Published in final edited form as:
Health Psychol. 2008 November ; 27(6): 811–818. doi:10.1037/0278-6133.27.6.811.

Trajectories of Smoking from Adolescence to Early Adulthood and their Psychosocial Risk Factors

Darce M. Costello,
Wesleyan University

Lisa C. Dierker,
Wesleyan University

Bob L. Jones, and
Carnegie Mellon University

Jennifer S. Rose
Wesleyan University

Abstract

This study used semi-parametric group-based modeling to explore patterns of persistence and change in smoking behavior as well as risk factors associated with the developmental course of smoking from age 13 to 25. Drawing on data from the National Longitudinal Study of Adolescent Health, six distinct trajectories were identified: *nonsmokers, experimenters, stable light smokers, quitters, late escalators*, and *stable high smokers*. Baseline risk factors that were associated with greater likelihood of membership in all of the smoking trajectory groups compared to nonsmokers included alcohol use, deviance, peer smoking, and (with the exception of the *late escalators*) drug use. Deviance, peer smoking, alcohol and drug use also distinguished the likelihood of membership among several of the five smoking trajectory groups. The results add to basic etiologic research on developmental pathways of smoking in adolescence and young adulthood by providing evidence of heterogeneity in smoking behavior and prospectively linking different patterns of risk factors with the probability of trajectory group membership.

Keywords

smoking trajectories; smoking risk factors; adolescence; longitudinal; person-centered analysis

Introduction

Despite the well known negative health consequences of cigarette smoking, a recent study examining health risk behaviors among U.S. high school students found that more than half (58%) had tried smoking, and 22% were current smokers (Centers for Disease Control and Prevention, 2004). Further, while the prevalence of lifetime and current smoking among high school students had been declining since the late 1990s (Centers for Disease Control and Prevention, 2005), new data indicate the *rate* of decline in smoking has slowed, raising concern that these downward trends may not continue (Johnston, O'Malley, Bachman, & Schulenberg, 2006). Given that the majority (89%) of adult daily smokers first tried smoking by the time they were 18 (Lynch & Bonnie, 1994), along with evidence that smokers tend to increase their consumption of cigarettes after high school (Chassin, Presson, Pitts, & Sherman, 2000), developing effective smoking prevention and cessation programs for this population is a public health priority. This work can benefit from an understanding of (1) patterns of persistence and change (e.g., escalation or remission) in smoking behavior over time that may represent

different etiological processes (Colder et al., 2001), and (2) individual factors associated with the developmental course of smoking that are amenable to intervention (Chassin et al., 2000). The primary aim of this study was to identify distinct developmental trajectories of cigarette use spanning adolescence and early adulthood in a nationally representative sample, using empirically derived psychosocial risk factors for smoking to predict the probability of trajectory group membership.

Longitudinal study of the natural history of cigarette smoking has described a pattern of increased incidence in adolescence, followed by relative stability from the late teens onward (Chassin, Presson, Rose, & Sherman, 1996; Chen & Kandel, 1995). This line of research has contributed important information toward understanding the developmental course of smoking through its characterization of average smoking behavior over time. Increasingly, researchers have extended this framework by integrating person-centered and variable-centered approaches to longitudinal analysis in order to distinguish *multiple* developmental pathways reflecting previously unobserved individual heterogeneity in smoking behavior (Audrain-McGovern et al., 2004; Chassin et al., 2000; Colder et al., 2001; Jackson, Sher, & Schulenberg, 2005; Orlando, Tucker, Ellickson, & Klein, 2004; Stanton, Flay, Colder, & Mehta, 2004; White, Pandina, & Chen, 2002). This integrated strategy draws the person to the foreground of the analysis and illuminates the ways in which individual characteristics are organized into meaningful patterns that distinguish subgroups of people (Hart, Atkins, & Fegley, 2003; Magnusson, 1998, 2003). In addition, by linking risk factors to different smoking trajectories, a combined person- and variable-centered analysis strategy may inform the development of intervention and prevention programs that are tailored to different subgroups within the broader population (Bates, 2000).

One flexible framework for conducting such an integrated analysis is provided by a semi-parametric group-based modeling approach (Jones, Nagin, & Roeder, 2001; Nagin, 1999, 2005). This technique is a category of finite mixture models, which were developed to identify population subgroups following distinct developmental trajectories (Muthén & Muthén, 2000; Nagin, 1999). In contrast to conventional growth modeling, which “assumes that the population distribution of trajectories varies continuously across individuals” (Nagin & Tremblay, 2005, p. 84), finite mixture models permit the empirical identification of trajectory groups that may, in our analysis, reflect unique etiologies of tobacco use that diverge from the average trajectory that describes the population.

Support for the plausibility of distinct smoking trajectories comes from previous studies that have used a variety of person-centered techniques to identify several patterns of smoking behavior over time. These studies have reported evidence for three (White et al., 2002), four (Audrain-McGovern et al., 2004), five (Abroms, Simons-Morton, Haynie, & Chen, 2005; Colder et al., 2001; Guo et al., 2002), and six (Chassin et al., 2000; Orlando et al., 2004; Stanton et al., 2004) distinct smoking trajectory groups. Smoking trajectories that were common across these studies were (1) *nonsmokers* (among the studies that included nonsmokers in the analysis), (2) *light* or *experimental* smokers, and (3) *chronic* or *stable* smokers. An additional pattern described *late escalators*, whose cigarette use emerged in late adolescence and transitioned to a steady pattern of consumption in early adulthood (Chassin et al., 2000; Guo et al., 2002; Orlando et al., 2004; Stanton et al., 2004). Two of the studies that followed participants into young adulthood also reported a *quitter* trajectory (Chassin et al., 2000; Orlando et al., 2004). Other smoking trajectory groups were differentiated based on erratic patterns of cigarette use (Chassin et al., 2000), the rate at which use escalated (Audrain-McGovern et al., 2004; Colder et al., 2001), or nonsmokers’ intentions to smoke in the future (Abroms et al., 2005).

