The Natural History of Cigarette Smoking: Predicting Young-Adult Smoking Outcomes From Adolescent Smoking Patterns

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Assessed the magnitude of risk that adolescent cigarette smoking carries for adult smoking. Using a longitudinal, prospective design, results indicate that even infrequent experimentation in adolescence significantly raises the risk for adult smoking and that regular (at least monthly) adolescent smoking raises the risk for adult smoking by a factor of 16 compared to nonsmoking adolescents. Relative risk was also increased by an early onset of smoking and by a stable, uninterrupted course from experimentation to regular smoking. Relative risk did not significantly vary by age or sex. The continuity of smoking behavior between adolescence and adulthood supports the importance of primary prevention programs directed at adolescent populations.

Key words: adolescent smoking, young-adult smoking, relative risk

Cigarette smoking represents a significant public health problem and has been identified as the single largest preventable cause of disease and premature death in the United States (U.S. Surgeon General, 1989). Despite substantial rates of quitting since the Surgeon General's report in 1964, many adults continue to smoke cigarettes, with an estimated 30.4% of adults being regular smokers in 1985 (Fiore et al., 1989). Moreover, smoking rates in adolescents have also remained high. Rates of adolescent smoking prevalence rose to a peak in 1976-1977 and have been stable since 1981. Available national data show that 26.9% of high school seniors report smoking in the past month (Johnston, O'Malley, & Bachman, 1987).

Recent efforts to control smoking have been largely focused on the primary prevention of cigarette smoking among adolescents, mostly through school-based programs (Botvin & Wills, 1985; Flay, 1985; Perry, Killen, Telch, Slinkard, & Danaher, 1980). The rationale for such an approach is that smoking, once established, is a very difficult behavior to modify. Cessation programs have generally shown some short-term success.
but substantial relapse (see, e.g., Curry, Marlatt, Gordon, & Baer, 1988; Klesges et al., 1988). Accordingly, professionals have turned to primary prevention rather than cessation as the intervention of choice.

These prevention programs, however, rest on a central assumption about the natural history of smoking behavior. It is assumed that adolescent cigarette smoking does indeed raise risk for adult smoking—that is, that these potential adolescent smokers would actually become long-term regular adult smokers. There are theoretical reasons to believe that this could be the case. Once cigarette smoking is initiated, processes of physical and psychological dependence would ensure that a majority of adolescent smokers would remain smokers into adulthood. In addition, if the factors that motivate adolescent smoking also continue into adulthood, smoking behavior should similarly show a high degree of stability. For example, if the social-image benefits obtained from adolescent smoking also accrue to adults who smoke, then this stable motivation would produce a pattern of continuity between adolescent and adult cigarette smoking.

It is also possible, however, that adolescent cigarette smoking represents a period of benign experimentation with few consequences or implications for regular adult smoking. These adolescent smokers may naturally mature out of smoking such that the behavior is self-limiting. Thus, adult smokers (who may smoke for very different reasons than adolescents) would come equally from both adolescent smokers and nonsmokers alike. If this were true, then prevention programs (which are usually aimed at the motivations underlying adolescent smoking) might be ineffective at preventing ultimate adult smoking outcomes.

The best way to assess the relative risk of adult smoking given that one is an adolescent smoker is to carry out prospective longitudinal studies that follow the smoking patterns of a group of subjects from adolescence into adulthood. Past studies that have looked at this matter have reported retrospective data only (e.g., Kandel & Logan, 1984), have followed subjects only as far as the high school years (Ershler, Leventhal, Fleming, & Glynn, 1989), or have analyzed data only at the group level so that continuity for individuals was not addressed (O'Malley, Bachman, & Johnston, 1984). Thus, there are no studies that have assessed the relative risk for adult smoking that is associated with adolescent smoking. Assessing the magnitude of this risk is one of the primary purposes of the current prospective, longitudinal study.

There are several senses in which the term risk might be used. Many studies concerned with adolescent and adult substance use have talked about risk in terms of the percentage of adolescent users who continue on to adult use (cf. Donovan, R. Jessor, & L. Jessor's, 1983, study of problem drinking). This approach reports the absolute risk for adolescent users. Such a measure, however, can be misleading because it fails to indicate the
relative risk for users as opposed to nonusers. That is, it fails to take into account the risk that nonusers also face. We use the term risk in the sense of relative risk—the increase in the odds of adult smoking given adolescent smoking (Breslow & Day, 1980). This measure is particularly useful because it assesses the elevation in risk compared to the relevant level of background risk.

