DISCUSSION PAPER SERIES

IZA

IZA DP No. 2191

The Effect of Taxes and Bans on Passive Smoking

Jérôme Adda
Francesca Cornaglia

July 2006

Forschungsinstitut
zur Zukunft der Arbeit
Institute for the Study
of Labor

The Effect of Taxes and Bans on Passive Smoking

Jérôme Adda
University College London and Institute for Fiscal Studies

Francesca Cornaglia
University College London, CEP and IZA Bonn

Discussion Paper No. 2191
July 2006

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. 2191
July 2006

ABSTRACT

The Effect of Taxes and Bans on Passive Smoking*

This paper evaluates the effect of excise taxes and bans on smoking in public places on the exposure to tobacco smoke of non-smokers. We use a novel way of quantifying passive smoking: we use data on cotinine concentration – a metabolite of nicotine – measured in a large population of non-smokers over time. Exploiting state and time variation across US states, we show that excise taxes have a significant effect on passive smoking but smoking bans have contrasting effects on non-smokers. While bans in public transportation or in schools decrease the exposure of non smokers, bans in recreational public places perversely increase their exposure by displacing smokers to private places where they contaminate non smokers, and in particular young children. Bans affect socio-economic groups differently: we find that smoking bans increase the exposure of poorer individuals, while it decreases the exposure of richer individuals.

JEL Classification: I1

Keywords: passive smoking, bans, taxes

Corresponding author:

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

* We are grateful to a number of seminar participants and to David Card, Ken Chay, Christian Dustmann, Steve Machin, Costas Meghir, and Imran Rasul for helpful discussions and comments and to William Evans for supplying us with data on US tobacco prices and taxes. Funding through the ESRC is gratefully acknowledged.

1 Introduction

In the US, 15% of the population smokes regularly. Yet, detectable levels of tobacco related chemicals can be found in body fluids in 84% of non smokers of all ages.[1] A large medical and epidemiological literature has stressed the dangers of exposure to environmental tobacco smoke.[2] Passive smoking has been linked to a number of serious illnesses such as lung cancer or heart disease in the adult population. It causes about 35000 deaths per year from heart diseases and about 3000 lung cancer deaths (Environmental Protection Agency, 1992, American Cancer Society, 2003, IARC, 2004). Passive smoking affects particularly the health of young children and babies, causing asthma, bronchitis or sudden infant death syndrome. Exposure to smoke causes about 200,000 lower respiratory tract infections in young children each year, resulting in 10,000 hospitalizations (Environmental Protection Agency, 1992). Medical studies consistently find that smokers impose a negative externality on non-smokers. As a result, governments have come under pressure by the general public and by anti-tobacco groups to limit the exposure of non-smokers and generally to discourage smoking. Since the mid eighties, support for smoking bans in public places has steadily risen. The proportion of individuals supporting a total ban in restaurants has increased from 20% in 1985 to 54% in 2005.[3] Public intervention uses two instruments to discourage smoking: directly by limiting or banning smoking in public places, and indirectly by raising taxes on cigarettes.

The economic literature has focused on the effect of prices or taxes on *smokers*. Following the work of Becker and Murphy (1988), most papers estimate price elasticities both in the short and the long run.[4] The evidence in these papers suggests that prices have an effect on cigarette consumption. However, some recent papers dispute the effect of prices. DeCicca et al (2002) show that cigarette prices do not affect initiation at young ages. Adda and Cornaglia (2006) show that although taxes affect the number of cigarette smoked, smokers compensate

[1] See descriptive evidence in section 3.1
[2] See for instance Law et al (1997), Hackshaw et al (1997), He et al (1999), Otsuka (2001), Whincup et al (2004), for adults and Strachan and Cook (1997), Gergen et al (1998), Kriz et al (2000), Lam et al (2001), Mannino et al (2001) for children who all find that exposure to passive smoke is harmful for non-smokers health.
[3] Source: Gallup poll (http://poll.gallup.com/).
[4] See for instance the paper by Becker et al(1994), Chaloupka (1991), and references in Chaloupka and Warner (2000).

by smoking each cigarette more intensively. Few papers analyze the effect of bans on smoking. Among these, Evans et al. (1999) show that workplace bans decrease the prevalence of smoking in those who work.

While the literature on the effect of taxes or bans on smokers is quite large, there is hardly any evidence on the effectiveness of these measures on the population of *non-smokers.*[5] Yet, the debate in public circles and in the media on the effectiveness of different measures has recently intensified, and policies to ban smoking are often justified by the protection of non smokers rather than smokers[6]. There is to our knowledge no study evaluating the response of passive smoking to changes in excise taxes, or on the growing set of regulation and clean air acts passed in the last decade[7]. A main reason why there is hardly any work in the economic literature on the exposure of non-smokers to environmental smoke is the apparent difficulty of measuring passive smoking directly.

