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Differentiating Stages of Smoking Intensity Among Adolescents: Stage-Specific Psychological and Social Influences

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Researchers' understanding of the impact of sociocultural and psychological factors on the various stages of adolescent smoking uptake is limited. Using national data, the authors examined transitions across smoking stages among adolescents (N = 20,747) as a function of interpersonal, familial, and peer domains. Peer smoking was particularly influential on differentiating regular smoking, whereas alcohol use was most influential on earlier smoking. Although significant, depression and delinquency were attenuated in the context of other variables. Higher school grade was more likely to differentiate regular smoking from earlier smoking stages, whereas African American ethnicity and connectedness to school and family were protective of smoking initiation. Results lend support for an interactional approach to adolescent smoking, with implications for stage-matched prevention and intervention applications.

Smoking rates among adolescents have risen over the past decade, with prevalence of current cigarette smoking (i.e., at least 1 cigarette in the past 30 days) among high school students increasing from 27.5% in 1991 to a high of 36.4% in 1997 (Centers for Disease Control and Prevention [CDC], 1998), with a slight decrease in 1999 to 34.8% (CDC, 2000). Among high school seniors, 23% report daily smoking (Johnston, O'Malley, & Bachman, 2000), with 8.9% of seniors smoking more than 10 cigarettes per day (CDC, 2000). Research suggests that nearly all first use of tobacco occurs by the age of 18 (U.S. Department of Health and Human Services [U.S. DHHS], 1994), with considerable stability in smoking status from adolescence to adulthood (Chassin, Presson, Pitts, & Sherman, 2000; Chassin, Presson, Sherman, & Edwards, 1990; Derzon & Lipsey, 1999). Furthermore, teens who begin smoking regularly at a younger age are more likely to be nicotine dependent in adulthood (Stanton, 1995). Although a third of high school seniors smoking a pack per day believe they will quit within 5 years, the median cessation age is 33 years for men and 37 years for women (Pierce & Gilpin, 1996). Similar to adult smokers, many adolescents report frequent unsuccessful quit attempts and withdrawal symptoms on nicotine abstinence (Colby, Tiffany, Shiffman, & Niaura, 2000) and, according to the 1994 Surgeon General's report (U.S. DHHS, 1994), most adolescents reporting daily smoking are nicotine dependent.

Leventhal and Cleary (1980) originally described smoking as a complex behavior that evolves through several stages. Smoking in adolescence is commonly conceptualized as progressing through a sequence of developmental stages characterized by different stages of smoking frequency and intensity (see Mayhew, Flay, & Mott, 2000, for review), often culminating in nicotine dependence (Colby et al., 2000). Basic definitions of smoking have been summarized as preparation, initial trying, experimentation, regular use, and addictive use (Flay, 1993; Flay, Ockene, & Tager, 1992). The preparation stage involves formation of beliefs and attitudes about smoking prior to ever trying a cigarette. Initial trying refers to experimentation with the first few cigarettes. Experimentation is characterized by irregular use of cigarettes, with a gradual increase in the frequency of smoking in various situations. Regular use refers to smoking on a regular, although still infrequent, basis, such as every weekend or weekdays before or after school. Addictive

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use refers to adolescent smoking that occurs on a regular basis and is driven by cravings for nicotine, regular daily smoking, and experience of withdrawal symptoms (Colby et al., 2000).

Although investigators have underscored the theoretical importance of conceiving of youth smoking as a series of developmental stages (Flay, 1993), there remains some discussion over the staged versus continuous nature of smoking uptake (Mayhew et al., 2000). Opponents of stage conceptualizations argue that research to date has consisted primarily of arbitrary cutpoints for smoking-stage boundaries. Proponents argue that stage conceptualizations reflect the dynamic phenomenon of developmental changes taking place and have important implications for controlling smoking among adolescents (Flay, Hu, & Richardson, 1998). Furthermore, although many previous studies have assumed that stage movement is influenced in a linear fashion by explanatory factors (an artifact of statistical methods that have heretofore been available), it is possible to evaluate whether a particular variable exerts differential influence on early stages of smoking, with diminishing influence as smoking stage advances. This has tremendous implications for prevention efforts, as primary prevention strategies may best be implemented by focusing on stages of preparation and initial trying, whereas secondary prevention efforts involve getting experimenters and regular users to quit. For example, sparse research concerning factors that are associated with higher levels of smoking, such as family conflict, could be used to increase the efficacy of teen smoking-cessation programs (Flay et al., 1998). Stage conceptualizations of smoking provide the structure for investigations of which variables, whether at the individual or environmental level, distinguish various levels of cigarette use and may serve to allow for better identification of which individuals may progress to higher stages of cigarette use and possible dependence.

Although multiple psychosocial risk and protective factors have been linked to youth smoking and its progression from initial to regular use, including various sociodemographic, contextual, behavioral, psychological, and biological variables (Hawkins, Catalano, & Miller, 1992; U.S. DHHS, 1994), few studies have investigated the salience of variables at particular smoking stages (see Mayhew et al., 2000). However, several recent studies have highlighted factors that appear to vary along the smoking continuum. For example, onset of smoking has consistently been linked to susceptibility to smoking (Pierce, Choi, Gilpin, Farkas, & Merritt, 1996), deviance and antisocial behavior (C. Reynolds & Nichols, 1976), and other drug use (McGee & Stanton, 1993). Flay et al. (1998) found that transition from trial to experimental smoking was related to friends' smoking, cigarette offers by friends, smoking intentions, grade, and alcohol and marijuana use. Experimentation and regular smoking have been associated with individual-level variables of gender and race (Flay, d'Avernas, Best, Kersell, & Ryan, 1983), affect regulation (Pallonen, Prochaska, Velicer, Prokhorov, & Smith, 1998), concerns about weight gain (Robinson, Klesges, Zbikowski, & Glaser, 1997), positive attitudes toward smoking (Pallonen et al., 1998), as well as parent and family influences (Flay et al., 1998; Mayhew et al., 2000).

Research investigating the etiology of adolescent drug use (Jessor & Jessor, 1977; Magnusson, 1988) proposes an interactional approach to drug use and abuse, in which individuals are thought to develop in reciprocal interaction with their environment (Brook, Cohen, Whiteman, & Gordon, 1992). Based originally on Bandura's (1977) social learning theory, the assumption is that adolescents' acquisition of behaviors and values is based in large part on the complex web of interpersonal social relationships in which the individual is fixed. Adolescent problem behaviors, including drug use, are proposed to be a result of interactions between intrapersonal variables, such as personality, attitudes, and behaviors, and the environmental system, including both family and peers (Jessor & Jessor, 1977; Kandel & Davies, 1992; Petraitis, Flay, & Miller, 1995). Incorporating this interactional approach into a stage conceptualization of smoking, we are able to work within a theoretical framework involving the adolescent and interactions with his or her surrounding environment, namely family and peers, investigating how these may be related to smoking behaviors.

