Brief Report: Social Risk Factors Predict Cigarette Smoking Progression Among Adolescents with Asthma

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Objective To compare smoking progression in adolescents with and without asthma and to compare their psychosocial risk factors. Methods Participants were 1,507 adolescents with asthma and 1,507 healthy matched controls from Waves I and II of the Add Health Project assessed at baseline and again 1 to 2 years later at follow-up. Three levels of smoking progression (defined as smoking more frequently and/or intensely over time) were identified: (a) Late Experimenters (never smokers at baseline, ever smokers at follow-up), (b) Early Experimenters (ever smokers at baseline, current/current frequent smokers at follow-up), and (c) Early Smokers (current smokers at baseline, current frequent smokers at follow-up). Results Twenty percent of adolescents experienced progression in their smoking behavior; those with and without asthma were equally likely to progress. Among adolescents who progressed, 37% were Late Experimenters, 42% were Early Experimenters, and 21% were Early Smokers. Exposure to friends who smoked was a consistent and powerful social risk factor for smoking progression among adolescents with asthma—more so than among adolescents without asthma. This effect was intensified among Late Experimenters by the presence of a positive history of parent smoking. Conclusions Findings underscore the importance of addressing cigarette smoking behavior and its social risk factors among adolescents with asthma in both clinical and public health contexts, during early adolescence, and through research on this topic.

Key words adolescents; asthma; psychosocial risk factors; smoking.

Cigarette smoking is harmful to all individuals, especially those affected by chronic illness. To date, the pediatric psychology literature has largely focused on cigarette smoking behaviors among healthy youth, with scant attention paid to youth with chronic conditions. The few studies that have emerged with a focus on youth with illnesses, including asthma (Tercyak, 2003; Zbikowski, Klesges, Robinson, & Alfano, 2002), diabetes (Frey, Guthrie, Loveland-Cherry, Park, & Foster, 1997; Tercyak, 2004), and survivors of childhood cancer (Emmons et al., 2002; Tyc, Hadley, & Crockett, 2001) suggest alarming similarities in smoking prevalence rates between adolescents with and without chronic illnesses.

Recently, Tercyak (2003) reported on the prevalence of smoking among adolescents with asthma who took part in the National Longitudinal Study of Adolescent Health (Add Health Project). There, the lifetime prevalence of smoking was 56% among adolescents with asthma and their prevalence of current smoking was 20%. No differences were observed between adolescents with and without asthma with respect to lifetime smoking prevalence, though adolescents with asthma were more likely to be current smokers than were their healthy counterparts. Psychosocial risk factors for smoking were highly similar between the two groups, and included exposure to family members and friends who smoked, as well as concurrent depression. These
results are in line with those obtained by Zbikowski et al. (2002), who also found high smoking prevalence rates among adolescents with asthma.

At question is whether cigarette smoking behavior progresses in adolescents with asthma over time, and whether psychosocial risk factors identified by Tercyak (2003) and Zbikowski et al. (2002) are predictive of progression in this population. Adolescent smoking progression (defined as smoking more frequently and/or intensely over time) has been the subject of a number of investigations (e.g., Dierker, Avenevoli, Goldberg, & Glantz, 2004; Turner, Mermelstein, & Flay, 2004; van den Bree, Whitmer, & Pickworth, 2004). Understanding the behavioral epidemiology of cigarette smoking among individuals with asthma is critical so that more effective smoking prevention and intervention programs can be designed and implemented. To address this issue, two successive waves of Add Health Project data were analyzed: Wave I data collected at baseline (1994–1995) and Wave II data collected at follow-up (1996). (Data for this study were obtained through a contractual arrangement with the proprietors of the Add Health Project Waves I and II data sets.) It was hypothesized that adolescents with asthma would progress in their smoking behavior at a rate similar to their healthy peers. Further, that psychosocial risk factors for smoking identified at baseline (exposure to parents' and friends' smoking, depression) would predict smoking progression in adolescents with asthma as powerfully as in adolescents without asthma.

