

# Regular smokeless tobacco use is not a reliable predictor of smoking onset when psychosocial predictors are included in the model

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Tomar (*Nicotine & Tobacco Research*, 5, 545–553) analyzed the CDC's Teenage Attitudes and Practices Survey (TAPS) and reported smokeless tobacco may act as a starter product for or gateway to cigarettes. Regular smokeless tobacco users at baseline were said to be 3.45 times more likely than never users of smokeless tobacco to become cigarette smokers after 4 years (95% *CI* = 1.84–6.47). However, this analysis did not take into account well-known psychosocial predictors of smoking initiation. We reanalyzed TAPS to assess whether including psychosocial predictors of smoking affected the smokeless tobacco gateway effect. Experimenting with smoking, *OR* = 2.09 (95% *CI* = 1.51–2.90); below average school performance, *OR* = 9.32 (95% *CI* = 4.18–20.77); household members smoking, *OR* = 1.49 (95% *CI* = 1.13–1.95); frequent depressive symptoms, *OR* = 2.19 (95% *CI* = 1.25–3.84); fighting, *OR* = 1.48 (95% *CI* = 1.08–2.03); and motorcycle riding, *OR* = 1.42 (95% *CI* = 1.06–1.91) diminished the effect of both regular, *OR* = 1.68 (95% *CI* = .83–3.41), and never regular smokeless tobacco use, *OR* = 1.41 (95% *CI* = .96–2.05), to be statistically unreliable. Analyzing results from a sample of true never smokers (never a single puff) showed a similar pattern of results. Our results indicate that complex multivariate models are needed to evaluate recruitment to smoking and single factors that are important in that process. Tomar's analysis should not be used as reliable evidence that smokeless tobacco may be a starter product for cigarettes.

## Introduction

Tomar (2003) used the Teenage Attitudes and Practices Survey (TAPS) to explore whether smokeless tobacco is a risk factor for or gateway to cigarette smoking. Using a sample of adolescent males who were never smokers at initial survey, Tomar investigated whether having used smokeless tobacco at baseline made one more likely to smoke at follow-up, controlling for age and race. He reported that those who used smokeless tobacco regularly were 3.45 times more likely to smoke and argued that smokeless tobacco may be a starter product for later cigarette smoking.

We believe Tomar's analysis is problematic and that his findings may be due to spurious correlation. He did not account for important psychosocial predictors of smoking (U.S. Department of Health and Human Services [USDHHS], 1994), many of which are available in the TAPS data. The 1994 surgeon general's report on preventing youth smoking notes that several factors are associated with cigarette smoking and smokeless tobacco use: depressive symptoms, low knowledge of long-term consequences, risk-taking behavior, self-image, and subjective expected utility of use (USDHHS, 1994). Other authors have noted that adolescent drug use is associated with a host of interpersonal and intrapersonal factors (e.g., Petraitis, Flay, & Miller, 1995). Also, Miller (1994) found that some adolescents are unwilling to try any substances, whereas others are willing to try any available substances: This means that correlated use can be the result of both "nonusing" individuals who are unlikely to use any drugs and "using" individuals who are likely to use many different drugs.

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In light of past research demonstrating strong relations between psychosocial factors and both smokeless tobacco and cigarette use, not including these factors in the model linking smokeless tobacco and subsequent cigarette use results in a misspecified model. Omitted variables are not a problem when descriptive statistics are the goal or when the omitted variables are not correlated with the predictors of interest. However, when covariates are correlated with the predictor(s) in the model, and these covariates are not included in the analysis, the misspecified model produces biased estimates (James, 1980). Therefore, when the goal is to draw statistical conclusions regarding the importance of a predictor, model specification is critical. Because the causal gateway argument depends on accurate statistical inference and comparison among multiple potential putative causes, it is important to work toward a correct model specification. No model is ever perfectly specified, but a model that includes reliable predictors of a response is closer to the true model than one that does not.

Tomar's never-smoker group included people who indicated some past experience with cigarettes (i.e., at least a puff). Among these individuals, smokeless tobacco and cigarette initiation are confounded. These individuals' early experiences with cigarettes may influence their later decisions to smoke, independent of their exposure to smokeless tobacco (Kozlowski & Harford, 1976). In this group, the individuals' initial puffs are just as likely to be a determinant of later smoking as is their use of smokeless tobacco. A more stringent test of how well smokeless tobacco use predicts subsequent cigarette smoking would be to start with individuals who have *never* had a puff on a cigarette.