On the basis of an interactional approach, we proposed in this study to use advanced statistical methods that permit modeling of the multivariate influence of variables within three domains—intrapersonal, family, and peer—on various transitions between smoking stages, without assuming that transition across stages is a linear function of the variables. Within the intrapersonal domain, we included sociodemographic variables of gender, ethnicity, age, grade, and poverty level, as well as measures of depression, delinquency, and alcohol use. Depression has consistently been associated with adolescent smoking (Patton et al., 1996; Windle & Windle, 2001), although causality remains less clear (Brown, Lewinsohn, Seeley, & Wagner, 1996; Kendler et al., 1993; Wu & Anthony, 1999). Delinquency and acting-out behaviors have been found to be strongly related to cigarette use, as well as to drug use in general (Brook et al., 1992; Kandel & Davies, 1992), with conduct disordered symptoms predictive of substance use and abuse in adolescents, regardless of their gender (Disney, Elkins, McGue, & Iacono, 1999). Recently, Ellickson, Tucker, and Klein (2001) found that those 7th graders who smoked, whether experimentally or more regularly, were at greater risk for behavior problems at school, delinquent behaviors, and relational violence in the 12th grade than were nonsmokers.

Within the family domain, we have included measures of parental smoking, as well as connectedness with family. Previous research has found parental smoking to be a strong influence on adolescent smoking. Conrad, Flay, and Hill (1992) found that among 7 of 13 studies reviewed, parental smoking significantly predicted smoking onset among youth. Although parental smoking may play a role in predicting trying, experimenting with, and regular smoking, more recent research suggests parental-smoking effects may be strongest for the transition to regular smoking (Flay et al., 1998), influencing smoking initiation only indirectly. Similarly, family communication about tobacco use predicted smoking escalation in teens but not initiation of smoking (Ennett, Bauman, Foshee, Pemberton, & Hicks, 2001), and family conflict significantly predicted transition from experimental to regular use of cigarettes (Flay et al., 1998).

Peer group often serves as a major influence on adolescent's substance use, an importance possibly explained by selective association and socialization by peers (Kandel, 1980). Within the peer domain, we included measures of peer daily smoking and school connectedness. Peer substance use has been shown to distinguish adolescent light from moderate use of illegal substances (Kandel & Davies, 1992). With respect to smoking, friends' smoking has been shown to both directly and indirectly

affect adolescent smoking initiation and, more specifically, transition from trial to experimental use (Flay et al., 1998).

Variables chosen for analyses were included on the basis of an existing relationship in the adolescent smoking literature, as well as their clinical relevance. Through cross-sectional analyses, we were able to examine whether each of these variables had a uniform influence in differentiating between stages or whether their influence was stronger or weaker at particular stages. It is important to note that, although we have opted to evaluate smoking as a staged variable, the statistical analyses we incorporated presume an underlying continuum to smoking behavior. Using data from the National Longitudinal Study of Adolescent Health (Add Health; Berman, Jones, & Udry, 1997), a population-based sample, we examined the interplay of intrapersonal, family, and peer-level variables and their influences on adolescent smoking at its various stages. On the basis of previous findings (Flay et al., 1998), we hypothesized that peer (peer smoking, school connectedness) variables may have more of an impact on earlier stages of initiation or experimentation, whereas intrapersonal (sociodemographics, depression, delinquency, alcohol use) and family (parental smoking, family connectedness) variables would be more important at later stages of use. We also examined gender differences among these predictors.

Method

Study Sample and Survey Procedure

The sample was drawn from the restricted use Add Health data set. Permission was granted by the original study principal investigators for use of the data, and human participants' approval was obtained from the hospital Institutional Review Board.

Participants consisted of 20,747 adolescents who were originally drawn from a national school survey of 90,118 adolescents. Participants completed in-home interviews administered between April and December of 1995. Adolescents were randomly selected and stratified by gender, grade, and school; they were from Grades 7 through 12 from 80 high schools and 52 feeder middle schools. These high schools were chosen from an original sample of all United States high schools that included an 11th grade and enrolled 30 or more students. The 80 high schools were systematically selected from the original sampling frame, with stratification by region, urbanicity, ethnicity, school type, and school size, to ensure that they were nationally representative.

Study interviewers read and recorded responses for items deemed to be minimally intrusive or sensitive. To maximize confidentiality, adolescents self-reported items deemed to be highly sensitive onto a laptop computer using audio-computer-assisted interview technology. Additional details about the survey design may be obtained elsewhere (Berman, Jones, & Udry, 1997).

Measures

Smoking stage. Consistent with previous literature (CDC, 1993; Flay, 1993; Flay et al., 1992) smoking stage was defined on the basis of smoking frequency and recency. *Never smokers* were defined as those adolescents who denied ever trying a puff or two of cigarettes. *Experimental smokers* were defined as those who endorsed trying cigarettes, although denied smoking within the past 30 days or ever smoking regularly (i.e., daily smoking). *Intermittent smokers* were defined as those who reported smoking between 1 and 29 out of the past 30 days. *Regular/Established smokers* were defined as those who reported smoking on a daily basis within the past 30 days. *Ex-smokers* were defined as those who reported quitting smoking; endorsed regular, daily past smoking; and denied smoking within the past 30 days. However, in regression analyses reported, the ex-smoker category was omitted because of small counts that led to convergence problems with the estimation algorithms.

Sociodemographic variables. Variables including gender, ethnicity, poverty, age, grade, and grade repetition were assessed. Although the race and ethnicity questions contained within the Add Health study allowed adolescents to choose multiple racial and ethnic backgrounds, a categorical race–ethnicity variable was created using the following logic: A respondent was classified as White, African American, or Asian if he or she marked that category only and did not claim a Hispanic background. A respondent was classified as Hispanic if he or she claimed a Hispanic background, regardless of racial background. A race category of Other was used for all other responses, therefore allowing racial categorizations of White, African American, Hispanic, Asian, and Other. Poverty level was assessed with parental report of total household income before taxes in 1994. Poverty was then defined as total household income below 1.5 times the U.S. Census Bureau 1994 poverty thresholds adjusted for household size and number of related children under 18 years of age (Goodman, 1999). Similar to Goodman (1999), we categorized poverty level in the following way: below 1.5 times the poverty threshold, 1.5 to less than 2.5 times the poverty threshold, 2.5 to less than 4 times the poverty threshold, and 4 times the poverty threshold and higher, according to 1994 census data obtained in figures located at www.census.gov/hhes/poverty/threshld/thresh94.html.