This paper fills this gap. We propose a way of measuring passive smoking directly in non-smokers. We use a unique data set, which reports a direct measure of exposure to passive smoking: cotinine concentration in body fluids. Cotinine is a by-product of nicotine, and is a good marker of exposure to second hand smoke, which has been used routinely in the medical and epidemiological literature.[8] Using cotinine measures for analysing changes in exposure to passive smoking has several advantages. First, one can detect even small effects in exposure to environmental smoke; second, cotinine measures are sensitive to changes in exposure; third, it is a more reliable and objective measure than self-reported exposure which has been used as a measure of passive smoking. An alternative measure would be to use changes in smoking related diseases. However, most of these diseases are not specific to smoking and they usually take several years to develop. This makes it difficult to correctly identify the effect of anti-smoking policies. Cotinine is therefore a straightforward and

[5] One exception is the effect of maternal smoking on birth weight, see for instance Rosenzweig and Schultz (1983) and Evans and Ringel (1999).
[6] See for instance ASH (2005) for a summary of the case for smoke free public places.
[7] A search in EconLit for the key words “passive smoking” generates only 4 hits that are unrelated to the issue discussed here.
[8] The epidemiological literature has examined the issue of passive smoking, mostly from its health consequences. This literature has produced a measure of passive smoking by analyzing the concentration of cotinine, a metabolite of nicotine, in blood, saliva or urine samples. The amount of cotinine is a good marker of the exposure to environmental smoke (Jarvis et al 1984). The epidemiological literature has also tried to characterize the socio-economic groups that are more prone to exposure to environmental smoke (Pirkle et al, 1996; Howard et al, 1998; Siegel, 1993; Jarvis et al, 2001; Whitlock et al, 1998; Jarvis et al, 2000; Strachan and Cook, 1997).

precise measure of passive smoking and particularly suited to evaluate policies aimed at reducing smoking.

Our analysis proceeds in several steps. First, using cotinine levels for a large and representative sample of non-smokers over time, including very young children, we document the extent of passive smoking in the US. We evaluate the effect of increases in excise tax that took place in the US over the last decade on exposure to environmental tobacco smoke (ETS). Our analysis exploits changes over time in regulations on smoking in public places across different states. We find that changes in tobacco taxes have a significant effect on the exposure to environmental smoke. We find a tax-elasticity of passive smoking of about -0.3 to -0.4, which is three to four times higher than the tax-elasticity of cigarette consumption. The effect is particularly sizable for children who are exposed to their parents' smoke. This suggests that excise taxes are an efficient tool to curb passive smoking as smokers cut down on cigarettes smoked in company of non-smokers, especially children.

Second, we analyse the impact of smoking bans. Bans on smoking in public places have on average *no* effects on non-smokers. However, we show that bans have different effects when imposed in different public places. While on average bans in public transports, shopping malls or schools decrease the exposure of non-smokers, bans in bars, restaurants or recreational facilities appear to *increase* their exposure. We hypothesise that such bans displace the smoking to places where non-smokers are more exposed, especially young children. We test this hypothesis by contrasting the effect of smoking bans on children living in smoking and non smoking families and by investigating the seasonal effects of bans on these two groups. The evidence support the hypothesis of a displacement of smokers to places shared with non-smokers who then get more exposure to tobacco smoke.

Moreover, bans have contrasting effects on different social and demographic groups. We find evidence that smoking regulations increases the exposure of poorer individuals, while it decreases the exposure of individuals in higher socio-economic position. This suggests that smoking regulations may increase health inequalities between socio-economic groups. Finally, we provide an estimation of the health and economic costs of anti-smoking measures.

The remainder of the paper is structured as follows. Section 2 presents the theoretical framework used for analyzing the effect of passive smoke exposure, and outlines the estimation strategy. Section 3 contains a description of our data set. In Section 4, we investigate the effect of different state interventions on passive smoking, measured by the cotinine concentration present in non-smokers. Finally, Section 5 concludes and discusses the implications of our results.

2 Methodology

This section discusses our framework for analyzing the effect of tax changes and smoking regulations on passive smoking. In particular, we define our measure of passive smoking and describe our identification strategy.

2.1 Cotinine as a Proxy for Smoking Intake

The effect of anti-tobacco policies depends on the interactions between smokers and non-smokers and these interactions can change with policies. It is therefore not straightforward to infer the effect of government interventions on *non-smokers* by looking at the effect of these interventions on *smokers* (i.e. measuring the change in prevalence, or the change in the number of cigarettes smoked). Passive smoking should be measured *directly* in non-smokers. In order to analyze the effect of state interventions on non-smoker we need a measure of the amount of tobacco smoke inhaled by non smokers. We use as a proxy the cotinine concentration in body fluids. Cotinine is a metabolite of nicotine. While nicotine is unstable and is degraded within a few hours of absorption, cotinine has a half-life in the body of about 20 hours and is, therefore, a biological marker often used as an indicator of passive smoking.[9] It can be measured in, among other things, saliva or serum.