We explored the TAPS data in more depth, looking at additional predictors of smoking to see if taking them into account affected the relationship between smokeless tobacco use and cigarette use. We also refined the model to look at the role of smokeless tobacco in the progression of true never smokers to established smoking.

## Method

### *Subjects*

Detailed methodological descriptions of the TAPS (Moss, Allen, Giovino, & Mills, 1992) are available elsewhere (Moss et al., 1992; Tomar, 2003; Tomar & Giovino, 1998), and are not repeated here. Our sample consisted of males aged 12–18 years in 1989 who were interviewed by telephone and who had not smoked a cigarette ( $n=2,683$ ). Data were weighted to provide estimates of the U.S. population.

Although we had access to the full TAPS dataset and followed the procedures outlined in Tomar (2003) precisely, we were unable to replicate exactly the  $n$

numbers that Tomar reported. We are unsure why this occurred. We tried multiple methods and calculated multiple tobacco use variables in order to approximate his results. These differences are related to the selection of the subsample. When we examined the entire sample, our response frequencies matched those in the TAPS documentation. We are confident that our replication is adequate, in that we basically reproduced the results reported by Tomar (2003).

### *Definitions of tobacco use*

We attempted to use categorizations of tobacco use (cigarette and smokeless tobacco) identical to those reported by Tomar (2003). Smokeless tobacco users were classified as never users, never regular users (used smokeless tobacco at some time but did not consider themselves regular users), and regular users, based on a calculated variable provided in the TAPS dataset. Smoking status was defined as current smokers (smoked in last 30 days and smoked 100 cigarettes in lifetime), former smokers (smoked 100 cigarettes in lifetime but none in last 30 days), or nonsmokers (smoked less than 100 cigarettes in lifetime). These definitions comprised a variable in the TAPS dataset.

### *Two definitions of never smoker status*

Tomar (2003) limited his predictive analyses to those adolescents who had not smoked a cigarette at baseline. In his never-smoker group, however, 474 subjects answered yes to the subsequent question, "Have you ever experimented with cigarettes, even a few puffs?" For these people, smokeless tobacco and past experience with cigarettes are confounded. We found an association between smokeless tobacco use and experimentation with cigarettes: 41.8% of regular smokeless tobacco users (95%  $CI=29.5-54.1$ ), 34.5% of never regular users (95%  $CI=29.7-39.2$ ), and 14.7% of never users (95%  $CI=13.1-16.2$ ) reported having experimented with cigarettes,  $\chi^2(2)=66.08$ ,  $p<.0001$ . In this group, there is no way to discern whether smokeless tobacco use or cigarette experimentation occurred first. To perform a more stringent test of gateway effects, we performed a second set of analyses using individuals who said they had never experimented with cigarettes ( $n=2,211$ ).

### *Definitions of psychosocial predictors of smoking*

At the initial interview, subjects were asked whether anyone in their household smoked (yes, no). Self-reported performance in school also was assessed (much better than average, better than average, average, below average). Depressive symptoms were assessed by responses to the question "During the past year, how often have you felt unhappy, sad, or

depressed?" (often, sometimes, rarely, never). Finally, subjects were asked whether in the past year they had been in a physical fight and whether they had ridden a motorcycle (both yes–no response options). These last two variables can indicate risky behavior.

### *Analyses*

SUDAAN v. 8.0.1 was used to perform cross-tabulation and logistic regression, accounting for the complex survey design. We used logistic regression in two populations of never smokers. The first regression used the same group that Tomar analyzed (i.e., never smoked a cigarette). The second analysis focused on a group that never experimented with cigarettes (i.e., not even a puff). Candidate psychosocial predictors were then entered one at a time to assess overall changes in the model. We also undertook an alternative method of analysis, creating principal component scores from 15 candidate psychosocial variables and using those scores in a logistic regression model. Because these results were so similar to our other logistic regression models, they are not discussed here (results available on request).