Intrapersonal variables. Depression was evaluated with a 17-item scale (Resnick et al., 1997) consisting of items assessing past week symptoms of depression, including poor appetite, anhedonia, distraction, depressed mood, and loneliness. Principal-components analysis yielded a single-factor solution with good internal consistency (Cronbach's $\alpha = .86$).

Delinquency was assessed with an 11-item scale (Resnick et al., 1997) consisting of items assessing such past-year behaviors as destroying property, lying to parents, stealing, running away from home, and skipping school. Principal-components analysis yielded a single-factor solution with good internal consistency (Cronbach's $\alpha = .85$).

Alcohol use was assessed with the question, "During the past 12 months, on how many days did you drink alcohol?" Original response choices were "Never in the past 12 months," "one or two days in the past year," "once a month or less," "2 or 3 days a month," "1 or 2 days a week," "3 to 5 days a week," and "every day or almost every day." However, given extremely small cell sizes in several frequent-use categories, we combined the four higher categories. Therefore, responses were grouped into the following: "Never in the past 12 months," "1 or 2 days out of the past year," "1 day per month or less," and "2 or more days per month."

Family variables. Parent smoking was coded as positive if any of the adolescents' biological or resident mother or father endorsed current smoking, as reported by residential parent. Family connectedness was assessed with the Parent–Family Connectedness scale (Resnick et al., 1997), which consists of 13 items assessing the adolescent's general relationship with and feelings toward his or her family members. Internal consistency was good ($\alpha = .73$; Resnick et al., 1997).

Peer variables. Peer daily smoking was assessed with the question, "Of your 3 best friends, how many smoke more than one cigarette per day?" School connectedness was assessed by an eight-item scale designed to assess perceptions of school atmosphere and the adolescent's relationship with peers and teachers (Resnick et al., 1997). Internal consistency was good ($\alpha = .75$; Resnick et al., 1997).

Statistical Analyses

As can be seen from Figure 1, 97.4% of the population of interest can be classified into one of four smoking stages (1 = never smoker, 2 = experimental smoker, 3 = intermittent smoker, 4 = regular/established smoker) that are ordered by degree of smoking and presumed to reflect an

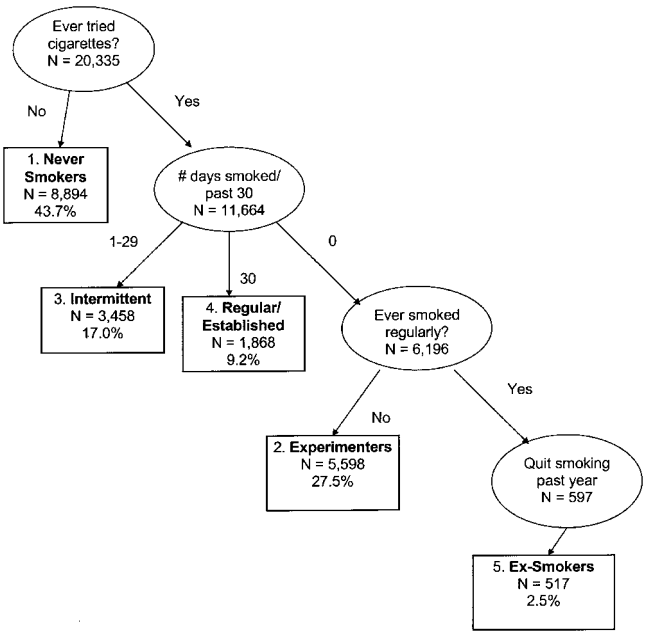

Figure 1. Branch diagram of Add Health adolescent smoking stage categorization and corresponding sample sizes.

underlying dimension of nicotine dependence. Given the small sample size and to focus on the four-category ordinal outcome alone, we chose not to further analyze the last smoking category (5 = ex-smoker) within this study.

A flexible parametric model for analyzing ordinal outcomes is the cumulative odds ordinal regression model (Agresti, 1990), which assumes that the stages in which we classify smoking transitions correspond to a discretization of an underlying continuous smoking scale. Further, when the latent variable is taken to have a logistic distribution in the population of interest and the covariates have uniform impact on all the thresholds of this underlying dependence scale, the model is called a proportional odds model. Generalizations of this model allow a subset of the covariates to have threshold-specific effects (Peterson & Harrell, 1990), thereby allowing exploration of the salience of different predictors on various stages of smoking. Improvements in model fit resulting from adoption of a partial proportional odds model can be assessed by likelihood ratio tests.

In coding the data, we chose to standardize all five continuous covariates (age, depression, delinquency, family, and school connectedness) by subtracting off the median and dividing them by the distance between the median and the third quartile. Exponentiating the corresponding regression coefficient gives us the change in the odds that the response will be in a stage greater than k ($k = 1, 2, 3$) rather than a stage no higher than k, when the value of the covariate is increased from its median to its third quartile.

Following the advice given in Hedeker and Mermelstein (1998), we discretized with quartile splits all continuous covariates for which we wished to assess the nonproportionality assumption, so as to ensure that probability estimates fell inside the (0, 1) interval. Those predictors for which nonproportionality could be rejected were subsequently reentered in their original form and tested for possible nonlinearity. Ordinal-categorical predictors (grade, poverty, alcohol use, peer smoking) were coded using first-to-zero contrasts. Race was coded with White as the baseline group. Binary predictors (parental and peer smoking, cigarette availability, female gender) were coded as 0 = *no*, 1 = *yes.*

To this point, our model description has assumed a simple random-sampling scheme, in which participants are drawn independently from the population of interest. However, because individual participants were actually sampled in clusters—schools in this case—allowance had to be made for the fact that participants in the same cluster may be more alike than those of different clusters. Between-cluster heterogeneity was partly accommodated by allowing the intercept to vary from school to school according to a normal random-effects distribution. The resulting mixed effects ordinal regression model was fit using the well-tested program MIXOR (Hedeker & Gibbons, 1996), an updated copy of which can be obtained at no cost from www.uic.edu/~hedeker/mix.html.

Results

Sample Characteristics

On the basis of defined stages of adolescent smoking, Figure 1 presents a branch diagram of stage classification and the corresponding sample sizes. Table 1 presents the demographic and key variable characteristics of the total sample ($N = 20{,}747$), as well as the conditional distribution of smoking stage at each level of the predictors. The mean age of participants was 16.16 years ($SD = 1.72$) and ranged from 12 to 18 years. The sample was evenly distributed between genders (49.5% male). Just over 50.0% of the sample was White, with 22.6% African American, 17.0% Hispanic, 7.1% Asian, and 2.9% other (consisting primarily of Native Americans). Continuous predictors have been discretized with quartile splits. By including the ex-smokers in our tabulation, we were able to assess for each covariate whether reducing the five-category nominal response to a four-category ordinal outcome resulted in percentage losses that differ dramatically by level. It is of note that the percentage of ex-smokers remained low and stable across variables, rarely going outside a 1%–4% range.

