates that negative mood may foster smoking, our findings add to the literature by suggesting that the absence (or low level) of positive mood also may contribute to smoking (Watson et al., 1988). For these prospective models, neither perceived family emotional support nor actual parental smoking significantly predicted changes in smoking or depressive symptoms.

In interpreting the findings of this study and in recognizing that different predictors may be associated with different levels or stages of smoking (e.g., onset, escalation, maintenance, and cessation), it is important to note that our findings are based on our emphasis on heavy and persistent smoking and on persistently high levels of depressive symptoms. As discussed previously in this article, we propose that (a) evolving, reciprocal relationships between smoking and depressive symptoms are likely to reside in more traitlike, neural and cognitive structures and in associated coregulatory processes that maintain and foster this dynamic relationship (e.g., Carmody, 1989; Parrott, 1995) and (b) lower, or less intense, levels of smoking and depression may not be sufficient to establish a stable and dominant coregulatory system. The severity

and persistence operational definitions of smoking and depressive symptoms were such that each index prospectively predicted depressive symptoms and cigarette use, respectively, within the context of multiple other predictor variables. Neither low-to-moderate levels of these severity and persistence indexes, nor continuously measured (single occasion) variables of cigarette use and depressive symptoms, were statistically significant predictors in the regression models. We believe that an emphasis on the severity and persistence of cigarette use and depressive symptoms in adolescents is needed to complement the much more extensive research on smoking onset or on single-occasion measures of depressive symptoms and cigarette use. With regard to smoking, Nordstrom and Kinnunen (2000) reported that heavier smokers are at more risk than light smokers for long-term smoking; thus, early (childhood and adolescent) intervention with such heavier smokers may reap substantial health benefits.

Our findings with the regression-based statistical model provided relatively straightforward results on predictors of change in cigarette use and depressive symptoms. The findings with the LGC model provided useful information in two ways not provided by the regression model. First, by modeling intraindividual change trajectories in mean levels, and not just individual differences in rank-order stability, the functional form or shape of cigarette use across the four waves of measurement was quadratic, which suggests an initial acceleration in cigarette use followed by a deceleration between Times 3 and 4. This decelerating trend has been reported in other domains such as delinquency (e.g., Windle, 2000) and speculatively may reflect a maturational process among some adolescents who have moved beyond the experimental substance-use phase (including smoking) and are preparing for college or other post–high-school activities. Nevertheless, this decelerating trend was not pervasive across adolescents and was at its lowest rate of deceleration among those adolescents previously smoking at the highest levels. This information was not evident based on the regression analyses. Second, although the description of findings related to the predictors of the linear and quadratic components was somewhat cumbersome because of paradoxes associated with the measurement of change (e.g., Kessler & Greenberg, 1981), they nonetheless provide an additional perspective on the dynamic processes involved when trying to measure change phenomena (e.g., Bryant et al., 1997; Collins & Horn, 1991). It is conceivable that the heterogeneity of intraindividual trajectories of smoking among adolescents may reflect subgroup homogeneity (e.g., stable high smokers, linearly increasing smokers, and experimenters who manifest a high rate of deceleration) that would be better represented with alternative statistical models (e.g., mixture-distribution models). Some of the "noise" associated with the predictors of change in the LGC model may be reduced by such a subgroup formulation and may provide clarity on the predictors of cigarette use, which may vary contingently with specific (subgroup) patterns of mean-level changes.

There are several important intervention implications that follow from our findings. First, interventions aimed at treating cigarette use–nicotine dependence among teens need to recognize that, as with adult smokers, there is substantial co-occurrence between smoking behaviors and depressive symptoms among this age group. Consequently, to maximize treatment effectiveness, it is important that smoking interventions assess levels of internalizing problems (such as depressed affect, low self-esteem, and suicidal

ideation) and, where appropriate, include components that address both the smoking behaviors and the concomitant internalizing symptoms (e.g., Thompson, Horn, Herting, & Eggert, 1997). Second, whereas prior levels of cigarette use and depressive symptoms predict increases in levels of cigarette use and depressive symptoms, respectively, both dispositional (e.g., temperament) and social (e.g., peers) influences also contribute to increases and decreases in these behaviors across time. Hence, interventions may need to target the multilevel influences (e.g., pharmacologic vulnerabilities, temperament, and peer influences) that account for the maintenance or escalation of levels of smoking and depressive symptoms across time. Third, our findings indicate that as a complement to primary prevention programs aimed at eliminating smoking onset among early adolescents, secondary and tertiary intervention programs are needed for middle adolescents who have already initiated smoking and are escalating to or maintaining higher levels of use.

The current study has several limitations. First, the sample is predominantly White, suburban, and middle class; thus, the generalizability of the findings to other adolescent ethnic groups is not known and merits study. Second, the data were collected by means of self-reports, which may introduce monomethod bias and affect the resulting findings. A multimethod protocol would have strengthened tests of the proposed models. Third, some of the constructs (e.g., adolescent cigarette smoking and parental regular smoking) were measured with a small number of items that may have imposed restrictions on the reliability of the assessed construct. Fourth, although the depressive and smoking groups were formed in such a way as to represent serious and persistent levels of depressive symptoms and smoking behaviors, these group formations should not be equated with the diagnostic categories of major depressive disorder and nicotine dependence, respectively. It would be beneficial for research to include major depressive disorder and nicotine dependence in studies using multiple predictor models and investigating the directionality of the depression–smoking relationship. Similarly, it would be advantageous to assess other psychiatric disorders (e.g., anxiety disorders) to include as potential confounding variables in specified models. Fifth, although we accounted for substantial amounts of variance in the dependent variables, other predictors (e.g., coping skills, family history of depression, depressive attributional style, and sibling and peer smoking behaviors) are needed to more fully account for additional variation in the outcomes of interest. Despite these limitations, our findings provide strong support for prospective linkages between depressive symptoms and smoking behaviors among middle adolescents. More effective interventions for adolescent smoking and depression may benefit by incorporating this information into treatment protocols.

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