Nearly all Americans now recognize that smoking causes lung cancer and other serious diseases; however, cigarette smoking has not yet been eradicated in this country. This might be taken as evidence that beliefs about the health risks of smoking do not influence smoking onset or quitting. In this paper, we report new evidence that perceiving smoking to entail greater health risks reduces the likelihood that a young person will begin to smoke. This evidence suggests that public health campaigns should continue to focus on this theme to bolster resistance to smoking onset among young people.
choose not to try to quit, and why do young people continue to start smoking at alarming rates?

One possible answer to this question is that beliefs about the health risks of smoking do not motivate people to protect themselves by avoiding cigarette use. Instead, perhaps smoking onset and cessation are driven by a host of other factors, including peer modeling, parental modeling, sibling modeling, the subjective norms created by parents, and more. Interestingly, remarkably little scientific work has explored the question of whether beliefs about the health risks of smoking actually influence behavior. We help to redress this shortcoming of the literature by offering new evidence here on this issue.

We begin below by describing the findings of past representative sample survey studies assessing Americans’ perceptions of the health risks of smoking. Then, we review the available evidence on the causal impact of health beliefs on smoking onset. To complement this evidence, we report the results of new statistical analyses of longitudinal survey data gauging the impact of health risk beliefs on smoking onset among adolescents. Taken together, this work offers a justification for continued efforts to promote public recognition of the health risks of smoking.

Prior survey studies of public perceptions of the health risks of smoking

Many surveys of nationally representative samples of American adults have been conducted during the past 5 decades on perceptions of smoking and health issues, sponsored by government agencies, private organizations dedicated to public health promotion, and tobacco companies. These studies indicate that during the 1950s, large portions of Americans failed to assert that smoking had health risks, and this fraction has fallen precipitously. Nonetheless, even today, nontrivial portions of the American public do not recognize that smoking is risky.

One relevant series of surveys was conducted by the Gallup Organization and asked respondents, “Do you think that cigarette smoking is or is not one of the causes of lung cancer?” A second question asked respondents whether smoking is or is not one of the causes of heart disease. A third question asked about throat cancer. In another series of surveys, the Gallup Organization asked more general questions, not mentioning specific diseases: “Do you think cigarette smoking is or is not harmful to your health?” or “Do you think cigarette smoking is harmful, or not?” The proportions of respondents failing to say that smoking causes a specific disease or that smoking is risky are plotted in Figure 1.

Three principal patterns are apparent in these data. First, all four trend lines manifest consistent decreases over time, meaning that Americans increasingly embraced the views of health professionals on these issues. Second, none of the trend lines reach zero, meaning that a relatively small group of holdouts continued to resist the view of smoking as entailing health risks. And third, the most general view of risk has been accepted more widely than any specific risk. That is, fewer people denied all
health risks than denied risks specific to lung cancer, heart disease, or throat cancer. Thus, there is still progress to be made in educating the public about these specific risks. And according to these figures, more Americans accept the risk of lung cancer than accept the risk of heart disease.

The same finding was echoed in a 1993 survey done by the Gallup Organization and sponsored by SmithKline Beecham, and this survey identified health effects of smoking that were denied by even larger groups. Respondents were asked, “Does smoking cause or make these conditions worse?”, and respondents were then read a list of medical problems. Eighty-four percent of respondents answered “yes” for lung cancer, and 75% answered “yes” for heart disease, comparable to the surveys described above. In addition, 66% answered “yes” for oral cancers and 65% answered “yes” for stroke. Thus, 16%, 25%, 34%, and 35% of respondents, respectively, failed to acknowledge these health effects of smoking.

Recent surveys suggest that the percentages of people denying health risks have continued to be small but not zero after the ends of the time series shown in Figure 1. For example, in 2001, the Gallup Organization asked a national sample, “Do you feel that cigarette smoking is a major cause of lung cancer, a minor cause, or that science

Figure 1 Proportions of Americans who failed to assert that smoking is dangerous to human health: Gallup Organization surveys.
hasn’t yet been able to tell just what the relation is between cigarette smoking and lung cancer?'' Eighteen percent of respondents said that either “science hasn’t been able to tell” or “don’t know” or “refused.” Likewise, two surveys done by Harris Interactive by telephone in 2000 and 2001 found 11% and 12% of respondents, respectively, saying “no” or “don’t know” in response to more personalized forms of the question: “Do you believe that smoking increases your risk of getting lung cancer?” or “Do you believe that smoking increases your risk of getting lung cancer or not.”

Are these beliefs behaviorally consequential? Should any effort be devoted to further public education to press the proportions of Americans denying these risks to zero?

The effects of beliefs about the health consequences of smoking on smoking onset

Review of existing evidence

If health beliefs are causes of smoking onset, then people who smoke should, on average, believe that smoking is less risky than people who do not smoke. And indeed, many studies show that, as compared to people who do not smoke cigarettes, people who do smoke are less likely to believe that smoking causes health problems for people in general (e.g., Bauman & Chenoweth, 1984; Benthin, Slovic, & Severson, 1993; Grønhaug & Kangun, 1979; Halpern & Warner, 1994; Hansen & Malotte, 1986; Harrison et al., 1996; Loken, 1982; Murray, Prokhorov, & Harty, 1994; Reppucci, Revenson, Aber, & Reppucci, 1991; Swinehart, 1966; Wang, Fitzhugh, Cowdery, & Trucks, 1995; Williams & Clarke, 1997; for a review, see Weinstein, 2001). And Chassin, Corty, Presson, Olshavsky, Bensenberg, and Sherman (1981) and Presson et al. (1984) found that people who believed that smoking has less adverse impact on health were more likely to say they intended to smoke cigarettes in the future. Only four studies found no difference between smokers and nonsmokers in the extent to which they believed smoking causes undesirable health effects (Burns & Williams, 1995; Grube, McGree, & Morgan, 1986; McKenna, Warburton, & Winwood, 1993; Schneider & Vanmastrigt, 1974).