Although it is difficult to make direct comparisons across these studies due to differences in their methods (e.g., how they measured smoking, age at initial assessment and length of follow-up, whether or not they included nonsmokers in the analysis), analytic techniques (e.g., semi-parametric group-based modeling, latent growth mixture modeling, piecewise growth mixture modeling), and sample characteristics (e.g., urban versus suburban, racially-ethnically diverse versus homogeneous, and high risk versus community samples), taken together, these findings suggest that there is a fair amount of heterogeneity in smoking behavior across adolescence and young adulthood that may be represented by integrating variable- and person-centered approaches to analyzing developmental trajectories.

Along with describing multiple patterns of smoking trajectories, most of these studies have explored various smoking risk profiles by linking correlates of smoking to trajectory group membership. An array of predictors has been examined, ranging from the individual (e.g., self-efficacy, locus of control, academic achievement, sports participation, beliefs about smoking), the peer group (e.g. peers' problem behaviors), the family (e.g., family structure, parenting practices, parental smoking), and the school (e.g., school attachment) contexts. Characteristics that have differentiated among the trajectory groups that are of greatest public health concern (i.e., those that reflect stable patterns of smoking that emerge in early or later adolescence compared to patterns of experimental, light, or no smoking) include depression (Audrain-McGovern et al., 2004; Stanton et al., 2004), deviance (Orlando et al., 2004; Stanton et al., 2004), alcohol and other drug use (Audrain-McGovern et al., 2004; Orlando et al., 2004; Stanton et al., 2004), and peer smoking (Audrain-McGovern et al., 2004; Chassin et al., 2000; Orlando et al., 2004; Stanton et al., 2004).

Building upon existing research that has mapped a variety of prototypical trajectories characterizing common patterns of smoking behavior, our study aimed to extend this work in the following ways: (1) by using a nationally representative sample, we sought to discover whether previously identified smoking trajectory groups were characteristic of the general population of U.S. adolescents, (2) by following adolescents from the time they were most likely to start smoking (i.e., before age 18) into early adulthood, when patterns of regular smoking are established, we included the most informative age group for mapping the health risk behaviors that were the focus of our inquiry, and (3) by incorporating into our trajectory analysis established risk factors for smoking that may aid the development of targeted prevention and intervention programs, we evaluated whether they were associated with individuals' propensity to follow a particular smoking trajectory.

Method

Participants

The data for this analysis were drawn from the National Longitudinal Study of Adolescent Health (Add Health), a nationally representative, probability-based survey examining a broad range of health-related attitudes and behaviors of U.S. adolescents who were in grades 7 through 12 between September 1994 and April 1995 (Chantala, 2003). From a primary sampling frame of all high schools in the United States with an 11^{th} grade and at least 30 students, a systematic random sample of 80 high schools was selected proportional to enrollment size, and stratified by geographic region, urbanicity, school type (i.e., public, private, parochial), and ethnic mix. Fifty-two "feeder" schools that sent their students to those high schools without 7^{th} or 8^{th} grades were randomly selected proportional to the percent of their contribution to the high school's entering class. Up to three rounds of data are available for 10 age cohorts (born between 1974 and 1983). At Wave 1, a confidential in-school self-administered questionnaire was given to all students in grades 7 through 12 who were in school on the day of the survey. A representative sample of 12,105 students (stratified by grade and gender) was randomly selected from the more than 90,000 students who completed the in-

school survey, and these students participated in a 90-minute in-home interview between April and December 1995. A second round of interviews (Wave 2) was conducted with the Wave 1 participants one year later. The third round of interviews (Wave 3) was conducted five years later, between 2001 and 2002.

Analytic Sample

The Add Health study collected up to three rounds of data from adolescents in multiple overlapping age cohorts. This approach provided an opportunity to examine developmental trajectories spanning early adolescence to young adulthood by "linking" the cohorts together based on age in a *cohort-sequential design* (Miyazaki & Raudenbush, 2000). Our initial analytic sample consisted of the 6,504 participants from the public use dataset. We excluded 175 participants who were younger than age 13 at Wave 1 and 53 participants who were older than age 25 at Wave 3, as we were concerned they were too few in number to be representative of their respective age cohorts. Sixty four participants were missing smoking outcome measures at all assessment points and were not included in our analysis. As is true of conventional growth models, group-based trajectory models cannot accommodate cases with missing covariates, so 423 participants were set aside for this reason. We considered data imputation as an alternative to excluding these cases, however typical strategies for handling missing data (e.g., multiple imputation) may not be appropriate in this situation, as their assumption of a single population is at odds with the group-based trajectory modeling approach's hypothesis of multiple subgroups within a population (Colder et al., 2001). Therefore, we conducted our analysis with the remaining 5,789 participants (89% of the public use sample) with complete data. We used chi-square analysis and t-tests to compare participants with complete data to those with missing data, and examined effect sizes in addition to significance tests to evaluate the potential for bias. We found the participants in the analytic sample were more likely to be white (Cramer's V=.10), female (Cramer's V=.05), and from families with more highly educated parents (Cramer's V=.05). In addition, participants with complete data reported lower levels of baseline alcohol use (Cramer's V=.06), deviance (Cohen's d=.15), depression (Cohen's d=.11), smoking behavior (Cohen's d=.11), and had fewer best friends who smoked (Cramer's V=.07). Thus, although there were baseline differences between the two groups, the small effect sizes (i.e., <.2; Cohen, 1988) suggested they were minimal.