The use of cotinine has several advantages. First, cotinine is related to the exposure to cigarette smoke.

[9] The elimination of cotinine is slow enough to allow comparing measurements done in the morning or in the afternoon.

Figure 1 plots the relationship between the total number of cigarettes smoked in the household and the cotinine level observed in the body fluids of non smokers sharing the house with smokers.

[Figure 1]

The relationship between the number of cigarettes smoked in the household and the cotinine level in non smokers living with smokers is upward sloping. Second, cotinine – and nicotine from which it is derived- is a good proxy for the intake of health threatening substances in cigarettes. The nicotine yield of a cigarette is highly correlated with the level of tar and carbon monoxide, which causes cancer and asphyxiation.[10,11] Cotinine is, therefore, a good indicator of health hazards due to exposure to passive smoking. Third, cotinine levels reveal rapidly variations in exposure due to changes in policy, which is not the case with other markers such as tobacco related diseases which take time to develop. Finally, there is minimal measurement error, compared with self-declared exposure to cigarettes.

The novelty of our analysis is to use cotinine concentration in non smokers to evaluate the effect of public intervention aimed at reducing tobacco exposure.

2.2 Overall Effect of Smoking Restrictions and Taxes

We consider the following econometric model of exposure to environmental smoke for a non smoker indexed by *i*, in state *s* and in period *t*:

$$\text{Cot}_{ist} = \alpha_0 + \beta \log tax_{st} + \gamma R_{st} + \alpha_1 X_{ist} + \delta_s + \lambda_t + u_{ist} \qquad (1)$$

[10] Based on our data set (the National Health and Nutrition Examination Survey), 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.

[11] The main health impacts of exposure to environmental tobacco smoke (ETS) are lung cancer (more than 50 epidemiological studies have examined the relationship between passive smoking and lung cancer; for a review see NHS Scotland, 2005), coronary heart diseases, respiratory disorders, and ETS in pregnancy can lead to low birth weight and poor gestational growth.

where Cot_{ist} is the cotinine concentration (expressed in ng/ml); tax_{st} is the state excise tax on tobacco (adjusted for inflation) in a given state and period; R_{st} is a measure of restrictions on smoking in the state at the period of interest; X_{ist} is a vector of individual characteristics that affect exposure such as age, sex, occupation or race. It also includes (detrended) state GDP as it may be correlated both with smoking and with excise taxes if they are used as a tool to raise state revenue; δ_s is a set of state of residence dummies, while λ_t is a set of year dummies. The coefficients of interest are the effect of taxes and the effect of smoking restrictions on cotinine measures. We relate exposure to excise taxes as this is the relevant policy variable from a public health point of view.

The coefficients β and γ measure both the direct and indirect effects of taxes and smoking bans. Smoking bans have a direct effect on non-smokers as they guarantee a smoke-free environment. Changes in excise taxes operate indirectly as they can only have an effect through the behaviour of smokers. To some extent, this indirect effect is also present in smoking bans given that they may induce changes in the way smokers smoke. To interpret the effect of bans on non-smokers, it is necessary to understand the effect on smokers and the extent to which smokers and non smokers cohabit and interact.

The identification of the effect of taxes and regulation comes from variation across states and time, and not from cross-sectional differences in the level of state regulations or taxes, which are taken into account by state dummies. Our identification relies on the exogeneity of *changes* in taxes and regulation *within* states, but not on the heterogeneity in levels of regulations and exposure to passive smoking.

The coefficients of interests would be biased despite the number of controls in equation (1), if changes in unobserved factors affect both changes in exposure to passive smoking and smoking regulations. This could occur for at least two reasons. First, introducing tougher smoking regulations in states where prevalence of smoking is on the decrease could be politically easier as the median voter would shift towards a non-smoker. If this is the case, the estimate of smoking regulations would be biased downwards. Another possibility is that tougher regulations are more likely to be enforced on health grounds in states where smoking is on the increase, or in relative increase compared to the rest of the country. In this case, estimates would be biased upwards.

We control for both these potential biases by including in X_{ist} the (lagged) prevalence rate of smoking at state level. The prevalence rate would be the obvious control variable if the endogeneity comes through a shift in the political power of non-smokers. It is also a key variable to monitor smoking trends in relation to health issues related to smoking and is easily observable by policy regulators. On the contrary, more direct measures of exposure, such as cotinine concentrations, are much more difficult to obtain and are more difficult to monitor.