Weinstein (1980, 1982, 1987, 1998) reported that people typically believe that they are at less risk personally of experiencing an undesirable life circumstance than are other people. This highly robust finding makes clear the importance of recognizing the distinction between people’s beliefs about the health risks of smoking to people in general and their beliefs about the health risks of smoking for themselves personally. A series of studies show that smokers are less likely to believe that smoking causes undesirable health effects for them personally than are nonsmokers (Benthin et al., 1993; Grønhaug & Kangun, 1979; Hansen & Malotte, 1986; Leventhal, Glynn, & Fleming, 1987; Marshall, 1990; Murray et al., 1994; Urberg & Robbins, 1984; Virgili, Owen, & Severson, 1991). These findings suggest that personalized
health beliefs may partly determine smoking behavior. Also consistent with this logic, Levitt (1971) and Kahn and Edwards (1970) found that the most frequently given reason by nonsmokers to not smoke was to avoid the undesirable health effects.

Even if a person is completely convinced that smoking substantially increases his or her risk of experiencing health problems, this belief may have no impact on his or her smoking behavior if he or she attaches no personal importance to those health effects (see, e.g., Petty & Krosnick, 1995). Consistent with this logic, Mettlin (1973) reported that people who attached more importance to the effects of smoking on health were indeed less likely to smoke.

One very robust finding at first appears to challenge the general conclusion that beliefs about the health effects of smoking are partial causes of smoking behavior. Gerrard, Gibbons, Benthin, and Hessling (1996), Harrison et al. (1996), Lee (1989), McKenna et al. (1993), McMaster and Lee (1991), Strecher, Kreuter, and Kobrin (1995), Weinstein (1987), and Williams and Clarke (1997) found that smokers were more likely than nonsmokers to say that they themselves would get a smoking-related disease during their lifetimes. Likewise, Hurd and McGarry (1995) and Schoenbaum (1997) reported that smokers were less likely to believe they would live to ages 75 and 85 than nonsmokers said of themselves. And Greening and Dollinger (1991) showed that smokers said “a person like me” was more likely to die of cancer, stroke, or emphysema than did nonsmokers. At first glance, these results seem to conflict with results showing that smokers think the health risks of smoking are less than nonsmokers do.

But it is important to recognize that the questions used to measure beliefs about likelihood of experiencing illnesses and life expectancy in these studies did not mention cigarettes or smoking. That is, these questions did not measure people’s perceptions of the impact of smoking on their own health or likelihood of death. And it turns out that smokers do not in fact see themselves to be more likely to experience smoking-related diseases uniquely. Instead, smokers perceive themselves to be more likely than nonsmokers perceive themselves to experience a wide range of undesirable physical conditions, including ones clearly unrelated to smoking, for example, getting the flu (Swinehart, 1966), having “an accident” or developing arthritis (Harrison et al., 1996), or become sterile, getting venereal disease, and developing cirrhosis of the liver (McKenna et al., 1993).

It appears that these perceptions are grounded in observable real-life events and general risk factors. For example, smokers report having had more recent hospitalizations, more recent visits to doctors, more chronic health conditions, and more restricted physical activity than do nonsmokers (Halpern & Warner, 1994). And smokers perceive themselves as having higher levels of risk factors other than smoking. For example, Reppucci et al. (1991) found that smokers reported experiencing more stress than did nonsmokers. Thus, although the rates at which smokers report experiencing health problems and expect to experience health problems exceed the rates reported by nonsmokers, this is not confined to smoking-related health problems.
Ambiguity in this evidence
Most of the evidence just reviewed is consistent with the claim that beliefs about the health risks of smoking are determinants of smoking onset. Most important among this evidence is the correlation between health beliefs and smoking behavior, which is consistent with the notion that the former cause the latter. However, there is a theoretical basis for expecting that at least some of the correlation between health beliefs and smoking behavior is not in fact due to causal impact of beliefs on behavior. Rather, this correlation may be due to post hoc rationalization of smoking behavior. As Festinger (1957) argued, cigarette smokers are likely to find it very uncomfortable to hold simultaneously the beliefs that they smoke cigarettes regularly and that this behavior is damaging to their health. One way to reduce the discomfort associated with holding these beliefs simultaneously is to deny or underestimate the health risks of smoking.

Festinger’s (1957) idea may partly explain the robust correlation observed between smoking/nonsmoking status and belief in the health effects of smoking. However, evidence reported by McMaster and Lee (1991) suggests that smokers may reduce this cognitive dissonance by a different cognitive mechanism that Festinger also identified: downplaying the importance of the health risk by believing that health problems caused by smoking can be caught early and cured and that other risks posed in life are more threatening. Likewise, Loken (1982) reported that smokers perceived the health consequences of smoking to be less undesirable than did nonsmokers. Thus, it appears that smokers cope with their cognitive dissonance at least partly by this mechanism, though they may also strategically downplay their perceptions of the likelihood of undesirable health consequences following from smoking.

Regardless of whether or how smokers cope with their cognitive dissonance, it is also possible that preexisting differences between smokers and nonsmokers in their beliefs about the health consequences of smoking may partly determine whether these individuals subsequently become smokers or not. To test this directly, data should be collected over time from a group of nonsmokers, initially measuring their beliefs about the health effects of smoking, and then assessing whether these beliefs predict who later become smokers. This would be strong evidence of causal impact of one factor on another (Finkel, 1995; Kessler & Greenberg, 1981).

Some studies reported that people who initially believed smoking was more dangerous to their health were more likely to initiate smoking later (e.g., Bauman & Chenoweth, 1984; Chassin, Presson, Pitts, & Sherman, 2000). But other studies produced evidence of such lagged effects in some tests but not others (e.g., Chassin, Presson, Sherman, & Edwards, 1991), and still other investigations found no impact of health beliefs on subsequent onset (Collins et al., 1987).