Procedure

During the in-home survey, an interviewer read the questions aloud and recorded the respondent's answers using a laptop computer-assisted personal interview (CAPI) system. Portions of the survey pertaining to potentially sensitive information (e.g., questions about suicidality and substance use) were administered using an audio computer-assisted self-interview (ACASI) that allowed the participants (rather than the Add Health interviewer) to enter their responses directly into the computer.

Measures

Background characteristics

Age: We calculated adolescents' age in years at Wave 1 by subtracting their birth date from their interview date. At Waves 2 and 3, Add Health provided a calculated age variable. However, the value of the Wave 2 age variable was incorrect for 17 adolescents, and we calculated their age following the same method used at Wave 1. We centered age by subtracting 19.25 from each observed value so the model intercept would have a meaningful value (i.e., expected average smoking at the approximate midpoint of the developmental period under study) (Singer & Willett, 2003) and to reduce collinearity among the variables in the model

(Cohen, Cohen, West, & Aiken, 2003). The sample mean ages were 15.99 (SD=7.99), 15.97 (SD=6.61) and 21.74 (SD=7.31) across the three data collection waves.

Race-ethnicity: We created an indicator of race-ethnicity based on the six non- mutually exclusive choices offered in the Wave 1 in-home survey. The order of precedence for determining the value of the race-ethnicity indicator was: Hispanic/Latino (11.73%), Black/ African American (14.94%), Native American (2.11%), Asian American (3.27%), Other (0.76%), and non-Hispanic/Latino White (67.2%). For example, if adolescents said they were Hispanic/Latino, Asian American, and Other, we coded their race-ethnicity indicator as Hispanic/Latino.

Gender: The Wave 1 measure of biological sex was coded with values of 1 for males (50.09%) and 2 for females (49.91%).

Parent education: In order to minimize the number of cases lost due to missing data, the maximum value of resident mother's or father's highest level of education at Wave 1 was used as a proxy measure of socioeconomic status. Based on the distribution of the original variables, parent education was recoded to reflect the following categories: (1) less than high school or not known (13.23%), (2) high school, GED, business, trade, or vocational school (32.22%), (3) some college, business, trade, or vocational secondary school (20.16%), (4) college or university graduate (22.62%), and (5) professional training beyond a 4-year college or university (11.77%).

Risk factors

Depression: Depression symptoms were measured at Wave 1 with 20 items corresponding to the Center for Epidemiological Studies-Depression Scale (CES-D) (Radloff, 1977). Using a 4-point scale (where 0 = "never or rarely" and 3 = "most of the time or all of the time"), adolescents indicated how often they had experienced various emotional states (e.g., "felt depressed," "felt happy") during the past week. Responses were summed to create a composite score (α=.87) for each adolescent that ranged from 0 to 56, with a mean of 12.08 (SD=12.11).

Deviance: Involvement in deviant behavior was assessed at Wave 1 with 11 questions asking the adolescents whether they had engaged in a variety of delinquent acts (e.g., painted graffiti, deliberately damaged other's property, lied to their parents, stole from a store, ran away from home, drove a car without the owner's permission). We computed a mean score (α=.78) for each respondent (Resnick et al., 1997) that ranged from 0 to 10.91, with a mean of 0.40 (SD=1.58).

Alcohol use: At Wave 1, adolescents indicated how frequently they drank alcohol in the past 12 months, selecting from choices that ranged from "every day or almost every day" (1) to "never" (7). We recoded this variable so that higher values represented more frequent alcohol use. Over half of adolescents (52.64%) reported no alcohol use in the past 12 months.

Other drug use: We used adolescents' responses to Wave 1 measures that indicated whether they had ever used marijuana, cocaine, inhalants or other drugs to create a dichotomous measure of any drug use (0=no, 1=yes). Most (70.36%) of the adolescents reported no drug use.

Peer smoking: Adolescents indicated at Wave 1 how many of their three best friends smoked at least one cigarette per day. More than half (54.56%) of the adolescents reported no smoking among their three best friends.

Outcome measure

Smoking: At each time point, participants reported their quantity (number of cigarettes) and frequency (number of days) of smoking in the past 30 days. Because the distributions of both items were skewed, with most participants reporting no smoking, we categorized each variable as follows: (1) quantity: 0, 1–5, 6–10, 11–20, 20+; (2) frequency: 0, 1–5, 6–10, 11–15, 16–20, 21–25, 26–29, 30. Then, we multiplied the categorical quantity and frequency variables to obtain a single outcome measure that ranged from 0 to 28. This approach permitted us to succinctly capture diverse patterns of smoking behavior (e.g., someone who smoked one cigarette per day for 30 days versus someone who smoked 30 cigarettes on one day and did not smoke again that month) that resulted in similar levels of exposure to nicotine and tobacco smoke. The mean values at each assessment were 2.17 (SD=11.73), 2.68 (SD=12.64), and 4.65 (SD=14.45).

Analytic Strategy

We used a semi-parametric group-based method (Jones et al., 2001; Nagin, 1999, 2005) to empirically identify smoking trajectories from age 13 to 25. We estimated zero-inflated Poisson (ZIP) trajectory models with a user-written SAS procedure, PROC TRAJ (Jones et al., 2001). We included sample weights to account for the Add Health study's design effects and to obtain parameter estimates that reflect accurately the population of interest.