### *Model quality – predicted probabilities*

Appropriate procedures for assessing model fit and diagnostics for logistic regression models in complex survey data are not yet available in standard statistical packages. In particular, an  $R^2$ -like measure of the predictive power of the model would be quite useful. Unfortunately, quantities like  $R^2$  are not useful for logistic regression, and their use is discouraged (Hosmer & Lemeshow, 2000). In order to assess model quality with respect to variance explained, we computed the predicted probabilities of 1993 current smoking for sample members, as suggested by Korn & Graubard (1999). If a model is useful, it should have a reasonably large range of predicted probabilities. That is, some covariate patterns should correspond to a low probability of making a transition and other covariate patterns should have a relatively high probability of the transition. A model with a large range of predicted probabilities accounts for a correspondingly large amount of the variance in the response. If a model predicts only a small range of probabilities, it probably does not do a good job of accounting for the variance in the response. Here we report the minimum and maximum predicted probabilities under four of the models.

## **Results**

Because we replicated the basic descriptive statistics of the sample on cigarette and smokeless tobacco use presented in Table 1 in Tomar (2003) to within .1

percentage points, they are not reported here. Descriptive statistics on the psychosocial predictor variables, by smokeless tobacco status and smoking status, are presented in Table 1.

### *Logistic regression models of smoking status at follow-up*

Many variables influence adolescent smoking initiation (USDHHS, 1994). We attempted to create a multivariate model of smoking initiation, including smokeless tobacco use, to put the gateway effect in context (Tables 2 and 3). First, we replicated Tomar's findings – limiting our analysis to those who were never smokers (i.e., had not smoked a whole cigarette) at baseline and predicting their smoking status at follow-up with smokeless tobacco use status, controlling for age and race (Table 2). Although we were able to reproduce Tomar's odds ratio for regular users, our odds ratio for never regular users was nonsignificantly higher. This difference may be the result of our having used different versions of SUDAAN (7.5 vs. 8.0) or because of the sample replication issue discussed earlier. However, we judged that our replication was acceptable for further analyses.

Table 3 shows a series of models including psychosocial predictor variables. Model 1 assessed the impact of having experimented with cigarettes on later smoking. Such experimentation is quite a significant predictor of current smoking at follow-up,  $OR=2.37$  (95%  $CI=1.75-3.22$ ). At the same time, the effect of smokeless tobacco dropped from 3.45 to 2.69. We then entered school performance, a known predictor of adolescent smoking (e.g., Choi, Pierce, Gilpin, Farkas, Berry, 1997; USDHHS, 1994), into Model 2. Again, this predictor was significant and further diminished the effect of smokeless tobacco. As successive predictors (household member smoking, depressive symptoms, fighting, and motorcycle riding) were added, the effect of smokeless tobacco continued to diminish. By Model 3, regular smokeless tobacco use was not a statistically significant predictor of smoking status, and by Model 6 neither smokeless tobacco use category significantly predicted current smoking at follow-up.

### *A more stringent test of gateway effects*

To reduce the possibility of confounding from early cigarette experiences, we ran a series of analyses parallel to those discussed above in which our base population was males who never puffed on a cigarette (Table 4). The addition of school performance alone eliminated the effect of regular smokeless tobacco use on smoking. Including additional psychosocial predictors of smoking further diminished the nonsignificant effect of regular smokeless tobacco use and

**Table 1.** Predictors of smoking by baseline smokeless tobacco status and follow-up smoking status, male nonsmokers at baseline<sup>a</sup>