Cotinine is constantly eliminated by the body, although at a slow rate. Some of the variation in cotinine levels depends on the timing of the examination during the day. To the extent that the timing of the examination is uncorrelated with changes in taxes and level of regulation in the state, we do not expect a bias in the coefficient of interest. The same argument can be made for biological diversity in the speed at which cotinine is cleared from the body.

The model is estimated by OLS, and standard errors are adjusted for heteroskedasticity and clustered at state level. This correction accounts for the presence of a common random effect at the state level. We therefore allow for serial correlation in the error term following Bertrand et al (2004) who show that difference-in-difference estimations can be seriously biased in the presence of autocorrelation.

2.3 Effect of Restrictions in Different Public Places

In equation (1) we use an overall measure of the severity of bans within a state. Smoking restrictions in public places may have different impacts on non-smokers, depending on where they are enforced. We therefore consider the following model:

$$\text{Cot}_{ist} = \alpha_0 + \beta \log tax_{st} + \sum_j \gamma_j R_{j,st} + \alpha_2 X_{ist} + \delta_s + \lambda_t + u_{ist} \qquad j = GO, PT, SM, WP, Sch. \qquad (2)$$

where tax_{st} is the tax of tobacco (adjusted for inflation) in a given state and period; GO is a measure of restrictions on smoking in bars, restaurant and other recreational places ("going out"); PT restrictions in public transport; SM restrictions in shopping malls; WP restrictions in the work place; Sch restrictions at school; X_{ist} is a vector of individual and state characteristics as in equation (1); δ_s is a set of state of residence dummies, while λ_t is a set of year dummies. As in (1), the identification of the effect of taxes and regulation comes from changes within states.

2.4 Measuring Displacement Effects

Excise taxes and smoking restrictions may induce a reallocation of smoking across different places and different moments throughout the day. This displacement of smoking may benefit or harm non smokers depending on where it occurs. These effects are captured in the two specifications proposed above, but we cannot disentangle the direct and indirect effects of anti-smoking policies in a straightforward way.

To demonstrate the importance of displacement we need to evaluate the effect of such policies for groups of individuals for whom a direct effect is less likely to occur. We first discuss how taxes and bans can lead to a displacement of smoking and we then propose a formal statistical test. Our goal is not to fully decompose the effect of policies as direct or indirect effects, but our aim is to demonstrate that some policies can induce perverse effects. For instance, smokers can change locations if smoking is not allowed as well as, to some extent, the period within a day during which cigarettes are smoked. Tighter regulations may therefore induce a perverse displacement of smoking. The literature has shown the negative effect of taxes (and prices) on the demand for cigarettes[12]. However, this does not mean that regular smokers reduce smoking in a uniform way: during the day, some cigarettes may in fact be easier to cut down. If smoking is a social activity, a smoker may reduce the number of cigarettes consumed when alone or at home, and not those consumed in company of other adults. In this case, adult non smokers may not benefit at all from a rise in excise taxes, whereas children would be less exposed.

[12] See for instance Becker et al (1994), Chaloupka (1991), and references in Chaloupka and Warner (2000).

We now outline our identification strategy. We first highlight the methodological approach in a general setting. Suppose the following model holds for individual *i*:

$$Cot_i = \alpha_0 + \alpha_1 x_i + \alpha_2 D_i + \alpha_3 x_i D_i + u_i \qquad i = 1, \ldots N$$

where D_i is an indicator variable and x_i is a measure of public intervention with $E(x_i \mid D_i) = E(x_i)$, $E(x_i u_i \mid D_i) = E(x_i u_i)$ and $E(u_i D_i) = 0$. We place no restriction on the covariance between x_i and u_i, i.e. we allow for the possible endogeneity of x_i. Denote $\widehat{\alpha}_{1,N}$, $\widehat{\alpha}_{2,N}$ and $\widehat{\alpha}_{3,N}$ the OLS estimators of the parameters α_1, α_2 and α_3.

<u>Proposition:</u>

Under the assumptions detailed above, when N tends to infinity,

$$\underset{N \to \infty}{p\lim}(\widehat{\alpha}_{1,N} - \alpha_1) \neq 0$$

$$\underset{N \to \infty}{p\lim}(\widehat{\alpha}_{2,N} - \alpha_2) = 0$$

$$\underset{N \to \infty}{p\lim}(\widehat{\alpha}_{3,N} - \alpha_3) = 0$$

For a proof, we refer the reader to appendix A. The proposition above states that even if x_i is endogenous, OLS consistently estimates the effect of x_i for individuals with characteristics D_i. The intuition behind this result is that the coefficient α_3 captures the differential impact of x_i across groups characterized by D_i and by assumption, the endogeneity affects both groups in the same way. Hence, although the effect of public intervention may be biased due to endogeneity, this bias is of the same magnitude for both groups and therefore differenced out when considering differential effects.