Method
Among the more than 90,000 adolescents enrolled in the Add Health Project, some 20,000 adolescents and their parents completed extensive in-home interviews about adolescent health and well-being (Udry & Bearman, 1998). Taking an hour or more to complete, these interviews were individually administered in person in adolescents’ homes. Special care was taken to assure participants that their responses would remain confidential. For example, on sensitive topics adolescents listened to tape-recorded questions and entered their answers directly into a laptop computer.

Participants
Wave I
During the Wave I parent in-home interview, parents reported on a number of parent-, child-, and family-specific factors, including if the child currently experiences the health problem of asthma or emphysema (yes/no). On the basis of parents’ responses to this question, 2,073 affected adolescents were identified. (The in-home interview did not distinguish between asthma and emphysema as different forms of chronic obstructive pulmonary disease. However, it is assumed that affected adolescents were diagnosed with asthma and not emphysema as emphysema usually develops in adulthood, often after many years of exposure to cigarette smoke and other air pollutants.)

Adolescents included in the control sample were randomly selected via a computer program from the Wave I data set after the cases of adolescents with asthma had been identified. The computer program sought to exactly match controls to cases on four vital statistics: (a) gender (male or female), (b) race (Hispanic, Black or African American, Asian, Native American, Other, White), (c) age (11–21 years), and (d) the U.S. census region in which the school the adolescent attended lies (west, midwest, south, northeast), respectively. A total of 2,039 out of 2,051 cases (99%) could be matched in this manner. Limitations in the Add Health Project data set prevented a complete determination of the health status of these individuals, other than that they did not have asthma. However, adolescents with diabetes (n = 78) could be excluded before the selection of the sample of controls as the Wave I in-home parent interview asked parents to identify whether their adolescent was affected with diabetes.

Wave II
Cases accessioned in the manner described above were then screened to determine whether they also had relevant data (i.e., smoking behavior data) at Wave II. Of the 2,039 cases, 1,507 (74%) answered a critical tobacco use screening item regarding lifetime cigarette use since the date the Wave I interview was conducted. There were no discernible demographic differences at baseline between adolescents who could or could not be included in the longitudinal analysis. Only these 1,507 cases and their matched controls were retained for analysis.

Procedure
In the Add Health Project, the sampling frame included all schools in the United States meeting certain eligibility criteria (N = 26,666). A systematic random sample of 80 high schools and 52 middle schools was then selected with unequal probability of selection for participation. Incorporating systematic sampling methods and implicit stratification into the Add Health Project design ensured that the sample was representative of U.S. schools with respect to region of country, urbanicity, school type, ethnicity, and school size. Overall, 79% of schools agreed to participate (Resnick et al., 1997).
Measures
The following variables/scales were extracted/created from the Waves I and II adolescent in-home interviews (Tercyak, 2003).

Wave I: Environmental Smoking Exposure
Two items assessing resident mother and resident father lifetime cigarette smoking (yes/no), and one item assessing the number of best friends who smoke at least one cigarette per day (none, one, two, or three), were included in the analysis.

Wave I: Depression Symptoms
Resnick et al. (1997) created a reliable (Cronbach $\alpha = .86$) multi-item scale of psychological distress/depression symptoms over the past week for the Add Health Project data set. This 19-item 4-point Likert scale (0, “never or rarely”; 1, “sometimes”; 2, “a lot of the time”; 3, “most of the time or all of the time”) is based, in large part, on the Center for Epidemiologic Studies-Depression Scale, which is commonly used with adolescents (Myers & Winters, 2002); higher item-summed scores indicate greater depression symptoms. Sample items include “You felt depressed” and “You felt sad.” To ease the interpretability of this variable, a median-split procedure determined those adolescents with depression symptoms falling at or above the median (“high”) from those with depression symptoms falling below the median (“low”).

Waves I and II: Cigarette Smoking
Adolescent cigarette smoking was assessed by a series of standard epidemiological items regarding the frequency and intensity of their smoking behavior/lifetime and current cigarette smoking. These items were “Have you ever tried cigarette smoking, even just one or two puffs?” and “During the past 30 days, on how many days did you smoke cigarettes?”