Odds Ratios

As assumed in the cumulative odds ordinal regression model, stages of smoking represent the discretization of the underlying measure of smoking. In Table 2, we present crude odds ratios for each of three contrasts: (a) experimental, intermittent, and regular/established versus never smokers; (b) intermittent and regular/established versus never and experimental smokers; and (c) regular/established versus never, experimental, and intermittent smokers. These contrasts represent various cutpoints along this underlying smoking scale, which is presumed to reflect nicotine dependence. The crude odds ratios are useful in examining bivariate relationships between the outcome and the response and in screening covariates for failure of the proportional odds assumption. Because odds ratios for the baseline categories are all one by definition, the first row for each variable contains baseline odds rather than odds ratios. For example, Table 2 shows that students who did not repeat a grade in high school were 1.19 times as likely to be experimental, intermittent, or regular/established smokers than never smokers; 0.35 times as likely to be intermittent or regular/established smokers than never or experimental smokers; and 0.09 times as likely to be regular/established smokers than never, experimental, or intermittent smokers. Reexpressing these results in the probability scale using the relationship $p = \text{odds}/(1 + \text{odds})$, we find that the corresponding probabilities are .54 for being an experimental, intermittent, or regular/established smoker; .26 for being an intermittent or regular/established smoker; and .08 for being a regular/established smoker. Taking differences, we find that of these students, 46% were never smokers, 28% experimen-

Table 1
Percentage of Demographic and Key Variable Characteristics of Adolescents by Smoking Stage

Variable	Total sample (N = 20,745)		Never smoker (n = 8,894)	Experimenter (n = 5,598)	Intermittent (n = 3,458)	Reg/estab (n = 1,868)	Ex-smoker (n = 517)
	n	%					
Race							
White	10,455	50.4	36.9	24.8	19.8	13.6	3.1
Black	4,669	22.6	55.3	30.8	11.0	1.7	1.0
Hispanic	3,525	17.0	43.7	30.1	15.9	5.6	2.3
Asian	1,467	7.1	51.3	25.8	13.4	5.5	1.8
Other	608	2.9	34.9	26.5	19.1	14.6	3.5
Gender							
Male	10,263	49.5	42.8	26.5	16.9	9.2	2.3
Female	10,480	50.5	43.0	27.5	16.4	8.8	2.7
Age							
1st quartile	4,986	24.1	55.7	25.5	13.0	2.9	1.3
2nd quartile	5,135	24.8	42.0	27.6	18.1	8.0	2.5
3rd quartile	5,398	26.0	37.6	28.6	17.9	11.0	3.2
4th quartile	5,209	25.1	37.0	26.2	17.6	13.8	3.0
Grade							
7	2,716	13.1	59.5	24.2	11.5	2.1	0.9
8	2,718	13.1	48.8	26.7	16.0	4.5	2.1
9	3,620	17.4	41.6	27.0	17.6	9.4	2.6
10	3,967	19.1	39.3	28.9	18.0	8.9	3.0
11	3,809	18.4	37.9	27.4	18.0	12.0	2.7
12	3,356	16.2	37.4	28.0	16.9	12.9	3.1
Repeated grade							
No	17,864	88.5	43.7	27.5	16.5	8.2	2.3
Yes	2,316	11.5	38.4	25.1	17.2	12.6	3.6
Poverty level							
<1.5× poverty threshold	4,680	22.6	43.0	28.3	15.5	8.6	2.1
1.5 to <2.5 poverty threshold	3,469	16.7	41.7	27.3	15.5	10.6	2.5
2.5 to <4 poverty threshold	4,023	19.4	42.3	27.1	17.7	8.8	2.8
≥4× poverty threshold	3,166	15.3	43.9	24.7	18.5	8.5	3.0
Depression							
1st quartile	5,153	25.0	54.5	25.2	11.0	6.0	2.0
2nd quartile	5,151	25.0	44.6	28.2	15.8	7.7	2.3
3rd quartile	5,149	25.0	40.9	27.8	17.7	9.3	2.5
4th quartile	5,153	25.0	31.8	27.1	22.5	13.3	3.2
Delinquency							
1st quartile	5,405	27.0	67.8	21.0	6.2	2.9	1.4
2nd quartile	4,922	24.6	48.1	29.5	13.3	6.2	1.8
3rd quartile	4,671	23.3	33.9	30.1	20.8	10.9	3.0
4th quartile	5,024	25.1	22.0	29.6	27.4	15.4	4.0
Alcohol use							
Never drank alcohol	8,930	43.0	69.0	21.4	6.1	1.8	0.8
No drinks in past 12 mos.	1,921	9.3	40.4	38.7	10.6	4.9	3.8
1 or 2 days/past 12 mos.	3,506	16.9	29.0	37.7	19.9	9.0	3.5
1 day/month or less	2,494	12.0	17.9	31.8	29.7	15.1	4.2
>2 days/month	3,646	17.6	12.7	22.2	34.5	24.9	4.0
Maternal smoking							
No	10,672	53.4	49.9	26.2	14.2	5.4	2.4
Yes	9,673	46.6	35.4	27.9	19.3	12.7	2.6
Paternal smoking							
No	7,379	45.7	50.1	25.6	14.5	5.8	2.3
Yes	11,256	54.3	38.8	27.7	18.2	10.6	2.7
Family connectedness							
1st quartile	5,078	24.7	30.9	27.2	22.0	14.4	3.3
2nd quartile	5,611	27.3	37.5	29.4	18.8	9.8	2.9
3rd quartile	5,626	27.4	48.3	27.6	14.1	6.6	2.2
4th quartile	4,230	20.6	57.8	23.3	11.3	4.6	1.3
Peer smoking							
No peers who smoke	11,144	53.7	57.8	30.8	7.9	1.1	1.8
1 peer who smokes	4,230	20.4	35.0	29.8	24.1	6.2	3.6
2 peers who smoke	2,405	11.6	20.9	21.9	32.0	19.4	4.2
3+ peers who smoke	2,525	12.2	13.2	12.2	30.8	39.7	2.5
School connectedness							
1st quartile	5,025	25.0	30.7	26.7	21.2	15.8	3.3
2nd quartile	5,024	25.0	41.1	27.8	17.9	8.9	2.7
3rd quartile	5,025	25.0	48.3	28.3	14.9	5.2	2.1
4th quartile	5,025	25.0	53.5	26.3	12.6	4.7	1.7

Note. Percentages may not add up to 100% because of missing data as well as rounding errors. Quartiles of quasi-continuous variables may not contain exactly 25% of the sample because of "clumping." Reg/estab = regular/established smokers; mos. = months.