In the context of passive smoking, let D_i be a variable indicating whether an individual belongs to a particular group for whom there is no direct effect of a given anti-smoking policy, perhaps because this individual does not go to certain public places. We define the displacement effect as the differential impact of a policy on this group.

We extend model (2) to

$$\text{Cot}_{ist} = \alpha_0 + \beta \log tax_{st} + \tilde{\beta} \log tax_{st} D_i + \sum_j \gamma_j R_{j,st} + \sum_j \tilde{\gamma}_j R_{j,st} D_i + \alpha_1 X_{ist} + \delta_s + \lambda_t + u_{ist} \qquad (3)$$

where j=GO, PT, SM, WP, Sch.

We test for displacement by testing whether $\tilde{\beta}$ or $\tilde{\gamma}_j$ are zero. In section 4, we propose to test the existence of displacement effect and we discuss in detail which groups we consider.

3 The Data and Descriptive Statistics

3.1 Exposure to Passive Smoking

We use data from the National Health and Nutrition Examination Survey (NHANES III and NHANES 1999-2002). NHANES is a nationwide representative sample of the US civilian population. It provides information, from 1988 to 1994 and from 1999 to 2002, for 51,835 individuals, aged zero and above. The data set reports information on the age, sex, race, health, education and occupation of the individual, as well as information at the household level such as family composition, income or geographical location. In addition, the cotinine concentration in both smokers and non smokers (aged four and above), and the number of cigarettes smoked in the household are reported. This last information allows distinguishing between non smokers that are exposed to passive smoke at home and non smokers that live in smoke-free households.

From the available sample we select non-smoking individuals. We drop all individuals who report them-selves as smoker or report consuming cigarettes, cigars, pipe, snuff or chewing tobacco. We also drop all individuals who have a cotinine level in excess of 10 ng/ml. This rule is often use in epidemiological studies to distinguish smokers from non smokers.[13] It represents about 5% of the declared non smokers. In total, we observe 29667 non-smokers with a valid measure of cotinine concentration.[14]

[Table 1]

[13] See Jarvis et al, 1987. This threshold also constitutes the upper level of exposure of younger children (aged 6 or less) for whom we can presumably assume that they are genuinely non smokers. The distribution of cotinine is very skewed and mainly concentrated in the 0 - 2 ng/ml region which contains more than 90% of the sample.

[14] All valid cotinine measures below the detection threshold (0.035 ng/ml), were set to the threshold value.

Table 1 provides a summary statistic of the data set. Column 1 refers to the whole sample, columns 2 and 3 provide descriptive statistics for non-smokers living in household where the other members either smoke or not. The average cotinine concentration is equal to 0.44ng/ml. 84% of the sample has a cotinine concentration higher than the detectable threshold of 0.035ng/ml, while 14% have a value higher than 1ng/ml. The amount of cotinine in non smokers living in a non smoking household is more than five times lower than the amount of cotinine present in individuals living with smokers (0.26 n/ml in non-smokers living in non-smoking households compared to a level of 1.47 n/ml in individuals living with smokers). Individuals living in households with smokers have almost all detectable levels of cotinine, and are much more likely than non smokers living in non smoking households to have a concentration of cotinine above 1ng/ml.

3.2 Excise Taxes and Smoking Restrictions to Tobacco Exposure

We merge information on state level excise taxes and smoking regulations to the NHANES datasets. The data on excise taxes are from the Tax Burden on Tobacco, published by The Tobacco Institute until 1998 and updated by Orzechowski and Walker (2001). It reports taxes by state and year. We deflate taxes using the consumer price index. Most of the variation is cross-sectional, where taxes can vary by about 80%. There is differential variation over time and across states that we exploit to identify the effect of taxes. Figure 2 plots the excise taxes over time (1988-2002) in each of the US states. Taxes have on average increased by 2 cents per year.

[Figure 2]

We also merge information on smoke free laws in the different US states to the NHANES datasets. Regulations on smoking bans in the US are obtained from the ImpacTeen web site, based on state clean air acts.[15] This data set reports the regulation in place by year and by state in different locations. The data set provides information on the severity of the restrictions and on the place where the restriction is enforced (e.g. government worksites,

[15] http://www.impacteen.org. Note that restrictions on smoking are measured at state level, combining both state level regulations and an aggregation of county level restrictions.

private work sites, public transits, schools, and restaurants). We refer the reader to Appendix A for a more detailed description of the dataset. We recode the severity of the restriction into four categories: zero if no restrictions; one if smoking is restricted to designated areas; two if smoking is restricted to separate areas; three if there is a total ban on smoking.[16] Figure 3 displays the average level of restrictions on smoking, by US states over the period 1991-2000. The restriction index is the average, by year and state, of all levels of restriction in all places.