However, the results of those studies are problematic for a number of reasons. Most importantly, all studies’ measures of respondents’ beliefs about the health consequences of smoking did not assess the perceived increase in risk due to smoking. For example, Collins et al. (1987) averaged responses to three questions asking,
“If you smoke cigarettes, will you get lung disease?”, “If you smoke cigarettes, will you have heart trouble?”, and “If you smoke cigarettes, will you be out of breath?” Although these items may be viewed as assessing the perceived probabilities of experiencing various health problems given smoking, no measures were taken of the perceived probabilities of those health problems given not smoking, so these measures did not permit the assessment of the perceived increase in risk associated with smoking. All the measures of health beliefs analyzed by Bauman and Chenoweth (1984) were similarly worded, as were most of the measures of health beliefs used by Chassin et al. (1991, 2000; e.g., “If I smoke cigarettes, I will live a long time.”). Given the wording of these items, their associations with subsequent smoking onset could be attributable to an acquiescent response bias (yielding reports of higher perceived probability of undesirable health outcomes) or general life pessimism (which would yield more pessimistic answers to all questions, regardless of whether they were specifically addressed to smoking or not).

Other items used in these investigations to measure health beliefs did not explicitly and specifically ask about perceptions of the health effects of smoking at all (e.g., “If you are young and healthy, cigarette smoking is not dangerous.” or “The anti-smoking ads twist the facts to make cigarette smoking look worse for your health than it really is.”). And the measure of smoking onset used by Collins et al. (1987) was computed by standardizing and averaging responses to a variety of questions asking about how many cigarettes one has smoked in his or her lifetime, how long it has been since the respondent last smoked a cigarette, how long it will be before he or she thinks he or she might smoke again, how much he or she currently smokes, and more. Thus, it is difficult to interpret analyses predicting this measure as offering straightforward empirical assessments of the predictors of smoking onset.

New longitudinal evidence on health beliefs and smoking onset

Overview
In light of the ambiguities in existing longitudinal evidence, we revisited the surveys conducted by Chassin et al. (2000) and (a) built a measure of health beliefs using questions that explicitly gauged perceptions of the causal impact of smoking on health and controlled for acquiescence response bias, (b) used a measure of the value respondents placed on health to test interactions between beliefs about the health effects of smoking and value placed on health (e.g., Fishbein & Ajzen, 1975), (c) built an array of measures of other suspected causes of smoking onset among adolescents, and (d) used longitudinal panel data to assess the impact of health beliefs on smoking onset controlling for other potential causes.

Each year from 1980 through 1983, 6th to 12th graders attending public schools in a midwestern county completed self-administered questionnaires in school (for details on the data collection, see Chassin, Presson, Sherman, & Edwards, 1990; Rose, Chassin, Presson, & Sherman, 1996). Each questionnaire included a wide array of
measures, tapping smoking status as well as many potential predictors of it (for question wordings and coding, see Appendix A).

**Analysis strategy**
The data set allowed us to identify the predictors of smoking onset during three time intervals: between Wave 1 (1980) and Wave 2 (1981), between Wave 2 and Wave 3 (1982), and between Wave 3 and Wave 4 (1983). Therefore, for each of the first three waves, we created a set of variables representing the posited antecedent conditions of smoking onset between that wave and the next one. We focused only on respondents who were nonsmokers at the time of an initial wave in an adjacent pair, so we predicted smoking onset; respondents who were smokers at an initial wave were dropped from these analyses. The data from the three adjacent wave pairs were stacked to yield one large data set.¹

A total of 2,264 respondents provided suitable data for only one of the three wave pairs (e.g., people who were nonsmokers at two consecutive waves and provided no data for the third or fourth waves; people who were nonsmokers in 1980, became smokers in 1981, and remained smokers in 1982 and 1983). Another 1,155 respondents provided data for two wave pairs (e.g., people who were nonsmokers in 1980 and 1981 and were smokers in 1982 and 1983). And another 752 respondents provided data for all three wave pairs (e.g., people who were nonsmokers in 1980, 1981, and 1982, and became smokers in 1983).

Logistic regressions were conducted separately for each gender, predicting smoking onset with variables measured in the first wave of the adjacent wave pair: a main effect of health beliefs, the interaction between health beliefs and value placed on health, main effects of friends’ smoking, friends’ subjective norm, motivation to comply with friends, parents’ smoking, parents’ subjective norm, motivation to comply with parents, older siblings’ smoking, dummy variables for absent mother, absent father, and absent older siblings, and age of the respondent. Some two-way interactions were also tested: Friends’ Smoking × Motivation to Comply With Friends, Friends’ Subjective Norm × Motivation to Comply With Friends, Parents’ Smoking × Motivation to Comply With Parents, and Parents’ Subjective Norm × Motivation to Comply With Parents. Of these interactions, only Parents’ Smoking × Motivation to Comply With Parents exerted a significant effect on smoking onset. All the other interaction terms were not significant predictors of smoking onset and were therefore excluded from the regressions we report.²

**Results**
The first two columns of Table 1 show parameter estimates for regressions conducted separately with boys and girls. The last column shows parameter estimates for a regression using both genders combined together, testing the significance of coefficient differences between the genders. Specifically, the last 19 rows of coefficients test interactions of gender with each of the other predictors in the model.
### Table 1  Logistic Regression Coefficients Predicting Smoking Onset in Boys and Girls