Table 2
Cumulative Odds Ratios: Fixed Effects Univariate Regression Models

Covariate	Contrast		
	2, 3, 4 vs. 1	3, 4 vs. 1, 2	4 vs. 1, 2, 3
Race			
White[a]	1.58	0.54	0.17
Black	0.50	0.28	0.11
Hispanic	0.75	0.54	0.37
Asian	0.55	0.45	0.36
Other	1.09	1.02	1.07
Gender			
Male[a]	1.23	0.38	0.11
Female	1.00	0.94	0.92
Grade			
7[a]	0.64	0.16	0.02
8	1.51	1.70	2.46
9	2.03	2.46	5.45
10	2.22	2.47	5.16
11	2.37	2.87	7.20
12	2.41	2.85	7.84
Repeat grade			
No[a]	1.19	0.35	0.09
Yes	1.20	1.34	1.73
Poverty level			
$<1.5\times$[a] poverty threshold	1.22	0.34	0.10
1.5 to <2.5 poverty threshold	1.05	1.11	1.25
2.5 to <4 poverty threshold	1.04	1.12	1.01
$\geq 4\times$ poverty threshold	0.97	1.16	0.98
Depression			
1st quartile[a]	0.78	0.21	0.07
2nd quartile	1.49	1.54	1.24
3rd quartile	1.72	1.87	1.54
4th quartile	2.54	2.89	2.33
Delinquency			
1st quartile[a]	0.44	0.10	0.03
2nd quartile	2.32	2.51	2.27
3rd quartile	4.14	4.95	4.28
4th quartile	7.48	8.29	6.50
Alcohol use			
Never drank alcohol[a]	0.42	0.09	0.02
No drinks in past 12 mos.	3.19	2.18	2.73
1 or 2 days/past 12 mos.	5.47	4.81	5.20
1 day/month or less	10.19	10.02	9.51
>2 days/month	15.30	18.91	17.94
Maternal smoking			
No[a]	0.92	0.26	0.06
Yes	1.84	1.94	2.56
Paternal smoking			
No[a]	0.92	0.27	0.06
Yes	1.58	1.60	2.09
Family connectedness			
1st quartile[a]	2.06	0.63	0.18
2nd quartile	0.75	0.68	0.64
3rd quartile	0.49	0.43	0.41
4th quartile	0.33	0.31	0.28
Peer smoking			
No peers who smoke[a]	0.69	0.10	0.01
1 peer who smokes	2.49	4.68	6.97
2 peers who smoke	5.08	12.01	25.94
3+ peers who smoke	9.08	27.76	70.64
School connectedness			
1st quartile[a]	2.07	0.64	0.20
2nd quartile	0.64	0.61	0.51
3rd quartile	0.48	0.41	0.28
4th quartile	0.39	0.34	0.25

Note. 1 = never smokers; 2 = experimental smokers; 3 = intermittent smokers; 4 = regular/established smokers; mos. = months.
[a] Baseline cumulative odds.

tal, 18% intermittent, and 8% regular/established smokers. Up to rounding error, these proportions are exactly equal to those reported in the corresponding row of Table 1 after rescaling them to sum to unity so as to account for the exclusion of the never smokers. For students that did repeat a grade, the odds of being experimental, intermittent, or regular/established smokers rather than never smokers were 1.43 (i.e., 20% higher than for those that did not repeat a grade). Similarly, their odds of being intermittent or regular/established smokers rather than never or experimental smokers were 0.47 (i.e., 34% higher than for those that did not repeat a grade). Their odds of being regular/established smokers rather than never, experimental, or intermittent smokers were 0.16 (i.e., 73% higher than for those that did not repeat a grade). Converting these odds to the probability scale as described above, we find that they agree with the entries of Table 1, after adjusting for the exclusion of the never smokers (i.e., 41% of these students were never smokers, 27% experimental, 19% intermittent, and 13% regular/established smokers), indicating that repeating a grade is positively associated with increased smoking behavior.

Regression Analyses

As shown in Table 3, using the intercept row as a common baseline for all variables, four ordinal categorical predictors (grade, alcohol use, number of peers smoking daily, school connectedness) were found to violate the proportional odds assumption at the 5% level of significance and remained in the final model coded as nominal predictors using first-to-zero contrasts. Further, there were no significant differences between the two lowest levels of school connectedness or between its two highest levels, so this variable was turned into a binary predictor using median splits. No evidence of nonproportionality was present in the data for three

Table 3
Cumulative Odds Ratios: Mixed Effects Multivariate Regression Model

	Contrast					
	2, 3, 4 vs. 1		3, 4 vs. 1, 2		4 vs. 1, 2, 3	
Covariate	OR	CI	OR	CI	OR	CI
Intercept[a]	0.27	0.23–0.32	0.031	0.024–0.042	0.002	0.001–0.003
Race						
White	1.00		1.00		1.00	
Black	0.66	0.58–0.75	0.46	0.37–0.56	0.20	0.12–0.34
Hispanic			0.76	0.65–0.88		
Asian			0.55	0.39–0.77		
Other			0.95	0.70–1.29		
Gender						
Male	1.00		1.00		1.00	
Female			0.89	0.77–1.04		
Grade						
7	1.00		1.00		1.00	
8	1.13	0.95–1.34	1.27	1.03–1.55	1.89	1.02–3.48
9	1.21	1.00–1.47	1.26	1.02–1.55	2.96	1.82–4.83
10	1.26	1.03–1.54	1.15	0.88–1.50	3.06	1.79–5.21
11	1.14	0.91–1.42	1.18	0.93–1.50	3.90	2.37–6.44
12	1.07	0.87–1.31	1.10	0.88–1.38	4.68	2.82–7.76
Depression (std.)			1.14	1.07–1.22		
Delinquency (std.)			1.15	1.12–1.17		
Alcohol use						
Never drank alcohol	1.00		1.00		1.00	
No drinks in past 12 mos.	2.97	2.47–3.57	1.87	1.47–2.38	1.80	1.09–2.98
1 or 2 days/past 12 mos.	4.27	3.68–4.95	3.49	2.90–4.18	2.43	1.78–3.30
1 day/month or less	6.39	5.39–7.56	5.84	4.79–7.13	3.30	2.33–4.68
>2 days/month	8.87	7.44–10.56	9.27	7.75–11.08	4.34	3.14–6.00
Maternal smoking			1.04	0.92–1.18		
Maternal Smoking × Female Gender			1.36	1.11–1.65		
Paternal smoking			1.26	1.14–1.39		
Family connectedness (std.)			0.91	0.86–0.98		
Peer smoking						
No peers who smoke	1.00		1.00		1.00	
1 peer who smokes	1.69	1.48–1.93	3.16	2.70–3.70	3.54	2.20–5.71
2 peers who smoke	2.65	2.15–3.27	6.41	5.31–7.74	9.76	6.40–14.89
3+ peers who smoke	3.64	2.91–4.54	12.70	10.36–15.58	23.92	17.19–33.27
Low school connectedness	1.16	1.03–1.30	1.17	1.02–1.35	1.59	1.29–1.95