In addition to the issue of risk of adolescent smoking, the present study has two other specific aims. First, we examine the risk associated with different ages of onset of adolescent smoking. Theoretically, we expect that earlier onset of use will carry greater relative risk for adult smoking than will later onset. Adolescents who begin smoking at earlier ages are likely to be those with particularly powerful motives and, therefore, are more likely to continue smoking. In fact, earlier age of onset has been associated with more continued use of cigarettes (Ershler et al., 1989) and other substances (e.g., Brill & Christie, 1974).

Second, we examine the degree of risk associated with different levels of adolescent smoking. Many adolescents are experimental rather than regular smokers. Is it simply the act of smoking that raises risk for adult smoking, or is the amount of smoking an important risk predictor? Four groups of adolescents (never smokers, triers, ex-smokers, and regular smokers) are compared in terms of their relative risk for later adult smoking.

METHOD

Subjects

Subjects were participants in an ongoing longitudinal investigation of cigarette smoking (cf. Chassin et al., 1981). The original adolescent sample consisted of all consenting 6th to 12th graders in a Midwestern county school system who were present in school on the day of testing. All 6th- to 12th-grade classrooms (excluding special-education classrooms) in the county school system were surveyed annually between 1980 and 1983. Mean ages of the sample were 14.5, 14.3, 14.0, and 14.1 years, respectively, over the 4 years of measurement (range = 10 to 21 years in each year).

For the current analyses, potential subjects were drawn from those cohorts who had completed high school by the time of latest data collection (1987–1988) and, thus, were considered to be “adults” at follow-up. For these adult subjects, there was a potential pool of 5,799 individuals who had been assessed at least once during their adolescence (between 1980 and 1983). At the time of follow-up, 25 of these subjects were found to be decreased, and 175 refused participation; 4,156 provided data (a follow-up rate of 72%).
Demographic figures show that the subjects were predominantly White (96%), were equally divided by sex (49% men, 51% women), and had an average age of 21.8 years. More than 99% of the subjects were between the ages of 18 and 26. Seventy-one percent had never been married, and 26% were currently married. Nearly 58% of those sampled had completed at least some college by the time of follow-up, and many (32%) were still students. Forty-three percent had a high school education. For nonstudents, occupational status ranged from 29% in factory, crafts, and labor occupations to 39% in professional, technical, and managerial positions.

At follow-up, the overall rate of smoking (at least weekly) was 26.7%. National data (Fiore et al., 1989) report some current cigarette use among 29.4% of White adults measured in 1985. Our slightly lower prevalence may be due to differences in the definition of smoking combined with the slightly higher than average education level of our sample. A further comparison can be made with the Monitoring the Future Study (Johnston et al., 1987), which found a daily smoking rate of 25.9% among a sample of high school seniors measured 1 to 9 years after high school (compared to a 23.3% daily smoking rate in our sample). Thus, smoking prevalence in our sample is fairly representative of recent national trends.

Procedure

The adolescent data were collected using group-administered questionnaires in classroom groups. Questionnaires were administered by members of the research team who were unconnected to the school system. Subjects were assured of confidentiality.

Follow-up data were collected between 1987 and 1988, resulting in a 4- to 8-year follow-up interval. Subjects were located by using public school records, city directories, voter registration lists, Department of Motor Vehicles records, local university directories, alumni records, high school reunion committees, and local newspaper stories. In addition, subjects were tracked through siblings and relatives, through informal peer friendship networks (using “trackers” hired from each high school class), and by placing advertisements in the local newspaper. After location, subjects received mailed questionnaires and were paid $10 for their participation.

If questionnaires were not returned, follow-up “reminder” postcards and phone contacts were attempted approximately 1 month after the initial mailing. Short telephone interviews were used to collect data from subjects who failed to return questionnaires. Two hundred thirty cases were obtained in this manner. Of this subsample, 81 subjects eventually returned completed questionnaires. This allowed us to test the comparability of self-reported smoking across the two methods of data collection. Pearson
product-moment correlations between items reported by questionnaire and by telephone ranged from .84 to 1.00 (Cramer's $V$ ranged from .65 to 1.00). Due to the high degree of similarity in reporting, the telephone data were included in analyses.