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Boys</th>
<th>Girls</th>
<th>Model with interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Friends’ smoking</td>
<td>4.87**</td>
<td>4.20**</td>
<td>4.20**</td>
</tr>
<tr>
<td>Friends’ subjective norm</td>
<td>2.62**</td>
<td>4.49**</td>
<td>4.49**</td>
</tr>
<tr>
<td>Mother’s smoking</td>
<td>0.07</td>
<td>-0.09</td>
<td>-0.09</td>
</tr>
<tr>
<td>Mother’s Smoking × Motivation to Comply With Parents</td>
<td>1.53</td>
<td>1.57*</td>
<td>1.57*</td>
</tr>
<tr>
<td>Father’s smoking</td>
<td>-0.03</td>
<td>0.07</td>
<td>0.07</td>
</tr>
<tr>
<td>Father’s Smoking × Motivation to Comply With Parents</td>
<td>0.35</td>
<td>1.60*</td>
<td>1.60*</td>
</tr>
<tr>
<td>Parents’ subjective norm</td>
<td>-.68</td>
<td>0.55</td>
<td>0.55</td>
</tr>
<tr>
<td>Older brother’s smoking</td>
<td>1.05**</td>
<td>0.07</td>
<td>0.07</td>
</tr>
<tr>
<td>Older sister’s smoking</td>
<td>-.32</td>
<td>0.59*</td>
<td>0.59*</td>
</tr>
<tr>
<td>Health beliefs</td>
<td>1.81*</td>
<td>-1.68</td>
<td>-1.68</td>
</tr>
<tr>
<td>Value placed on health</td>
<td>0.24</td>
<td>0.84</td>
<td>0.84</td>
</tr>
<tr>
<td>Health Beliefs × Value Placed on Health</td>
<td>0.53</td>
<td>3.46*</td>
<td>3.46*</td>
</tr>
<tr>
<td>Motivation to comply with parents</td>
<td>-1.78*</td>
<td>-1.75*</td>
<td>-1.75*</td>
</tr>
<tr>
<td>Motivation to comply with friends</td>
<td>0.19</td>
<td>-0.42</td>
<td>-0.42</td>
</tr>
<tr>
<td>No mother</td>
<td>-0.55</td>
<td>1.47*</td>
<td>1.47*</td>
</tr>
<tr>
<td>No father</td>
<td>-0.15</td>
<td>0.17</td>
<td>0.17</td>
</tr>
<tr>
<td>No older brother</td>
<td>0.22</td>
<td>0.42</td>
<td>0.42</td>
</tr>
<tr>
<td>No older sister</td>
<td>-0.07</td>
<td>0.12</td>
<td>0.12</td>
</tr>
<tr>
<td>Age of respondent</td>
<td>0.79*</td>
<td>2.18**</td>
<td>2.18**</td>
</tr>
<tr>
<td>Gender of respondent</td>
<td>—</td>
<td>—</td>
<td>2.16*</td>
</tr>
<tr>
<td>Gender × Friends’ Smoking</td>
<td>—</td>
<td>—</td>
<td>0.66</td>
</tr>
<tr>
<td>Gender × Friends’ Subjective Norm</td>
<td>—</td>
<td>—</td>
<td>-1.87*</td>
</tr>
<tr>
<td>Gender × Mother’s Smoking</td>
<td>—</td>
<td>—</td>
<td>0.16</td>
</tr>
<tr>
<td>Gender × Mother’s Smoking × Motivation to Comply With Parents</td>
<td>—</td>
<td>—</td>
<td>-0.04</td>
</tr>
<tr>
<td>Gender × Father’s Smoking</td>
<td>—</td>
<td>—</td>
<td>-0.09</td>
</tr>
<tr>
<td>Gender × Father’s Smoking × Motivation to Comply With Parents</td>
<td>—</td>
<td>—</td>
<td>-1.25</td>
</tr>
<tr>
<td>Gender × Parents’ Subjective Norm</td>
<td>—</td>
<td>—</td>
<td>-1.23</td>
</tr>
<tr>
<td>Gender × Older Brother’s Smoking</td>
<td>—</td>
<td>—</td>
<td>0.98*</td>
</tr>
<tr>
<td>Gender × Older Sister’s Smoking</td>
<td>—</td>
<td>—</td>
<td>-0.91*</td>
</tr>
<tr>
<td>Gender × Health Beliefs</td>
<td>—</td>
<td>—</td>
<td>3.49*</td>
</tr>
<tr>
<td>Gender × Value Placed on Health</td>
<td>—</td>
<td>—</td>
<td>-0.60</td>
</tr>
<tr>
<td>Gender × Health Beliefs × Value Placed on Health</td>
<td>—</td>
<td>—</td>
<td>-2.92*</td>
</tr>
<tr>
<td>Gender × Motivation to Comply With Parents</td>
<td>—</td>
<td>—</td>
<td>-0.03</td>
</tr>
<tr>
<td>Gender × Motivation to Comply With Friends</td>
<td>—</td>
<td>—</td>
<td>0.61</td>
</tr>
</tbody>
</table>

(continued)
The parameter estimates are largely as would be expected based upon conventional understandings of the instigators of smoking onset. The more peers who smoked, the more likely subsequent smoking onset was (boys: $b = 4.87$, $p < .01$; girls: $b = 4.20$, $p < .01$). Respondents whose friends believed they should smoke were more likely to manifest onset than respondents whose friends thought they should not smoke, more so for girls ($b = 4.49$, $p < .01$) than for boys ($b = 2.62$, $p < .01$; gender difference: $b = -1.87$, $p < .05$).