Note. 1 = never smokers; 2 = experimental smokers; 3 = intermittent smokers; 4 = regular/established smokers; OR = odds ratio; CI = 95% confidence interval; std. = standard; mos. = months.
[a] Baseline cumulative odds.

other ordinal predictors (depression, delinquency, and family connectedness), which were recoded into their continuous form and tested for linearity, a hypothesis that could not be rejected for any one of them. For ethnicity, the proportional-odds assumption could be rejected for African Americans ($p < .05$) but not the other four racial groups (White, Hispanic, Asian, other), meaning African American ethnicity differentially affected the three smoking contrasts investigated. Proportional odds also seemed to hold for parental smoking. Finally, repetition of a school grade ($p = .72$) and poverty ($p = .49$) had no additional explanatory power in the presence of the remaining predictors and were dropped from the final model. Age was strongly correlated with grade and was dropped from the model because of collinearity concerns. It is interesting that gender was not significant when originally entered as a main effect alone ($p = .48$), but it became so once interactions were added with maternal smoking.

Therefore, all exponentiated regression coefficients presented in Table 3 can be interpreted as odds ratios relative to a *predominantly low-risk* baseline group of White seventh-grade students who never drank alcohol, had no parents or peers who smoked, were highly connected at school, and had median values of depression, delinquency, and family connectedness: a student profile corresponding to setting all final model covariates to zero. These are conditional and not marginal odds ratios that should only be interpreted within the context of schools with the same overall level of cigarette use. However, the intraclass correlation coefficient (ICC = .011, 95% confidence interval [CI] = 0.004–0.020) is small enough to suggest that little will be lost by ascribing them a population-averaged interpretation.[1]

The cutpoints of the standard logistic density that distinguished the four stages of smoking progression were estimated at 1.31 (95% CI = 1.13–1.49) for never smokers in our baseline group, 3.46 (95% CI = 3.18–3.74) for experimental smokers, and 6.45 (95% CI = 5.85–7.06) for intermittent smokers. Assuming that these students attend schools with an average overall level of cigarette use, one can calculate the odds that they will be in any particular stage of smoking. These can then be used to obtain point estimates and 95% confidence intervals for the probabilities of being in each stage of smoking progression: 78.7% for never smokers (95% CI = 75.7%–81.7%), 18.2% for experimental smokers (95% CI = 15.4%–21.1%), 2.9% for intermittent smokers (95% CI = 2.1%–3.7%), and just 0.2% for regular/established smokers (95% CI = 0.1%–0.3%). Almost four fifths of this low-risk group appear to have been never smokers, with experimental smokers predominating among the remaining fifth. Profiles of students showing higher degrees of cigarette use can be guessed at by examining each of the significant covariates in turn, while keeping in mind that odds ratios less than 1 are protective because they make higher levels of use less likely. Specific results for significant variables follow.

1. Ethnicity: All sizable minority groups had odds ratios significantly different from those of White students. As seen in Table 3, being African American was far more protective in lowering the odds of a transition to regular/established smoking (80% decrease) than to either smoking initiation (34% decrease) or experimentation (54% decrease). Being Hispanic lowered the odds of higher stage transitions by 25% across the board, whereas being Asian reduced them by almost 50%.

2. Grade: The relationship between the outcome and school grade of the student was quadratic in the log-odds scale and both statistically and clinically more significant for the third cutpoint than for the first two. That is, students in higher grades were more likely to transition to regular/established smoking. Whereas both experimental and intermittent smoking seemed to increase initially with advancing grade and then to tail off, regular/established smoking showed a strong positive association with grade, with Grade 12 students more than 4 times as likely to be in this category than corresponding Grade 7 students.

3. Alcohol use: Alcohol use was strongly predictive of teenage smoking, but it seemed to have a larger impact on the transition from never smokers to experimental or intermittent smokers than on regular/established smokers. Smoking initiation was more than 9 times as likely among students drinking at least twice a month than it was among abstinent students and regular/established smoking was more than four times as likely among students drinking at least twice a month than it was among abstinent students.

4. Gender and parental smoking: Paternal smoking increased the odds of an adolescent being in a higher rather than a lower smoking stage by 26% across each transition point for both genders. Maternal smoking had no effect on male offspring but raised the odds ratio for female offspring by 36% throughout. As a result, girls with mothers who did not smoke were 11% less likely to initiate smoking than their male counterparts (95% CI = 0.77–1.04) but became 26% more likely to do so if their own mothers smoked (95% CI = 1.10–1.44).

5. Peer smoking: By far the strongest predictor of smoking progression was peer smoking. Although it seemed to have less of an effect than alcohol use on transition to smoking experimentation, students having at least two friends who smoke were more than 6 times as likely to transition from experimental to intermittent smoking and almost 10 times as likely to transition to regular/established smokers. For the 12% of our total sample with at least three peers who smoke, the odds of regular/established smoking were 24 times higher than for the 54% of students with no peers who smoke.

6. School connectedness: Low school connectedness had a weak, but statistically significant, effect on raising the odds of smoking initiation and experimentation, but it had a stronger effect on accelerating the transition to regular/established smoking, with odds 59% higher for students with low as compared with high connectedness.

7. Family connectedness: Strong family ties were mildly protective in the sense that students at the third quartile of connectedness were 9% less likely to make any particular transition to a higher smoking stage than were those at the median.

8. Depression and delinquency: Both of these predictors emerged as statistically significant risk factors, with the odds of any particular transition to a higher smoking stage about 14% higher for those at the third quartile of the predictors compared with those at the median.

One systematic aspect of Table 3 is that all adjusted odds ratios have been uniformly attenuated toward 1 (i.e., toward independence, relative to the crude odds ratios appearing in Table 2).

[1] Interested readers may obtain additional information from Elizabeth E. Lloyd-Richardson on statistical methods used.

