



# Smoking Risks in Spain: Part III—Determinants of Smoking Behavior

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## *Abstract*

Using original survey data from Spain, this paper assesses the determinants of smoking behavior. This study examines the effect on smoking of the most diverse set of risk measures ever considered: lung cancer, relative lung cancer risks, lung disease, heart disease, relative heart disease risks, lost life expectancy to smokers, and various risk measures for passive smoking. Smoking measures include cigarette smoking, the number of cigarettes smoked, and pipe and cigar smoking. Primary smoking risks have a more consistent negative effect on smoking than perceived passive smoking risks.

**Key words:** cigarettes, smoking, risk perception

**JEL Classification:** D81, I10, K2

## **1. Introduction**

Why do people smoke? Antismoking advocates often claim that it is because smokers are ignorant of the risks. Yet the empirical results suggest that there appears to be widespread awareness. A second possibility is that risk beliefs do not affect individual behavior. This hypothesis will be explored using the most detailed

smoking risk data ever assembled. Our study is based on data from a national survey in Spain.

In the companion papers by Antoñanzas et al. (2000) and Rovira et al. (2000) we found that not only was there substantial risk awareness in Spain, but also a tendency to overestimate risks. These results parallel those found in the United States by Viscusi (1990, 1991, 1992, 1998). The data for Spain include a diverse set of measures of risk beliefs. For private smoking risks, there is information on perceptions of lung cancer risks, relative lung cancer risks, lung disease risks, heart disease risks, relative heart disease risks, and life expectancy losses. Moreover, there is also extensive data on perceptions of the hazards of passive smoking. Considering each set of risk measures will make it possible to disentangle the extent to which smoking decisions are responsive to perceived risks to the smoker as well as to the perceived risks to others.

The role of demographic factors that drive smoking behavior will also be of substantial interest, particularly with respect to education. The better-educated segment of society in the United States is less likely to smoke. However, this may not be the case in Spain, as smokers have higher levels of education than nonsmokers. Is this result due to differences in risk beliefs by education or differences in tastes and the social acceptability of smoking? In Antoñanzas et al. (2000) we found that the better educated had lower risk beliefs for risks to the smoker, and in Rovira et al. (2000) we found similar results for environmental tobacco smoke. Exploration of the demographic determinants of smoking will enable us to disentangle the source of the different composition of the smoking population in Spain as opposed to that in the United States.

The Spanish data also afford an especially rich characterization of the effect of different risk variables on smoking behavior. In addition to information on lung cancer risk beliefs, the survey also includes data on perceptions of heart disease risk and life expectancy losses due to smoking. These risk variables are available not only for primary risks to the smoker but also for secondary risks posed to others. Thus, it will be possible to assess the effect of perceptions of environmental tobacco smoke risk beliefs on smoking behavior as well as primary smoking risks.

While people may have beliefs regarding the potential hazards of smoking, do these beliefs also influence smoking behavior? If they do not, then this failure to have a behavioral impact might serve as an indicator that people do not internalize these risk beliefs in terms of their own behavior. This paper examines the effect of the different risk perception measures on smoking rates. In every instance there is a substantial significant effect of smoking risk perceptions on the probability that the respondent smokes. Moreover, an exploratory analysis taking into account the potential endogeneity of smoking risk beliefs and smoking status, using instrumental variables methods, generates even larger negative effects of smoking risk perceptions on the probability of smoking.

Section 2 summarizes the basic aspects of the data set, and Section 3 assesses the effect of smoking risk beliefs on the propensity to smoke cigarettes as well as other discrete choices, such as whether to be a nonsmoker. Section 3 considers the

quantity of cigarettes smoked decision and the linkage to risk beliefs. The role of risk beliefs for passive smoking and their effect on smoking rates and smoking intensity is the subject of Section 4. Section 5 concludes the analysis.

## 2. Sample characteristics

The sample for this article consists of respondents contacted by telephone interviews in Spain in 1997. The details of this random national survey are discussed in Antoñanzas et al. (2000). Table 1 summarizes the sample characteristics for the full sample and different smoking group subsamples. Of the 2,571 respondents, 844 are current smokers, 30 smoke cigars or pipes, 513 are former smokers, and 1,214 never have smoked. Almost half the sample is between the ages of 25 and 50, and the sample is evenly divided between men and women.

The differences by smoking status are especially noteworthy. Smokers tend to be younger than the other groups, as there are very few smokers over the age of 50—only 18 percent versus a sample average of 39 percent. Smokers in Spain have the highest level of education of any of the groups, as smokers average more than one year more of schooling than those who have never smoked. The role of educational background is particularly surprising given the smoking patterns in the United States, which has become increasingly concentrated outside the college educated ranks. American smoking behavior formerly was a pursuit of people in the higher income brackets, which is a phenomenon that still holds true in Spain. Blue-collar workers are, however, more likely to be smokers, which is consistent with the pattern in the U.S.

The data set also includes several regional variables as well as information on other consumptive behaviors that could pose risks or capture risk-related tastes. Smokers, for example, are much more likely to prefer whiskey to beer and are much less likely to abstain from drinking whiskey or beer. Coffee drinking is also more prevalent among smokers.

The smoking status breakdown indicates a higher rate of smoking than in the U.S. Overall, one-third of the respondents are smokers, and an additional one-fifth are former smokers. Those who smoke average 14 cigarettes per day.

The set of risk variables relies extensively on questions that pose the smoking risk in terms of the number of risk cases per 100 population. As discussed in Viscusi (1992), this formulation of the risk question is an effective way of eliciting risk beliefs in a telephone interview. Moreover, the risk metric is a meaningful quantitative variable that provides a well ordered measure of the degree of risk belief. Similarly, the life expectancy loss question also provides a quantitative index of the degree of smoking risk. The survey text is reported in Antoñanzas et al. (2000), which analyzes the primary risk data.