For the purposes of the data analysis, three cigarette smoking dependent variables were created from Wave I: lifetime cigarette use (ever tried cigarette smoking, even one or two puffs), current cigarette use (smoked cigarettes on 1 or more of the past 30 days), and current frequent cigarette use (smoked cigarettes on 2 or more of the past 30 days). Definitions for each of these variables were adapted from the Centers for Disease Control and Prevention’s Youth Risk Behavior Surveillance System (Grunbaum et al., 2004). At Wave II, identical variables were created and analyzed. However, these variables were specific to the frequency and intensity of smoking behaviors that had occurred since the date of the Wave I interview.

Data Analysis
The sample’s baseline demographic characteristics, and smoking exposure and depression variables (independent psychosocial risk predictor variables) were described in univariate fashion, stratified by illness status. Equivalency between the groups of adolescents with and without asthma was confirmed with bivariate statistics. Next, three groups of adolescent cigarette smokers, distinguished by their smoking frequency and intensity, were created. Group I, labeled “Late Experimenters,” consisted of adolescents with a negative history of lifetime smoking at baseline who progressed to a positive lifetime history by follow-up. Group II, labeled “Early Experimenters,” was comprised of adolescents with a positive history of lifetime smoking at baseline who progressed to current smoking or current frequent smoking by follow-up. And Group III, labeled “Early Smokers,” was made up of adolescents who were currently smoking at baseline who progressed to current frequent smoking at follow-up. Bivariate analyses then tested for differences in smoking progression (dependent variable) based on the independent variables of interest; those with significant ($p < .05$) associations with progression were then tested in a multivariate fashion by using logistic regression. These adjusted equations generate odds ratios (OR) (with 95% confidence intervals [CI]) estimating each variable’s influence on smoking behavior progression 1 to 2 years later.

Results
Univariate and Bivariate Analyses
The sample’s M (SD) age was 15.1 (1.6) years (16.1 [1.6] years at follow-up). There were no differences in the distributions of gender (51% male), race (54% White), or region (25% northeast, 26% midwest, 32% south, 17% west) between adolescents with and without asthma; their ages at follow-up were also comparable. Thus, the case-control matching procedure was successful.

Regarding cigarette smoking, 596 adolescents (20%) experienced progression in the frequency and/or intensity of their smoking behavior from baseline to follow-up, 671 (22%) experienced regression, and 1,747 (58%) were unchanged. Adolescents with and without asthma were equally likely to experience progression (Table I), regression, or to remain unchanged. Within the group of adolescents who experienced progression, 219 (37%) fell into Group I and were Late Experimenters, 252 (42%) fell into Group II and were Early Experimenters, and 125 (21%) fell into Group III and were Early Smokers.
Among Late Experimenters (Group I) with asthma, male adolescents, those exposed to parents who smoked, and those exposed to friends who smoked were more likely to progress. Specifically, males were 82% more likely to progress than females, adolescents exposed to parents who smoked were 65% more likely to progress, and adolescents exposed to friends who smoked were 63% more likely to progress than were adolescents without those exposures. Among Late Experimenters (Group I) without asthma, only the effect of baseline exposure to friends who smoked was significant, OR = 1.91, 95% CI = 1.21, 2.99, p = .005.

For both Early Experimenters (Group II) and Early Smokers (Group III) with asthma, exposure at baseline to friends who smoked was associated with their smoking progression. For Early Experimenters, those with friends' exposure were 92% more likely to progress. For Early Smokers, those with friends' exposure were over 2 times more likely to progress. No variables were associated with smoking progression among adolescents without asthma in Groups II and III.

**Multivariate Analysis**

Because bivariate analyses suggested that more than one independent variable was associated with progression in smoking among Late Experimenters with asthma only, a multiple logistic regression analysis examining the effects of exposure to parents and friends who smoked (controlling for adolescent gender) was conducted. Both main effects and their interaction term were tested. The results indicated that adolescents with exposure from parents were 65% more likely to progress (OR = 1.65, 95% CI = 1.07, 2.54, p = .03) and adolescents with exposure from friends were 58% more likely to progress (OR = 1.58, 95% CI = 1.02, 2.44, p = .04); adolescents with exposure to both parents' and friends' smoking were the most likely to progress (p = .01). Unadjusted follow-up contingency tables revealed 10.4% of Late Experimenters progressed when exposed to parent smoking only, 5.4% progressed when exposed to friends smoking only, and 17.4% progressed when exposed to both risk factors at baseline.