**Measures**

The data of interest in the current study are self-reported smoking status in adolescence and at adult follow-up. In adolescence, smoking status was assessed with a single item that divided subjects into abstainers (never smoked a cigarette, not even a single puff), triers (have smoked one or two "just to try" but not in the last month), ex-smokers (no longer smoke but in the past was a regular smoker), and regular smokers (smoked monthly or more). To improve the validity of self-reported smokers status, a bogus pipeline procedure (Evans, Hansen, & Mittelmark, 1977) was used in Years 2 through 4. Subjects were told that chemical analysis of saliva could detect smoking and were then asked to lick a strip of paper and place it in an envelope marked with their subject number. After providing this saliva specimen, subjects self-reported their smoking status.

An adult follow-up, smoking status was assessed with an identical item except that regular smoking was defined as at least weekly smoking.

**RESULTS**

**Attrition Bias**

To assess the extent of attrition bias, we compared those lost to follow-up ("dropouts") with those who were successfully located ("stayers") in terms of their initial adolescent data. Stayers and dropouts were compared on 29 variables for each of the 4 initial years of data (using discriminant function analyses followed by univariate $F$ tests). These variables included personality and perceived environment items from R. Jessor and S. L. Jessor's (1977) problem behavior theory, attitudes and beliefs about cigarette smoking, and smoking in the adolescent's social environment (see Chassin et al., 1981, for a complete description). Results indicate that the two groups significantly differed on 23 variables for both Year 1 and Year 2, 20 variables for Year 3, and 22 variables for Year 4 (all discriminant function analyses showed multivariate differences between groups). For all years, dropouts were more likely to have been cigarette smokers ($p < .0001$), to have had friends who smoked ($p < .0001$), to have come from prosmoking environments ($p < .0001$), and to have an external locus of control ($p < .0001$), higher attitudinal tolerance for deviance ($p < .05$), and lower levels
of parental expectations for academic and general success \( (p < .05) \). It should be noted that, although the discriminant functions for each year were statistically significant, the canonical correlations were small in magnitude (.19 for Year 1, .20 for Year 2 and Year 3, and .25 for Year 4). Nevertheless, the consistent pattern of the results suggests that dropouts were more "deviance prone" in R. Jesser and S. L. Jesser's (1977) sense of the term. Therefore, as in any study requiring multiple measurement, caution should be used in generalizing these results, in this case because more deviant adolescents are likely to be underrepresented in the sample.

The Validity of Self-Reported Smoking

To verify the validity of self-reported smoking in the adult follow-up sample, a substudy was conducted. A subsample of adult subjects was drawn randomly from those who had returned completed questionnaires and whose address was within the same city in which the study was based. One hundred seventy-three subjects were contacted and asked to participate in an additional questionnaire study. One hundred forty subjects (80.9%) agreed to participate, and 133 (76.9%) arrived to complete the protocol. On arriving at the laboratory, subjects were given a short questionnaire to report on their tobacco use (including use in the past 4 hr). Subjects were then asked to help us test "a new machine" that could measure the amount of carbon monoxide (CO) in expired air (i.e., they were given an unannounced bioassay). After providing a separate informed consent for the bioassay, subjects gave a breath sample using a MiniCO device (Model 1000, Catalyst Research Corp., Owen Mills, MD).

Of the 133 potential subjects for this validation, the data for 20 subjects were discarded (in 15 cases, the bioassay was mistakenly announced in advance; in 4 cases, the MiniCO malfunctioned; in 1 case, the wrong subject arrived for testing). Pearson product--moment correlations were calculated between the CO readings and self-reported smoking items. Correlations ranged from a low of .65 to a high of .80.