The final risk question pertains to smoking-related diabetes risk. There is no such established relationship in the scientific literature, as this question is intended to capture whether people have virulent antismoking beliefs that smoking is

Table 1. Table of means and standard errors

	Full sample		Current smokers		Cigar/pipe smokers		Former smokers		Never smokers	
	Mean	S.E.	Mean	S.E.	Mean	S.E.	Mean	S.E.	Mean	S.E.
Age 18-25	0.143	0.007	0.181	0.013	0.067	0.046	0.060	0.011	0.151	0.010
Age 25-50	0.473	0.010	0.637	0.017	0.267	0.082	0.460	0.022	0.363	0.014
Male	0.471	0.010	0.552	0.017	1.000	—	0.657	0.021	0.336	0.014
Years of schooling	9.955	0.068	10.637	0.106	10.200	0.667	10.035	0.159	9.448	0.103
Blue collar	0.425	0.010	0.540	0.017	0.367	0.089	0.357	0.021	0.373	0.014
Head of household	0.400	0.010	0.396	0.017	0.900	0.056	0.624	0.021	0.309	0.013
Town population between 100,000 and 1 million	0.311	0.009	0.350	0.016	0.300	0.085	0.292	0.020	0.292	0.013
Town population greater than 1 million	0.099	0.006	0.126	0.011	0.100	0.056	0.092	0.013	0.083	0.008
Metro area	0.475	0.010	0.547	0.017	0.433	0.092	0.435	0.022	0.442	0.014
Prefers whiskey to beer	0.188	0.008	0.284	0.016	0.400	0.091	0.216	0.018	0.110	0.009
Not a whiskey or beer drinker	0.413	0.010	0.289	0.016	0.300	0.085	0.337	0.021	0.530	0.014
Coffee drinker	0.736	0.009	0.835	0.013	0.700	0.085	0.708	0.020	0.678	0.013
No habit, maybe smoking	0.086	0.006	0.014	0.004	0.133	0.063	0.097	0.013	0.132	0.010
Current smoker	0.328	0.009	1.000	—	0.133	0.063	—	—	—	—
Former smoker	0.200	0.008	—	—	0.667	0.088	1.000	—	—	—
Never smoker	0.472	0.010	—	—	0.200	0.074	—	—	1.000	—
Cigarettes per day	4.578	0.174	14.001	0.357	1.200	0.745	—	—	—	—
Diabetes risk for smokers $\times$ 100	23.916	0.703	17.559	1.067	18.364	7.684	23.968	1.657	28.585	1.061
Lung cancer risk for smokers $\times$ 100	49.669	0.545	46.261	0.975	40.192	5.170	47.760	1.255	52.821	0.759
Lung disease risk for smokers $\times$ 100	54.255	0.550	51.195	0.981	47.609	5.434	53.349	1.271	56.768	0.771
Heart disease risk for smokers $\times$ 100	44.665	0.552	42.008	0.956	32.043	4.627	43.420	1.268	47.054	0.792
Loss in life expectancy due to smoking	10.936	0.172	8.481	0.310	6.800	1.635	11.456	0.361	12.423	0.240
Lung cancer risk for passive smokers $\times$ 100	24.660	0.437	21.316	0.724	17.926	2.955	22.652	0.961	27.840	0.651
Heart disease risk for passive smokers $\times$ 100	24.775	0.449	21.736	0.746	15.261	3.071	23.036	0.983	27.669	0.670
Loss in life expectancy due to passive smoking	5.563	0.146	4.059	0.241	2.300	0.975	5.657	0.327	6.568	0.216
Relative lung cancer risk	11.473	0.379	9.398	0.588	11.864	4.119	11.044	0.830	13.104	0.592
Relative heart disease risk	9.184	0.351	7.331	0.528	12.579	4.689	8.533	0.741	10.797	0.562
Number of observations	2,571		844		30		513		1,214	

dangerous without knowledge of the specific diseases. It also reflects possible inattention to the survey task. Those who have never smoked assess the diabetes risk as 11 percent greater than do current smokers.

Smoking risk beliefs for lung cancer and heart disease are quite high. In each instance, people believe that these risks are almost a 50-50 proposition. Smokers have somewhat lower risk beliefs, but they are still very high. Smokers' mean assessment of the lung cancer probability equals 0.46, the lung disease probability 0.51, and the heart disease probability 0.42. The assessed relative risks of lung cancer and heart disease for smokers as compared to nonsmokers also exceed 1.0. Smokers also believe that they will lose 8.5 years of life.

The estimated perceived cancer risk levels dwarf the actual risk values. Estimates based on studies by the U.S. Surgeon General reported in Viscusi (1992) indicate that the lung cancer mortality risk due to smoking is in the range of 0.06 to 0.13. Even the upper bound of this range is less than a third as great as the lung cancer risk beliefs of current smokers in Spain.

The relative lung cancer risk question is less instructive because it compounds knowledge of smoking related lung cancers and lung cancer for the nonsmoking population as well. Nevertheless, the relative lung cancer risk belief of smokers of 9.4 is quite high. The scientific reference point suggests that the relative lung cancer risk level depends on gender. Consider first the relative risk ratios, which do not take into account the size of the smoking population, as did the survey question. Results from the 1959–1964 American Cancer Society study (CPS-I) indicate that the relative risk of lung cancer for current smokers is 11.35 for males and 2.69 for females. The 1982–1986 follow-up of the American Cancer Society study (CPS-II) found a much higher relative cancer risk value of 22.36 for male smokers and 11.94 for female smokers. These values are for smokers age 35 years or more with a history of regular cigarette smoking.<sup>1</sup> To convert these values into the relative number of lung cancer cases, which is what the survey asked, one must multiply these values by 0.69 to establish the appropriate scientific risk reference point. Thus, there is little evidence of underassessment of relative risks overall. Moreover, the questions themselves are difficult because people may not properly understand that the background risk of lung cancer is in fact quite low, as cigarette smoking is the dominant contributor to lung cancer.

Smokers' assessed relative risk of heart disease of 7.3 greatly exceeds scientific estimates of the risk as well. Consider the relative risks of coronary heart disease for current smokers with a history of regular cigarette smoking who are age 35 or higher. These values pertain to relative risk probabilities so that conversion to relative number of cases requires multiplication by 0.69. Evidence from CPS-I indicates that for current male smokers this relative risk value is 1.8, and for current female smokers this value is 1.40. Evidence from CPS-II is quite similar, with the risk for males of 1.94 and females of 1.78.<sup>2</sup> Once again note that these estimates must be multiplied by 0.69 to make them ratios of the total number of smokers getting heart disease relative to the number of nonsmokers with heart

disease. The mean relative risk assessments for heart disease are many times greater than the actual relative risks.

Similarly, the life expectancy results indicate substantial overestimation of risk. Smokers believe that cigarettes shorten their life by 8.5 years, whereas estimates using reports by the U.S. Surgeon General indicate a loss of 3.6 to 7.2 years.<sup>3</sup> By analyzing a variety of these personal risk belief measures it will be possible to assess how various private risk beliefs affect smoking behavior.

The final set of statistics in Table 1 presents the perceptions of the risks of environmental tobacco smoke (ETS). Even for smokers, the estimates of these risks are well beyond even any upper bound estimates in the scientific literature. Smokers assess these risks as being much less than do never smokers, but they still believe the risks are considerable. Smokers believe that the lifetime lung cancer risk to others from ETS is 21 percent, whereas scientific estimates peg the annual risk at under 1/100,000.<sup>4</sup> The heart disease risk from ETS is 22 percent of the population, and ETS is believed to shorten the life expectancy of nonsmokers by four years. If this anticipated mass carnage in fact took place, it would be comparable to the Black Death plague of 14th century Europe, where approximately one-third of all Europeans died from the disease.<sup>5</sup> Clearly, the public is unable to distinguish the risks of primary and secondary smoke, as these are viewed as being comparable orders of magnitude. Rovira et al. (2000) explore the level and determinants of ETS risk beliefs in detail for this Spanish data set.