Parental influence also appeared, in textured ways. Girls whose mothers and fathers smoked were more likely to manifest onset, but only if they were high in motivation to comply with their parents (mothers: $b = 1.57$, $p < 0.05$; fathers: $b = 1.60$, $p < 0.05$). Although mothers’ and fathers’ smoking had no statistically significant impact on boys, either as a main effect ($b = 0.07$, $ns$; $b = -0.03$, $ns$, respectively) or in interaction with motivation to comply ($b = 1.53$, $ns$; $b = 0.35$, $ns$, respectively), the interactions of gender with these effects are not significant (main effect: $b = 0.16$, $ns$; $b = -0.09$, $ns$; interaction: $b = -0.04$, $ns$). Therefore, it is most appropriate to conclude that the main effects and two-way interaction for mothers’ and fathers’ smoking in the column 3 regression apply to boys as well, meaning that boys also were especially likely to begin smoking if either of their parents smoked and if they were highly motivated to comply with their parents. Parents’ wishes about respondents’ smoking behavior had no impact on onset for either gender (boys: $b = -0.68$, $ns$; girls: $b = 0.55$, $ns$).

Siblings also influenced smoking onset. Having older brothers who smoked instigated smoking onset among boys ($b = 1.05$, $p < .01$) but not among girls ($b = 0.07$, $ns$; gender difference: $b = 0.98$, $p < .05$). Having older sisters who smoked instigated smoking onset among girls ($b = 0.59$, $p < .10$) but not among boys ($b = -0.32$, $ns$; gender difference: $b = -0.91$, $p < .10$).

Controlling for these effects, beliefs about the health consequences of smoking had impact on onset, though differently for boys and girls. For boys, believing that smoking was more likely to cause health problems inhibited smoking onset ($b = 1.81$, $p < .01$). For girls, however, beliefs about health consequences were not statistically significant ($b = 0.20$, $ns$).

The parameter estimates are largely as would be expected based upon conventional understandings of the instigators of smoking onset. The more peers who smoked, the more likely subsequent smoking onset was (boys: $b = 4.87$, $p < .01$; girls: $b = 4.20$, $p < .01$). Respondents whose friends believed they should smoke were more likely to manifest onset than respondents whose friends thought they should not smoke, more so for girls ($b = 4.49$, $p < .01$) than for boys ($b = 2.62$, $p < .01$; gender difference: $b = -1.87$, $p < .05$).

Parental influence also appeared, in textured ways. Girls whose mothers and fathers smoked were more likely to manifest onset, but only if they were high in motivation to comply with their parents (mothers: $b = 1.57$, $p < 0.05$; fathers: $b = 1.60$, $p < 0.05$). Although mothers’ and fathers’ smoking had no statistically significant impact on boys, either as a main effect ($b = 0.07$, $ns$; $b = -0.03$, $ns$, respectively) or in interaction with motivation to comply ($b = 1.53$, $ns$; $b = 0.35$, $ns$, respectively), the interactions of gender with these effects are not significant (main effect: $b = 0.16$, $ns$; $b = -0.09$, $ns$; interaction: $b = -0.04$, $ns$). Therefore, it is most appropriate to conclude that the main effects and two-way interaction for mothers’ and fathers’ smoking in the column 3 regression apply to boys as well, meaning that boys also were especially likely to begin smoking if either of their parents smoked and if they were highly motivated to comply with their parents. Parents’ wishes about respondents’ smoking behavior had no impact on onset for either gender (boys: $b = -0.68$, $ns$; girls: $b = 0.55$, $ns$).

Siblings also influenced smoking onset. Having older brothers who smoked instigated smoking onset among boys ($b = 1.05$, $p < .01$) but not among girls ($b = 0.07$, $ns$; gender difference: $b = 0.98$, $p < .05$). Having older sisters who smoked instigated smoking onset among girls ($b = 0.59$, $p < .10$) but not among boys ($b = -0.32$, $ns$; gender difference: $b = -0.91$, $p < .10$).

Controlling for these effects, beliefs about the health consequences of smoking had impact on onset, though differently for boys and girls. For boys, believing that smoking was more likely to cause health problems inhibited smoking onset ($b = 1.81$, $p < .01$). For girls, however, beliefs about health consequences were not statistically significant ($b = 0.20$, $ns$).

The parameter estimates are largely as would be expected based upon conventional understandings of the instigators of smoking onset. The more peers who smoked, the more likely subsequent smoking onset was (boys: $b = 4.87$, $p < .01$; girls: $b = 4.20$, $p < .01$). Respondents whose friends believed they should smoke were more likely to manifest onset than respondents whose friends thought they should not smoke, more so for girls ($b = 4.49$, $p < .01$) than for boys ($b = 2.62$, $p < .01$; gender difference: $b = -1.87$, $p < .05$).

Parental influence also appeared, in textured ways. Girls whose mothers and fathers smoked were more likely to manifest onset, but only if they were high in motivation to comply with their parents (mothers: $b = 1.57$, $p < 0.05$; fathers: $b = 1.60$, $p < 0.05$). Although mothers’ and fathers’ smoking had no statistically significant impact on boys, either as a main effect ($b = 0.07$, $ns$; $b = -0.03$, $ns$, respectively) or in interaction with motivation to comply ($b = 1.53$, $ns$; $b = 0.35$, $ns$, respectively), the interactions of gender with these effects are not significant (main effect: $b = 0.16$, $ns$; $b = -0.09$, $ns$; interaction: $b = -0.04$, $ns$). Therefore, it is most appropriate to conclude that the main effects and two-way interaction for mothers’ and fathers’ smoking in the column 3 regression apply to boys as well, meaning that boys also were especially likely to begin smoking if either of their parents smoked and if they were highly motivated to comply with their parents. Parents’ wishes about respondents’ smoking behavior had no impact on onset for either gender (boys: $b = -0.68$, $ns$; girls: $b = 0.55$, $ns$).

Siblings also influenced smoking onset. Having older brothers who smoked instigated smoking onset among boys ($b = 1.05$, $p < .01$) but not among girls ($b = 0.07$, $ns$; gender difference: $b = 0.98$, $p < .05$). Having older sisters who smoked instigated smoking onset among girls ($b = 0.59$, $p < .10$) but not among boys ($b = -0.32$, $ns$; gender difference: $b = -0.91$, $p < .10$).