We relied on several recommended criteria to assess model fit (Nagin, 1999, 2005). The Bayesian Information Criteria (BIC) is a standard method for deciding whether additional classes or predictors of trajectory group membership result in a better fitting model. Although definitive thresholds have yet to be established, the model with the larger BIC value is generally favored. The posterior probabilities of group membership "collectively measure a specific individual's likelihood of belonging to each of the model's *J* trajectory groups" (Nagin, 2005, p. 78), and provide another measure of how well the estimated model fits the observed data. Ideally, individuals' posterior probabilities equal 1 for their most likely trajectory group and 0 for the remaining trajectory groups, however a minimum average posterior probability of .70 for all trajectory groups is considered evidence of acceptable model fit (Nagin, 2005). Finally, we were guided by previous research in deciding whether the selected model was interpretable from a substantive and theoretical standpoint.

Results

Previous research led us to expect that we would find between three and six smoking trajectory groups in this sample. We began by specifying a three-group model and systematically increased the number of groups to eight. The six-group model (Table 1) had the largest BIC (−22383.13), suggesting it represented a better fit to the data than the other models. Adding to the six-group model demographic characteristics (i.e., gender, race, and parent's education) and risk factors (i.e., depression, deviance, peer smoking, alcohol, and any drug use) further improved the model fit (BIC=−18506.05). We tested for possible interactions suggested by previous studies (e.g., to examine whether associations between smoking and the risk factors varied as a function of gender or race), however adding these parameters did not improve the model fit.

We considered an average posterior probability of .70 for all groups as a minimum indicator of adequate model fit (Nagin, 2005). This threshold was met by each of the estimated smoking trajectory groups, with values ranging from .76 to .90, and suggested that the six-group model did a reasonable job of correctly classifying individuals with similar smoking profiles.

Finally, we evaluated the six-group model in terms of its substantive interpretability. The fitted model described distinct smoking trajectories that were in line with those identified by previous research and that reflected theoretically meaningful patterns of smoking behavior. Based on these collective criteria, we selected the six-group model (with covariates) as our final model. The predicted trajectories are depicted in Figure 1. The *nonsmokers* (an estimated 47.9% of the population, average posterior probability of membership = .85) represented the largest group in the sample and followed a constant (intercept-only) trajectory. The *experimenters* (an estimated 21.5% of the population, average posterior probability of membership = .76) showed a slight linear increase in smoking quantity-frequency over time. The *stable light smokers* (an estimated 7.3% of the population, average posterior probability of membership = .81) were characterized by a low level of smoking throughout the study period that followed a negative quadratic trajectory. The *quitters* (an estimated 6.3% of the population, average posterior probability of membership = .82) followed a negative quadratic trajectory, with increasing levels of smoking to age 19, followed by a decline to age 25. The *late escalators* (an estimated 9.6% of the population, average posterior probability of membership = .88) displayed smoking levels that were similar to *experimenters* until age 16, that then shifted to a pattern of increasing consumption into early adulthood that followed a positive quartic trajectory. Finally, the *stable high smokers* (an estimated 7.4% of the population, average posterior probability of membership = .90) showed a relatively steady pattern of cigarette use from adolescence into early adulthood that reflected negative quartic change.

Predictors of trajectory group membership

The six-group model with covariates allowed us to examine the associations between the probability of trajectory group membership and the selected risk factors while simultaneously estimating the parameters that defined the trajectory groups themselves. We used measures from Wave 1 to establish a temporal order between the predictors and the smoking trajectories that formed the outcome (Nagin, 2005). We fitted a series of multinomial logistic regression models in which we varied the reference group. The estimated odds ratios and 95% confidence intervals are presented in Table 2 and describe the odds of trajectory group membership relative to the reference group for each baseline risk factor (controlling for the other variables in the model).

Compared to *nonsmokers*, baseline risk factors that were associated with greater likelihood of membership in all of the smoking trajectory groups included alcohol use, deviance, peer smoking, and (with the exception of the *late escalators*) drug use. Compared to *experimenters*, adolescents reporting higher levels of drug use and peer smoking were more likely to be classified as *quitters*, *stable high*, or *stable light smokers*, whereas alcohol use was associated with a lower probability of being classified as a *quitter* or *late escalator*. In addition, adolescents reporting higher levels of deviance were more likely to be classified as *stable high smokers* than *experimenters*. Compared to *stable light smokers*, adolescents reporting higher levels of deviance and peer smoking were more likely to be classified as *stable high smokers* whereas peer smoking and drug use were associated with a lower probability of being classified as a *late escalator*. Compared to *quitters*, adolescents reporting higher levels of peer smoking were more likely to be classified as *stable high smokers* whereas peer smoking and drug use were associated with a lower probability of being classified as a *late escalator*. Finally, compared to *late escalators*, adolescents reporting higher levels of alcohol use, drug use, or peer smoking were more likely to be classified as *stable high smokers*.

Discussion

The goals of our analysis were to identify distinct developmental trajectories of cigarette use spanning adolescence and early adulthood, and to extend previous research by examining key

risk factors associated with the propensity to follow particular smoking trajectories in a representative sample of U.S. adolescents. Echoing previous studies, we observed evidence of heterogeneity in longitudinal patterns of smoking that, in our analysis, was best described by six smoking trajectory groups. Along with the *nonsmoker*, *stable light* or *experimenter*, and *stable high* smoking trajectories reported by other studies (e.g., Abroms et al., 2005; Audrain-McGovern et al., 2004; Chassin et al., 2000; Colder et al., 2001; Orlando et al., 2004), we also noted trajectories of *quitters* and *late escalators*. Overall, our findings were most similar to those detailed by Chassin and colleagues (2000), except that we empirically identified a *stable light* rather than an a priori *erratic* smoking trajectory group.

