DISCUSSION PAPER SERIES

IZA DP No. 1849

Taxes, Cigarette Consumption and Smoking Intensity

Jérôme Adda
Francesca Cornaglia

November 2005

Forschungsinstitut
zur Zukunft der Arbeit
Institute for the Study
of Labor

Taxes, Cigarette Consumption and Smoking Intensity

Jérôme Adda
University College London
and Institute for Fiscal Studies

Francesca Cornaglia
University College London,
CEP and IZA Bonn

Discussion Paper No. 1849
November 2005

IZA

P.O. Box 7240
53072 Bonn
Germany

Phone: +49-228-3894-0
Fax: +49-228-3894-180
Email: iza@iza.org

IZA Discussion Paper No. 1849
November 2005

ABSTRACT

Taxes, Cigarette Consumption and Smoking Intensity*

This paper analyses the compensatory behavior of smokers. Exploiting data on cotinine concentration – a metabolite of nicotine – measured in a large population of smokers over time, we show that smokers compensate tax hikes by extracting more nicotine per cigarette. Our study makes two important contributions. First, as smoking more intensively a given cigarette is detrimental to health, our results question the usefulness of tax increases. Second, we develop a model of rational addiction where agents can also adjust their intensity of smoking and we show that the previous empirical results suffer from severe estimation biases.

JEL Classification: I1

Keywords: taxes, smoking, cigarettes, addiction

Corresponding author:

Francesca Cornaglia
Department of Economics
University College London
Gower Street
London WC1E 6BT
United Kingdom
Email: f.cornaglia@ucl.ac.uk

* We are grateful to a number of seminar participants and to David Card, Ken Chay, Christian Dustmann, Martin Jarvis, Steve Machin, Adriana Lleras-Muney, Costas Meghir, Bernard Salanié and three anonymous referees for helpful discussions and comments and to William Evans for supplying us with data on US tobacco prices and taxes. Funding through the Nuffield Foundation is gratefully acknowledged.

I. Introduction

The economic literature on smoking has considered cigarettes mostly as a homogenous consumption product. In this setting, individuals decide on how many cigarettes to smoke, conditional on prices, income and possibly past and future consumption. A number of empirical studies, surveyed in Chaloupka and Warner (2000), have analyzed the effect of prices on quantities smoked. They have consistently found a significant price effect on quantities, which differs in the short run and the long run, due to addiction (see for instance Becker et al 1994, and Gruber and Kosegi 2001). These studies have been influential for the design of public health policies aimed at reducing smoking, by advocating higher excise taxes.

Our paper makes two contributions to that literature. First, we show that smokers compensate increases in excise taxes by smoking more intensively a given cigarette. Hence the effect of taxes on smoking uncovered in the literature so far is partly misleading. This type of public intervention may not achieve the health benefits that are to be expected based on the observation of the number of cigarette smoked. Second, we show that ignoring compensatory behaviors leads to severe bias in the estimation of the rational addiction model.

Cigarettes are not a homogenous product. They vary in their tar and nicotine yields and in size. Moreover, studies in epidemiology and social medicine have shown that smokers can regulate the amount of nicotine they extract from a given cigarette. This can be done by varying the number of puffs, by varying the degree and the length of inhalation or by blocking the ventilation holes on the filter. While the effect of prices or taxes on the number of cigarettes smoked has been extensively documented in the literature, little attention has been devoted to the other margins of adjustment: cigarettes differ in

observable characteristics, and may be smoked in different ways. [1] An important exception is the work by Evans and Farrelly (1998) and by Farrelly et al (2004) who analyze the effect of prices on the type of cigarette smoked. They find that smokers compensate tax increases by buying cigarettes with higher tar and nicotine yield. Another exception is the contribution by Harris (1980) in which he presents a theoretical model predicting the compensating behavior.

In this paper, we explore the effects of public intervention through excise taxes on the intensity of smoking, defined as the amount of nicotine extracted per cigarette smoked. We exploit a new source of data, which reports not only the number of cigarettes smoked and their type, but also the cotinine concentration (a marker of nicotine intake) in saliva, for a large number of smokers over time in the US. Analyzing the variations in cotinine concentrations in body fluids allows us to take into account compensating behaviors: cotinine concentrations are influenced not only by the size or the type of the cigarette smoked but also by the way the cigarette is smoked. The use of cotinine concentration is novel in the economic literature. As a proxy for nicotine intakes, it is an important variable for the study of addiction, which has been the focus of the literature. It is also a good measure of the exposure to tar and carcinogens, which have important health effects.

We first develop a simple model where an agent can choose the number of cigarettes and the intensity with which they are consumed. To achieve a particular level of nicotine in the body, there is a trade-off between more cigarettes, which are costly, and more effort to extract nicotine. This model supports the compensation mechanism that has been described in the epidemiological literature. [2] Our empirical findings are consistent with this explanation. We find that increases in taxes over the nineties have led to an increase

[1] The epidemiological literature has paid more attention to compensatory smoking behavior. For instance, Kozlowski et al (1980) show that smokers of low yield cigarettes block ventilation holes in the filter. Jarvis et al (2001) show that smokers compensate low yield cigarettes by smoking more intensively. Russell et al (1975), Gritz et al (1983) and DeGranpre et al (1992) use experiments in which they restrict the access to cigarettes or vary the nicotine yield of cigarettes and show that smokers compensate. Wagenknecht et al (1990), McCarthy et al (1992), Caraballo et al (1998) document the differences in smoking intensity across ethnic groups.

[2] See note 1.

in the intensity of smoking. Our results control for a number of observed characteristics, including state and year fixed effects. We find that a one percent increase in taxes increases the smoking intensity by about 0.4%. We also show that other factors contribute to higher smoking intensity, such as socio-economic position and race. Individuals higher up the social hierarchy tend to smoke less intensively. African-Americans extract more nicotine per cigarette, a fact which is consistent with differences in lung cancer rates by race.

Our findings have two main implications. First they question the effect of public intervention through increases in excise taxes. If on the one hand higher excise taxes lead to more quits and, perhaps, to lower initiation rates (although DeCicca et al, 2002, question this fact), our results suggest that they also lead smokers to increase their intake of nicotine (and therefore tar) which may be detrimental to their health. Smoking more intensively a cigarette up to the filter leads the smoker to inhale more dangerous chemicals, and has been shown to cause cancer deeper into the lung (Thun et al 1997).

Second, our findings have implications for the estimation of dynamic models of smoking. The models of rational addiction, empirically derived in Becker et al (1994) and Chaloupka (1991), ignore compensatory behaviors and use the number of cigarettes smoked as a dependent variable. If the focus of the literature is on the health effects of smoking, or on addiction due to nicotine, then cotinine concentration is a better proxy for smoking than the number of cigarettes consumed. By ignoring smoking intensity, these models are mis-specified. We evaluate the bias introduced by estimating a model of rational addiction, which ignores compensatory behaviors, when the true model consists of an agent that chooses both the number of cigarettes and the intensity in order to optimize the consumption of nicotine. We show that the standard rational addiction models in which compensatory behaviors are ignored are subject to bias. We derive the bias and evaluate it empirically and we show that it can be substantial, potentially invalidating the results in the literature.

The remainder of the paper is structured as follows. Section II, develops a simple model of smoking and smoking intensity to motivate the empirical analysis. We also outline the estimation strategy. Section II.D contains a description of our data sets and some descriptive statistics. Section III, investigates the relationship between smoking, smoking intensity and excise taxes. Section III.B, is about other determinants of smoking intensity. Section IV evaluates the bias in the rational addiction literature, when the choice of smoking intensity is ignored. Finally, Section V concludes and discusses the implications of our results.

II. Model, Methodology and Data

A. A Simple Model of Smoking and Smoking Intensity

To fix ideas, we present a simple model of smoking and smoking intensity. We develop a more elaborate and dynamic model in Section IV.A.