Controlling for these effects, beliefs about the health consequences of smoking had impact on onset, though differently for boys and girls. For boys, believing that smoking was more likely to cause health problems inhibited smoking onset ($b = 1.81$, $p < .01$). For girls, however, beliefs about health consequences were not statistically significant ($b = 0.20$, $ns$).
But among girls, health effect beliefs and value placed on health interacted: The conjunction of believing that smoking was more likely to cause health problems and attaching substantial value to health inhibited onset ($b = 3.46, p < .05$). The main effect of health effect beliefs was significantly stronger among boys than among girls ($b = 3.49, p < .05$), and the interaction of health effect beliefs with value placed on health was marginally significantly stronger among girls than among boys ($b = -2.92, p < .10$). This is very strong evidence of causal impact of health beliefs on smoking onset and suggests that the process operates slightly differently for boys than for girls.

**Discussion**

The analyses reported here are relatively unusual in the smoking literature: Lagged effects using longitudinal survey data have rarely been estimated. Rather, researchers have offered usually reported cross-sectional correlations and made assumptions about the direction of causality at work. But we have shown here that it is possible to yield clearer support for a particular direction of causality using survey data collected on multiple occasions from the same individuals, some of whom experienced transitions out of the nonsmoking category and into the category of smokers (see also Tucker, Ellickson, & Klein, 2003; Wang et al., 1999).

This evidence reinforces presumptions widely held among health professionals about the primary instigators of smoking onset early in the life cycle. Peers, both via their behavior and subjective norms, are the most powerful instigators of smoking onset. But family forces are not irrelevant during this time period. Parents who smoked inspired their children to begin smoking, and parents’ expressed (or unexpressed) desires regarding child behavior were apparently inconsequential. That is, children acted as if parents instructed: “Do as I do, not as I say.”

Our evidence on parental influence may offer an explanation for the puzzle addressed by Avenevoli and Merikangas (2003): “Why is the effect of parental smoking weak?” (p. 13). We discovered that parental smoking behavior itself was not directly related to child smoking. Impact of parental smoking behavior was only apparent in light of the interaction involving motivation to comply with parents. Parental smoking was consequential among children who wished to comply with their parents but not among those who lacked such motivation. Most past studies reviewed by Avenevoli and Merikangas failed to measure and test interactions involving motivation to comply with parents when examining the effect of parental smoking. Perhaps taking this interaction into account will reveal more consistently powerful parental influence in future studies.

Siblings also inspired smoking onset, but in a gender-specific fashion. Boys were induced to start smoking by older brothers, and girls were induced to start smoking by older sisters. These findings are consistent with cross-sectional evidence showing the same gender specificity: boys’ smoking status was associated only with older
brothers’ smoking, and girls’ smoking status was associated only with older sisters’ smoking (e.g., Wang et al., 1995).

The gender specificity in the impact of older sibling’s behavior may occur partly because of gender segregation in sharing of material goods. For example, if two same-gender siblings share a bedroom, it may be easier for the younger sibling to borrow the older sibling’s cigarettes. Sharing bedrooms seems less likely for opposite gender siblings during adolescence. Thus, sibling influence may occur partly because of simple practicalities regarding obtaining cigarettes. But the gender-specific nature of this influence may also occur because younger boys and girls look up to older same-gender siblings as role models, so the process of behavior adoption may be driven by the desire to emulate specific other family members of the same gender.

Controlling for all these social influences, we found evidence consistent with the conclusion that beliefs about health consequences are causes of cigarette smoking onset as well. Believing that smoking is less damaging to health apparently allowed a young person to begin smoking more readily. But this process unfolded differently for boys and girls. For girls, beliefs about health effects were an insulating factor only when coupled with high value placed on health. Some girls place much more value on their health than others. Among those who valued their health, believing that smoking entailed health risks inhibited smoking onset. But among girls who did not value their health at all, beliefs about health risks had no effect at all on onset.

Boys manifested a simpler reasoning process. Beliefs about health effects were an insulating factor, in and of themselves. Believing that smoking entailed health risks lowered the likelihood of smoking onset regardless of value placed on health. The failure of this interaction to appear is not an artifactual result of no variance in value placed on health among boys. So future research might usefully investigate the reasons why value placed on health seems inconsequential in this domain and that beliefs about the health risks of smoking are more universally insulating for boys.

Our results have a number of interesting implications for health communication strategies that might be implemented in the future. First, according to the national survey data we reviewed, the belief that smoking cigarettes entails health risks is not held universally by American adults. And beliefs that smoking entails specific risks involving lung cancer, heart disease, and throat cancer are even less widely held. Thus, because these beliefs appear to inhibit smoking onset, it seems worthwhile to continue to educate the public about the state of evidence on these health risks.

Recent years have seen a substantial change in the role tobacco companies have played in this educational process. In contrast to decades during which these firms questioned the convincingness of research evidence on health risks of smoking, they now publicly endorse the view of most health professionals. For example, as of September, 2005, the Philip Morris Web site stated, “Philip Morris USA agrees with the overwhelming medical and scientific consensus that cigarette smoking causes lung cancer, heart disease, emphysema, and other serious diseases in smokers. Smokers are far more likely to develop serious diseases, like lung cancer, than non-smokers.
And in countries such as Canada and the UK, as well as the United States, cigarette packages or cartons universally contain large messages warning of the health effects. However, these messages are not necessarily reaching nonsmokers before they consider smoking or just before they begin to smoke. Therefore, it may not be sensible to assume that the public has been sufficiently convinced of the health risks of smoking. Especially among young people, efforts at convincingly illustrating the health risks of smoking may yield valuable payoffs in terms of decreased onset rates. Some of the currently implemented health education campaigns focused on smoking (e.g., sponsored by the Legacy Foundation) have chosen not to emphasize beliefs about the health risks of smoking and have instead focused on other strategies. Our evidence suggests that added attention to fostering accurate health risk beliefs may be worthwhile.