### 3. The effect of risk beliefs on discrete smoking status decisions

From an economic standpoint, one should be less likely to smoke the higher is one's assessed probability of an adverse outcome associated with smoking, *ceteris paribus*. This result is borne out in Viscusi (1992) for lung cancer, for which there is a strong powerful relationship. Table 2 reports probit estimates where the dependent variable is the 0–1 variable for whether the respondent is currently a smoker. Coefficients are transformed to reflect the influence of the variables on the marginal probability of smoking. The explanatory variables consist of demographic and taste variables considered earlier, as well as each of the three principal risk measures elicited in the survey. For heart disease and lung cancer, both absolute risk and relative risk variables are included. In each case, two sets of results are reported. The first set consists of probit estimates. The second set of estimates reports exploratory instrumental variables estimates where the instrument used for the risk variable is the response to whether smoking is related to diabetes. These IV estimates should be regarded as exploratory analyses to investigate the robustness of the OLS results.

More specifically, Table 2 reports the estimates for the probit model where the estimates are undertaken using the two stage procedure developed by Rivers and Quang (1988) in the case of the instrumental variables estimates. In the first stage, the endogenous variable is regressed on all the exogenous variables included or

Table 2. Smoking equation (probit model)

	OLS (1)	IV (2)	OLS (3)	IV (4)	OLS (5)	IV (6)	OLS (7)	IV (8)	OLS (9)	IV (10)	OLS (11)	IV (12)
Age 18-25	0.322 (0.042)**	0.400 (0.046)**	0.320 (0.043)**	0.370 (0.045)**	0.308 (0.042)**	0.266 (0.044)**	0.325 (0.042)**	0.454 (0.051)**	0.310 (0.045)**	0.230 (0.050)**	0.312 (0.046)**	0.218 (0.053)**
Age 25-50	0.290 (0.025)**	0.313 (0.025)**	0.300 (0.025)**	0.308 (0.025)**	0.279 (0.024)**	0.219 (0.030)**	0.294 (0.025)**	0.335 (0.027)**	0.300 (0.026)**	0.238 (0.031)**	0.302 (0.027)**	0.236 (0.033)**
Male	0.058 (0.023)*	0.004 (0.028)	0.053 (0.024)*	0.014 (0.026)	0.053 (0.023)*	0.017 (0.025)	0.057 (0.023)*	0.006 (0.027)	0.052 (0.024)*	0.059 (0.024)*	0.059 (0.025)*	0.063 (0.025)*
Years of schooling	-0.005 (0.003)	-0.012 (0.004)**	-0.005 (0.003)	-0.010 (0.004)*	-0.006 (0.003)*	-0.016 (0.004)**	-0.005 (0.003)**	-0.009 (0.003)**	-0.007 (0.003)*	-0.012 (0.004)**	-0.006 (0.004)**	-0.015 (0.004)**
Head of household	0.023 (0.025)	-0.059 (0.033)	0.032 (0.026)	-0.011 (0.029)	0.038 (0.025)	0.026 (0.025)	0.035 (0.025)	-0.044 (0.028)	0.029 (0.027)	0.028 (0.027)	0.029 (0.028)	0.020 (0.028)
Prefers whiskey to beer	0.111 (0.028)**	0.153 (0.031)**	0.090 (0.028)**	0.114 (0.029)**	0.105 (0.027)**	0.103 (0.027)**	0.108 (0.028)**	0.147 (0.030)**	0.110 (0.029)**	0.122 (0.030)**	0.093 (0.030)**	0.094 (0.030)**
Not a whiskey or beer drinker	-0.035 (0.023)	-0.030 (0.023)	-0.045 (0.023)	-0.037 (0.024)	-0.034 (0.023)	0.006 (0.025)	-0.036 (0.023)	-0.010 (0.024)	-0.038 (0.024)	-0.030 (0.024)	-0.046 (0.025)	-0.023 (0.026)
Coffee drinker	0.125 (0.023)**	0.103 (0.024)**	0.125 (0.023)**	0.120 (0.023)**	0.127 (0.022)**	0.068 (0.029)*	0.126 (0.023)**	0.086 (0.026)**	0.126 (0.024)**	0.109 (0.025)**	0.123 (0.025)**	0.109 (0.025)**
No habit, maybe smoking	-0.159 (0.042)**	-0.168 (0.041)**	-0.148 (0.045)**	-0.153 (0.044)**	-0.159 (0.041)**	-0.186 (0.037)**	-0.154 (0.042)**	-0.207 (0.036)**	-0.150 (0.047)**	-0.162 (0.045)**	-0.146 (0.049)*	-0.147 (0.048)
Lung cancer risk for smokers	-0.168 (0.036)**	-1.653 (0.421)**										
Heart disease risk for smokers	-0.132 (0.037)**	-1.231 (0.328)**										
Loss in life expect. due to smoking					-0.009 (0.001)**	-0.057 (0.014)**						
Lung disease risk for smokers							-0.175 (0.036)**	-1.893 (0.468)**				
Ln relative lung cancer risk									-0.152 (0.060)*	-2.042 (0.495)**		
Ln relative heart disease risk											-0.141 (0.071)*	-2.447 (0.623)**
R-squared	0.1304	0.1345	0.1281	0.1320	0.1446	0.1486	0.1311	0.1355	0.1228	0.1282	0.1205	0.1258
Observations	2,452	2,452	2,333	2,333	2,569	2,569	2,468	2,468	2,195	2,195	2,076	2,076

Dependent variable: dummy for current smoker; Instrument: dummy variable for "smoking related to diabetes"  
 \* Significant at 5% level (t-stat > 1.96); \*\* significant at 1% level, both are two-tailed tests. Coefficients on indicators for living in a city with more than 100,000 but less than 1 million inhabitants, living in a city with more than 1 million inhabitants, blue collar job and living in a metropolitan area are not reported.

excluded in the structural equation. In the second stage, a probit model is estimated in which the dummy dependent variable is regressed on the endogenous variable, the exogenous variables included in the structural equation, and the residuals from the first-stage.