	Smokeless tobacco use status, 1989, % (SE)			Smoking status, 1993, % (SE)		
	Regular (n=58)	Never regular (n=378)	Never (n=2,245)	Current (n=250)	Former (n=37)	Never (n=2389)
Someone in household smokes						
Yes (n=1,111)	59.1 (6.9)	48.7 (2.67)	40.1 (1.2)	56.0 (3.15)	36.1 (8.32)	40.4 (1.13)
No (n=1,568)	40.9 (6.9)	51.3 (2.67)	59.9 (1.2)	44.0 (3.15)	63.9 (8.32)	59.6 (1.13)
During the past year have you been in a physical fight?						
Yes (n=805)	49.6 (6.37)	40.1 (2.68)	27.9 (.99)	44.0 (3.28)	37.4 (7.93)	28.7 (.97)
No (n=1,864)	50.4 (6.37)	59.9 (2.68)	72.1 (.99)	56.0 (3.28)	62.6 (7.93)	71.3 (.97)
Have you ever experimented with cigarettes (even a few puffs)?						
Yes (n=472)	41.8 (6.27)	34.5 (2.44)	14.7 (.78)	33.3 (3.13)	32.7 (8.34)	16.2 (.80)
No (n=2,208)	58.2 (6.27)	65.5 (2.44)	85.3 (.78)	66.7 (3.13)	67.3 (8.34)	83.8 (.80)
How do you do in school?						
Far above average (n=485)	5.6 (2.98)	14.3 (1.88)	19.5 (.87)	6.8 (1.59)	10.9 (5.35)	19.7 (.86)
Above average (n=994)	20.8 (5.67)	36.1 (2.64)	37.6 (1.04)	34.5 (3.23)	38.1 (8.00)	37.1 (1.03)
Average (n=1,128)	63.9 (6.34)	47.0 (2.74)	40.8 (1.16)	50.1 (3.45)	44.4 (8.40)	41.3 (1.10)
Below average (n=63)	9.7 (3.65)	2.6 (.83)	2.1 (.33)	8.6 (1.90)	6.6 (3.58)	1.8 (.31)
During the past year, how often have you felt unhappy, sad, or depressed?						
Never (n=522)	18.3 (5.25)	15.9 (2.03)	19.7 (.86)	13.5 (2.16)	14.8 (6.38)	19.9 (.85)
Rarely (n=1,156)	53.6 (6.05)	49.4 (3.03)	42.3 (1.16)	40.9 (3.18)	44.4 (8.71)	43.8 (1.18)
Sometimes (n=748)	19.6 (4.53)	23.9 (2.35)	29.4 (1.04)	29.8 (2.84)	31.5 (7.72)	28.1 (1.03)
Often (n=238)	8.5 (4.20)	10.8 (1.66)	8.6 (.61)	15.9 (2.46)	9.3 (4.42)	8.3 (.55)
In the past year have you ridden on a motorcycle or minibike either by yourself or with someone else driving?						
Yes (n=1,130)	67.6 (7.06)	67.6 (2.54)	37.0 (1.14)	56.3 (3.39)	50.7 (8.76)	40.5 (1.12)
No (n=1,542)	32.4 (7.06)	32.4 (2.54)	63.0 (1.14)	43.7 (3.39)	49.3 (8.76)	59.5 (1.12)

<sup>a</sup>Data derived from the Teenage Attitudes and Practices Survey 1989–1993.

diminished the effect of never regular smokeless tobacco use. By Model 4, neither category was a significant predictor of smoking.

*Predicted probabilities as an index of model specification*

We output predicted probabilities from our logistic regression model to see how well each model distinguished between nonsmokers and current smokers. The broader the range, the better job the model did in separating nonsmokers from smokers. In Tomar’s model, predicted probabilities ranged from .037 to .26, with a median of .087. For our Model 6, in contrast,

probabilities ranged from .006 to .69, with a median of .064. That is, our most complex model had roughly twice the range of predicted probabilities as Tomar’s model. Concretely, this means that we distributed the sample over a much broader range of probabilities, yielding more precise and refined prediction than did Tomar’s model. When we restricted the sample to true never smokers, predicted probabilities in Tomar’s model ranged from .037 to .22 (median=.072), whereas those in our model ranged from .005 to .64 (median=.058). Again, the range of predicted probabilities was twice that of Tomar’s, providing a much better predictive model for distinguishing nonsmokers and current smokers in 1993.

**Table 2.** Tomar (2003) results and our replication<sup>a</sup>

	Tomar (2003)		Replication	
	n	OR (95% CI) <sup>b</sup>	n	OR (95% CI) <sup>b</sup>
Smokeless tobacco use				
Regular	60	3.45 (1.84–6.47)	58	3.45 (1.84–6.47)
Never regular	373	2.01 (1.38–2.93)	378	2.11 (1.46–3.05)
Never used	2,232	1.00 (referent)	2,245	1.00 (referent)

<sup>a</sup>Data derived from the Teenage Attitudes and Practices Survey 1989–1993.

<sup>b</sup>Odds ratios are adjusted for age and race (values not shown).