[Figure 3]

Over the nineties, regulations have become more stringent. Moreover, the proportion of states with no restriction in any places falls from 50% in 1991 to 36% in 2001. Similarly, in 1991 only 27% of the states had at least a total ban on smoking in one public space, whereas the figure is 51% in 2001.

[Table 2]

Our identification strategy relies on within state variation in excise taxes and smoking regulation. Table 2 presents descriptive statistics for taxes and regulations. In particular, the last column presents the within-state standard deviation, which is important to interpret the magnitude of the effects presented in the next section. While the range of our regulation variable is between zero (no ban) and three (full ban), a one standard deviation within a state correspond to a change of about 0.2 to 0.3 for most of the regulations we consider. In other words, we never observe in the data a state going from no ban to a complete restriction on smoking.

3.3 Trends in Passive Smoking

In the US the cotinine concentration in non-smokers has halved over the nineties, from about 0.8 ng/ml in 1988 to 0.4 ng/ml in 2002 (Figure 4). This remarkable trend may indicate that policies regulating smoking have been successful. This decrease in passive smoking can also be observed in non smokers at the upper end of the distribution of exposure (Figure 5). Over

[16] See appendix A for a detailed description of the regulations variables.

this period, the proportion of individuals with a cotinine level in excess of 1 ng/ml has decreased from 21% to 11%.[17]

[Figure 4 and Figure 5]

Next, we separate non smokers who share their household with smokers, from non smokers who live in “smoke free” households. Figure 6 plots the cotinine concentration in non-smokers living in non smoking households from 1988 to 2000. Figure 7 shows, for the same time period, the cotinine concentration of non smokers sharing the house with smokers.

[Figure 6 and Figure 7]

The level of cotinine has been halved in non smokers living with non smokers over the period of analysis (1988-2000), from about 0.4 ng/ml to 0.2 ng/ml (). However, policies have been less successful in reducing exposure of those who live with smokers. In the period considered (1988-2002) the concentration of cotinine in non-smokers living with smokers does not show a similar trend (Figure 7). Despite the increasing level of severity in regulations and higher excise taxes, this evidence suggests that tobacco exposure of non smokers living in smoking households did not decrease. [18]

4 Empirical Results: Passive Smoking and State Intervention

[17] We arbitrarily look at the cotinine level of 1 ng/ml, which corresponds to the 15% upper percentile.

[18] An alternative interpretation is that of a change in composition in the pool of smokers. If higher taxes and tougher regulation encourage proportionally more light smokers to quit, the sample of non smokers in smoking household will shift towards a population more exposed to passive smoking. This would bias upward the effect of taxes or regulations. As a robustness check, we have also done the analysis by re-weighting the sample so that each year becomes comparable, in terms of observables, to the first year of our sample. This methodology is developed in DiNardo et al (1996) to study changes in wage inequality and relies on a change in composition which can be corrected by matching on observables. In this way, we are comparing groups of individuals who are similar in a number of observable characteristics. We reweighted the sample by matching on a number of observable characteristics (sex, race, age group and income group). We found that the results are comparable to the analysis presented above.

4.1 Passive Smoking and Anti-Smoking Policies

Figures 8 and 9 plot the average cotinine concentration by state and year against the level of excise tax and overall severity of smoking bans. The relationship between exposure and policy variables shows a clear negative correlation. This could indicate that part of the decrease in exposure is due to successful anti-smoking policies. However, as argued in section 2, one must be cautious to interpret this as a causal link, due to omitted factors correlated both with exposure and anti-smoking regulations. Not surprisingly, exposure is highest in the tobacco states such Kentucky or Tennessee where taxes are low and few bans are in place and lowest in California or Utah which have higher levels of taxes and tighter bans.

[Figure 8 and Figure 9]

Figure 10 plots the cotinine concentration together with the excise tax and regulations in three selected states, California, New York and Texas. All variables are presented in deviation to state and (nationwide) time means. These three states have contrasted anti-smoking policies, with a sharp rise in bans and taxes in California, an increase in taxes in New York, but no relative tightening of bans and finally a relative decline in taxes and bans in Texas.

The first striking thing to observe is that cotinine concentration appears to be uncorrelated with changes in smoking bans within states. The increase in smoking bans which took place in California in the mid nineties does not appear to have affected the exposure of non-smokers. Nor does the relative softening of bans in Texas change the exposure in that state.

[Figure 10]

Taxes, on the other hand, appear to have an effect on exposure. In California, the relative exposure increased slightly during the nineties when taxes where on a relative decrease. The sharp rise in 1999 is associated with a marked decline in exposure.

We now turn to a statistical analysis. We first analyse the impact of taxes and bans on passive smoking in the whole sample of non smokers. The results are presented in Table 3. We consider first the effect of taxes on cotinine concentration in non smokers (columns 1 and 2), then of bans (columns 3, 4 and 5), and finally of both taxes and bans together (columns 6 and 7)[19].