This method is known as two-stage conditional maximum likelihood. This estimation procedure makes it possible to test statistically whether the endogenous variable is indeed endogenous. If it is not endogenous, then the coefficient on the residuals will be equal to zero when uncorrected standard errors are used. Thus, in terms of notation,

$$\text{Smoking}^* = a \text{ Risk} + Z_1 B + u \text{ (Probit)}, \quad (1)$$

and

$$\text{Risk} = ZD + v \text{ (First-stage regression)}, \quad (2)$$

where  $\text{Smoking}^*$  is not observed, only the dummy variable,  $\text{Smoking} = 1$  if smoker and 0 otherwise, is. Risk is the endogenous variable (the smoking risk assessment),  $Z_1$  is the vector of exogenous variables (all variables except for the risk assessment) and is a subset of  $Z$ , which also contains the instrumental variables, and  $B$  and  $D$  are column vectors. The instrumental variable is diabetes risk beliefs, which is not included in the Probit equation. The normally distributed errors are represented by  $u$ . When the coefficient on the residuals in the second stage is equal to zero, the standard errors are the usual probit standard errors.

All the effects reported in Table 2 have been converted into marginal probabilities of the influence. Thus, an increase in the lung cancer risk beliefs by 10 per 100 would decrease the smoking probability by .02 for the ordinary least squares estimates and by .17 for the IV estimates. Thus, there is a much stronger relationship shown by the IV estimates, which are consistently greater for each of the variables.<sup>6</sup>

The probit estimates reported at Table 2, column 1, imply that if all individuals had an assessment of the lung cancer risk involved with smoking equal to zero, the probability of smoking in the referred population would increase 7 percentage points, from 33% to 40%. The counterfactual based on the IV estimates implies that smoking rates would be as high as 94%, which is unrealistically high.

This pattern is remarkably consistent for all risk measures in Table 2. Heart disease risks, lost life expectancy, overall lung cancer risk, relative lung cancer risk, and relative heart disease risk are all negatively related to smoking behavior. Moreover, the IV estimates are always higher than the OLS estimates.

Notwithstanding the higher risk beliefs of those who are young, it is noteworthy that the younger age groups in Spain are more likely to smoke than their older counterparts. This result may be due in part to the fact that respondents over age 50 have often given up smoking or may not in fact be represented in the sample if they are severely ill or have experienced premature mortality. Males are more



likely to smoke in the OLS estimates, but these effects are not statistically significant in the IV estimates. Gender differences in risk taking, as found in Hersch (1996), more generally may be due in part to differences in risk beliefs. The results for education indicate that the better educated respondents are less likely to smoke controlling for other factors such as risk beliefs, but in some cases the effect is not statistically significant. Thus, we have a curious pattern with respect to education. As was shown in Table 1, smokers have higher levels of education. However, the better educated also have lower risk beliefs than do the less well educated. When it comes to the ultimate smoking decision, the higher smoking rates for those with more education do not stem from the independent influence of educational status alone. More likely contributory factors to the education effect are whether the respondent is a male or has other taste-related characteristics related to bearing risk. This negative influence of education is consistent with theoretical predictions to the extent that more education increases one's lifetime wealth. Since health is a normal good with a strong positive income elasticity, one would expect cigarette smoking to decline as one's lifetime wealth rose.

The risk-related taste variables reflect the fact that there is consistent risk taking behavior across a variety of domains. Respondents who prefer whiskey to beer and who drink coffee each are more likely to smoke.

The determinants of the composition of the smoking population are of interest as well. It is instructive to break out the cigarette smokers from those who smoke cigars or pipes. Table 3 presents the probit results for the probability of smoking cigarettes. Because cigarette smokers are the dominant component of the smoking population, these results closely parallel those in Table 2. The OLS estimates for

Table 3. Probit model for cigarette smokers who do not smoke cigars

	Coefficients (standard errors)					
	(1) OLS	(2) IV	(3) OLS	(4) IV	(5) OLS	(6) IV
Lung cancer risk for smokers	-0.163 (0.036)**	-1.612 (0.420)**				
Heart disease risk for smokers			-0.130 (0.037)**	-1.198 (0.327)**		
Loss in life expectancy due to smoking					-0.888 (0.116)**	-5.610 (1.354)**
Observations	2,452	2,452	2,333	2,333	2,569	2,569

\* Significant at 5% level (t-stat > 1.96); \*\* significant at 1% level, both are two-tailed tests. Coefficients on living in a city with more than 100,000 but less than 1 million inhabitants, living in a city with more than 1 million inhabitants, blue collar job, and living in a metropolitan area are not reported. Age 18–25, age 25–50, male, years of schooling, head of household, prefers whiskey to beer, coffee drinker, and no habit maybe smoking are not reported.

each of the risk variables is statistically significant, as are the instrumental variables estimates, which also are considerable larger.

The results for those who smoke cigars or pipes in Table 4 are somewhat different in that only the lost life expectancy variable is statistically significant at the usual levels, with the lung cancer risk being statistically significant at the 90 percent level (two-tailed test for the instrumental variables estimates). The weakness of the results for pipe and cigar smokers in all likelihood is attributable to the much smaller sample size of people in Spain who smoke pipes or cigars rather than cigarettes.

Table 5 assesses the determinants of being a former smoker, which are of particular interest because these people once believed that smoking was sufficiently attractive, but subsequently quit. The fact that these people quit smoking might suggest that they had high risk beliefs, whereas their initial smoking decision would have been consistent with lower risk beliefs. The probit results in Table 5 indicate that on balance there is no significant net effect of any of the risk belief variables on being a former smoker. Somewhat strikingly, this is the only smoking group for whom none of the risk belief variables is consequential.

Being a former smoker appears to be driven instead by other demographic and taste-related influences. Younger respondents are less likely to be former smokers, in part because they have just begun smoking. Males are more likely to be former smokers, as are heads of household. People who do not engage in other risky activities such as drinking whiskey or beer are less likely to be former smokers, perhaps in part because they never smoked at all.

Table 4. Probit model for cigar and pipe smokers

	Coefficients (standard errors)					
	(1) OLS	(2) IV	(3) OLS	(4) IV	(5) OLS	(6) IV
Lung cancer risk for smokers	-0.007 (0.013)	-0.334 (0.173)				
Heart disease risk for smokers			-0.019 (0.012)	-0.187 (0.116)		
Loss in life expectancy due to smoking					-0.093 (0.038)*	-1.105 (0.550)*
Observations	1,157	1,157	1,105	1,105	1,209	1,209

Note: No female in this data set smokes cigars. Therefore, female observations have to be dropped out from the Probit regression.

\* Significant at 5% level (t-stat > 1.96); \*\* significant at 1% level, both are two-tailed tests. Coefficients on living in a city with more than 100,000 but less than 1 million inhabitants, living in a city with more than 1 million inhabitants, blue collar job, and living in a metropolitan area are not reported. Age 18-25, age 25-50, male, years of schooling, head of household, prefers whiskey to beer, coffee drinker, and no habit maybe smoking are not reported.