**Table 3.** Multiple logistic regression models predicting current smoking at follow-up among those who have not smoked a cigarette at baseline <sup>a,b</sup>

	Model 1 <i>OR</i> (95% <i>CI</i> ) <sup>c</sup>	Model 2 <i>OR</i> (95% <i>CI</i> ) <sup>c</sup>	Model 3 <i>OR</i> (95% <i>CI</i> ) <sup>c</sup>	Model 4 <i>OR</i> (95% <i>CI</i> ) <sup>c</sup>	Model 5 <i>OR</i> (95% <i>CI</i> ) <sup>c</sup>	Model 6 <i>OR</i> (95% <i>CI</i> ) <sup>c</sup>
Smokeless tobacco use						
Regular	2.69 (1.36–5.33)	2.10 (1.07–4.13)	1.92 (.97–3.78)	2.07 (1.04–4.09)	1.84 (.91–3.70)	1.68 (.83–3.41)
Never regular	1.76 (1.22–2.54)	1.66 (1.15–2.41)	1.61 (1.12–2.33)	1.65 (1.14–2.38)	1.54 (1.06–2.24)	1.41 (.96–2.05)
Never used	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Experimented with cigarettes (≥ 1 puff)	2.37 (1.75–3.22)	2.23 (1.62–3.06)	2.14 (1.55–2.96)	2.13 (1.54–2.94)	2.10 (1.51–2.91)	2.09 (1.51–2.90)
How do you do in school?	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Far above average		1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Above average		2.54 (1.48–4.36)	2.48 (1.44–4.27)	2.53 (1.46–4.37)	2.48 (1.44–4.27)	2.41 (1.40–4.16)
Average		3.14 (1.81–5.44)	3.06 (1.76–5.32)	3.01 (1.72–5.27)	2.89 (1.65–5.06)	2.80 (1.60–4.91)
Below average		11.56 (5.43–24.62)	11.22 (5.21–24.19)	9.83 (4.55–21.27)	9.68 (4.42–21.18)	9.32 (4.18–20.77)
Someone in household smokes			1.59 (1.21–2.09)	1.54 (1.17–2.03)	1.52 (1.15–2.00)	1.49 (1.13–1.95)
			1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Depressive symptoms						
Never				1.00 (referent)	1.00 (referent)	1.00 (referent)
Rarely				1.27 (.82–1.97)	1.20 (.77–1.88)	1.18 (.76–1.85)
Sometimes				1.54 (1.00–2.37)	1.45 (.94–2.23)	1.42 (.92–2.20)
Often				2.41 (1.37–4.24)	2.20 (1.24–3.88)	2.19 (1.25–3.84)
Fight in past year					1.54 (1.13–2.09)	1.48 (1.08–2.03)
					1.00 (referent)	1.00 (referent)
Rode motorcycle in past year						1.42 (1.06–1.91)
						1.00 (referent)

<sup>a</sup>Data derived from the Teenage Attitudes and Practices Survey 1989–1993. Analysis limited to males who had never smoked at baseline.<sup>b</sup>Current smoking defined as having smoked at least 100 cigarettes and smoking on at least one of the 30 days preceding the follow-up interview.<sup>c</sup>Odds ratios are adjusted for age and race (values not shown).

**Table 4.** Multiple logistic regression models predicting current smoking at follow-up among those who have never experimented with cigarettes at baseline <sup>a,b</sup>

	Model 1 OR (95% CI) <sup>c</sup>	Model 2 OR (95% CI) <sup>c</sup>	Model 3 OR (95% CI) <sup>c</sup>	Model 4 OR (95% CI) <sup>c</sup>
Smokeless tobacco use				
Regular ( <i>n</i> =34)	3.41 (1.31–8.89)	2.04 (.73–5.69)	2.14 (.74–6.21)	1.97 (.69–5.65)
Never regular ( <i>n</i> =253)	1.77 (1.13–2.79)	1.69 (1.06–2.68)	1.66 (1.05–2.64)	1.59 (1.00–2.53)
Never used ( <i>n</i> =1,922)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
How do you do in school?				
Far above average		1.00 (referent)	1.00 (referent)	1.00 (referent)
Above average		2.51 (1.27–4.95)	2.63 (1.33–5.19)	2.49 (1.26–4.91)
Average		3.72 (1.92–7.18)	3.77 (1.94–7.34)	3.63 (1.86–7.07)
Below average		15.99 (6.51–39.28)	14.21 (5.62–35.93)	14.17 (5.58–36.00)
Depressive symptoms				
Never			1.00 (referent)	1.00 (referent)
Rarely			2.00 (1.08–3.68)	1.92 (1.04–3.54)
Sometimes			2.27 (1.29–4.01)	2.25 (1.27–3.99)
Often			3.81 (1.84–7.88)	3.70 (1.79–7.66)
Someone in household smokes				1.52 (1.10–2.11)
				1.00 (referent)

<sup>a</sup>Data derived from the Teenage Attitudes and Practices Survey 1989–1993. Analysis limited to males who had never experimented with cigarettes at baseline (not even a puff).