Integrating person- and variable-centered approaches in our analysis enabled us to examine how smoking risk factors were organized in meaningful patterns that distinguished subgroups of smokers. By incorporating risk factors into the trajectory model, we were able to test hypotheses relating these risk factors to the probability of trajectory group membership (Nagin, 2005). In line with findings reported by Orlando and colleagues (2004), risk factors that were associated with higher odds of membership in all of the smoking trajectory groups compared to *nonsmokers* included alcohol use, deviance, peer smoking, and (with the exception of the *late escalators*) drug use. Further, although they did not control for the effects of deviant behavior, Audrain-McGovern et al. (2004) also noted peer smoking, alcohol, and drug use were positively linked to membership in smoking versus nonsmoking trajectories.

Along with differentiating *nonsmokers* from the other smoking trajectories, deviance, peer smoking, drug and alcohol use distinguished the likelihood of membership in several of the smoking trajectory groups. Consistent with previous research, *experimenters* and *late escalators* displayed similarly low risk profiles in terms of peer smoking, deviance (Chassin et al., 2000; Orlando et al., 2004), and drug use (Orlando et al., 2004; Stanton et al., 2004). In contrast with other studies, we found that frequency of alcohol use in the past 12 months was associated with a higher probability of membership in the *experimenters* versus the *late escalators* group. Some studies have reported no differences between these groups in terms of binge drinking (Orlando et al., 2004) or alcohol use in the past month (Stanton et al., 2004), whereas Audrain-McGovern et al. (2004) found that adolescents who reported having at least one drink in their lifetime were less likely to be in the *experimenters* group.

When compared to *quitters*, *stable light*, and *stable high smokers*, higher odds of membership in the *late escalators* group were associated with lower levels of drug use and peer smoking. In contrast, the higher risk profiles of *quitters*, *stable light*, and *stable high smokers* were similar, with the exception that *stable high smokers* were likely to have more friends who smoke than *quitters* or *stable light smokers* and to report higher levels of deviance than *stable light smokers*. Given these differences in risk profiles, it is striking that, by the end of the developmental period we examined, these six trajectories coalesced to form two distinct pathways, with the *stable high smokers* and *late escalators* exhibiting high levels of smoking quantity-frequency, and the *experimenters*, *stable light smokers*, and *quitters* displaying smoking quantity-frequency levels that appear indistinguishable. A comparable divergence has been noted in trajectory analyses that followed participants into early adulthood (Chassin et al., 2000; Orlando et al., 2004). From a public health perspective, the *late escalators* and *stable high* trajectory groups are of particular concern, given their high levels of cigarette consumption in early adulthood. Yet the risk factors associated with these two trajectories are markedly different, with *late escalators* showing a profile of lower risk in terms of peer smoking, alcohol and drug use compared to the *stable high smokers*. The dynamic nature of risk offers one potential explanation for these results. The risk factors in our model were measured at Wave 1 and, over time, members of the *late escalators* trajectory may have exhibited a pattern of increasing risk. Alternatively, it is possible that the influence of unmeasured protective factors present at baseline diminished during the transition to early adulthood (Orlando et al., 2004).

In addition to the representative nature of the sampling frame, the Add Health data set offered several advantages for addressing the questions examined in this study. The multiple age cohorts allowed us to use a longitudinal cohort-sequential design to estimate smoking trajectories encompassing the most informative developmental period for mapping the health risk behaviors that were the focus of inquiry. In addition, Add Health included assessments of empirically derived risk factors for smoking. Finally, multiple measures of cigarette use allowed us to develop a comprehensive outcome variable that encompassed smoking quantity and frequency. We note, however, that our findings may be compared directly only to those studies that used a similar outcome measure (e.g., White, Johnson, & Buyske, 2000; White et al., 2002).

The strengths of this study should be considered along with its limitations. While our results lend support to previous research that identified distinct smoking trajectories and associated risk factors, our analysis has not identified specific processes or mechanisms that might explain the different patterns we observed. Thus, an extension to our study could examine whether these risk factors set into motion a sequence of events associated with stability or change in smoking behavior over time (Compas, Hinden, & Gerhardt, 1995). For example, an adolescent's deviant behavior might elicit parent or teacher intervention, which could then lead to changes in other risk behaviors (including smoking). In addition, although we considered risk factors that have been shown to differentiate among various smoking trajectory groups, other risk factors found in the adolescent smoking literature (e.g., health beliefs, social competence, exposure and receptivity to tobacco advertising) were not available in this data set.

Further, the range of average posterior probabilities (.76 to .90) that we examined to determine how well the estimated model fitted the observed data all met the minimum threshold of .70 (Nagin, 2005), but were somewhat lower than the range of values (.79 to .99) reported in previous studies (Orlando et al., 2004; Stanton et al., 2004; White, Nagin, Replogle, & Stouthamer-Loeber, 2004). Although it is difficult to make direct comparisons to these studies due to differences in outcome measures, analytic methods, and number and type of trajectory groups identified, this difference may signal some misclassification of individuals to their most likely group by our final model.

Our results also leave unanswered questions regarding potential associations between the trajectory groups we identified and the subsequent development of nicotine dependence. The Fagerström Test for Nicotine Dependence (FTND) (Heatherton, Kozlowski, Frecker, & Fagerström, 1991) administered at Wave 3 to assess current nicotine dependence was presented only to respondents who indicated they had (a) ever smoked at least once a day for 30 days, and (b) smoked at all in the past 30 days. Because these criteria excluded nearly one third of the smoking trajectory members in our analytic sample, we were unable to explore that line of inquiry. Finally, because Add Health is a school-based study, these results may not represent the experiences of U.S. adolescents who were not attending (or were absent from) school when the initial rounds of data collection occurred.