Table 5. Probit model for former smokers

	Coefficients (standard errors)					
	(1) OLS	(2) IV	(3) OLS	(4) IV	(5) OLS	(6) IV
Age 18–25	–0.104 (0.025)**	–0.103 (0.027)**	–0.105 (0.025)**	–0.102 (0.027)**	–0.097 (0.025)**	–0.100 (0.025)**
Age 25–50	–0.023 (0.020)	–0.023 (0.021)	–0.040 (0.021)	–0.039 (0.021)	–0.028 (0.020)	–0.035 (0.024)
Male	0.087 (0.020)**	0.086 (0.023)**	0.087 (0.021)**	0.083 (0.023)**	0.094 (0.019)**	0.090 (0.021)**
Years of schooling	0.003 (0.003)	0.003 (0.003)	0.003 (0.003)	0.003 (0.003)	0.004 (0.003)	0.003 (0.003)
Head of household	0.107 (0.022)**	0.105 (0.029)**	0.110 (0.023)**	0.105 (0.025)**	0.105 (0.021)**	0.104 (0.021)**
Prefers whiskey to beer	–0.004 (0.021)	–0.003 (0.023)	0.005 (0.022)	0.007 (0.023)	–0.005 (0.021)	–0.005 (0.021)
Not a whiskey or beer drinker	–0.054 (0.019)**	–0.054 (0.019)**	–0.046 (0.020)*	–0.046 (0.020)*	–0.049 (0.019)**	–0.045 (0.021)*
Coffee drinker	–0.034 (0.022)	–0.035 (0.023)	–0.030 (0.023)	–0.031 (0.023)	–0.037 (0.022)	–0.044 (0.027)
No habit, maybe smoking	–0.008 (0.035)	–0.008 (0.036)	0.005 (0.038)	0.004 (0.038)	–0.008 (0.034)	–0.013 (0.035)
Lung cancer risk for smokers	0.017 (0.030)	–0.016 (0.330)				
Heart disease risk for smokers			0.021 (0.031)	–0.095 (0.260)		
Loss in life expectancy due to smoking					0.178 (0.092)	–0.338 (1.062)
Observations	2,452	2,452	2,333	2,333	2,569	2,569

\* Significant at 5% level (t-stat > 1.96); \*\* significant at 1% level, both are two-tailed tests. Coefficients on indicators for living in a city with more than 100,000 but less than 1 million inhabitants, living in a city with more than 1 million inhabitants, blue collar job and living in a metropolitan area are not reported.

This possibility is borne out by the results in Table 6 for the probability that the respondent never smoked. Each of the two younger age groups is less likely to have never smoked. Since the excluded category consists of those who are over age 50, it is the older respondents who are most likely to have never smoked as compared to these younger groups. Age-related increases in quit rates may account for some of this difference. Engaging in some smoking behavior does, however, increase with age through the middle age ranges. The lowest probabilities of never smoking are observed for those in the intermediate age range of 25–50.

The demographic profile of smokers also is consistent with what one would expect in terms of the gender roles. Males are less likely to have never smoked, which is consistent with the gender risk preferences found in Hersch (1996). Heads

Table 6. Probit model for "never smoked"

	Coefficients (standard errors)					
	(1) OLS	(2) IV	(3) OLS	(4) IV	(5) OLS	(6) IV
Age 18–25	–0.179 (0.039)**	–0.238 (0.041)**	–0.164 (0.040)**	–0.206 (0.040)**	–0.163 (0.039)**	–0.126 (0.041)**
Age 25–50	–0.256 (0.027)**	–0.276 (0.028)**	–0.244 (0.028)**	–0.251 (0.028)**	–0.237 (0.026)**	–0.175 (0.032)**
Male	–0.150 (0.025)**	–0.098 (0.030)**	–0.144 (0.026)**	–0.104 (0.029)**	–0.151 (0.025)**	–0.116 (0.027)**
Years of schooling	0.003 (0.004)	0.011 (0.004)*	0.003 (0.004)	0.008 (0.004)*	0.003 (0.003)	0.013 (0.005)**
Head of household	–0.154 (0.027)**	–0.075 (0.037)*	–0.164 (0.028)**	–0.121 (0.031)**	–0.164 (0.026)**	–0.152 (0.027)**
Prefers whiskey to beer	–0.129 (0.029)**	–0.163 (0.031)**	–0.114 (0.030)**	–0.137 (0.030)**	–0.120 (0.029)**	–0.118 (0.029)**
Not a whiskey or beer drinker	0.091 (0.026)**	0.086 (0.026)**	0.092 (0.026)**	0.085 (0.026)**	0.086 (0.025)**	0.046 (0.028)
Coffee drinker	–0.097 (0.028)**	–0.074 (0.029)*	–0.101 (0.028)**	–0.096 (0.029)**	–0.097 (0.028)**	–0.030 (0.034)
No Habit, maybe smoking	0.075 (0.050)	0.089 (0.050)	0.047 (0.051)	0.056 (0.051)	0.070 (0.049)	0.114 (0.050)*
Lung cancer risk for smokers	0.166 (0.040)**	1.602 (0.444)**				
Heart disease risk for smokers			0.109 (0.041)**	1.254 (0.0345)**		
Loss in life expectancy due to smoking					0.707 (0.125)**	5.676 (1.427)**
Observations	2,452	2,452	2,333	2,333	2,569	2,569

\* Significant at 5% level (t-stat > 1.96); \*\* significant at 1% level, both are two-tailed tests. Coefficients on indicators for living in a city with more than 100,000 but less than 1 million inhabitants, living in a city with more than 1 million inhabitants, blue collar job and living in a metropolitan area are not reported.

of household are less likely to have never smoked. The head of household effect is almost identical in magnitude to the effect of being a male respondent. The education variable is never statistically significant in the OLS regressions, though it is in the instrumental variables regressions, in which case higher rates of education increase the probability that the respondent has never smoked. The alcohol and coffee variables perform in the expected manner. Respondents who prefer whiskey to beer are less likely to have never smoked, as are coffee drinkers. Respondents who are not whiskey or beer drinkers are more likely to have never smoked.

The smoking risk belief variables have coefficients that are consistently statistically significant and of a magnitude that is almost identical to the coefficients in the smoking equation, but of opposite sign. Thus, the risk variables have roughly

equivalent but opposing influences on smoking behavior and on being a never smoker, but there is essentially no consistent influence on the intermediate category of former smokers, which seems consistent with their mixed status. The positive risk belief effect for never smokers and the negative risk belief effect for current smokers reflects the two sets of influences embodied in the former smoker estimates, where these imply no net risk belief effect.