<sup>b</sup>Current smoking is defined as having smoked at least 100 cigarettes and smoking on at least one of the 30 days preceding the follow-up interview.

<sup>c</sup>Odds ratios are adjusted for age and race (values not shown).

### Discussion

Tomar (2003) used the longitudinal component of the TAPS survey to evaluate progression to smoking at follow-up (4 years later) in 12–18-year-olds who were never smokers at baseline. His analysis found that smokeless tobacco use at baseline was a predictor of smoking at follow-up. Tomar’s model, however, was misspecified in that it did not assess available psychosocial predictors of smoking. Additionally, the inclusion of individuals who had taken a puff on a cigarette in the sample introduced confounding.

Our reanalysis showed that smokeless tobacco use was not a statistically significant predictor of subsequent smoking when other variables were included in the model. Our model was also better able to differentiate smokers from nonsmokers through a greater range of predicted probabilities. Given typical publication criteria, it is unlikely that any positive smokeless tobacco results would have been featured in a publication on predictors of smoking; in fact, smokeless tobacco use would likely have been dropped from the prediction model. This raises serious doubts about the appropriateness of citing the Tomar (2003) results as evidence of smokeless tobacco effects on smoking initiation.

### Removal of confounded subjects

Changing the base sample from never smokers (never smoked a whole cigarette) to true never smokers (never even had a puff) provided a less confounded means to assess gateway effects. That is, by limiting the sample to those who had never had a puff on a cigarette, we were able to eliminate the influence of

any early cigarette smoking experiences on later decisions to smoke. The effect of smokeless tobacco use on smoking was approximately the same as in Tomar’s model in this group when smokeless tobacco was the only variable in the model; this effect was quickly made nonsignificant by the addition of school performance, an established correlate of adolescent smoking (Choi et al., 1997). Thus, a more stringent test of potential gateway effects showed that the trajectory from smokeless tobacco use to *subsequent* cigarette smoking was not a reliable effect.

### No guarantee of causality

The landmark 1964 surgeon general’s report set forth the rules for establishing causality from epidemiological data. In sum, (a) a clear and consistent relationship between the cause and outcome must be established, (b) other plausible causes must be eliminated or taken into account, (c) temporal precedence must be established, wherein the cause precedes the effect, and (d) a plausible mechanism of causation must be noted (U.S. Department of Health, Education, and Welfare, 1964). Although longitudinal data such as from TAPS allow for the establishment of temporal precedence, they do not guarantee that any of the other criteria will be met. Certainly, this is true of Tomar’s analysis. When other plausible explanations of smoking at follow-up were included in the model, smokeless tobacco use became less important.

Two specific limitations of the TAPS data are that (a) the baseline sample covered a wide age range and (b) a large gap existed between the measurement

periods, during which undetected processes could intervene to influence smoking. Regarding the first limitation, individuals across a broad age span (12–18 years at baseline) probably differ in their likelihood of smoking. Particularly interesting are those 18-year-olds who had never smoked, given that 90% of recruitment to smoking occurs by age 18 years (USDHHS, 1994).

Regarding the second limitation, a span of 4 years is a long time in adolescent development, and a great deal could have happened that was more related to Time 2 smoking than the Time 1 level of almost any variable. For example, participants may have acquired smoking friends after the initial survey, who influenced their decision to smoke, but had not retained them by the time of the second interview. In fact, when we ran a logistic model with only age, race, smokeless tobacco use, and number of male friends who smoke at follow-up, the number of male friends who smoke was a sizable predictor of smoking at follow-up,  $OR=2.7$  (95%  $CI=2.4-3.0$ ), whereas smokeless tobacco was not statistically significant,  $OR=2.1$  (95%  $CI=.92-4.8$ ). Process information also would allow insight into reasons some smokeless tobacco users move to cigarettes (i.e., elucidating plausible mechanisms). Another reason that process information is important is that the relationship between a predictor and a dependent variable is not necessarily static (Collins & Graham, 2002). That is, the number of male friends who smoke in 1989 may be somewhat predictive of 1993 smoking status, but perhaps the number of male friends who smoke in 1992 is much more predictive of 1993 smoking status. This lack of process information inhibited our ability to make causal inferences from these longitudinal data.