Therefore, taking into account the other variables has weakened the univariate relationships between smoking contrasts and each of the variables of interest. Another noteworthy aspect of the multivariate analysis is that the odds ratios that remain furthest away from 1 after adjusting for the other covariates in the model (i.e., those representing the strongest covariate effects) all seem to be systematically associated with the transition to regular/established smoking. Multiplying the respective odds ratios, we find that Grade 12 White males who drink regularly, report no parental smoking, but have at least three peers who smoke have odds almost 500 times ($4.68 \times 4.34 \times 23.92 = 486$) higher of becoming a regular/established smoker than do those in the baseline group, which has been deliberately chosen to have the lowest risk profile among White students, given the model predictions. This figure can be further interpreted by translating it into the probability scale: Changing the odds in favor of regular/established smoking from 0.002 to 0.972 (0.002×486) increases its prevalence from 0.2% (0.002/1.002) to 49% (0.972/1.972) as we move from the baseline group to this new student profile. Although this represents a very large absolute increase in prevalence, it still leaves the odds of regular/established smoking among this extremely high-risk group at slightly less than even, a far cry from the staggering odds one might have expected from the odds ratios alone.[2]

Discussion

In general, the findings are consistent with an interactional approach to drug use and abuse, with variables from intraindividual, peer, and family domains predictive of adolescent smoking behavior. Using advanced statistical methods to model the multivariate influence of explanatory variables representing each of these three domains and to examine whether influences were stronger or weaker at particular contrast points between stages, we found support for a stage conceptualization of smoking, in that more variables appear to strongly differentiate later smoking stages, or the regular/established smokers, from the never, experimental, and intermittent smokers. These variables included grade and peer daily smoking, ethnicity (particularly African Americans), and school connectedness. Alcohol use was more influential at early smoking stages, differentiating never smokers from experimental, intermittent, and regular smoking. Hispanic or Asian minority status and family connectedness were found to be protective across all three smoking cutpoints, whereas paternal smoking, elevated depressive symptoms, or delinquency increased risk across all smoking stages. The only notable gender difference was related to maternal smoking, which exerted a greater influence on daughters across all smoking cutpoints.

We hypothesized that peer variables would have more of an impact on earlier stages of initiation or experimentation, whereas intrapersonal and family variables would be more important at later stages of use. Although previous research has indicated peer smoking is an important predictor of smoking initiation (Killen et al., 1997; Robinson et al., 1997) and continued use (Stanton, Lowe & Silva, 1995), our results suggest that peer influence, including both friend daily smoking and low connectedness to school, is particularly salient to later smoking transitions and, to a lesser extent, to distinguishing experimental from intermittent, more regular smokers. There is some debate over whether this influence may be inflated because of adolescents' selection of a drug-using peer group and projection of drug use onto peer group as a result of their own drug-using habits (Bauman & Ennett, 1994a). Curran, Stice, and Chassin (1997) have proposed bidirectional influences of peer socialization and peer selection. Both of these mechanisms were supported in their longitudinal study of adolescent alcohol use: Peer alcohol use predicted subsequent adolescent use, and adolescent use predicted increases in peer use. A similar relationship may be operating in this study, with adolescents influenced to smoke by their friends who smoke and at the same time increasingly identifying and affiliating with peers who smoke. Moreover, lowered school connectedness may be a salient risk factor for escalation to regular smoking, which is consistent with research that shows commitment to school to be protective against escalation to regular marijuana use (Kandel & Davies, 1992). Lack of connectedness to school may represent a precursor for increased tolerance for deviance, alienation from all but a circumscribed group of peers, and subsequent substance use (Brook et al., 1992).

Alternatively, the assessment of peer smoking in this study was based on how many friends smoke cigarettes daily, reflecting the high end of the spectrum of peer influence. This lends itself to underestimating how experimental use of cigarettes may influence susceptibility to smoking in youth. Further research may indicate that having peers in earlier, more experimental smoking stages may be more influential at early stages of smoking, as found in Flay et al. (1998). Moreover, measurement of peer influence is limited by the adolescents' self-report of their peers' behavior. Adolescent smokers, in particular experimental and regular smokers, tend to overestimate the prevalence of smoking among peers and erroneously inflate the correlation between self and friends' behavior (Urberg, Shyu, & Liang, 1990). Regardless, what remains clear is the significant influence of peers on adolescent cigarette use across all stages.

We proposed that sociodemographics, depression, delinquency, and alcohol use would have a stronger influence on later smoking stages. Consistent with previous literature, ethnicity was found to be an important determinant of whether adolescents had ever tried a cigarette (CDC, 2000). Although minority adolescents are less likely to smoke than their White counterparts, among adults, African Americans have higher smoking prevalence rates than do Whites (CDC, 1994). One possible explanation for the resiliency seen in minority adolescents may involve the strong family ties characteristic of many ethnic groups. Ethnic groups have traditionally relied on their immediate and extended families for support (Aponte & Barnes, 1995) and are more likely to report a strong, connected family environment (Alexander, Allen, Crawford, & McCormick, 1999). These strong family ties may serve to decrease adolescent susceptibility to peer pressure and peer influences.

[2] This illustrates why we have chosen to also emphasize the baseline odds estimates in our presentation: An examination of the adjusted odds ratios allows one to gauge in isolation the potential influence of each of the predictors of interest, but too heavy a reliance on them might exaggerate the practical significance of the independent variables for outcomes that are unlikely to begin with. Rather, the interested reader ought to interpret them in context by combining them with the baseline odds to produce prevalence estimates for risk profiles of particular interest and evaluate whether their potential has been realized by assessing changes in absolute rather than relative risk.

Alternatively, given this family structure, strong antitobacco messages within the family may be attended to more by ethnic minorities, with African American, Hispanic, and Asian American teens more likely than White teens to report strong antismoking messages from family members (Mermelstein & The Tobacco Research Network, 1999). Further consideration should also be given to the possibility of African American–White differences in self-report of tobacco use, as Bauman & Ennett (1994b) suggest that African Americans may underreport their smoking behaviors.

Grade was strongly associated with regular/established smoking. This is consistent with recent research that found that those who had experimented with smoking by Grade 7 were at greater risk for academic difficulties and a wide range of behavioral problems, including substance use, and were more likely to continue to exhibit these behavior problems 5 years later (Ellickson et al., 2001). This suggests early substance use may be indicative of both coexisting behavioral problems and predictive of continued engagement in these behaviors. As Ellickson et al. (2001) suggest, prevention efforts aimed at youth who have already begun to smoke may need to widen their net of intervention, addressing multiple problem behaviors that often become evident prior to Grade 7. The relative importance of school versus family at various periods of development may be particularly influential on adolescent behaviors and should be further explored.