The role of risk beliefs is consistent and strong in the two principal areas of interest—cigarette smoking and abstaining from cigarettes. These results hold even using instrumental variables estimates that are attempts to account for measurement error in the risk belief variable. Indeed, this adjustment increases the magnitude of the effect. What these results suggest is that people not only have risk beliefs that are substantial, but that these risk beliefs also appear to be influential in affecting the smoking decision.

#### **4. The determinants of smoking intensity**

Smoking is not a discrete activity that poses the same risk to all smokers. The extent of one's smoking and the types of cigarettes one smokes also will be influential in determining the health implications of smoking to the smoker. Although the survey from Spain did not include information on cigarette brands, it does include data on the number of cigarettes that respondents smoke per day. For nonsmokers, this number is zero, leading to the use of a Tobit estimation procedure for the equations in which the number of cigarettes smoked per day is the dependent variable.

Table 7 reports these Tobit estimates for a baseline equation as well as for three equations including the principal risk variables. The number of cigarettes smoked per day is highest for those age 25–50 and somewhat less for those age 18–25. Because the comparison group is those age 51 and over, these results suggest that it is the young and middle aged segments of the population who consume the greatest number of cigarettes. The gender-related results are consistent with the earlier findings. Men tend to smoke more cigarettes per day than do women, as do people who are the heads of household.

Smoking is not, however, the exclusive pursuit of blue-collar workers and the less well educated as it seems to be in the United States. Indeed, blue-collar status has no statistically significant effect in any of the equations on the number of cigarettes smoked. Years of schooling also has no statistically significant influence in Equation 1, which does not include any risk belief variables. However, once one takes into account the level of risk beliefs, education has a negative effect on the number of cigarettes smoked. Thus, one obtains the expected negative education smoking linkage once one controls for the correlation of risk beliefs and other background factors.

The taste variables also perform in a consistent manner following that in the earlier findings. People who prefer whiskey to beer, indicating a greater predilec-

Table 7. Tobit model for number of cigarettes per day

	Coefficients (standard errors)			
	(1)	(2)	(3)	(4)
Constant	-30.488 (2.598)**	-24.138 (2.775)**	-24.614 (2.791)**	-23.740 (2.668)**
Age 18-25	13.558 (2.119)**	14.483 (2.116)**	14.659 (2.149)**	13.557 (2.101)**
Age 25-50	16.157 (1.451)**	16.514 (1.470)**	17.210 (1.508)**	15.682 (1.438)**
Male	4.899 (1.242)**	5.105 (1.241)**	4.584 (1.258)**	4.681 (1.231)**
Years of schooling	-0.300 (0.174)	-0.414 (0.176)*	-0.446 (0.180)*	-0.411 (0.173)*
Town population between 100,000 and 1 million	1.540 (1.361)	1.792 (1.355)	2.282 (1.391)	1.529 (1.349)
Town population greater than 1 million	4.651 (1.986)*	4.563 (1.998)*	6.721 (2.047)**	4.256 (1.968)*
Blue collar	1.099 (1.172)	1.063 (1.171)	1.648 (1.186)	0.911 (1.161)
Head of household	3.219 (1.338)*	2.548 (1.343)	2.881 (1.369)*	3.156 (1.328)*
Live in a metropolitan area	2.618 (1.317)*	2.412 (1.310)	1.340 (1.350)	2.356 (1.305)
Prefers whiskey to beer	4.632 (1.355)**	4.873 (1.349)**	3.750 (1.369)**	4.623 (1.342)**
Not a whiskey or beer drinker	-2.187 (1.268)	-1.746 (1.267)	-2.198 (1.287)	-1.835 (1.259)
Coffee drinker	9.315 (1.450)**	8.647 (1.435)**	8.414 (1.454)**	8.821 (1.440)**
No Habit, maybe smoking	-10.074 (3.395)**	-11.150 (3.491)**	-10.408 (3.525)**	-10.529 (3.392)**
Lung cancer risk for smokers		-0.099 (0.019)**		
Heart disease risk for smokers			-0.088 (0.020)**	
Loss in life expectancy due to smoking				-0.448 (0.062)**
Observations	2,569	2,452	2,333	2,569

Notes: \* significant at 5% level; \*\* significant at 1% level. Both are two-tailed tests.

tion toward hazardous consumption, tend to smoke more cigarettes. Coffee drinkers also tend to smoke more cigarettes. People who report no such habits of alcohol drinking or coffee drinking tend to smoke fewer cigarettes, as one would expect.

The three risk belief variables all perform in a consistent manner. Higher lung cancer risk beliefs diminish the number of cigarettes smoked per day, with an effect that is very similar in magnitude to the influence of the heart disease risk variable. Similarly, the loss in life expectancy due to smoking also diminishes the

number of cigarettes smoked per day. People respond to higher risk beliefs by both reducing smoking probabilities and reducing cigarettes consumed if one is a smoker.

The role of risk beliefs in relationship to the number of cigarettes smoked is also apparent in the distributional breakdowns in Table 8. The first column gives the level of the risk broken into different risk intervals. The first set of statistics reports the mean number of cigarettes per day for each of the specified lung cancer risk intervals as well as the standard error of these mean values. The final two columns of the table present the mean and the standard error of the mean of the number of cigarettes per day smoked by respondents in each of the heart disease risk intervals. In each instance, there is a general downward trend in the mean number of cigarettes smoked as the risk level increases. For example, the greatest number of cigarettes smoked is for the category of respondents who believe that the risk is less than .05 for lung cancer and heart disease. The main pattern in the table that does not bear out this relationship is the final entry for respondents who believe that the risk equals 1.0, but these estimates have a large standard error that is reflective of the comparatively small number of respondents who believe that lung cancer or heart disease is a certain outcome.