Suppose that the utility of an agent depends on nicotine, n, a composite good, q, and on the intensity of smoking, i. The intensity of smoking is defined as the amount of nicotine extracted per cigarette. The agent gets a disutility of increasing smoking intensity for at least two reasons. First, the last part of the cigarette tastes worst than the first part, given that the tobacco has been heated up by smoke. Second, for a given intake of nicotine, an increased intensity means that the agent smokes fewer cigarettes per day. This leads to peaks and troughs in nicotine levels during the day, which are both unpleasant. If price was not an issue, the agent would prefer to smoke often but with lower intensity.

The problem of the agent is:

$$\max_{n,i} u(n,i,q) \quad \text{subject to} \quad \begin{cases} n = c.i \\ y = pc + q \end{cases} \tag{1}$$

where p is the relative price of tobacco, c the number of cigarettes, i the intensity of smoking, q a composite good, and y is total income. The agent maximizes the utility by choosing both the amount of nicotine and the intensity. There is a trade-off between

smoking in a less intensive way and the number of cigarettes to purchase. We assume that the utility function is increasing and concave. Solving for the first order conditions and totally differencing with respect to prices, intensity and the number of cigarettes, we get:[3] $\frac{dc}{dp} \leq 0$ and $\frac{di}{dp} \geq \text{or} \leq 0$. An increase in prices leads to a reduction in the number of cigarette consumed. The effect on the intensity of smoking is ambiguous and depends on the level of nicotine, relative to preferences. When the nicotine level (*n)* is large (i.e. $n > -(\partial u / \partial n)/(\partial^2 u / \partial n^2)$), then $\frac{di}{dp} \geq 0$ and $\frac{di}{dy} \leq 0$. This means that these smokers compensate higher prices by smoking more intensively. Individuals who can afford spending more on cigarettes smoke less intensively. If nicotine intake is the outcome of interest, either because of its addictive nature or because of the health effects of other substances inhaled at the same time, then a regression of the number of cigarettes on prices will miss the real effect. We are more likely to observe compensatory behaviors in regular or heavy smokers.

We next outline our empirical methodology to uncover evidence of compensatory behavior.

B. Cotinine as a Proxy for Smoking Intake

The empirical implementation of the model presented in the previous section requires the observation of both the number of cigarettes smoked, and the amount of nicotine consumed. Nicotine consumption is not easy to measure. Nicotine can be measured in blood samples, but it is unstable, and it is metabolized within few hours after consumption. Nicotine is, however, transformed into cotinine, a more stable component.

Cotinine has a half-life in the body of about 20 hours and is, therefore, a biological marker often used as an indicator of regular smoking.[4] It can be measured in, among

[3] Appendix A provides details on the computation of the results.

[4] The elimination of cotinine is slow enough to allow comparing measurements done in the morning or in the afternoon. As our data set provides the time of the examination, we present more evidence on this point in section III.

other things, saliva or serum. Cotinine is also a good proxy for the intake of health threatening substances in cigarettes. The nicotine yield of a cigarette is, in fact, highly correlated with the level of tar and carbon monoxide, which causes cancer and asphyxiation.[5] Cotinine is, therefore, a good indicator of health hazards due to smoking.

The novelty of our analysis is to use cotinine concentration to evaluate models of smoking, and the effect of public intervention.

C. Methodology

The empirical section tests and compares the effect of excise taxes on the number of cigarettes smoked as well as on the amount of nicotine consumed. We use taxes instead of prices as the latter are potentially endogenous. Moreover, from a policy point of view, the relevant variable is the excise tax. For an individual *i*, living in state *s* and in period *t*, we relate the log number of cigarettes to excise taxes in that state and year. The regression also conditions on a set of individual characteristics, X_{ist}, as well as state and time dummies:

(2) $$\log c_{ist} = \alpha_0 + \alpha_1 \log tax_{st} + \alpha_2 X_{ist} + \alpha_s + \alpha_t + u_{ist}$$

Similarly, we relate the log concentration of cotinine to the same variables:

(3) $$\log \cot_{ist} = \beta_0 + \beta_1 \log tax_{st} + \beta_2 X_{ist} + \beta_s + \beta_t + v_{ist}$$

The (log) intensity is given by log cot_{ist} -log c_{ist}. If taxes have similar relative effects on the number of cigarette smoked and on the concentration of cotinine ($\alpha_1=\beta_1$), the intensity of smoking does not react to tax changes. The effect of taxes on smoking is identified through within state variations once an aggregate time effect has been removed. We adjust the standard errors for clustering at state and year level.

Classical measurement error affecting smoking (especially salient for the number of cigarette smoked) does not bias the estimation of the effect of taxes, as the variable in

[5] Based on our data set, which report for some years the nicotine, tar and carbon monoxide yield of each cigarette, the correlations between nicotine and both tar and carbon monoxide are high, 0.96 and 0.85.

question is the dependent one. If the measurement error is not classical, then the estimation is unbiased provided that the measurement error is uncorrelated with taxes, once state and time effects have been controlled for.

The estimation of models (2) and (3) by ordinary least squares may be biased if selection occurs. It may be possible that individuals with low smoking intensity are more likely to quit smoking as taxes increase. As taxes increase, the composition of the pool of smokers would shift towards more intensive smokers. If this is the case, the OLS coefficient of the effect of taxes on intensity would be biased upward. To control for this endogeneity, one may use an instrument affecting smoking participation but not smoking intensity directly. Finding such an instrument proves, however, to be a difficult task.

We deal with the effect of a potential change in composition in two ways. First, we assess the robustness of our OLS results by restricting our sample to individuals less likely to quit. We use either individuals who started smoking at an early age, or heavy smokers. The assumption is that those individuals are either more addicted to tobacco or have higher preferences for smoking. Second, we treat the selection out of smoking more formally. We bound the OLS coefficients using worst case bounds as in Manski (1994). This methodology does not require any identification restrictions. It incorporates information on the likelihood of quitting between two periods as well as on the distribution of smoking intensity in those two periods. The method allows investigating whether taxes have an effect on intensity, over and above any selection effects. We refer the reader to Appendix B for a formal description.

D. The Data

We use data from the National Health and Nutrition Examination Survey (NHANES III and NHANES 1999-2000). This is a large repeated cross-sectional survey, representative of the US non institutionalized civilian population. It provides information from 1988 to 1994 and 1999-2000[6], for approximately 20,000 individuals, aged seventeen and above. The data set reports information on the age, sex, race, education and occupation of the individual, as well as information at the household level such as family composition, income, geographical location or welfare eligibility. The smoking habits are recorded,

[6] No data was collected between 1995 and 1998.

including the self-reported number of cigarettes smoked per day, time since quitting and consumption of other tobacco goods. In addition, the cotinine concentration is measured by taking saliva samples. The data set also reports details on the cigarettes smoked, the nicotine, the tar and the carbon monoxide concentration, as well as the brand name after 1999. The later waves contain less information on individual characteristics, which restricts our analysis in some cases. Hence our analysis mainly relies on data from 1988 to 1994 (NHANES III).

[Figure 1**]**

We merge information on cigarette excise taxes. We use information from the Tax Burden on Tobacco, which reports taxes by state and year. We deflate the variable using the consumer price index. Figure 1 displays the state excise tax, adjusted for inflation, between 1988 and 1994 for each state. Most of the variation is cross-sectional, where taxes can vary by about 80%. Taxes vary from about three cents in North Carolina to 55 cents in Minnesota in 1988. Taxes have increased over the period of analysis from 28 cents per pack to 34 cents. There are differential variations over time across states that we exploit to identify the effect of excise taxes.