**Discussion**

The results of this study suggest that adolescents with asthma were just as likely as adolescents without asthma to progress in their smoking behavior over a 1- to 2-year interval beginning in midadolescence. This appeared to be true regardless of whether or not adolescents with asthma had a negative or positive lifetime history of smoking progression.

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**Table I. Smoking Progression Among Adolescents with Asthma**

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Variable present</th>
<th>Variable absent</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All (n = 1,507)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male gender</td>
<td>767</td>
<td>20.3</td>
<td>18.1</td>
<td>1.15</td>
<td>ns</td>
</tr>
<tr>
<td>White race</td>
<td>817</td>
<td>20.1</td>
<td>18.3</td>
<td>1.12</td>
<td>ns</td>
</tr>
<tr>
<td>Parents smoke</td>
<td>1,033</td>
<td>21.2</td>
<td>15.0</td>
<td>1.53</td>
<td>1.14–2.05</td>
</tr>
<tr>
<td>Friends smoke</td>
<td>634</td>
<td>21.3</td>
<td>17.8</td>
<td>1.25</td>
<td>ns</td>
</tr>
<tr>
<td>High depression</td>
<td>836</td>
<td>19.9</td>
<td>18.5</td>
<td>1.09</td>
<td>ns</td>
</tr>
<tr>
<td>Group I (n = 684)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male gender</td>
<td>356</td>
<td>21.1</td>
<td>12.8</td>
<td>1.82</td>
<td>1.20–2.74</td>
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<tr>
<td>White race</td>
<td>343</td>
<td>17.5</td>
<td>16.7</td>
<td>1.06</td>
<td>ns</td>
</tr>
<tr>
<td>Parents smoke</td>
<td>421</td>
<td>19.7</td>
<td>12.9</td>
<td>1.65</td>
<td>1.07–2.55</td>
</tr>
<tr>
<td>Friends smoke</td>
<td>167</td>
<td>22.8</td>
<td>15.3</td>
<td>1.63</td>
<td>1.06–2.52</td>
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<tr>
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<td>322</td>
<td>18.9</td>
<td>15.5</td>
<td>1.28</td>
<td>ns</td>
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<td>Group II (n = 202)</td>
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<td></td>
<td></td>
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<tr>
<td>Male gender</td>
<td>111</td>
<td>54.1</td>
<td>58.2</td>
<td>0.84</td>
<td>ns</td>
</tr>
<tr>
<td>White race</td>
<td>110</td>
<td>57.3</td>
<td>54.4</td>
<td>1.13</td>
<td>ns</td>
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<tr>
<td>Parents smoke</td>
<td>147</td>
<td>58.5</td>
<td>49.1</td>
<td>1.46</td>
<td>ns</td>
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<tr>
<td>Friends smoke</td>
<td>81</td>
<td>65.4</td>
<td>49.6</td>
<td>1.92</td>
<td>1.08–3.44</td>
</tr>
<tr>
<td>High depression</td>
<td>115</td>
<td>59.1</td>
<td>51.7</td>
<td>1.35</td>
<td>ns</td>
</tr>
<tr>
<td>Group III (n = 141)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male gender</td>
<td>55</td>
<td>38.2</td>
<td>45.4</td>
<td>0.74</td>
<td>ns</td>
</tr>
<tr>
<td>White race</td>
<td>88</td>
<td>46.6</td>
<td>35.9</td>
<td>1.56</td>
<td>ns</td>
</tr>
<tr>
<td>Parents smoke</td>
<td>109</td>
<td>45.9</td>
<td>31.3</td>
<td>1.86</td>
<td>ns</td>
</tr>
<tr>
<td>Friends smoke</td>
<td>89</td>
<td>49.4</td>
<td>30.8</td>
<td>2.20</td>
<td>1.07–4.52</td>
</tr>
<tr>
<td>High depression</td>
<td>88</td>
<td>42.1</td>
<td>43.4</td>
<td>0.95</td>
<td>ns</td>
</tr>
</tbody>
</table>

Group I: Adolescents with a negative history of lifetime smoking at baseline who progressed to a positive lifetime history by follow-up (Late Experimenters); Group II: Adolescents with a positive history of lifetime smoking at baseline who progressed to current smoking or current frequent smoking by follow-up (Early Experimenters); Group III: Adolescents who were currently smoking at baseline who progressed to current frequent smoking at follow-up (Early Smokers)

**Independent Predictor Variables Associated with Smoking Progression**

Analyses of independent psychosocial risk predictor variables associated with smoking progression are presented in Table I and in the text below.