Level of Adolescent Smoking as a Risk Factor for Adult Smoking

The major question of the current study was whether adolescent cigarette smoking represented a significant risk factor for adult smoking. To answer this question, we performed a multivariate analysis to predict adult smoking status, dichotomized as nonsmoker versus current regular smoker (at least weekly). Adult smoking status was predicted from adolescent smoking status (never, trier, and regular—at least monthly—smoking), sex, age (middle school student vs. high school student), and the two-way interactions among these variables. This was done using a categorical modeling
TABLE 1

Multivariate Analysis to Predict Adult Smoking Status, Using Adolescent Smoking Status, Sex, and Grade, by Year of Measurement

<table>
<thead>
<tr>
<th>Possible Predictor of Adult Smoking Status</th>
<th>Chi-Square Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1980⁺</td>
</tr>
<tr>
<td>Adolescent smoking status</td>
<td>377.45**</td>
</tr>
<tr>
<td>Sex</td>
<td>.47</td>
</tr>
<tr>
<td>Grade</td>
<td>32.90**</td>
</tr>
<tr>
<td>Adolescent Smoking Status × Sex</td>
<td>1.99</td>
</tr>
<tr>
<td>Adolescent Smoking Status × Grade</td>
<td>18.35**</td>
</tr>
<tr>
<td>Sex × Grade</td>
<td>.69</td>
</tr>
</tbody>
</table>

⁺n = 3,238. ⁻n = 2,656. ³n = 1,844.
*⁺p < .05. **⁺p < .01.

(CATMOD) analysis (within SAS; SAS Institute, Inc., 1985). Three modeling analyses were performed (for Years 1, 2, and 3 of measurement—Year 4 had no middle school subjects who were adults at follow-up).

In each year, there was a significant effect of adolescent smoking status on adult smoking status (see Table 1). This indicates that adolescent smoking does indeed significantly raise the risk for adult smoking. There were also consistent significant effects for grade such that original middle school students showed higher smoking prevalence at follow-up than did original high school students (smoking rates of 26.8% vs. 23.1% in the Year 1 data, 27.0% vs. 24.1% in the Year 2 data, and 25.5% vs. 23.5% in the Year 3 data). There were no significant effects of subject sex and no consistent interaction effects. Isolated interactions emerged within a single year of measurement (adolescent smoking by sex in Year 2 and adolescent smoking by grade in Year 1). Because these were isolated, unreplicated effects, however, they are not considered reliable and are not interpreted further.

To assess the magnitude of the relative risk that adolescent smoking carried for later adult smoking, we first calculated the percentages of adult smokers for each adolescent smoking status for each year of measurement (see Table 2). Outcomes for initial ex-smokers are also presented, although they were not included in the CATMOD analysis. Because there were no consistent significant interactions involving sex or grade in that multivariate analysis, these data are collapsed across sex and grade level. The relative risk statistic¹ (Breslow & Day, 1980; Fleiss, 1973) was then calculated for each adolescent smoking status (compared to adolescent never smokers) for

¹Relative risk is evaluated by the observed odds ratio: \( (p_1 \times q_0) / (p_0 \times q_1) \), where \( p_1 \) and \( p_0 \) are the probabilities of exposure to the risk factor among those who are diseased and disease free and \( q_1 \) and \( q_0 \) are the corresponding probabilities of nonexposure among those diseased and nondiseased (see Breslow & Day, 1980).
TABLE 2
Percentages of Adolescents Who Became Adult Regular Smokers, by Adolescent Smoking Status and Year of Measurement

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Never smoker</td>
<td>13.4</td>
<td>12.1</td>
<td>10.7</td>
<td>10.6</td>
</tr>
<tr>
<td>Trier</td>
<td>24.9</td>
<td>26.0</td>
<td>25.1</td>
<td>23.5</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>42.5</td>
<td>46.2</td>
<td>43.9</td>
<td>42.9</td>
</tr>
<tr>
<td>Regular smoker</td>
<td>72.4</td>
<td>72.0</td>
<td>69.6</td>
<td>71.5</td>
</tr>
</tbody>
</table>

Each year of measurement. These relative risk statistics (with 95% confidence intervals) are displayed in Figure 1. Note that the relative risk statistic associated with adolescent never smoking is always set to 1. The relative risk statistics thus reflect the elevation in risk for each level of adolescent smoking over and above the risk that is associated with adolescent nonsmoking.

As can be seen in Figure 1, each level of adolescent exposure to cigarette smoking significantly raised the risk for adult regular smoking; chi-square tests of association ($df = 1$) ranged from 33.94 for ex-smokers in 1983 to 437.92 for regular smokers in 1980 (all $ps < .001$). Even trying a few cigarettes during adolescence more than doubled the relative risk for adult regular smoking. Adolescent regular (at least monthly) smokers had a risk for adult smoking that was raised by a factor of at least 16 in the 4 years of measurement.