Table 1 provides a summary statistic of our data sets. Smokers are categorized according to their self-reported smoking status. We restrict our sample to individuals aged seventeen and above. Younger individuals are not asked the number of cigarette they smoke. We also restrict our sample to regular smokers of cigarettes only, and we discard those who smoke a pipe, cigars or use chewing tobacco or snuff (64 individuals).[7]

[Table 1]

The average number of cigarettes smoked over the observation window is about 19. The average level of cotinine concentration is about 230ng/ml. The average cotinine concentration per cigarette is therefore around 12ng/ml. Cotinine levels in non-smokers

[7] There is no information in NHANES III on the use of nicotine patches. In NHANES 99-00, only 15 out of 9965 use a nicotine patch, only half of them are active smokers.

are small at about 0.4ng/ml. The exposure one gets from passive smoking is tiny compared to the average level of cotinine in smokers. Smokers come proportionally more from low or medium education groups, as well as from unskilled and manual occupations. Men, as well as young individuals, are more likely to smoke.

Figure 2 plots the average quantity of cotinine concentration (ng/ml) in saliva as a function of the number of cigarettes consumed. Cotinine levels are increasing with the number of cigarettes smoked. The relationship is linear up to 10 cigarettes a day and then levels off. Heavy smokers smoke more frequently but less intensively, to keep the concentration of nicotine at a high level while avoiding an overdose. A nicotine overdose induces unpleasant effects such as nausea, headaches and dizziness (see for instance the British National Formulary, 2004, for evidence).

[Figure 2]

The graph also plots the 10% and 90% percentiles of cotinine concentration within each level of cigarette consumption. A striking feature in the graph is the large heterogeneity in cotinine levels. Some smokers reporting 5 to 9 cigarettes per day can absorb more nicotine than most heavy smokers. Conversely, heavy smokers can absorb no more nicotine than light smokers under half a pack a day. This heterogeneity can come from three different sources, measurement error in the number of cigarette smoked, individual variation in the metabolism of nicotine, and behavioral differences in the way a cigarette is smoked. Note that, as discussed in section C, measurement error and heterogeneity in metabolism are not a likely source of bias in our analysis as there is little reason to believe that they are correlated with state excise taxes, especially conditional on state and time dummies.

III. Determinants of Smoking Intensity

A. The Effect of Taxes on Smoking and Smoking Intensity

We now evaluate the effect of taxes on consumption using as a measure of smoking, either the quantities of cigarette smoked, the cotinine levels or the intensity of smoking defined as the cotinine levels per cigarette.[8]

As outlined in section II, we first present the estimates of model (2) and (3) using OLS. We then present bounds of the effect of taxes on smoking intensity to evaluate the robustness of the OLS results to a selective change in the composition of the pool of smokers.

1. OLS Results

All the regressions control for age, sex, race, education levels, household size, other smoking members in the family, as well as year and state dummies. Table 2 presents the results for the elasticity of smoking intensity, the number of cigarettes and cotinine levels. We present the effect of other characteristics in section IV.B.

[Table 2]

A one percent increase in taxes leads to 0.47% increase in the intensity of smoking (column 1). The tax elasticity of the number of cigarettes is estimated at -0.20, while the elasticity of cotinine is positive, but not significantly different from zero.[9] The compensatory behavior apparent in the data is in line with the implication of the model presented in section II.A. Smokers seem to adjust not only the number of cigarettes smoked but also the amount of nicotine they extract.

The model also predicts that compensatory behaviors are more likely for those with higher levels of cotinine. Column 2 reports the results for those with a cotinine

[8] One can convert tax elasticities into price elasticities by noting that a one percent increase in taxes leads to a 0.15% increase in prices over the period of analysis.

[9] As a robustness check, we also estimate the tax elasticity using data on cigarette sales instead of self-reported data. We find a similar elasticity equal to -0.25 (0.05), over the period 1985-2001.

concentration higher than the median (about 200ng/ml). Interestingly, we find that the elasticity of the intensity of smoking is higher at around 0.55, which means that heavier smokers are more prone to compensatory behaviors.

Columns 3 to 5 in Table 2 assess the robustness of these results. The third column includes additional conditioning variables. We add other possible determinants of cotinine concentration. The first one is height as taller individuals may have a lower concentration of cotinine per cigarette smoked. Height is arguably exogenous to compensatory behavior. Another possible choice of measure is body mass index, but smoking intensity may be correlated with diet and other health behavior affecting weight. However, including the body mass index in the model does not change the results. The regression in column 3 also controls for the time and the day of the examination. Individuals surveyed in the morning may have lower levels of cotinine. The results are not different from the previous regression in column 1. As argued in section II.C, the OLS regression is unbiased if there is no selection into or out of smoking. The selection into smoking is simple to control for, given that we have information on the age of smoking initiation. We estimate our model including only those who started smoking before 1988, the first year of our survey. Column 4 presents these results. The estimate of the elasticity of smoking intensity is equal to 0.43, which is not different from the result in column 1. Column 5 conditions the sample on early starters. Individuals who started smoking early in life are less likely to quit smoking, either because they are more addicted, or because their preference towards smoking is higher. We include only individuals who started smoking before age 17 (the median starting age in our sample).[10] The results are comparable to those in column 1, suggesting that a change in the composition in the pool of smokers is not a likely explanation to the effect of excise taxes on smoking intensity.[11]

[10] Using information in our data set, we checked that early starters are less likely to quit. Results available upon request. Controls include age, sex, education, occupation, race, state of residence and year effects.
[11] As a robustness check we interacted the taxes with years. We did not find a clear pattern indicating a change in the composition of smokers over time.

2. A Non Parametric Approach to Selection

We use the methodology developed in Manski (1994) to bound the effect of taxes on the intensity of smoking. We compare the distribution of the intensity of smoking within states, across two periods of time. As some smokers have quit, we bound the intensity in the second period using worst case bounds, where all quitters could be of either a low or a high intensity type. The test compares the intensity in the first period to the upper bounds in the second period.

We pool all states which have seen an increase in taxes over the period 1988-1990 and 1990-1994 as the computation of the cumulative distribution of smoking intensity is rather data demanding.[12]

[Figure 3]

Figure 3 presents the cumulative distribution of smoking intensity in the first period (labeled 1989) together with the bounds for the second period. For an intensity of smoking of 18 ng/ml and above, there is clear evidence that the intensity increased between these two periods, as the cumulative distribution function is above the upper bound. Below that level, the test is inconclusive. The evidence suggests that the increase in smoking intensity is not due to selection but driven by higher excise taxes. As predicted in Section II.A, compensatory behaviors appear to be more important in heavy smokers.

3. Compensatory Behavior: Cigarette Type and Behavioral Adjustment

The previous results establish that smokers partly compensate increases in taxes. This can be done either by changing the type of cigarettes smoked and/or by changing the way cigarettes are smoked. Our measure of smoking intensity encompasses both types of behavior. The analysis conducted so far does not allow looking at the effect of taxes on behavior. We do this now by separating the importance of the type of the cigarette and

12 We could also compare the distribution of smoking intensity in states which have seen a decrease in taxes to see if smokers decreased their intensity of smoking. However, the sample size is not big enough to compute a meaningful test.

the behavioral adjustment. We conduct such decomposition using the NHANES data for the year 1999-2000 (Table 3). The survey also reports for these years additional characteristics of the cigarette smoked, in particular the length of the cigarette and the nicotine yield. A caveat is that, due to the limited time span, we can not control for state fixed effects, as there is very little variation left when both time and state fixed effects are present. Instead, the regression includes four regional dummies (NorthEast, MidWest, South, and West, as defined by the US Census).

[Table 3]

Columns 1 and 2 in Table 3 provide such decomposition, Column 1 reports the average elasticity for that time period. On this sample we also find significant evidence of compensatory behavior, although the magnitude is smaller than for the period 1988-1994. The elasticity of smoking intensity is estimated at 0.11. Controlling for cigarette characteristics, the effect of taxes on smoking intensity is reduced to 0.10 (Column 2). This suggests that (a small) part of the effect on smoking intensity is mediated through changes in the type of cigarettes smoked as suggested by Evans and Farrelly (1998) and Farrelly et al (2004). However, the results also suggest that smokers change the way they smoke a given cigarette.