Table 9 presents comparable results for the mean number of cigarettes smoked per day as a function of the life expectancy loss. The relationship for the full sample is quite flat because high levels of life expectancy loss that are anticipated decrease smoking altogether. Focusing only on current smokers in the final set of estimates in Table 9 indicates that there is a slight decline in the number of cigarettes smoked per day, but it is nowhere near as dramatic as is apparent for the

*Table 8.* The conditional means of cigarettes per day as a function of lung cancer and heart disease risk perception

Current smoker	Lung cancer risk		Heart disease risk	
	Mean	Standard error	Mean	Standard error
0 ≤ Risk < 5	21.73	3.32	20.58	3.38
0 ≤ Risk < 10	16.24	2.27	14.79	2.11
0 ≤ Risk < 20	18.12	1.50	16.03	1.37
0 ≤ Risk < 30	13.35	1.13	15.68	1.21
0 ≤ Risk < 40	12.78	1.28	13.62	1.20
0 ≤ Risk < 50	15.37	1.36	13.02	1.19
0 ≤ Risk < 60	13.06	0.97	12.88	0.74
0 ≤ Risk < 70	14.21	1.38	14.35	1.74
0 ≤ Risk < 80	12.27	1.25	10.78	1.25
0 ≤ Risk < 90	13.46	1.15	12.97	1.29
0 ≤ Risk < 100	13.02	1.45	13.34	1.66
Risk = 100	15.92	2.57	16.92	4.26
Total number in group	802		766	

Table 9. The conditional means of cigarettes per day as a function of life expectancy loss perception

Life expectancy loss for smokers	Total		Current smoker	
	Mean	Standard error	Mean	Standard error
Risk < 0	9.42	1.71	15.41	1.90
Risk < 1	8.81	0.63	15.41	0.84
Risk < 3	7.75	1.62	14.59	1.87
Risk < 5	5.13	1.70	13.67	2.74
Risk < 10	4.72	0.44	14.02	0.89
Risk < 15	4.35	0.34	14.12	0.82
Risk ≥ 15	3.35	0.30	14.41	0.92
Total number in group	2,571		844	

full sample overall. These overall tabulations are consistent with the empirical estimates in Table 7, which is that each of the different risk belief variables has a significant negative influence on the number of cigarettes people smoke per day.

### 5. Smoking behavior and environmental tobacco smoke

In most risky economic decisions the effects of the risks on the individual are the primary factors influencing behavior rather than effects on others. Indeed, the terminology “externalities” typically refers to effects on others that by their very nature will not be adequately incorporated into the structure of the economic decision maker, leading to a potential source of market failure.

The potential hazards associated with secondhand smoke differ in many respects from the standard economic textbook externality problem. Secondhand smoke is not akin to midnight dumping of hazardous wastes in secret locations. The smoke is visible and has a strong odor as well. Thus, people are aware of the presence of this externality. Moreover, the ones who are more likely to suffer from this externality are the ones who spend more time close to the smoker. It is very plausible that most of the effects of second-hand smoke on one’s relatives, children in particular, and friends are altruistically internalized by the smoker. There may also be a nuisance value of the externality that may actually have a larger economic value than the actual health effects.

In addition to being an externality that is known to those exposed to it, environmental tobacco smoke (ETS) has also been the object of substantial public attention. Spain, for example, features an on-package hazard warning dealing with environmental tobacco smoke: “Protect your children; do not allow them to breath tobacco smoke” and “Smoking harms those around you.” As in the United States, cigarette smoking has also been the subject of substantial public controversy and social pressure with respect to ETS exposures. The question to be explored in this



section is the extent to which it is the risk to the smokers as opposed to the passive smoking risk that influences smoking behavior. Sorting out these influences is difficult because of the substantial correlation between the primary smoking risk beliefs and the passive smoking risk beliefs explored in Rovira et al. (2000). The correlation between primary and passive lung cancer risks is 0.60; for primary and passive heart disease risks, the correlation is 0.61; whereas for primary and passive life expectancy losses the correlation is 0.63. Those numbers are in the same order of magnitude as the correlation between primary lung cancer and heart disease risk, 0.66.

Table 10 presents the probit estimates for the analog of the earlier results in Table 2. The specifications are identical to those before with the exception that it includes both the primary smoking risk variable as well as the passive smoking risk belief variable when each of these is present. Equation 1 in Table 10 reports the estimates for lung cancer risk beliefs. Each of these risk belief coefficients is statistically significant and has a negative effect on the propensity to smoke. Moreover, the magnitude of the coefficients is almost identical. Because the mean values of lung cancer are twice as great as the mean value of the perceived lung cancer risk for passive smokers, the primary smoking risk variable has a larger effect on the probability of smoking than does the ETS risk belief variable. The estimates imply that if primary lung cancer risk assessments were equal to zero, keeping passive risk constant, smoking rates would increase from 32.8% to 37.8%. If passive risk was zero, keeping primary risk constant, smoking rates would be 35.2%. If both risk assessments were zero, predicted smoking rates would be 40.2%. In terms of sorting out the different risk effects, one-third of the effect of lung cancer risk beliefs is through the passive smoking risk belief, while an additional two-thirds of the effect of individual risk perceptions is through the primary smoking risk belief variable. Comparison of these results with those in Equation 1 in Table 2 implies a similar relative influence based on the magnitude of the primary lung cancer smoking risk coefficient, which is  $-0.17$  in Table 2 as compared to  $-0.11$  once the passive smoking variable is included in the analysis.

The results for heart disease in Table 10 are similar, but the passive smoking risk coefficient is not statistically significant as it has a large standard error. Controlling for ETS risk beliefs, heart disease risk perceptions for the smoker still have a significant negative influence on smoking behavior. Much the same pattern of results is also reflected in the lost life expectancy estimates in Equation 3, as it is the primary risk to the smokers that is the statistically significant influential negative influence on smoking behavior.

Table 11 reports the parallel results for the number of cigarettes that the respondent smokes per day. Panel A reports the Tobit estimates for the determinants of the quantity of cigarette smoking, where the risk variable included pertains to lung cancer. The specifications analyzed parallel those for the probability of cigarette smoking, with the principal addition being that these results also explore whether being a household head affects the role of either personal risk beliefs or risk beliefs regarding the externality. In each of the four sets of

Table 10. Probit model for smoking

	Coefficients (standard errors)		
	(1)	(2)	(3)
Age 18–25	0.323 (0.043)**	0.324 (0.043)**	0.308 (0.042)**
Age 25–50	0.289 (0.025)**	0.301 (0.026)**	0.279 (0.024)**
Male	0.056 (0.023)*	0.050 (0.024)*	0.053 (0.023)*
Years of schooling	–0.007 (0.003)	–0.006 (0.003)	–0.006 (0.003)
Town population between 100,000 and 1 million	0.041 (0.026)	0.055 (0.027)*	0.036 (0.025)
Town population greater than 1 million	0.073 (0.041)	0.120 (0.044)**	0.067 (0.039)
Blue collar	0.016 (0.022)	0.032 (0.023)	0.019 (0.022)
Head of household	0.019 (0.025)	0.038 (0.026)	0.038 (0.025)
Live in a metropolitan area	0.049 (0.025)*	0.024 (0.026)	0.047 (0.024)*
Prefers whiskey to beer	0.111 (0.028)**	0.092 (0.028)**	0.105 (0.027)**
Not a whiskey or beer drinker	–0.037 (0.023)	–0.044 (0.024)	–0.034 (0.023)
Coffee drinker	0.123 (0.023)**	0.127 (0.023)**	0.127 (0.022)**
No habit, maybe smoking	–0.157 (0.043)**	–0.147 (0.045)**	–0.159 (0.041)**
Lung cancer risk for smokers	–0.110 (0.045)*		
Lung cancer risk for passive smokers	–0.124 (0.058)*		
Heart disease risk for smokers		–0.095 (0.048)*	
Heart disease risk for passive smokers		–0.085 (0.061)	
Loss in life expectancy due to smoking			–0.009 (0.001)**
Loss in life expectancy due to passive smoking			0.000 (0.002)
Observations	2,408	2,299	2,569