Overall, these findings add to basic etiologic research on developmental pathways of smoking in adolescence and young adulthood by providing evidence of heterogeneity in smoking behavior and prospectively linking different patterns of risk factors with the probability of trajectory group membership. Our results suggest there are different periods for and profiles of risk that have implications for both the timing and targeting of smoking interventions. Along with prevention efforts aimed at early and middle adolescents that might be most effective for *quitters*, *stable high*, and *stable light* smokers, the post-high school years represent an important intervention period for *late escalators*, whose smoking behavior increased after age 18. Despite their low risk profile in early adolescence, *late escalators'* smoking surpassed that of *stable*

high smokers in early adulthood, suggesting the need for a better understanding of how the etiological processes and mechanisms associated with smoking risk may evolve over time or emerge during different periods of development. Future research incorporating into the trajectory model characteristics that might change during the transition from mid-adolescence to early adulthood, that have the potential to influence smoking behavior and alter the trajectories we observed would be a logical extension to this analysis. Such an approach will contribute to the development of intervention and prevention programs that are tailored to different subgroups within the broader population of smokers.

Acknowledgments

Data analyses were supported in part by grant K DA15454-01 from the National Institute of Drug Abuse (Dierker), and an Investigator Award from the Patrick and Catherine Weldon Donaghue Medical Research Foundation (Dierker), and used data from the National Longitudinal Study of Adolescent Health. The Add Health study was designed by J. Richard Udry (PI) and Peter Bearman and funded by Grant P01-HD31921 from the National Institute of Child Health and Human Development to the Carolina Population Center, University of North Carolina at Chapel Hill, with cooperative funding from 17 other agencies. Persons interested in obtaining data files from The National Longitudinal Study of Adolescent Health should contact Add Health, Carolina Population Center, 123 West Franklin Street, Chapel Hill, NC 27516-2524 (http://www.cpc.unc.edu/addhealth).

References

Abroms L, Simons-Morton B, Haynie D, Chen R. Psychosocial predictors of smoking trajectories during middle and high school. Addiction 2005;100:852–861. [PubMed: 15918815]

Audrain-McGovern J, Rodriguez D, Tercyak K, Cuevas J, Rodgers K, Patterson F. Identifying and characterizing adolescent smoking trajectories. Cancer Epidemiology, Biomarkers & Prevention 2004;13:2023–2034.

Bates M. Integrating person-centered and variable-centered approaches of developmental courses and transitions in alcohol use: Introduction to the special section. Alcoholism: Clinical and Experimental Research 2000;24:878–881.

Centers for Disease Control and Prevention. Cigarette use among high school students-United States, 1991–2003. Morbidity and Mortality Weekly Report 2004;53:499–502. [PubMed: 15201841]

Centers for Disease Control Prevention. Annual smoking-attributable mortality, years of potential life lost, and productivity losses-United States, 1997–2001. Morbidity and Mortality Weekly Report 2005;54:625–628. [PubMed: 15988406]

Chantala K. Introduction to analyzing Add Health data. 2003Retrieved February 4, 2006, from http://www.cpc.unc.edu/projects/addhealth/files/analyze.pdf

Chassin L, Presson C, Pitts S, Sherman S. The natural history of cigarette smoking from adolescence to adulthood in a Midwestern community sample: Multiple trajectories and their psychosocial correlates. Health Psychology 2000;19:223–231. [PubMed: 10868766]

Chassin L, Presson C, Rose J, Sherman S. The natural history of cigarette smoking from adolescence to adulthood: Demographic predictors of continuity and change. Health Psychology 1996;15:478–484. [PubMed: 8973929]

Chen K, Kandel D. The natural history of drug use from adolescence to the mid-thirties in a general population sample. American Journal of Public Health 1995;85:41–47. [PubMed: 7832260]

Cohen, J. Statistical power analysis for the behavioral sciences. Hillsdale, NJ: Lawrence Earlbaum Associates; 1988.

Cohen, J.; Cohen, P.; West, S.; Aiken, L. Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences. Vol. 3rd ed.. Mahwah, NJ: Lawrence Erlbaum Associates; 2003.

Colder C, Mehta P, Balanda K, Campbell R, Mayhew K, Stanton W, Pentz W, Flay B. Identifying trajectories of adolescent smoking: An application of latent growth mixture modeling. Health Psychology 2001;20:127–135. [PubMed: 11315730]

Compas B, Hinden B, Gerhardt C. Adolescent development: Pathways and processes of risk and resilience. Annual Review of Psychology 1995;46:265–293.

Guo J, Chung I, Hill K, Hawkins D, Catalano R, Abbott R. Developmental relationships between adolescent substance use and risky sexual behavior in young adulthood. Journal of Adolescent Health 2002;31:354–362. [PubMed: 12359381]

Hart D, Atkins R, Fegley S. Personality and development in childhood: A person centered approach. Monographs of the Society for Research in Child Development 2003;68:1–108.

Heatherton T, Kozlowski L, Frecker R, Fagerström K. The Fagerström Test for Nicotine Dependence: A revision of the Fagerström Tolerance Questionnaire. British Journal of Addiction 1991;86:1119–1127. [PubMed: 1932883]

Jackson K, Sher K, Schulenberg J. Conjoint developmental trajectories of young adult alcohol and tobacco use. Journal of Abnormal Psychology 2005;114:612–626. [PubMed: 16351384]

Johnston, L.; O'Malley, P.; Bachman, J.; Schulenberg, J. Monitoring the Future National Results on Adolescent Drug Use: Overview of Key Findings, 2005. Bethesda, MD: National Institute on Drug Abuse; 2006. (NIH Publication No. 06–5882)

Jones B, Nagin D, Roeder K. A SAS procedure based on mixture models for estimating developmental trajectories. Sociological Methods and Research 2001;29:384–393.