B. Individual Determinants of Smoking Intensity

Section II.A showed that beside taxes, other individual characteristics such as income may influence the intensity of smoking. We now explore the determinants of smoking and smoking intensity in relation to individual characteristics, focusing especially on the effect of socio-economic position and on race differences. Table 4 presents the determinants of smoking using either the daily quantities of cigarettes, the cotinine concentration, or the ratio of cotinine over the number of cigarettes. All the estimations are based on models (2) and (3). For ease of interpretation, all the dependant variables are expressed in logs.

[Table 4]

1. Socio-Economic Position and Smoking Intensity

Section II.A emphasized the role of income. Income is unfortunately not well measured in NHANES, which may lead to attenuation bias. A second issue is that except in the case of borrowing constraints, permanent income would be more important than current income in explaining differences in smoking intensity. We therefore include in the model markers of permanent income and wealth, proxied by education levels, and the size of the house where the individual lives. This latter variable is the only one in NHANES which can proxy wealth. We also consider other variables which capture life style (e.g. living in urban area).

The first column of Table 4 displays the regression coefficients of the logarithm of the number of cigarettes smoked. Income and education are not significant, although the coefficients have an expected negative sign. Individuals living in larger houses tend to smoke less. The second column of Table 4 displays the effect of these variables on cotinine concentration. Individuals with higher education level have significantly less cotinine. A one year difference in education leads to a three percent difference in cotinine levels. As a result, although individuals with different education levels appear to smoke roughly the same quantity, they differ in the intensity of smoking. Individuals with higher education extract less nicotine from a given cigarette (column 3). An additional year of education leads to a two percent decrease in smoking intensity. The effect of the size of the house on cotinine levels is stronger than for the number of cigarettes. Individuals in larger houses smoke less intensively: for each additional room in the house, smoking intensity decreases by five percent. These results suggest that permanent income and wealth are correlated with smoking intensity as predicted in section II.A.

2. Smoking Intensity and Race

Race is an important determinant in cigarette smoking. While Whites smoke more than any other racial groups in terms of number of cigarettes[13], African-Americans have the highest level of cotinine. This means that they also have the highest intensity of smoking:

[13] Whites smoke 40% more than Hispanics, and about 5% more than African-Americans.

they extract 56% more nicotine per cigarette than Hispanics and Whites. The race difference in cotinine has been pointed out in the epidemiological literature, for instance in McCarthy et al. (1992), Benowitz et al. (1999) or Caraballo et al. (1998) (and the references within).

The race difference can be due to differences in the type of cigarettes (African-Americans smoke on average cigarettes which are heavier and mentholated)[14] or to the way they smoke a given cigarette. Table 4, Column 4, presents the results of the determinants of (log) of the intensity of smoking estimated from NHANES 1999-2000. The sample size is small (less than 600 observations), but the data set reports for these years additional information on the cigarette consumed, its length, nicotine yield and whether it has a filter or is mentholated. Including those characteristics does not change substantially the results. It implies that the race difference does not primarily operate through differences in the type of cigarettes.

The large difference in smoking intensity across race groups suggests that it should have a sizable effect on health if increasing the intensity of smoking is detrimental to health. The medical literature indeed indicates that African-American men have by far the highest incidence of lung cancer (e.g. Miller et al 1996, and Campbell et al 2000), despite the fact that Whites smoke more cigarettes than both African-Americans and Hispanics.[15] One concern might be that the lung cancer statistics relates to an older population with respect to the one for which we observe the smoking behavior. However, further inspection of our data indicates that the intensity of smoking among African-Americans is higher than for Whites, across all birth cohorts. If smoking habits are persistent, then differences in lung cancer rates by race may be explained by differences in smoking intensity in addition to differences in the number of cigarette smoked.

[14] From NHANES 1999-2000, the prevalence of mentholated cigarettes among smokers is 13% in Whites and 73% in African-Americans. The average nicotine yield is 1.06 mg for African-Americans and 0.87 mg for Whites.

[15] According to the National Cancer Institute (2004), the rate is about 120 per 100000, while the rate is at 79.4 for whites and only 46.1 for Hispanics.

3. Other Determinants

Our results show no significant differences between gender and age groups (Table 4). Smoking intensity increases with family size, which may reflect an income effect as the measures of income and socio-economic status have not been scaled by household size.

Those who attend church smoke about seventeen percent less, probably reflecting a better life style. These individuals have also lower cotinine concentrations, although the effect is of a smaller magnitude than for the number of cigarette smoked. As a result the smoking intensity is about eight percent higher for those who attend church. This may be due to compensatory effect in an environment where smoking is discouraged.

IV. Implications for the Estimation of Dynamic Models of Smoking

In the last two decades, the economic literature has devoted a lot of effort to model the addictiveness of tobacco and to evaluate its magnitude. It is an important issue as it allows measuring the short and long run effect of public policies, especially the response of smoking to a change in prices. The economic literature on smoking behavior uses the number of cigarettes consumed as a measure of smoking. In this section, we argue that this leads to misleading conclusions if the focus of that literature is on the effect of public intervention on health, or on the addictive nature of nicotine and other components of tobacco.

We evaluate the bias introduced by estimating a model of rational addiction using the number of cigarettes smoked (and therefore ignoring compensatory behavior) when the true model consists of an agent optimizing the consumption of nicotine by choosing the number of cigarettes to smoke and the intensity. We first derive a model that extends the framework in Becker and Murphy (1988) by allowing differences in smoking intensity. Then we quantify the bias using our data set.

A. Rational Addiction and Smoking Intensity

We extend the simple model outlined in section II.A. An agent derives utility through the consumption of nicotine, n_t, and a composite good, q_t. Because of addiction, the agent also derives utility from past nicotine consumption, n_{t-1}. In addition, the agent experiences a disutility of increasing the intensity of smoking, defined as the amount of nicotine taken out of a given cigarette.

The plan of the agent is:

(4) $$\max_{\{i_t, n_t, q_t\}} \sum_t \delta^t u(n_t, n_{t-1}, q_t, i_{t-1})$$

Subject to a budget constraint:

(5) $$a_t = Ra_{t-1} + y_t - p_t c_t - q_t$$

where a_t is savings, R is (one plus) the interest rate, y_t is current income, c_t is the number of cigarette purchased and p_t is the relative price of tobacco.

We assume a quadratic utility function as in Becker et al (1994). Appendix C derives the first order conditions. It can be written (after some simple algebra):

(6) $$n_t = \theta_0 + \theta_1 \frac{p_t}{i_t} + \theta_2 n_{t-1} + \theta_3 n_{t+1} + u_t$$

As in Becker et al (1994), this equation relates smoking (here the amount of nicotine) to past and future values of smoking, as well as current prices. The difference is that prices are adjusted for smoking intensity.

The parameters $\Theta=[\theta_0, \theta_1, \theta_2, \theta_3]$ are functions of the underlying parameters of the utility function and satisfy $\theta_1<0$, $\theta_2>0$ and $\theta_3>0$ (see Appendix C). An increase in prices leads to a decrease in smoking. Current smoking is reinforced by past and future smoking.

B. Bias when Estimating the Standard Rational Addiction Model

The standard rational addiction model predicts, from the first order condition, that the consumption of cigarettes can be expressed as (Becker et al 1994):

(7) $$c_t = \gamma_0 + \gamma_1 p_t + \gamma_2 c_{t-1} + \gamma_3 c_{t+1} + v_t$$

As in (6), the coefficients $\Gamma=[\gamma_0, \gamma_1, \gamma_2, \gamma_3]$ appearing in the first order condition depend on the underlying parameters of the utility function. This equation is usually estimated by instrumental variable techniques given the lagged and lead dependent variable structure (Chaloupka 1991, Becker et al 1994). The instruments for past and future consumption that are commonly used are leads and lags of prices or excise taxes.

The point of this section is to understand the biases introduced by the estimation of (7) when the underlying model is in fact one where agents adjust also their smoking intensity as in (6).