But even more can be done, according to our evidence. Our results suggest that the impact of health risk beliefs on smoking onset depends upon the value a girl places on her health. Among girls who value their health, establishing the belief that smoking entails health risks will put into place a powerful inhibitor of smoking onset. But among girls who do not place value on their health, efforts at fostering belief in health risks are likely to have no payoff in terms of reduced onset. Therefore, it would be worthwhile for health communication efforts to promote placing value on health in general. Any attempts to do so would be most effective if informed by a large literature documenting the causes of value placed on health. Therefore, our findings suggest additional work along these lines and the application of that work in public education campaigns, especially focused on young girls.

Another implication of our findings has to do with the gender matching apparent in sibling influence. As we suggested above, older same-gender siblings may facilitate smoking onset by providing behavioral opportunities. But teenage girls and boys may also be especially powerfully influenced by the behaviors and values of older same-gender role models. This reinforces the notion that educational messages might be best segregated by gender, having girls provide messages to girls and boys provide messages to boys. This can be done in classroom settings by having single-gender groups of students participate in health promotion exercises. And gender segregation can also be accomplished via the mass media, by placing ads featuring attractive same-gender sources in magazines that are read primarily either by girls or by boys.

It is interesting to note one other statistically significant effect in our regressions that has action implications. Among both boys and girls, motivation to comply with parents had significant negative impact on smoking onset (boys: $b = -1.78, p < .05$; girls: $b = -1.75, p < .05$). This effect was independent of parental smoking behavior and parents’ desires regarding their child’s smoking behavior. That is, simply being highly motivated to comply with parents’ wishes reduced smoking onset. Perhaps, this is because most of the things parents hope their children will do are incompatible with smoking behavior, so compliance-motivated children are busy doing other
things that minimize deleterious social contact and available time, thus reducing smoking onset as an unintentional byproduct. This finding suggests that public education campaigns designed to enhance motivation to comply with parents in particular (and with adults in general) might be another route to successful inhibition of smoking onset.

Another implication of our findings has to do with future research on the causes of smoking behavior. It is not uncommon to see publications of research predicting smoking status using an array of predictors (e.g., Alexander, Piazza, Mekos, & Valente, 2001; Epstein, Botvin, & Spoth, 2003). But it is also not uncommon to see such regression equations fail to include measures of the perceived health risk of smoking (e.g., Alexander et al., 2001; Epstein et al., 2003). Given the risk of spuriousness in such regressions, it is important to control for all other possible causes of the dependent variable. The present research findings suggest that health risk beliefs should always be among these control variables.

Conclusion

Public health efforts to encourage Americans to more accurately recognize the health consequences of smoking seem likely to have been consequential in shaping smoking behavior, leading to a reduction in the nation’s smoking rate and a consequent reduction in smoking-related morbidity and mortality.

Notes

1 This introduces some nonindependence into the data set that we used for our initial analyses. To assess the impact of this nonindependence, we repeated all of our analyses using only one wave of data per respondent. To do so, we randomly selected one wave for each respondent who had two or three waves of data suitable for our analyses. These analyses yielded similar results to those reported in the text.

2 Among boys, the following two-way interaction effects were not significant: Friends’ Smoking × Motivation to Comply With Friends ($b = -1.75, p > .10$), Friends’ Subjective Norm × Motivation to Comply With Friends ($b = -0.79, p > .50$), and Parents’ Subjective Norm × Motivation to Comply With Parents ($b = -1.11, p > .50$). Among girls, the following two-way interaction effects were not significant: Friends’ Smoking × Motivation to Comply With Friends ($b = -0.84, p > .60$), Friends’ Subjective Norm × Motivation to Comply With Friends ($b = -0.81, p > .70$), and Parents’ Subjective Norm × Motivation to Comply With Parents ($b = 2.23, p > .50$). These interactions were therefore removed from the regressions.

3 This cut-point was necessary to capture the shape of nonlinear moderation that we observed in the data. The majority of respondents (92%) were clustered in the high-value group.

4 A very small number of respondents transitioned from being a regular smoker at one wave to being a nonsmoker at the next wave (only 0.8% from Wave 1 to Wave 2, 0.9% from Wave 2 to Wave 3, and 0.6% from Wave 3 to Wave 4), so we were not able to study the predictors of smoking cessation.
Data from respondents who were aged 19 (2%) and 20 (0.1%) were not included in our analyses.

References


Appendix A

Question wording

Health beliefs

Three pairs of questions were used to build an index of beliefs about the health consequences of smoking. The first pair was “If I smoke cigarettes, I will live for a long time” and “If I do NOT smoke cigarettes, I will live for a long time,” the second pair was “If I smoke cigarettes, I will get lung cancer” and “If I do NOT smoke cigarettes, I will get lung cancer,” and the third pair was “If I smoke cigarettes, I will get heart disease” and “If I do NOT smoke cigarettes, I will get heart disease.” On all six items, the response choices strongly agree, agree, neutral, disagree, and strongly disagree were coded 1, .75, .5, .25, and 0, respectively. A difference score was computed for each corresponding pair of items: the response to the “If I do NOT smoke cigarettes” item was subtracted from the responses to the “If I smoke cigarettes” item. The average of the heart disease and the lung cancer difference scores was multiplied by $-1$, then averaged with the difference score for the long life items. This was done so that the items about negative health consequences would have the same weight as the positively worded item about longevity. In this health belief index, higher scores imply less endorsement of the negative consequences of smoking.