[Table 3]

Column (1) of Table 3 displays the effect of (log) excise taxes on passive smoking without controlling neither for state of residence nor for year of survey. The effect is identified here through variations through time and state differences. This is equivalent to the results presented in Figure 8, apart from the fact that the regression controls for age, sex and race. A standard deviation change in state taxes would lead to a reduction in exposure of about 0.02 ng/ml. Note that the average concentration of cotinine is equal to 0.44 ng/ml and that a one standard deviation in excise taxes represents about 25 cents. Thus each dollar increase in taxes reduces exposure by about 18%.

Column (2) controls for year of survey and state of residence. This eliminates state level characteristics and aggregate changes in passive smoking. The effect of taxes is stronger. One standard deviation change in taxes leads to a reduction in exposure of 0.03ng/ml. Thus each dollar increase in taxes reduces exposure by about 27%.

Column (3) displays the effect of regulations on smoking. The result reported in column (3) shows that a one standard deviation increase in regulations would decrease the cotinine concentration in non-smokers by 0.03 ng/ml. However, controlling for state effects reduces considerably the effect of regulations on passive smoking but we can still rule out a zero effect (Column 4). The apparent beneficial effect of smoking bans come mainly from the confounding by time effect (column 5). As shown in the previous section, smoking bans have become more prevalent through the eighties and nineties, and exposure decreased during that period. The evidence in Table 3 shows that the decrease occurred both in states with and without these regulations.

[19] Controlling in addition for income level does not change the results.

Columns 6 and 7 of Table 3 introduce both taxes and regulation in the model. The effects of excise taxes are larger than those estimated in column 2 (one standard deviation change in taxes leads to a reduction in exposure of 0.05ng/ml). This corresponds to an elasticity of about -0.3 to -0.4. This is higher than the tax elasticity of cigarette consumption. The price elasticity of smoking is usually estimated at around -0.5 (Chaloupka and Warner 2000), and the tax-price elasticity is around 0.17 during that period, which translate into a -0.08 tax elasticity of smoking.[20] The fact that passive smoking is more reactive to a change in taxes than smoking it-self is an indication that smokers do not cut down smoking uniformly, but are more prone to cut down on the cigarettes smoked when non-smokers are present. We look further into this result below when we break down the effect by age.

From column 6, regulations appear to have no overall effect. The 95% confidence interval for the effect of bans ranges from -0.006 to 0.014. Even if the effect is at the lowest part of that interval, the effect of regulations would be small. This appears to contradict previous epidemiological studies of bans, see for instance Hopkins et al (2001) for a review, and Travers et al. (2003) and Siegel et al (2004) for more recent contributions. The contradiction is, however, only apparent. Most of the epidemiological work finds that a smoking ban reduces the concentration of ETS in the places where the restrictions apply, but do not measure it directly in non smokers so they do not address the question of displacement. Second, when exposure is measured at the individual level, the study designs are often simple, relying on cross-sectional data or time series evidence. When we do not control for state or year effect, we also find a negative and significant effect of smoking bans (Table 3, columns (3 and 4)).

Column 7 of Table 3 includes the lagged prevalence rate. The results are remarkably stable. We interpret this as an indication that there is little endogeneity of taxes or bans once we control for state and time effects.

[20] Using NHANES 1988-2002, we estimate the tax elasticity of smoking at -0.16, controlling for state and time effects.

4.2 Distributional Effects of Smoking Regulations and Taxes

We now investigate whether state interventions affect individuals differently according to their socio-economic status. In many countries, public health authorities seek not only to improve the health of the population, but also to reduce health inequalities across socio-economic groups. We assess the effect of smoking regulations and changes in excise taxes by household income groups. We split our sample in three income groups of equal size and estimate separately the effect on passive smoking. The results are presented in Table 4.

[Table 4]

For the lowest income group, the effect of taxes is not significant, while the effect of regulations is positive and significant. A total ban would increase exposure by 0.06 ng/ml. An explanation of the increase in exposure can be due to larger displacement effects for low income individuals who are also more likely to live with smokers, as the prevalence of smoking is higher in poorer households. For intermediate levels of income, taxes have a significant and negative effect, while bans appear to have no effect. Finally, for non-smokers in high income households, introduction of smoking regulations decreases the exposure to tobacco smoke.

These results suggest that smoking regulations have a distributional effect, increasing the exposure and putting at risk the health of poorer section of the population while it benefits individuals in higher socio-economic position. The strengthening of smoking regulations could possibly lead to a widening in health disparities across socio-economic groups.

4.3 Passive Smoking in Different Public Places

Until now we have referred to cigarette smoking regulations regardless of the place where these regulations are enforced. Smoking bans may in fact apply to very different places.