For simplicity of notation, denote $\mathbf{X}=[1, p_t , c_{t-1}, c_{t+1}]$ and $\mathbf{Y}=[1, p_t/i_t, n_{t-1}, n_{t+1}]$ two Tx4 matrices which contain the explanatory variables used in each equations. Both equations can be written more compactly in matrix notations:

(8) $$n = \mathbf{Y}\Theta + \mathbf{u}$$

(9) $$\mathbf{c} = \mathbf{X}\Gamma + \mathbf{v}$$

Let $\boldsymbol{Z}$ be a Tx4 matrix of instruments consisting of lags and leads of excise taxes. The instrumental variable estimator of Γ can be written as:

(10) $$\hat{\Gamma}_{IV} = (\mathbf{Z}'\mathbf{X})^{-1}\mathbf{Z}'\mathbf{c}$$

Using the fact that $c=n/i$ and equation (8), we get:

(11) $$\hat{\Gamma}_{IV} = (Z'X)^{-1}Z'Y/i\Theta + (Z'X)^{-1}Z'u/i$$

The IV estimation of equation (9), as estimated in Becker et al (1994) and Chaloupka (1991), leads to biased estimates of the original parameters **Θ.** The bias is composed of two terms, one is multiplicative, and the other additive. The additive part is equal to zero

in expectation.[16] The multiplicative part is not equal to the identity matrix if compensatory behaviors exist. The multiplicative part of the bias involves the matrices:

$$Z'X = \begin{bmatrix} T & \sum tax_t & \sum c_{t-1} & \sum c_{t+1} \\ \sum tax_t & \sum tax_t p_t & \sum c_{t-1} tax_t & \sum c_{t+1} tax_t \\ \sum tax_{t-1} & \sum tax_{t-1} p_t & \sum c_{t-1} tax_{t-1} & \sum c_{t+1} tax_{t-1} \\ \sum tax_{t+1} & \sum tax_{t+1} p_t & \sum c_{t-1} tax_{t+1} & \sum c_{t+1} tax_{t+1} \end{bmatrix} \text{ and } Z'Y/i = \begin{bmatrix} \sum \frac{1}{i_t} & \sum \frac{p_t}{i_t^2} & \sum c_{t-1} \frac{i_{t-1}}{i_t} & \sum c_{t+1} \frac{i_{t+1}}{i_t} \\ \sum \frac{tax_t}{i_t} & \sum \frac{tax_t p_t}{i_t^2} & \sum c_{t-1} tax_t \frac{i_{t-1}}{i_t} & \sum c_{t+1} tax_t \frac{i_{t+1}}{i_t} \\ \sum \frac{tax_{t-1}}{i_t} & \sum \frac{tax_{t-1} p_t}{i_t^2} & \sum c_{t-1} tax_{t-1} \frac{i_{t-1}}{i_t} & \sum c_{t+1} tax_{t-1} \frac{i_{t+1}}{i_t} \\ \sum \frac{tax_{t+1}}{i_t} & \sum \frac{tax_{t+1} p_t}{i_t^2} & \sum c_{t-1} tax_{t+1} \frac{i_{t-1}}{i_t} & \sum c_{t+1} tax_{t+1} \frac{i_{t+1}}{i_t} \end{bmatrix}$$

Note that if the intensity of smoking is constant over time, the two matrices are equal[17] and there is a direct mapping between the parameters in both models.

The matrix Z'X is easily evaluated using our data set as it involves leads and lags of prices, taxes and cigarette consumption at one point in time. The first two columns of the matrix Z'Y/i are also straightforward to compute given the information on prices, taxes and smoking intensity. The last two columns involve the ratio of smoking intensity over two consecutive periods and require panel data on smoking intensity. We approximate the ratio of smoking intensity using the model in section III[18]. This allows us to evaluate the multiplicative bias that typically arises when estimating the standard rational addiction model.

We compute this bias using data on the number of cigarettes, cotinine levels and prices and taxes. We augment models (8) and (9) to control for individual and area characteristics. The control variables are age, sex, race, education, year and state specific

[16] The proof requires a second order Taylor approximation and is available upon request from the authors.
[17] The two matrices are equal up to a scalar multiplying the first two columns which will be subsumed into the constant term and the coefficient associated with prices.
[18] From equations (2) and (3), the log of the intensity of smoking is expressed as $\log i_{ist} = \phi_0 + (\beta_1 - \alpha_1) tax_{st} + \phi_2 X_{ist} + \phi_s + \phi_t + w_{ist}$. Taking the first difference of this equation over time, one gets an equation relating the ratio of smoking intensity to changes in taxes, individual characteristics and the parameters estimated in section III. The caveat is that the estimated equation derives from a static model, and not from the fully dynamic model specified in this section.

effects. The instruments are current excise taxes as well as one lag and lead of this variable.

[Table 5]

Table 5 displays the results for the matrix $(Z'X)^{-1}Z'Y/I$. The matrix is different from the identity matrix, which implies that the rational addiction literature suffers from biases. From Table 5, it is difficult to get a sense of its importance. We therefore compute the vector of coefficients $\hat{\Theta} = (Z'Y/i)^{-1}Z'X\hat{\Gamma}$ implied by a set of estimated coefficients taken from the literature on rational addiction smoking. The estimates of these effects are taken to be γ_1=-1.5, γ_2=0.5, γ_3=0.5.[19] These correspond to a model estimated with the number of cigarettes as a proxy for smoking, appearing to display addiction and forward looking behavior (as both γ_2 and γ_3 are positive). The implied coefficients $\hat{\Theta}$ for model (6) are displayed in Table 6.

[Table 6]

The implied coefficients $\hat{\Theta}$ are usually large and contradict the model. In particular, price effects are positive, and the effect of future smoking is large and negative. These results cast some doubts on the ability of the rational addiction model to explain smoking.

V. Conclusion

The novelty of our analysis is to use biomarkers to study health behaviors and how they respond to economic variables and government intervention. We focus on the intensity of smoking, defined as the ratio of cotinine concentration to the number of cigarettes smoked. This is a broad definition of smoking, which encompasses many compensating behaviors. We can, therefore, better analyze how smokers alter their behavior in response of changes in state level excise taxes.

[19] Chaloupka (1991) estimates the effect of prices, past and future smoking to be γ_1=-1.67, γ_2=.486, γ_3=0.338. In Becker et al (1994), similar estimates are found: γ_1=-1.39, γ_2=.42, γ_3=0.13

Using variation within states and over time in excise taxes, we show that smokers increase the intensity of smoking in response to increases in taxes. They adjust not only the number or the type of cigarette smoked (Evans and Farrelly 1998, and Farrelly et al 2004 show that smokers switch to cigarettes with a higher tar and nicotine yield), but also the nicotine intake per cigarette. Our results, therefore, question the usefulness of excise taxes as a tool to regulate smoking intakes, especially as the medical literature has shown that increasing the intensity of smoking is detrimental to health (Thun et al 1997).

The existence of compensatory behaviors implies that the estimation of models of smoking behavior, such as the rational addiction model, may be subject to misspecification bias. We characterize this bias, and evaluate its magnitude using the NHANES data set. We show that the bias can be substantial, invalidating previous results of the literature.

Our results call for more analysis using similar data to understand better the complexity of smoking, and its implication for health. The economic literature on smoking has much to gain from exploiting information on cotinine concentration. This new source of data can be used to better understand the process of addiction in which nicotine plays an important role. Cotinine may also be helpful in understanding differences in quitting rates across ethnic or socio-economic groups, because of differences in smoking intensities. Finally, a natural extension of the present work is the analysis of passive smoking, whose relevance for public health has been acknowledged by a large medical and epidemiological literature.

Appendix A : Static Model

Consider an agent with utility *u(n,I,q)*, where *n* is the consumption of nicotine, *I* is the intensity of smoking and *q* is a bundle of other goods. The intensity of smoking is the amount of nicotine taken out per cigarette, defined by the relationship *n=c.i*. The budget constraint is *y=pc+q*, where *p* is the relative price of a cigarette and *y* is income or total expenditures.