To assess whether the relative risk significantly varied over year of measurement, chi-square tests of homogeneity were performed (Fleiss, 1973). The chi-square tests of homogeneity ($df = 3$) were 2.77 for triers, .98 for ex-smokers, and .33 for regular smokers (all ns). Thus, there was no significant variation in risk across the 4 years of study.

The patterns of relative risk suggest a strong dose-response relation such that higher levels of adolescent exposure to smoking further increased the risk of adult regular smoking. To evaluate this pattern, we calculated a chi-square test for trend ($df = 3$; Breslow & Day, 1980), within which we weighted each level of adolescent exposure differentially (assigning weights of 0, 1, 2, and 3 to the four adolescent smoking levels). The importance of the chi-square test for linear trend can be evaluated relative to the overall chi-square test of association for that year, stratified by level of exposure. We performed this test for each year of adolescent measurement. In Year 1, the overall chi-square was 641.56, and the chi-square associated with the linear trend was 611.80. The comparable figures were 534.77 and 515.14 for Year 2, 352.93 and 332.98 for Year 3, and 333.51 and 322.46 for Year 4. Therefore, for each year, most of the overall variation in risk for adult
smoking can be accounted for by the linear increase in risk with dose of adolescent smoking.

Grade Effects in Adult Smoking Prevalence

Recall that the multivariate CATMOD analysis showed significant grade effects on adult smoking prevalence such that initial middle schoolers showed higher adult smoking prevalence than did initial high school students. One question that arises from this result is whether the same grade differences were present in the adolescent data. If the grade effects were a result of stable, historical factors that selectively impacted on certain cohorts of children, then these initial high school subjects should have shown lower smoking prevalence than the initial middle school subjects even in the adolescent years. To test this possibility, we examined the

FIGURE 1 Relative risk of adult regular smoking for each adolescent smoking status compared to adolescent never smokers for each year of measurement, with 95% confidence intervals.
original smoking data for these subjects who were in our adult follow-up sample. In adolescence, these high school students showed greater smoking prevalence (at least monthly smoking) than did middle school students (14.6% vs. 7.0% in 1980, 13.9% vs. 8.2% in 1981, and 12.8% vs. 6.7% in 1982). Thus, the pattern of age differences that was found in adolescence (older high school subjects smoking more) was reversed in young adulthood (the oldest subjects smoking less).

To further demonstrate that age differences in smoking prevalence found in young adulthood were not the result of stable, preexisting cohort differences, we can examine the adolescent data from 10th graders over the 4 years of adolescent measurement. The prevalence of regular (weekly) smoking in these four cohorts was 11.3%, 11.5%, 10.2%, and 9.5%. Therefore, during the adolescent years, the younger cohorts did not show greater smoking prevalence than the older cohorts. Thus, the higher smoking rates among younger cohorts in the adult data are not due to stable, preexisting cohort differences.

Another possible explanation for the age effects on adult smoking prevalence is differential attrition from the study such that initial high school smokers were more likely to be lost to follow-up than initial middle school smokers. Such a pattern of differential attrition could produce a lower adult smoking prevalence among initial high school students than among initial middle school students. This was not the case, however. In fact, we retained 65% of the Year 1 middle school smokers and 73% of the Year 1 high school smokers. Similarly, we retained 65.9% of the Year 2 middle school smokers and 73.8% of the Year 2 high school smokers. We retained 67.0% of the Year 3 middle school smokers and 74.5% of the Year 3 high school smokers.

Examining retention in general (without regard to adolescent smoking status) revealed the same pattern. There was 72.5% retention of Year 1 middle school subjects compared to 76.4% retention of Year 1 high school students. There was 72.3% retention of Year 2 middle school subjects compared to 77.1% of Year 2 high school subjects. There was 74.2% retention of Year 3 middle school subjects compared to 77.2% of Year 3 high school subjects. Therefore, differential attrition of middle school and high school smokers cannot explain the age effect on adult smoking prevalence.

Age of Onset Effects on the Risk of Adolescent Smoking for Adult Smoking

The group of smokers at any particular age or grade (i.e., smoking prevalence) is composed of both recent initiators and longstanding smokers. It is important to examine the incidence (or new cases) of smoking by grade
in terms of age effects on relative risk. It is possible that adolescents who initiate smoking at an early age are particularly committed to the behavior and that, therefore, early onset smoking carries higher risk for adult smoking.