Lynch, B.; Bonnie, R., editors. Growing Up Tobacco Free: Preventing Nicotine Addiction in Children and Youths. Washington, DC: National Academy Press; 1994.

Magnusson, D. The logic and implications of a person-oriented approach. In: Cairns, R.; Bergman, L., editors. Methods and Models for Studying the Individual. Thousand Oaks, CA: Sage; 1998. p. 33-64.

Magnusson D. The person approach: Concepts, measurement models, and research strategy. New Directions for Child and Adolescent Development: Person-Centered Approaches to Studying Development in Context, Fall 2003 2003:3–23.

Miyazaki Y, Raudenbush S. Testing for linkage of multiple cohorts in an accelerated longitudinal design. Psychological Methods 2000;5:44–63. [PubMed: 10937322]

Muthén B, Muthén L. Integrating person-centered and variable-centered analyses: Groth mixture modeling with latent trajectory classes. Alcoholism: Clinical and Experimental Research 2000;24:882–891.

Nagin D. Analyzing developmental trajectories: A semi-parametric group-based approach. Psychological Methods 1999;4:139–157.

Nagin, D. Group-Based Modeling of Development. Cambridge, MA: Harvard University Press; 2005.

Nagin D, Tremblay R. What has been learned from group-based trajectory modeling? Examples from physical aggression and other problem behaviors. The Annals of the American Academy of Political and Social Science 2005;602:82–117.

Orlando M, Tucker J, Ellickson P, Klein D. Developmental trajectories of cigarette smoking and their correlates from early adolescence to young adulthood. Journal of Consulting and Clinical Psychology 2004;72:400–410. [PubMed: 15279524]

Radloff L. The CES-D Scale: A self-report depression scale for research in the general population. Applied Psychological Measurement 1977;1:385–401.

Resnick M, Bearman P, Blum R, Bauman K, Harris K, Jones J, Tabor J, Beuhring T, Sieving M, Shew M, Ireland M, Bearinger H, Udry J. Protecting adolescents from harm. Findings from the National Longitudinal Study on Adolescent Health. JAMA 1997;278:823–832. [PubMed: 9293990]

Singer, J.; Willett, J. Applied Longitudinal Data Analysis: Modeling Change and Event Occurrence. New York: Oxford University Press; 2003.

Stanton W, Flay B, Colder C, Mehta P. Identifying and predicting adolescent smokers' developmental trajectories. Nicotine & Tobacco Research 2004;6:843–852. [PubMed: 15700920]

White H, Johnson V, Buyske S. Parental modeling and parenting behavior effects on offspring alcohol and cigarette use: A growth curve analysis. Journal of Substance Abuse 2000;12:287–310. [PubMed: 11367605]

White H, Nagin D, Replogle E, Stouthamer-Loeber M. Racial differences in trajectories of cigarette use. Drug and Alcohol Dependence 2004;76:219–227. [PubMed: 15561473]

White H, Pandina R, Chen P. Developmental trajectories of cigarette use from early adolescence into young adulthood. Drug and Alcohol Dependence 2002;65:167–178. [PubMed: 11772478]