We make the following assumptions on the first and second derivatives of the utility function: $u_1 \geq 0, u_{11} \leq 0, u_2 \leq 0, u_{22} \leq 0, u_3 \geq 0, u_{33} \leq 0$

We impose the utility function to be decreasing with intensity, at an increasing rate, as increasing the intensity of smoking is less pleasant at higher level of intensity. The first order conditions are:

(A-1)
$$\begin{aligned} cu_1 + u_2 &= 0 \\ iu_1 - pu_3 &= 0 \end{aligned}$$

First differentiating the system with respect to *c*,i, *p* and *y* gives:

(A-2)
$$\begin{aligned} dc[u_1 + nu_{11}] + di[c^2u_{11} + u_{22}] &= 0 \\ dc[i^2u_{11} + p^2u_{33}] + di[u_1 + nu_{11}] &= dp[u_3 - pcu_{33}] + pu_{33}dy \end{aligned}$$

Hence:

(A-3)
$$\frac{dc}{dp} = \frac{(u_3 - pcu_{33})(c^2u_{11} + u_{22})}{(u_1 + nu_{11})^2 - (c^2u_{11} + u_{22})(i^2u_{11} + p^2u_{33})}$$

Given that $(u_1 + nu_{11})^2 - (c^2u_{11} + u_{22})(i^2u_{11} + p^2u_{33})$ is negative for a maximum to exist, dc/dp<0.

(A-4)
$$\frac{di}{dp} = \frac{(u_1 + nu_{11})(u_3 - pcu_{33})}{(u_1 + nu_{11})^2 - (c^2u_{11} + u_{22})(i^2u_{11} + p^2u_{33})}$$

The effect of prices on intensity depends on the sign of $(u_1 + nu_{11})$. For values of n higher than $-u_1/u_{11}$, a smoker would increase the intensity of smoking when prices increases. Similarly, for these values of *n*, an increase in income leads to a decrease in the intensity of smoking:

(A-5)
$$\frac{di}{dy} = \frac{pu_{33}(u_1 + nu_{11})}{(u_1 + nu_{11})^2 - (c^2u_{11} + u_{22})(i^2u_{11} + p^2u_{33})}$$

Appendix B : Worst Case Bounds

We summarize here the methodology in Manski (1994). Let *i* denote the intensity exerted to smoke a cigarette and *X* a set of conditioning variables. Let *S* be an indicator which takes the value of 1 if *i* is observed and 0 otherwise. In our case, *S=1* indicates that the individual is still smoking, whereas *S=0* indicates that the person has quitted since the start of the sample. The probability of *S=1* given *X* is written as *P(X)*, the probability of not quitting. We write the conditional cumulative distribution of *i* given *X* in period *t* as $F_t(i|X)$.

We are interested in comparing $F_t(i|X)$ and $F_{t+1}(i|X)$, assuming that a price increase has taken place between period *t* and *t+1*. While in our data we can characterize the cumulative distribution $F_t(i|X)$, as we observe every smoker in that period, it is not the case for period *t+1*. Some smokers may have quitted, and if quitting is not random the cumulative distribution in period *t+1* reflects both the effect of taxes on smoking intensity and the change in the composition of the sample. However we can write

(B-1) $$F_{t+1}(i \mid X) = F_{t+1}(i \mid X, S=1)P(X) + F_{t+1}(i \mid X, S=0)[1 - P(X)]$$

The data in period t+1 identify the distribution $F_{t+1}(i|X,\ i=1)$ and $P(X)$, but not the distribution of the intensity of those who happened to quit between *t* and *t+1*, $F_{t+1}(i|X,\ i=0)$.

However, the inequality

$$0 \le F_{t+1}(i \mid X, S=0) \le 1$$

is always true. Using it in (A1) produces bounds

(B-2) $$F_{t+1}(i \mid X, S=1)P(X) \le F_{t+1}(i \mid X) \le F_{t+1}(i \mid X, S=1)P(X) + [1 - P(X)]$$

or

$$F_{t+1}^{low}(i \mid X) \le F_{t+1}(i \mid X) \le F_{t+1}^{high}(i \mid X)$$

Clearly, if *P(X)* is close to one, i.e. few smokers quit, then the bounds will be tight. If, however, the probability of quitting is large, then the bounds become wider and less informative.

We implement this procedure using two adjacent dates in the survey to minimize the effect of quitting. We control for observable characteristics such as age, sex, education level, race, as well as state fixed effects for the US. The probability *P(X)* is computed as the proportion of individuals who have not quitted from period *t* to *t+1*. We use information on the duration since quitting which is documented in the survey.

Higher taxes increase smoking intensity if

(B-3) $$F_{t+1}(i \mid X) \leq F_t(i \mid X)$$

a sufficient condition is

(B-4) $$F_{t+1}^{high}(i \mid X) \leq F_t(i \mid X)$$

although there could be cases where (B-3) is satisfied even if (B-4) is not.

Appendix C : Rational Addiction and Smoking Intensity

The first order conditions of model (4) are:

$$
\begin{aligned}
& i_t u_{1,t} + \delta i_t E_t u_{2,t+1} = \lambda_t p_t \\
& \lambda_t = E_t \lambda_{t+1} \\
& u_{3,t} = \lambda_t \\
& c_t u_{1,t} + \delta c_t E_t u_{2,t+1} + u_{4,t} = 0
\end{aligned}
\qquad \textbf{(C-1)}
$$

Where λ_t is the Lagrange multiplier on the budget constraints. Using a quadratic utility function, some straightforward algebra leads to:

$$n_t = \theta_0 + \theta_1 \frac{p_t}{i_t} + \theta_2 n_{t-1} + \theta_3 n_{t+1} + u_t \qquad \textbf{(C-2)}$$

With:

$$\theta_1 = \frac{\lambda_t \alpha_{qq}}{\alpha_{11}\alpha_{qq} - \alpha_{1q}^2 + \delta(\alpha_{22}\alpha_{qq} - \alpha_{2q}^2)} < 0$$

$$\theta_2 = \frac{\alpha_{12}\alpha_{qq} - \alpha_{1q}\alpha_{2q}}{\alpha_{11}\alpha_{qq} - \alpha_{1q}^2 + \delta(\alpha_{22}\alpha_{qq} - \alpha_{2q}^2)} > 0$$

$$\theta_3 = \delta\theta_2$$

REFERENCES

Becker G, Grossman M and K Murphy (1994) "An Empirical Analysis of Cigarette Addiction", *American Economic Review*; 84; 3; 396-418.

Becker G and K Murphy (1988) "A Theory of Rational Addiction", *Journal of Political Economy*, 96, 4, 675-699

Benowitz N, Perez-Stable E, Fong I, Modin G, Herrera B and P Jacob, III (1999) "Ethnic Differences in *N*-Glucuronidation of Nicotine and Cotinine", *Journal of Pharmacology and Experimental Therapeutics,* 291, 1196-1203

British National Formulary (BNF) Number 47 (2004), section 4.10. Pharmaceutical Pr; 1st edition. British Medical Association, Royal Pharmaceutical Society of Great Britain, London

Campbell D E, Greenberg E R, Polednak A P, DeShazer C, Bach P B, Cramer L D, Begg C B, and J L Warren (2000) "Racial Differences in the Treatment of Early-Stage Lung Cancer", *New England Journal of Medicine*, 342, 517-519

Caraballo R, Giovino G, Pechacek T, Mowery P, Richter P, Strauss W, Sharp D, Eriksen M, Pirkle J and K Maurer (1998) "Racial and Ethnic Differences in Serum Cotinine Levels of Cigarette Smokers", *JAMA,* 280, 135-139

Chaloupka, F (1991), "Rational Addictive Behavior and Cigarette Smoking", *Journal of Political Economy*, 99, 4, 722-742