For this analysis, we selected subjects who had at least 2 years of data, who reported regular (at least monthly) smoking at some time during our study period, and whose pattern of reported smoking status allowed us to identify uniquely the grade at which they began regular smoking. Table 3 presents the adult smoking outcomes (at least weekly smoker–nonsmoker) for each grade of onset.\(^2\) A multivariate CATMOD analysis was used to predict adult smoking status (current smoker–current nonsmoker) from adolescent grade of smoking initiation, sex, and the interactions between grade of smoking initiation and sex. This analysis showed a significant main effect of adolescent grade of smoking initiation, \(\chi^2(1, N = 1069) = 12.52, p < .01\), such that earlier grade of onset was associated with greater risk for adult smoking. There was no significant effect of sex, and there was no interaction between grade of onset and sex.

**Course of Adolescent Smoking Onset and Risk for Adult Smoking**

Another question of interest was whether the pattern and course of adolescent smoking onset affected risk for later adult smoking. Here the questions were whether slow (rather than fast) and stable (rather than erratic) patterns of smoking onset led to higher risk for later adult smoking.

\(^2\)These prevalence rates for adolescent regular smokers who became adult smokers are lower than are the ones in Table 2. This is because the current analysis uses only a subset of adolescent smokers, those for whom the grade of initial regular smoking could be uniquely identified.
To test whether fast versus slow onset patterns had greater risk for later adult smoking, we selected all subjects with at least 3 years of data who were never smokers or triers in Years 1 or 2 and who ended the study in Years 3 or 4 as a regular smoker. Those who made the transition from a never smoker to a regular smoker in 1 year were considered “fast” \((n = 22)\), and those who took longer than 1 year were considered “slow” \((n = 120)\). Of the fast onset group, 54.6% became adult regular smokers compared to 65% of the slow onset group. There was no significant elevation, however, in relative risk of slow onset compared to fast onset—relative risk = 1.55, \(\chi^2(1, N = 140) = .48\), ns.

To test whether erratic versus stable smoking onset affected the risk for adult smoking, we selected subjects who had 4 years of data and who were regular (at least monthly) smokers by Year 4. Those who showed 2 or more years of uninterrupted smoking were considered “stable” \((n = 42)\), and those who showed interruptions in smoking were considered “erratic” \((n = 91)\). Of the stable onset group, 78.6% were regular adult smokers compared to 54.9% of the erratic onset group. There was a significant elevation in relative risk for the stable compared to the erratic onset group—relative risk = 3.01, \(\chi^2(1, N = 133) = 15.87, p < .05\).

**DISCUSSION**

The findings of the current study convincingly demonstrate that exposure to cigarette smoking during adolescence does indeed substantially raise the risk for regular adult smoking. These prospective data confirm retrospective reports of the stability of cigarette smoking between adolescence and adulthood (Kandel & Logan, 1984). In addition to generally confirming previous speculation of raised risk associated with adolescent smoking, however, the current study is the first to allow a detailed examination of the magnitude of that risk. As indicated by our findings, the magnitude of this risk increases with the extent of exposure to smoking in adolescence. Even very light, experimental use (i.e., smoking only a few cigarettes) doubles the risk for adult smoking relative to no experimentation during adolescence, and monthly smoking in adolescence raises the risk for adult smoking by a factor of 16. The substantial elevation in risk for adult smoking as a function of adolescent smoking supports the importance of primary prevention interventions designed to deter the initiation of cigarette smoking during adolescence.

The current study found no substantial differences in the risk associated with adolescent smoking among male and female subjects. Given that this sample is of childbearing age, we might have expected females who smoked during adolescence to have quit at adult follow-up and, thus, shown lower
relative risk than their male peers. Available data, however, show high smoking prevalence among women in childbearing years (higher prevalence of female than male smokers in high school, college, and young adulthood; Johnston et al., 1987). Moreover, available data suggest that only a minority of pregnant smokers quit during pregnancy and that this quitting is usually a temporary phenomenon (Prager et al., 1983; Windsor & Orleans, 1986). If only a small percentage of women quit during pregnancy, and, if those who quit resume smoking after delivery, then this quitting would have only minimal impact on adult smoking among women in our sample.