Value placed on health

Four items measured the extent to which respondents valued health and longevity, all asking whether a series of life outcomes would be very bad, bad, not good or bad, good, or very good. Two life outcomes were desirable (“If I live for a long time, that will be ….” and “If I live a healthy life, that will be ….”), and responses to these items were coded to range from 1 (very good) to 0 (very bad). The other two life outcomes were undesirable (“If I get lung cancer, that will be ….” and “If I get heart disease, that will be ….”), and responses to these items were coded to range from 1 (very bad) to 0 (very good). Responses to the four items were then averaged to yield an index score that ranged from 1 (meaning placing maximal value on health and longevity) to 0 (meaning placing minimal value on health and longevity). This index was dichotomized such that all data points from .8 through 1 were recoded as 1 (high value placed on health), and all lesser values were recoded as 0 (low value placed on health).

Smoking onset

Respondents read six sentences and selected the one that best described their smoking behavior: “I have never smoked a cigarette, not even a few puffs”; “I have smoked one cigarette or a few cigarettes just to try, but I have not smoked in the past month”; “I no longer smoke but in the past I was a regular smoker”; “I smoke regularly but no more than one cigarette a month”; “I smoke regularly but no more than one cigarette a week”; “I smoke more than one cigarette a week.” People who said “never smoked,” “have not smoked in the past month,” or “no longer smoke” were coded 0 to identify them as nonsmokers, and people who said “smoke regularly but no
more than one cigarette a month,” “smoke regularly but no more than one cigarette a week,” or “smoke more than one cigarette a week” were coded 1 to identify them as current smokers.

Fewer than 2% of respondents said that they “smoke regularly but no more than one cigarette a month” (1.5% in Wave 1, 1.4% in Wave 2, 1.1% in Wave 3, and 1.1% in Wave 4) or “smoke regularly but no more than one cigarette a week” (1.3% in Wave 1, 1.5% in Wave 2, 1.1% in Wave 3, and 1.1% in Wave 4), so the vast majority of people classified as current smokers were those who said they smoked more than one cigarette a week. Among respondents classified as noncurrent smokers, a small minority said they no longer smoked but were a regular smoker in the past (6.2% in Wave 1, 5.5% in Wave 2, 5.1% in Wave 3, and 4.2% in Wave 4); these respondents were excluded from our analyses.4 Most people classified as noncurrent smokers had never smoked before or smoked just once or twice to try.

For each of the first three waves, we created a variable coded 0 for people who were nonsmokers at that wave and the subsequent wave and coded 1 for people who were nonsmokers at that wave and became smokers by the subsequent wave. This dichotomous variable measuring smoking onset constituted the core dependent variable used in the present analyses to study the predictors of smoking onset.

Friends’ smoking status
Respondents were asked how many of their five closest friends smoked cigarettes. Responses were coded 1 (all five friends smoked), .8 (four friends smoked), .6 (three friends smoked), .4 (two friends smoked), .2 (one friend smoked), and 0 (no friend smoked).

Motivation to comply with friends
Respondents indicated the extent to which they agreed or disagreed with the statement “Most of the time when my friends want me to do something, I go along with it.” Responses were coded 1 (strongly agree) or 0 (agree, neutral, disagree, and strongly disagree).

Friends’ subjective norm
Respondents indicated the extent to which they agreed or disagreed that “My friends think that I should smoke cigarettes” and “My friends think that I should NOT smoke cigarettes.” Responses to the latter item (coded to range from .5, strong disagreement, to 0, strong agreement) were added to responses to the former item (coded to range from .5, strong agreement, to 0, strong disagreement). The resulting index ranged from 1 (meaning a strong norm favoring smoking) to 0 (meaning a strong norm against smoking).

Parents’ smoking status
Respondents were asked whether their mother smoked cigarettes and whether their father smoked cigarettes. Offered response options included “yes,” “no,” or “I have no mother/father.” A dichotomous variable was created, coded 1 for respondents whose
mothers smoked and 0 for respondents whose mothers did not smoke or who had no mother. A second dichotomous variable was coded 1 for respondents whose fathers smoked and 0 for respondents whose fathers did not smoke or who had no father.

To separate for the effect of having a parent who did not smoke from the effect of not having a parent, another pair of dummy variables was created: one to identify respondents who did not have mothers (coded 1 for people with no mother and 0 for people with a mother present) and another to identify respondents who did not have fathers (coded 1 for people with no father and 0 for people with a father present).

Motivation to comply with parents
Motivation to comply with parents was measured by the extent of agreement or disagreement with the statement, “Most of the time when my parents want me to do something, I go along with it.” Responses were coded 1 (strongly agree) or 0 (agree, neutral, disagree, and strongly disagree).

Parents’ subjective norm
Respondents indicated the extent to which they agreed or disagreed that “My parents think that I should smoke cigarettes” and “My parents think that I should NOT smoke cigarettes.” Responses to the latter item (coded to range from .5, strong disagreement, to 0, strong agreement) were added to responses to the former item (coded to range from .5, strong agreement, to 0, strong disagreement). The resulting index ranged from 1 (meaning a strong norm favoring smoking) to 0 (meaning a strong norm against smoking).

Older siblings’ smoking status
Respondents were asked whether their older brother smoked cigarettes and whether their older sister smoked cigarettes (response choices: “yes,” “no,” or “I have no older brother/sister”). A dichotomous variable was coded 1 for respondents with an older brother who smoked and 0 for respondents whose older brothers did not smoke or who had no older brother. Similarly, a dichotomous variable was coded 1 for respondents with an older sister who smoked and 0 for respondents whose older sisters did not smoke or who had no older sister.

To control for the effect of not having older siblings, a pair of dummy variables was created: one to identify respondents who did not have older brothers (coded 1 for people with no older brother and 0 for people with an older brother), and another to identify respondents who did not have older sisters (coded 1 for people with no older sister and 0 for people with an older sister).

Age
Respondents reported their date of birth, which was used to compute age. Data from respondents who were aged 11 through 17 at the first wave in each wave pair were included in our analyses. The age variable represented age at the time of the second wave in each wave pair, ranging from 0 (age 12) to 1 (age 17).5