Chaloupka, F and K Warner, (2000), "The Economics of Smoking", Handbook of Health Economics, J Newhouse and A Cuyler editors

DeCicca P, Kenkel D and A Mathios (2002) "Putting out the Fires: Will Higher Taxes Reduce the Onset of Youth Smoking?", *Journal of Political Economy*, 110, 1, 144-169

DeGranpre RJ, Bickel WK, Hughes JR, and ST Higgins (1992) "Behavioral economics of drug self-administration. III. A reanalysis of the nicotine regulation hypothesis", *Psychpharmacology*, 108, 1-10

Evans W and M Farrelly (1998) "The Compensating Behavior of Smokers: Taxes, Tar, and Nicotine", *RAND Journal of Economics*, 29, 3, 578-595

Farrelly M, Nimsch CT, Hyland A, and M Cummings (2004) "The Effects of Higher Cigarette Prices on Tar and Nicotine Consumption in a Cohort of Adult Smokers", *Health Economics.* 13, 1, 49-58

Gritz ER, Rose JE and ME Jarvik (1983) "Regulation of tobacco smoke intake with paced cigarette presentation" *Pharmacology Biochemistry and Behavior*, 18, 3, 457-462

Gruber J and B Koszegi (2001) "Is Addiction 'Rational'? Theory and Evidence", *Quarterly Journal of Economics*, 116, 4, 1261-1303

Harris J (1980) "Taxing Tar and Nicotine", *American Economic Review,* 70, 300-311

Jarvis M J, Boreham R, Primatesta P, Feyerabend C, and A Bryant (2001), "Nicotine Yield from Machine-Smoked Cigarettes and Nicotine Intakes in Smokers: Evidence from a Representative Population Survey", *Journal of the National Cancer Institute*, 93, 2, 134-138

Kozlowski LT, Frecker RC, Khouw V, and MA Pope (1980) "The Misuse of "Less Hazardous" Cigarettes and its Detection: Hole-blocking of Ventilated Filters". *American Journal of Public Health*, 70, 11, 1202-1203

McCarthy WJ, Caskey NH, and ME Jarvik (1992) "Ethnic Differences in Nicotine Exposure", *American Journal of Public Health*, 82, 1171-1172

Manski C (1994) "The Selection Problem" in *Advances in Econometrics, Sixth World Congress* Vol 1. Editor C Sims, Cambridge University Press

Miller BA, Kolonel LN, Bernstein L, et al. (1996) "*Racial/Ethnic Patterns of Cancer in the United States, 1988-1992"*. Bethesda, Md: National Cancer Institute, NIH publication, 96-4104

National Cancer Institute (2004) http://www.cancer.gov/newscenter/healthdisparities

Russell MA, Wilson C, Patel UA, Feyerabend C, and PV Cole (1975) "Plasma Nicotine Levels after Smoking Cigarettes with High, Medium and Low Nicotine Yields", British Medical Journal, 2, 5968, 414-416

Thun MJ, Lally CA, Flannery JT, Calle EE, Flanders WD, and CW Heath, Jr (1997) "Cigarette smoking and changes in the histopathology of lung cancer", *Journal of the National Cancer Institute*, 89, 1580-1586

Wagenknecht, Cutter GR Haley NJ, Sidney S, Manolio TA, Hughes GH, and DR Jacobs (1990) "Racial differences in serum cotinine levels among smokers in the Coronary Artery Risk Development in (Young) Adults study" *American Journal of Public Health*, 80, 1053-1056

Table 1: Descriptive Statistics

	All	Smokers	Non-smokers
# of observations	20050	4641	13882
average # of cigarettes	10	18.8	0
	(9)	(12.2)	-
average level of cotinine (ng/ml)	78	230	0.44
	(140)	(138)	(0.87)
% education high	40	27	47
% education medium	49	63	41
% education low	11	10	11
average age	44	39	45
sex (% male)	47.7	51	43
% Professional and managerial	17	12.2	20
% skilled manual	27	27	28
% unskilled manual	24	34	20
% white	84.1	84	84.6
% income 1st quartile		18	14
% income 4th quartile		27	38

Note: Smokers only includes smokers of cigarettes. Non smokers excludes consumers of any tobacco product, and condition on a cotinine level of 10ng/ml or less.

Table 2: Elasticity of Smoking Intensity, of Number of Cigarettes and of Cotinine

	(1)	(2)	(3)	(4)	(5)
	Baseline[a]	Baseline[a] + Excluding cotinine level<200 ng/ml (median value)	Baseline[a] + Additional controls: height, day and time of examination	Baseline[a] + Excluding those started after 1988	Baseline[a] + Excluding late starters
Elasticity Smoking Intensity	0.47**	0.55**	0.46**	0.43**	0.34**
	(0.18)	(0.19)	(0.20)	(0.18)	(0.14)
Elasticity Number of Cigarettes	-0.20	-0.49**	-0.19	-0.13	-0.73**
	(0.37)	(0.21)	(0.34)	(0.38)	(0.27)
Elasticity Cotinine	0.28	0.06	0.27	0.30	-0.39
	(0.25)	(0.05)	(0.26)	(0.27)	(0.35)

[a] Baseline estimations use NHANES 1988-1994. All regressions control for age, sex, race, education, occupation, household size, passive smoking, year and state effect. Robust Standard errors clustered at state and year level. ** significant at the 5% level

Table 3: Compensatory Behavior: Cigarette Type and Behavioral Adjustment

	(1)	(2)
	NHANES 1999-2000	NHANES 1999-2000. Additional controls: cigarette length and nicotine yield
Elasticity Smoking Intensity	0.11**	0.10**
	(0.04)	(0.04)
Elasticity Number of Cigarettes	-0.15**	-0.15**
	(0.04)	(0.04)
Elasticity Cotinine	-0.03	-0.04
	(0.04)	(0.07)

All regressions control for age, sex, race, education, occupation, household size, passive smoking, year and region effect. Four regional dummies were included (NorthEast, MidWest, West, and South). Robust Standard errors clustered at region and year level. ** significant at the 5% level

Table 4: Determinants of Smoking as measured by log of cigarettes, log of cotinine concentration and log of cotinine concentration per cigarette smoked.

	(1)		(2)		(3)		(4)	
	Log(Cig)[a]		Log(Cot)[a]		Log(Cot/Cig)[a]		Log(Cot/Cig)[b]	
Men	-0.05	(0.040)	-0.11	(0.060)	-0.06	(0.040)	0.11	(0.120)
Age	0.05**	(0.006)	0.05**	(0.008)	-0.01	(0.007)	0.00	(0.021)
Age squared (*100)	-0.1**	(0.001)	-0.04**	(0.010)	0.01	(0.007)	-0.00	(0.022)
Log income	-0.02	(0.026)	-0.05	(0.035)	-0.03	(0.027)	0.03	(0.020)
Education (years)	-0.01	(0.007)	-0.03**	(0.009)	-0.02**	(0.007)	-0.04	(0.061)
Size of house (number of bedrooms)	-0.04**	(0.009)	-0.09**	(0.010)	-0.05**	(0.009)	-	-
White	0.39**	(0.094)	0.36**	(0.130)	-0.03	(0.100)	0.16	(0.129)
African-American	-0.05	(0.102)	0.51**	(0.140)	0.56**	(0.100)	0.64**	(0.140)
Family size	0.01	(0.010)	0.05**	(0.020)	0.04**	(0.010)	-	-
Attending church	-0.17**	(0.030)	-0.08**	(0.040)	0.09**	(0.030)	-	-
Living in urban area	-0.10**	(0.030)	-0.04	(0.041)	0.06*	(0.030)	-	-
Height (inches)	0.01*	(0.005)	0.01	(0.007)	-0.00	(0.006)	-0.01	(0.007)
Married	0.19**	(0.060)	0.10	(0.090)	-0.09	(0.070)	-0.02	(0.101)
Age started smoking	-0.02**	(0.003)	-0.03**	(0.004)	-0.00	(0.003)	-0.00	(0.010)
Filter							0.40	(0.372)
Nicotine Yield							0.76**	(0.190)
Length of Cigarette (centimeter)							0.06	(0.051)
Mentholated							0.09	(0.110)
Number of Observations	3424		3424		3424		590	

[a] Estimation done for years 1988-1994; [b] Estimation done for 1999. * significant at the 10% level; ** significant at the 5% level. Robust standard errors in parenthesis. Regression also controls for year and region effects.