Both depression and delinquency were significant risk factors for smoking across all stages, although contrary to hypothesis, they failed to exert a stronger influence at higher smoking stages. It is unclear what influences are responsible for this pattern, and it may be due to an unexplored interaction with family or peer influences. Depressive symptoms, particularly when combined with peer smoking, have been found to predict smoking initiation and experimentation in adolescents (Patton et al., 1996) and progression to daily smoking in young adults (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998). Smoking has also been found to increase adolescent risk of developing depression, even after controlling for other psychiatric disorders (Brown et al., 1996; Choi, Patten, Gillin, Kaplan, & Pierce, 1997). Although adolescents may self-medicate their affective disorders with nicotine, the role of parents and parenting styles, peer relations, and self-esteem may moderate the relationship between affect and smoking (M. W. Reynolds & Frank, 2000). Furthermore, the relationship between depression and cigarette smoking has been found to vary by ethnicity and socioeconomic status, with depression and cigarette use positively associated among White suburban high school students, but not among inner-city minority youth (Way, Stauber, Nakkula, & London, 1994). Given discrepancies in prevalence rates for depression and delinquency across males and females (American Psychiatric Association, 1994), one may expect to identify gender differences. However, consistent with previous research (Brown et al., 1996; Barber, Bolitho, & Bertrand, 1999), we did not find any significant interactions between gender and either depression or delinquency.

Contrary to hypothesis, alcohol use more strongly influenced early smoking stages than later stages. Research investigating adolescent risk-taking behaviors has found that alcohol use and smoking do not occur independently, nor do they occur in a random fashion (Igra & Irwin, 1996). It remains unclear whether the observed relationship between alcohol use and smoking experimentation is a result of the physiological affects of alcohol, peer modeling of alcohol or drug use, or perhaps an overarching tolerance for deviance that condones underage drinking and substance use. Nevertheless, this relationship provides insight into potential school- and individual-level intervention strategies worth exploration, such as comprehensive programs that address the use of multiple substances and are taught over an extended period of time across several developmental levels.

Although we hypothesized that family variables, namely parental smoking and family connectedness, would differentiate later smoking stages, both of these appear to significantly impact teen smoking across each smoking cutpoint, with no significant differences across smoking stages. We noted a gender difference with maternal smoking, which significantly affected girls' smoking only. These findings are consistent with research suggesting that girls may be more susceptible to the social influences of peers and family, namely parental smoking and family conflict (Flay et al., 1998). Similarly, strong family ties were mildly protective of smoking across any stage comparison. This is consistent with the theory of social control (Hischi, 1969), which asserts that weak attachment to parents is a predictor of smoking initiation and other problem behaviors in adolescence. Parent–adolescent communication related to tobacco use rules, consequences, and circumstances has been found to predict smoking escalation, but not initiation, and to vary on the basis of parents' tobacco use (Ennett et al., 2001). It is possible that stronger associations may have been found if we had assessed family relations relative to several specific family members and taken into account the context of family influences given differences in family structure.

We realize that the variables investigated in this study are pieces of the very large and complicated puzzle of adolescent smoking. Results provide support for stage conceptualizations of smoking, which provide structure for investigations of individual- and environmental-level variables that may distinguish various levels of cigarette use and also allow for better identification of which individuals may progress to higher stages of use and dependence. The relative contributions of risk and protective factors to smoking stages reported here are interesting in light of current conceptualizations of the development of adolescent risk behaviors. Igra and Irwin (1996) proposed that risk behaviors fulfill different functions and meanings at different stages of adolescent development. Therefore, the Grade 7–12 sample reported here may show differing relationships as compared with a younger cohort. This further underscores the importance of understanding contextual factors in the acquisition of behaviors associated with adolescent role transitions (Brooks-Gunn & Graber, 1994).

Limitations of this article include the fact that analyses were cross-sectional and therefore prevent us from determining causality. Future research should incorporate longitudinal investigations that allow for causal discussion of variables influencing smoking stages over time, as well as investigation of various trajectories of smoking uptake and nicotine dependence. The Add Health survey did not allow for in-depth assessment of constructs. In addition, the measurement of peer smoking failed to assess this predictor at different levels of influence (e.g., peers that have tried smoking or who are intermittent smokers). With the exception of poverty level and parental smoking, variables were based on adolescent self-report.

Although we were able to accommodate the clustering aspect of the data by school, MIXOR does not handle the probability

weights that are provided for each student in the Add Health database. It is possible to fit a hierarchical ordinal-regression model with fully nonproportional odds in Stata (StataCorp., 1999), which can also handle sample survey weights. This more copious output from Stata is available from Elizabeth E. Lloyd-Richardson. It leads to identical interpretations for all predictors presented in Table 3, with several minor exceptions.[3]

Although it is likely that many of these adolescents have made unsuccessful quit attempts and experienced withdrawal symptoms as a result of these quit attempts, the Add Health data rely solely on quantity–frequency data. Therefore, they do not allow for evaluation of tolerance and withdrawal symptoms, making it impossible to classify nicotine dependence. Nonetheless, research suggests a large majority of adolescents who smoke on a daily or near daily basis are nicotine dependent (Colby et al., 2000; Stanton, 1995; U.S. DHHS, 1994). Future research is needed to refine and clarify staging algorithms for youth smoking, allowing for greater correspondence with diagnostic criteria, as well as consideration of the role of biochemical assessments in staging algorithms.

Although previous research has assumed that movement between smoking stages is influenced in a linear fashion by various explanatory factors, we were able to evaluate whether a particular variable exerts differential influence on early stages of smoking compared with later smoking stages. Using advanced statistical analyses, we compared these stages along a continuum that may reflect progressive stages in which youth are more or less susceptible to internal or external influences toward smoking initiation, experimental use, or more regular use. Stage conceptualizations of smoking have tremendous implications for prevention efforts, both in allowing for identification of which individuals may progress to higher stages of use, and likely dependence, and in developing interventions tailored to an adolescent's previous experiences with smoking.

Comprehensive interventions that involve the integration of personality and intrapersonal background, familial level of support, and impact of peer group and that take into consideration an adolescent's smoking stage should be further explored in the development of both primary and secondary prevention strategies. For instance, teens who are already experimenting with smoking, are involved in alcohol use, and have several peers who smoke regularly may be more effectively targeted with secondary prevention efforts designed to halt the entrenchment of smoking. It is clear that the role of mediating and moderating variables on stages of youth smoking should not be underestimated and deserves further investigation.

[3] The protective effect of the Hispanic ethnic group is strengthened in both its clinical and practical significance, whereas parental smoking remains a risk factor but loses its significance.

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