A surprising finding concerned the significant effects of age on smoking prevalence, in that older subjects (initial high school students) showed lower smoking prevalence than did younger subjects (initial middle school subjects). Further analyses ruled out stable preexisting (adolescent) differences between these groups as an explanation and also ruled out differential attrition as an explanation. One possible interpretation of this finding is that it reflects the aging of our sample into a point in adulthood at which individuals stop smoking cigarettes. Research in other substance use behaviors suggest that use peaks between the ages of 18 and 25 and that in the early to mid-20s prevalence rates drop (at different ages depending on the substance; Kandel & Logan, 1984). This developmental trend has been interpreted to reflect an increased commitment to mainstream social values that comes with the assumption of adult roles (Kandel & Logan, 1984). In fact, in our data, the lowest adult smoking prevalence rates were found for the oldest two cohorts who were 25 to 26 years of age at follow-up. Our data are consistent with those from studies of other substance use behaviors. Caution is warranted, however, in interpreting these age effects because other studies of young-adult smoking behavior have not found decreases in smoking rates before age 25 (Kandel & Logan, 1984) or between ages 25 and 28 (Johnston et al., 1987).

There were also important effects in terms of relative risk associated with different ages of smoking onset. As expected, those who initiated smoking at earlier ages were more likely to be smokers in young adulthood. Adolescents who initiate smoking at earlier ages may be particularly committed to this behavior and, thus, less likely to quit smoking. This age of onset effect is consistent with that reported for marijuana use (Brill & Christie, 1974) and for cigarette smoking through the high school years (Ershler et al., 1989).

The idea that more committed adolescent smokers continue on to adult smoking is also consistent with our finding that a stable pattern of adolescent smoking carries greater relative risk for adult smoking than does an erratic pattern. Even temporary periods of smoking abstinence in adolescence decrease relative risk for adult smoking. This finding supports
the importance of adolescent cessation programs that produce even temporary interruptions in the behavior. Perhaps even temporary decreases in commitment to smoking can discourage the persistence of smoking behavior.

Important unanswered questions in the current study concern the implications of these natural history data for building an underlying model of smoking behavior. Although we can describe the magnitude of risk that different patterns of adolescent smoking behavior carry for adult smoking, there are alternative causal models of smoking behavior that might fit the current data. The data are consistent with the notion of smoking as an addictive behavior such that greater levels of exposure, uninterrupted exposure, and more years of exposure carry the greatest risk for continuing the behavior. Other models are possible, however. For example, adolescent smoking would raise the risk for adult smoking if the same factors that served as motives for adolescent smoking simply continued on through adulthood. The idea that smoking is simply motivated by the same factors in adolescence and adulthood seems unlikely given theories about cigarette smoking and other adolescent “problem behaviors” as ways of attaining premature transitions to adult status (R. Jessor & S. L. Jessor, 1977). Motives for adolescent smoking such as peer pressure and rebelliousness would not be expected to be powerful motives for adult smoking onset. The possibility of stable, continuing smoking motives cannot be ruled out by the current data, however.

Finally, our pattern of findings would also be produced if different causal factors motivated adolescent and adult smoking, but only if each set of factors affected the same segment of the population. For example, adolescent smoking might be motivated by social and peer factors, whereas adult smoking might be motivated more by stress management and negative affect reduction. Because those who smoked as adolescents already have smoking in their repertoires as a coping strategy, however, adolescent smokers would be more likely to use smoking behavior to cope with negative affect. This model would also produce continuity between adolescent and adult smoking. Each of these three underlying models (smoking as addiction, stable causal factors over age, and age-specific factors that affect the same segment of the population) are consistent with the current data.

In sum, the current study is the first to allow a detailed examination of the magnitude of risk that adolescent smoking carries for adult smoking as a function of extent of exposure, age of onset, sex, and pattern of smoking initiation. Although caution is necessary when generalizing from this predominantly White, well-educated, young-adult sample, the findings were clear-cut. Adolescent cigarette smoking (even experimental smoking) represents a considerable risk factor for adult smoking. Moreover, the magnitude of this risk does not vary with age or sex, although both earlier
age of onset and a stable progression of smoking increase relative risk for adult smoking. These data support the importance of primary preventive interventions targeted at adolescent populations.

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