Table 5: Biases in the Rational Addiction Model.

$(Z'X)^{-1}Z'Y/i$			
-0.08	-0.19	-2.15	8.82
-0.00	0.01	-0.02	0.05
0.01	0.04	1.20	0.36
0.01	-0.01	0.16	-0.78

Notes: Model controls for age, sex, education level, race, state and year fixed effects. A constant was included in the regression. Lags and leads of taxes were used as instruments.

Table 6: Implied Values for Θ in Rational Addiction Model.

	Estimated parameters in rational addiction model	Implied parameters in full model with smoking intensity
Price effects	$\gamma_1 = -1.5$	$\theta_1 = 0.16$
Past smoking	$\gamma_2 = 0.5$	$\theta_2 = 42.90$
Future smoking	$\gamma_3 = 0.5$	$\theta_3 = -0.91$

Notes: Model controls for age, sex, education level, race, state and year fixed effects. A constant was included in the regression. Lags and leads of taxes were used as instruments.

Figure 1: State Excise Taxes , 1988-1994

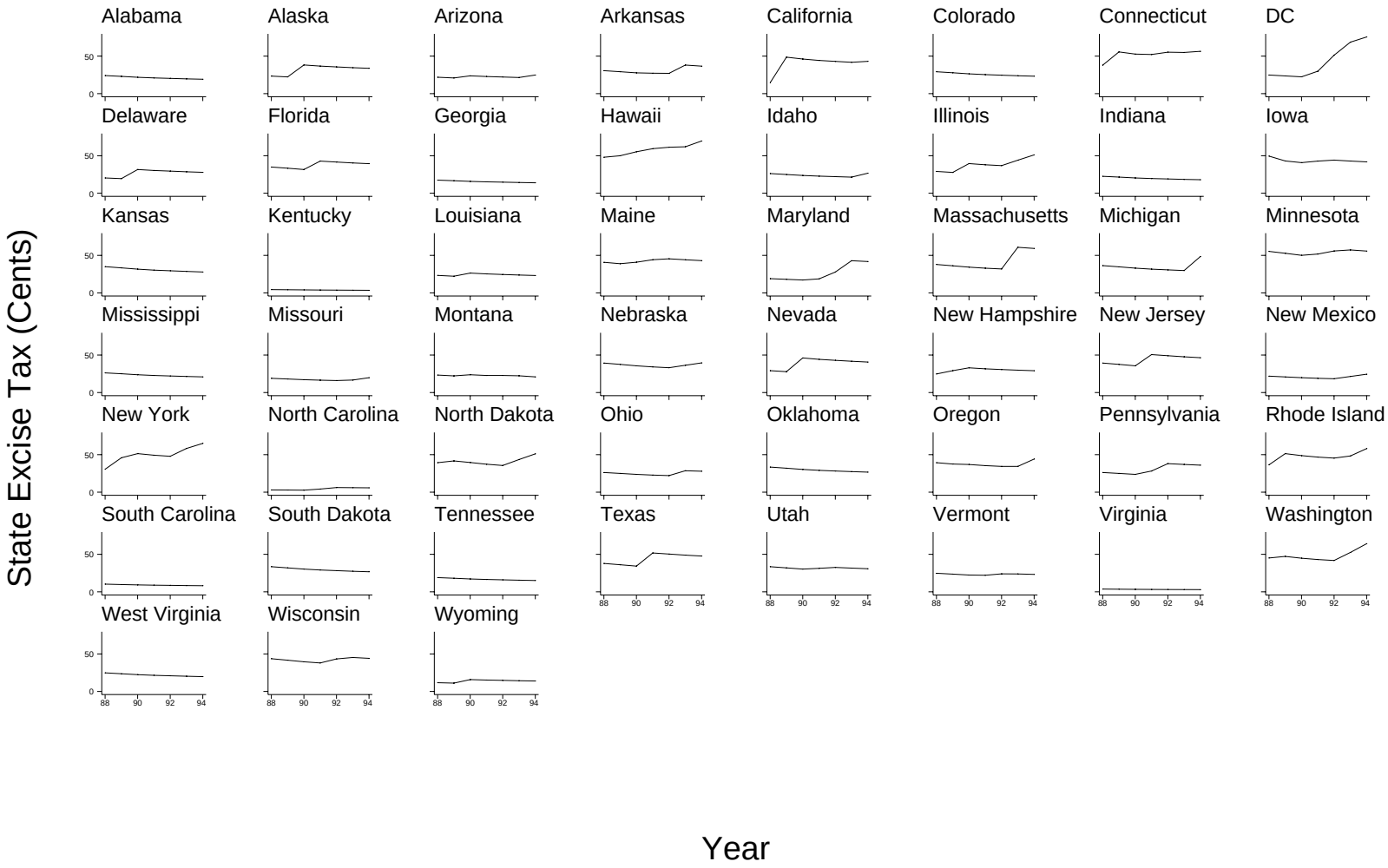

Figure 2: Cotinine Concentration and Cigarette Smoked.

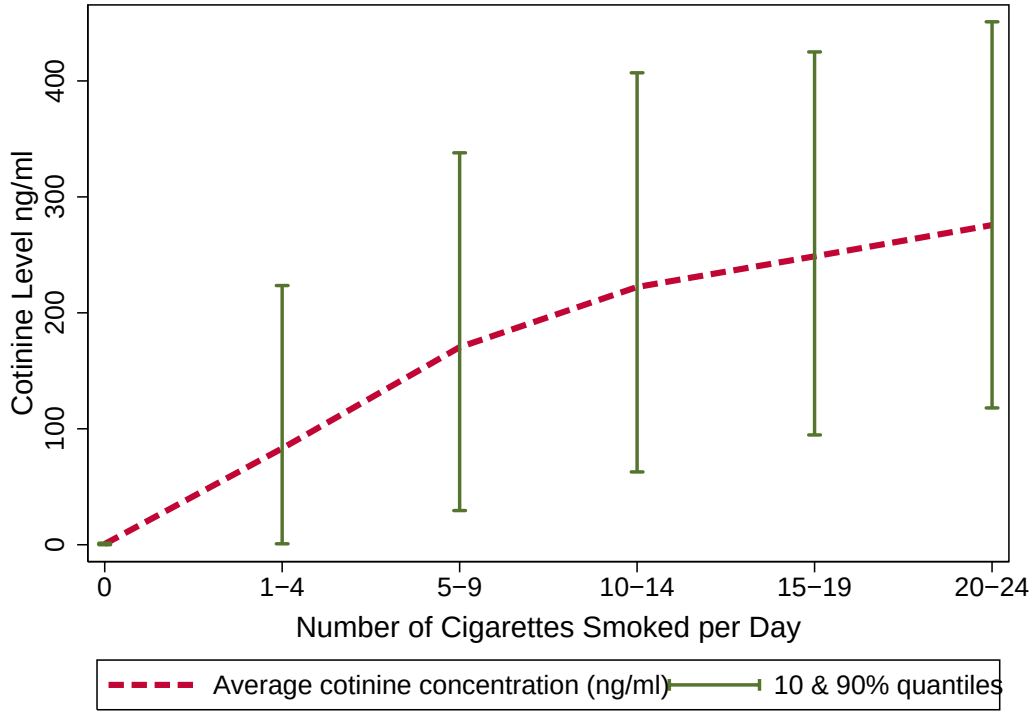

Figure 3: Worst Case Bounds for States in Which Taxes Increased.