Notes: \* significant at 5% level; \*\* significant at 1% level. Both are two-tailed tests.

estimates, the perceived lung cancer risk to the smoker has a statistically significant negative effect on the number of cigarettes smoked. The ETS lung cancer risk also has a negative influence on smoking behavior, controlling for the risk to the smoker. Similarly, interacting the lung cancer risk to passive smokers variable with whether the respondent is a household head has a significant effect in Equation 3,

Table 11. Tobit model for number of cigarettes per day

	Coefficients (standard errors)			
	(1)	(2)	(3)	(4)
<b>PANEL A</b>				
Lung cancer risk for smokers	-0.084 (0.018)**	-0.052 (0.022)*	-0.068 (0.019)**	-0.081 (0.023)**
Lung cancer risk for passive smokers		-0.070 (0.029)*		
Lung cancer risk for smokers × head of household				-0.006 (0.036)
Lung cancer risk for passive smokers × head of household			-0.086 (0.042)*	
Observations	2,448	2,404	2,404	2,448
<b>PANEL B</b>				
Heart disease risk for smokers	-0.070 (0.019)**	-0.043 (0.023)	-0.061 (0.020)**	-0.070 (0.024)**
Heart disease risk for passive smokers		-0.058 (0.030)		
Heart disease risk for smokers × head of household				0.000 (0.038)
Heart disease risk for passive smokers × head of household			-0.045 (0.041)	
Observations	2,329	2,295	2,295	2,329
<b>PANEL C</b>				
Loss in life expectancy due to smoking	-0.428 (0.056)**	-0.439 (0.073)**	-0.412 (0.061)**	-0.327 (0.070)**
Loss in life expectancy due to passive smoking		0.021 (0.089)		
Loss in life expectancy due to smoking × head of household				-0.259 (0.113)*
Loss life expectancy due to passive smoking × head of household			-0.070 (0.115)	
Observations	2,564	2,564	2,564	2,564

Notes: \* significant at 5% level; \*\* significant at 1% level. Both are two-tailed tests.

where the magnitude of this influence is only slightly larger than the comparable coefficient in Equation 2 without the household head interaction. The personal lung cancer risk to smokers does not alter its magnitude for the household heads in the sample, as is shown by the results in column 4.

Panel B presents comparable results for perceived risks of heart disease. These results are somewhat weaker than the estimates for lung cancer. However, the heart disease risk to smokers variable is consistently significant, though only based on a one-tailed test at the 95 percent confidence level for Equation 2. Likewise, the heart disease risk for passive smokers has a negative influence on the number of cigarettes smoked per day in Equation 2 (one-tailed test). The other interactions with household head are not statistically significant for heart disease.

In the case of the life expectancy results shown in Panel C, the personal loss in life expectancy to the smoker has a strong negative effect on the number of cigarettes smoked per day. The other variables are not consequential except for the interaction of the personal risks to smokers with being a head of the household. The inclusion of this interactive term in Equation 4 indicates that the role of the perceived life expectancy loss diminishes the number of cigarettes smoked per day by a greater amount if one is the household head.

## 6. Conclusion

Examining the determinants of smoking behavior provides a different context for assessing smoking than in the United States. The informational environment is different than in the United States, though there are many parallels, particularly in terms of having formal information provision on cigarette packaging. In addition, the rates of smoking in Spain are higher than the rates of smoking in the United States. Most importantly, the social demographics of the smoking population are quite different. Examining the determinants of smoking behavior consequently affords a glimpse into how risk beliefs alter smoking within a social context that is different than what prevails in the United States, which has been the focus of most of the existing literature.

The results presented here indicate that there is widespread awareness of a wide variety of risks to the smoker. Following the standard principles of economic theory, as the perceived risk of an activity increases, individuals' willingness to participate in the activity should diminish. This result is borne out quite strongly for a wide variety of smoking measures. Unlike existing studies that have been restricted to examination of lung cancer risks, mortality risks, and life expectancy loss, this study considered a wide range of risk measures including lung cancer risks, heart disease risks, lung disease risks, life expectancy loss, and relative risks of lung cancer and heart disease. Each of these risk beliefs had a consistent negative influence on smoking behavior. Moreover, because these data included information pertaining to smoking participation as well as the number of cigarettes smoked it was possible to assess how each of these magnitudes was influenced by the risk variables. Both cigarette smoking probabilities and the number of cigarettes smoked respond to risk beliefs.

Examination of the smoking trends also helped solve a puzzle in the Spanish smoking data. Unlike the United States, it is the better educated members of the Spanish population who are more likely to smoke. What the results here indicate is that, controlling for their level of risk beliefs and other demographic factors, better educated respondents are not more likely to smoke and do not smoke more cigarettes. Indeed, for many equations, the opposite result is the case.

Smokers' belief in the risks to others may also affect smoking behavior. These secondhand smoke consequences to others have a substantial significant effect on individual smoking rates and the extent of smoking. Particularly in the case of lung

cancer risk beliefs, if the person perceives that there will be a risk created by passive smoking, then there will be a negative influence on the extent of smoking behavior. These results suggest that smokers themselves exert a self-regulating effort to diminish the exposure of others to ETS. This self-regulating behavior may, of course, be attributable not only to attitudes of the smoker but also be the result of social pressures and public risk awareness efforts with respect to environmental tobacco smoke. Other ETS risk measures were not influential.

Overall, the results suggest a coherent picture of the Spanish smoking population. All measures of risk belief are high, often well beyond actual risk levels, and these beliefs deter smoking behavior. There also appears to be an effort to internalize the external risks of ETS for the widely publicized ETS lung cancer risk.

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### Notes

1. These statistics are from the U.S. Department of Health and Human Services (1989), p. 148–151.
2. These statistics are taken from the U.S. Department of Health and Human Services (1989), p. 148–151.
3. See Viscusi (1992), p. 80.
4. See Rovira et al. (2000) for documentation.
5. See *The Economist*, Dec. 31, 1999, p. 7 and “The Black Death; Plague and Economics,” pp. 33–34.
6. In the first-stage regressions, which are not reported, the relationship between the instrumental variable and the risk assessment is statistically significant at any usual level, with t-statistics always above 4.0. Hausman tests provide evidence that risk assessments are indeed endogenous with statistical significance at the 1% level.

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