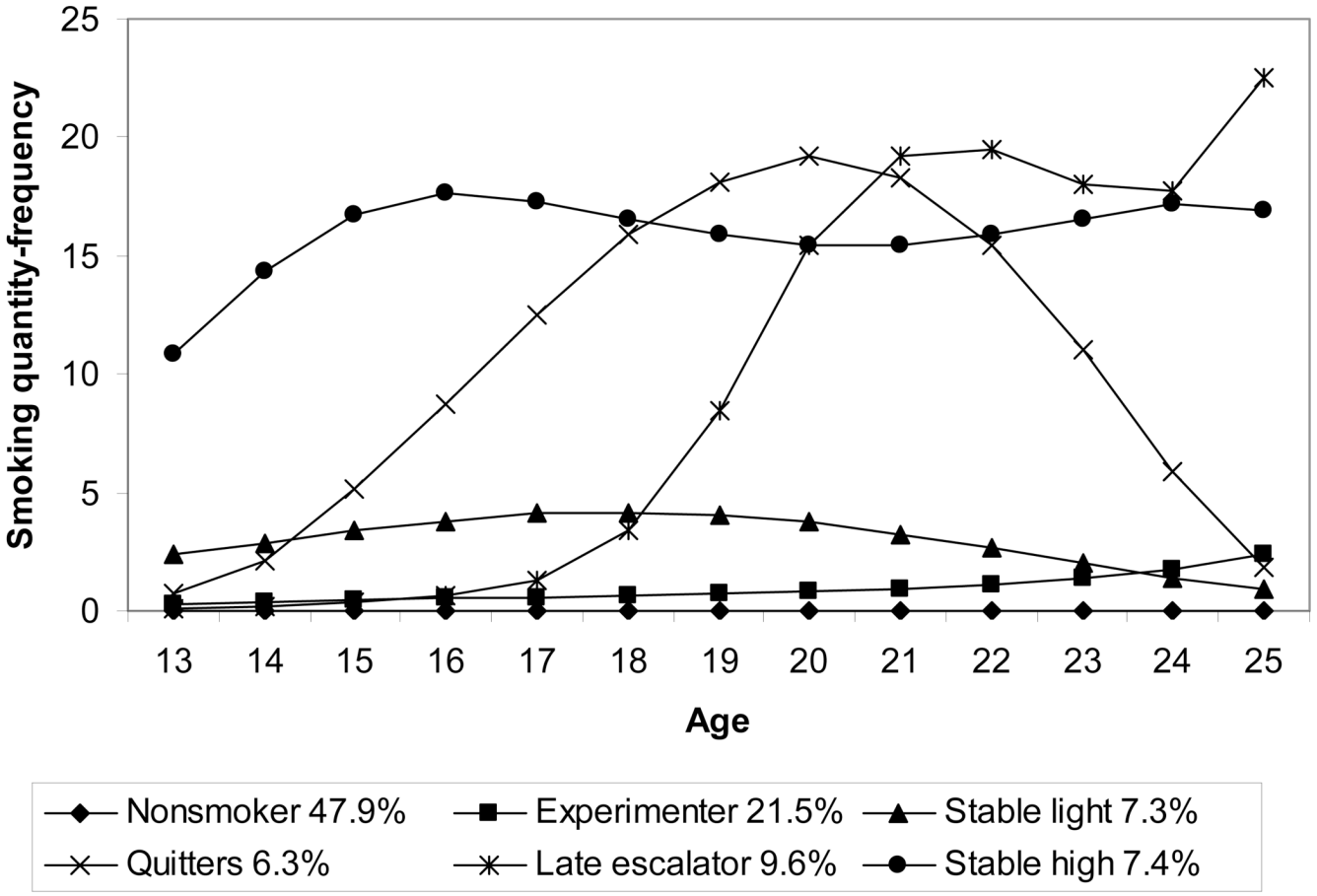

Figure 1.
Predicted smoking quantity-frequency for each smoking trajectory group in a sample of U.S. adolescents and young adults (*n*=5,789).

Table 1

Bayesian information criteria (BIC) for a series of non-parametric group-based models of smoking quantity-frequency

Model	BIC
Three groups	−23528.39
Four groups	−22866.30
Five groups	−22610.13
Six groups	−22383.13
Seven groups	−22446.44
Eight groups	—
Six groups (including covariates)	−18506.05

Note. The eight group model failed to converge.

Table 2

Adjusted odds ratios (95% CI) for predictors of smoking trajectory group membership in a sample of U.S. adolescents and young adults (*n*=5,789).

Reference group / Trajectory group	Alcohol use	Drug use	Depression	Deviance	Peer smoking
Nonsmokers					
Experimenters	1.86 (1.56–2.22)	1.85 (1.23–2.79)	1.00 (0.98–1.02)	2.54 (1.55–4.17)	1.54 (1.28–1.85)
Stable light	1.77 (1.46–2.15)	5.50 (3.18–9.52)	1.01 (0.98–1.03)	2.78 (1.69–4.56)	2.77 (2.22–3.46)
Quitters	1.53 (1.25–1.87)	4.34 (2.59–7.27)	1.01 (0.99–1.04)	2.48 (1.48–4.15)	2.82 (2.25–3.54)
Late escalators	1.53 (1.29–1.83)	1.28 (0.87–1.90)	1.01 (0.99–1.03)	2.32 (1.44–3.72)	1.54 (1.30–1.83)
Stable high	1.82 (1.51–2.20)	4.83 (3.06–7.63)	1.01 (0.99–1.04)	3.19 (1.95–5.21)	4.17 (3.39–5.13)
Experimenters					
Stable light	0.95 (0.81–1.11)	2.97 (1.77–4.98)	1.01 (0.98–1.03)	1.09 (0.93–1.29)	1.80 (1.49–2.17)
Quitters	0.82 (0.71–0.95)	2.34 (1.42–3.86)	1.01 (0.99–1.03)	0.97 (0.74–1.28)	1.83 (1.46–2.30)
Late escalators	0.82 (0.72–0.94)	0.69 (0.48–1.01)	1.01 (0.99–1.03)	0.91 (0.65–1.29)	1.00 (0.83–1.20)
Stable high	0.98 (0.87–1.10)	2.60 (1.75–3.88)	1.01 (0.99–1.03)	1.26 (1.08–1.45)	2.70 (2.23–3.28)
Stable light					
Quitters	0.86 (0.71–1.05)	0.79 (0.39–1.62)	1.00 (0.97–1.03)	0.89 (0.65–1.21)	1.02 (0.76–1.38)
Late escalators	0.87 (0.74–1.02)	0.23 (0.13–0.41)	1.00 (0.97–1.03)	0.83 (0.57–1.22)	0.56 (0.44–0.70)
Stable high	1.03 (0.89–1.18)	0.88 (0.50–1.54)	1.00 (0.98–1.03)	1.15 (1.01–1.30)	1.51 (1.21–1.88)
Quitters					
Late escalators	1.00 (0.84–1.20)	0.30 (0.17–0.50)	1.00 (0.97–1.02)	0.94 (0.62–1.41)	0.55 (0.44–0.68)

Reference group					
Trajectory group	**Alcohol use**	**Drug use**	**Depression**	**Deviance**	**Peer smoking**
Stable high	1.19 (0.99–1.43)	1.11 (0.62–2.00)	1.00 (0.97–1.03)	1.29 (0.97–1.71)	1.48 (1.13–1.94)
Late escalators					
Stable high	1.19 (1.03–1.37)	3.76 (2.40–5.89)	1.00 (0.98–1.03)	1.38 (0.95–1.99)	2.70 (2.18–3.35)
Stable high (see previous entries)					

Note. Models adjusted for gender, race, and mother's education.