Of the five variables tested, only exposure to parents who smoked was significantly associated with any level of progression among adolescents with asthma; those exposed to parents who smoked were 50% more likely to progress than were unexposed adolescents. Among adolescents without asthma, race and exposure to friends who smoked were associated with any progression. Specifically, White adolescents were 40% more likely to progress than were nonWhite adolescents, OR = 1.40, 95% CI = 1.08, 1.81, p = .01 and adolescents exposed to friends who smoked were almost 50% more likely to progress than were unexposed adolescents, OR = 1.47, 95% CI = 1.15, 1.90, p = .003.
smoking at baseline, or were currently smoking at baseline. Further, the results indicated that among adolescents with asthma, exposure at the beginning of this interval to friends' smoking was a significant and consistent social risk factor for smoking progression at three different levels of personal smoking behavior. This effect was intensified at the early stage of smoking experimentation by the presence of exposure to parents who smoked.

The results are consistent with prior cross-sectional work with adolescents with asthma suggesting social factors such as exposure to smoking among family members and friends may place adolescents at risk for smoking (Tercyak, 2003; Zbikowski et al., 2002). They are also consistent with longitudinal research findings that these risk factors exert an influence over time on the uptake and progression of adolescent smoking behavior (Chassin, Presson, Rose, & Sherman, 1996; Choi, Pierce, Gilpin, Farkas, & Berry, 1997). This study's unique contribution is in both the identification and replication of exposure to friends' smoking as a social risk factor for smoking progression among adolescents with asthma, and its synergistic interaction effect with exposure to parents' smoking on experimentation. This study did not demonstrate any effect of depression symptoms on smoking progression over time. It is unclear why social factors predicted cigarette smoking more consistently and powerfully among adolescents with asthma than among adolescents without asthma. One possible explanation as to why this may be the case draws upon previous findings suggesting adolescents with asthma have more positive attitudes toward cigarettes and smoking, stronger intentions to smoke, and positive self-image of smokers (Brook & Shiloh, 1993; Zbikowski et al., 2002). Another is that adolescents with asthma may more readily succumb to social influences, including offers from friends to smoke, to fit in with their peer group and avoid stigmatization as being different because of chronic illness (Zbikowski et al., 2002).

The results reported should be interpreted in light of a few limitations. First, changing patterns in the prevalence of cigarette smoking among U.S. high school students since the time these data were collected in the mid 1990s may limit the relevance of the findings. Second, adolescents' self-reported cigarette smoking status was not independently confirmed via biochemical or other means and may have been subject to biased reporting. Similarly, parental smoking history was not well detailed and it is possible that parents stopped smoking before the adolescent's birth. Third, the Add Health Project offers relatively crude assessments of smoking progression and depression; varying levels of smoking experimentation were not covered and clinical cut-offs for depression are unavailable. Finally, complete case ascertainment was not possible at Wave II because of missing data.

These limitations notwithstanding, the findings underscore the importance of addressing the problem of smoking among adolescents with asthma. Clinically, health care providers should routinely screen adolescents and their parents for tobacco use and offer them education, counseling, and assistance. Overt signs of smoking, such as the smell or presence of cigarettes, are also cues to action for providers. Providers should be aware of the social influences on adolescent smoking progression too and inquire about smoking among adolescents' peers. Enhancing self-efficacy may be key, along with providing social skills and cigarette refusal skills training, to reduce use and prevent or delay smoking progression among adolescents with asthma (Borrelli et al., 2002). From a public health standpoint, effective ways to reach adolescents with asthma or other chronic illnesses and deliver and tailor smoking prevention and cessation messages to this special population are not known. More research in these contexts seem warranted.

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References