The TAPS data also have limitations in terms of creating complete models of smoking initiation. Several authors have shown that exposure to tobacco advertising and promotion (e.g., Pierce, Choi, Gilpin, Farkas, Berry, 1998) is a predictor of smoking initiation, controlling for other factors. Because an advertising exposure variable was not included in TAPS, its effect could not be assessed. Information on all established predictors of smoking within the dataset would be needed for the development of a more comprehensive model of smoking initiation.

#### *Weak evidence for smokeless tobacco gateway effects*

A large study by Haddock and colleagues (2001) seems supportive of Tomar's findings, and they controlled for psychosocial predictors as we do here. Past and current smokeless tobacco users (at baseline) were each about 2.3 times more likely to have begun smoking at follow-up (Haddock et al., 2001). However, this study was limited in two major ways. First,

male military recruits are likely unrepresentative of males in the general population. Second, and more important, the definition of smoking changed from baseline to follow-up. At baseline, smokers were defined as having smoked at least one cigarette per day (the timeframe was not specified, but we assumed the past 7 days, consistent with the authors' other definitions), whereas at follow-up smoking was defined as having even a single puff on a cigarette in the preceding 7 days. This change in definition to a very loose criterion for smoking seems strange. Given potential confounding between previous smoking experience and smokeless tobacco use, the criteria ought to have been reversed, with an entry criterion of not having had a single puff and an outcome measure of one cigarette per day. Sticking with the prior definition might have resulted in no effect, given the lack of difference in cigarettes smoked per day among never users, past users, and current users of smokeless tobacco (Haddock et al., 2001; Table 2). Haddock and colleagues appear to have found a relationship between ever use of smokeless tobacco and smoking *experimentation*, but this is not evidence for an addiction-based gateway from smokeless tobacco to cigarettes.

Our finding of no smokeless tobacco gateway, controlling for other predictors, concurred with a 6-year prospective study ( $N=743$ ) by Griffin, Botvin, Doyle, Diaz, and Epstein (1999). In this study, regular chewing tobacco use in seventh grade was not associated with heavy smoking (pack-a-day) at follow-up (high school senior),  $OR=1.37$  (95%  $CI=.38-4.90$ ), controlling for school performance and experimentation with smoking, alcohol use, and marijuana use. Never regular use (one to five times) was associated with later smoking,  $OR=2.21$  (95%  $CI=1.11-4.42$ ), but this finding provides no evidence for a gateway effect. The authors concluded that those who experimented with *any* substances were more likely to be smoking. Hatsukami, Jensen, Boyle, Grillo, & Bliss (1999) found no relationship between smokeless tobacco use and later smoking. And another body of literature suggests effects in the opposite direction—that cigarette smoking can precede smokeless tobacco use (Cohen-Smith & Severson, 1999; Dent, Sussman, Johnson, Hansen, & Flay, 1987; Murray, Roche, Goldman, & Whitbeck, 1988). Other data suggest bidirectional relationships – that smokeless tobacco onset and cigarette onset can influence one another (e.g., Peterson, Marek, & Mann, 1989).

We also have found, looking at the order of tobacco product use in the 1987 National Health Interview Survey, that less than 25% of smokeless tobacco users were subject to potential gateway effects (Kozlowski, O'Connor, Edwards, & Flaherty, in press). That is, 75% of smokeless tobacco users either used smokeless tobacco exclusively or used cigarettes regularly before they ever used smokeless tobacco. Further, analysis

of age cohort effects in this sample indicated that an 18% *decrease* in smoking occurred at the same time as a 32% *increase* in smokeless tobacco use. Overall, the evidence for a unidirectional, causal smokeless tobacco-to-smoking gateway is weak at best.

*Nicotine is nicotine: Satisfied and unsatisfied nicotine users*

It makes sense that addiction to one form of a drug means that another form can satisfy that addiction. Cigarettes and smokeless tobacco both administer nicotine, by different methods. A bias appears to exist on the part of many scientists and policy makers in tobacco control that cigarettes represent the acme of nicotine delivery, while smokeless tobacco is a second-class product (i.e., less reinforcing than cigarettes). This bias may contribute to the readiness of these individuals to accept a gateway hypothesis. However, the use of any of these products delivers sufficient nicotine to sustain addiction (Benowitz & Henningfield, 2001; USDHHS, 1988). Nicotine users may have many reasons to switch between forms of administration. They may switch to satisfy nicotine needs (e.g., dissatisfaction with the pharmacological effects of smokeless tobacco) or simply because cigarette smoking has more social cache (e.g., more widespread use, greater social acceptability, no spitting). In our sample of adolescent males, the reason may be something as simple as dating someone who disapproves of smokeless tobacco use but does not mind smoking.

One might consider that users of smokeless tobacco who do not later use cigarettes represent a group of "satisfied customers." By *satisfied*, we mean that the user is receiving a desired level of pharmacological and behavioral effects from the product in a context that supports his or her use of the product. Those who use smokeless tobacco exclusively can hardly be considered victims of a gateway effect. There may also be situational consumers, who use smokeless tobacco and cigarettes depending on factors such as smoking restrictions. It is unlikely that the majority of such users would be gateway users (i.e., would not have come to cigarettes but for their use of smokeless tobacco).

If smokeless users who do not move to cigarettes are satisfied customers, then those who abandon smokeless tobacco for cigarettes can be considered "unsatisfied customers." In this group, gateway effects *could possibly* occur. However, numerous factors other than drug effects could predict the move of unsatisfied smokeless tobacco users to cigarettes. Context is likely a major determinant of tobacco product use (Kozlowski, 1982). Social learning reinforcement and peer influences are examples of contextual factors that might influence the type of nicotine delivery system chosen. Factors such as price, availability, and local

tobacco use norms also can influence use of smokeless tobacco or cigarettes (USDHHS, 1994). Various scenarios can be imagined in which individuals and groups of tobacco users are influenced in their choice of smokeless tobacco or cigarettes by myriad, complex contextual dynamics.

*Gateway effects and policy*

Undoubtedly a general pattern of progression through various substances of abuse exists (Kandel, 2002), but patterned associations among psychoactive substances are doubtless not invariant and can be the result of many noncausal factors (see Kandel & Jessor, 2002, for review). Arguments about smokeless tobacco products as gateways or starter products often neglect that a substantial portion of users *do not* progress to cigarette smoking. In the present study, only 23.9% of smokeless tobacco users went on to become regular smokers, which means that 76.1% did not. Clearly, smokeless tobacco use did not precede cigarette smoking for even a majority of smokeless tobacco users in this sample.

At least part of the appeal of gateway effects in policy settings may be its simplicity, focusing on single issues rather than complexities. If smokeless tobacco use were the main source of smoking initiation among adolescents, the means of addressing the problem through policy modification would be obvious. Policy makers might ban smokeless tobacco, attempt to restrict sales by increasing taxes, or raise the purchasing age. However, the policy implications of complex models of smoking initiation are less clear. For example, our results and others' (e.g., Choi et al., 1997; USDHHS, 1994) implicate school performance as a strong and consistent predictor of smoking. Perhaps the focus of smoking reduction policy could be to improve school quality through curriculum changes, smaller classrooms, and more dedicated and qualified teachers. In any case, such a policy would involve an array of complex factors, making it challenging to craft and evaluate. We believe that such an encompassing approach to analysis and policy making is needed.

## Conclusion

Recruitment to cigarette smoking is a complex, biobehavioral phenomenon in which multiple factors are influential (Kozlowski, Henningfield, & Brigham, 2001; USDHHS, 1994). Single-variable models can inflate the importance of predictors that are not significant when other covariates are included. Smokeless tobacco use is a minor predictor of later cigarette smoking in the TAPS sample, much less significant than prior smoking experimentation, school performance, and even motorcycle-riding experience. Even when the



sample was limited to those naïve to cigarettes, a causal gateway effect was made nonsignificant by the addition of a handful of other predictors.

Tomar (2003) concluded, "In summary, findings from this nationally representative cohort study suggest that smokeless tobacco may have been a starter product for cigarette smoking...." In light of our more complete multivariate analysis, we think that the more supportable conclusion is that smokeless tobacco use appears not to be an important predictor of smoking initiation in this nationally representative cohort. Unfortunately policy makers have already made use of Tomar's conclusions, to support that smokeless tobacco leads to cigarette smoking (e.g., Waxman & Durbin, 2002). To prevent smoking, a higher priority should likely be given to improving schools, reducing smoking by others in the household, and reducing depressive symptoms – at least, that is what our analyses of the TAPS dataset would lead us to believe.

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