Table 4 displays the effect of taxes and regulation on passive smoking considering separately different places where regulation may be enforced. In particular, we distinguish between places where individuals spend their leisure time, and called them “going out” (i.e. restaurants, recreational and cultural facilities), and public transportation, shopping malls, workplaces, and schools[21]. The first row of Table 5 reports the coefficient of (log) excise taxes. The other rows of the table report the regression coefficients of regulation in different places.

[Table 5]

Higher taxes lead to a reduction in cotinine concentration in non-smokers, while tighter regulations have different effects on the cotinine concentration depending on where they are enforced.

The effect of tighter smoking regulations in workplaces is not significantly different from zero. It seems therefore that there is no evidence of an effect of bans on non smokers’ exposure in such places. However, the precision of the estimates does not exclude the fact that a workplace ban could decrease exposure: it should be noted that the lower point of the confidence interval implies a reduction of about 0.16ng/ml for a total ban, a non trivial amount. Tighter regulations in public transportation do not seem to have an effect on reducing the exposure of non smokers. On the other hand, tighter regulations have an impact on the cotinine levels in non-smokers in schools (a one standard deviation change in state regulation in schools decreases cotinine levels by 0.04ng/ml in non-smokers) and in shopping malls (a one standard deviation increase in bans leads to a decrease in cotinine levels of about 0.3 ng/ml).

Most interesting is the observed impact of tighter regulations in public recreational places. We observe a significant increase in the cotinine level in non-smoking individuals when bans are enforced in public recreational places. A one standard deviation increase in bans in “going out” places lead to an increase in cotinine levels of more than 0.2 ng/ml. This effect cannot be explained by a direct effect of the ban on non-smokers, which would have decreased the exposure of those who spend time in such places. The only explanation for an

[21] See Appendix A for a more detailed description of the regulation data.

increase in exposure is through an indirect contamination due to the displacement of smokers towards non-smokers. We now investigate this point further.

4.4 Characterizing Displacement Effects

To uncover displacement effects due to tougher smoking regulations in places where people go out, we focus on non-smokers who would not be directly affected by such regulations. We focus our attention on children. First it is likely that children are less prone than adults to go to bars, restaurants and, perhaps, recreational public places. Second, the displacement effect should be larger for children whose parents are smoking. Third, the displacement effect should also be larger when people are more likely to be indoors, such as in winter, especially at a young age. We apply the methodology developed in section 2.4.

4.4.1 - Policy Impact by Age Group

In section 2.4, we listed the conditions under which we are able to get consistent estimates of the relevant parameter, regardless of whether taxes or anti-smoking regulations are endogenous. The first condition states that the average excise tax or smoking regulation is the same across age groups. This is essentially an assumption about random sampling. It would be violated if, for instance, more children had been sampled in earlier periods when taxes were lower. Given that we control for state and time fixed effects, it requires random sampling within state, which is the case given the design of NHANES, and the fact that we use weights to make the sample representative. The next assumption relates to the fact that age is exogenous to unobserved shocks to exposure. Finally, we assume that the (possible) endogeneity of taxes or anti-smoking regulations is the same across age groups. For instance, this would be violated if (within state) taxes were changed differently as a results of unobservable shocks to exposure according to changes in the relative size of different age groups.

We now proceed in analysing the effect of taxes and regulation on passive smoking across age groups. Table 6 separates non-smokers by age groups. Column (1) refers to the overall sample of non smokers. Columns (2) to (5) of Table 6 distinguish between four different age groups. The first age group is from 4 to 8, an age where children are mostly either at home or in school or day-care, and supervised by an adult. At that age, it is unlikely that any peers would be smoking. These individuals are therefore exposed either to ETS at home, where parents or other adults in the household smoke, or in public places. The second age group ranges from 9 to 12, an intermediate age group between early childhood and adolescence. The third age group ranges from 13 to 20. Exposure for these individuals would come from parents and also from peers. Finally, we group all individuals aged 21 or above into group 4. We have experimented with different cut-off ages, in particular with young and elder adults, and have found similar results.[22]

[Table 6]

The first row of Table 6 displays the effect of taxes by age groups. The effect of taxes decreases with age. Young children are the most sensitive to a change in taxes. For children aged 4 to 8, a one standard deviation in taxes decreases the cotinine concentration by 0.2 ng/ml. This corresponds to a tax elasticity of about -0.8. For older individuals, taxes have no significant effect on exposure to tobacco smoke. This is further evidence that cigarettes smoked in the presence of non-smokers and especially children are the first to be cut as a result of a change in taxes. This suggests that smoking is partly a social activity so that smokers derive more utility to smoke with other adults. An alternative explanation could be that adults with children are poorer and face liquidity constraints, which would make them more sensitive to a change in tobacco prices. The empirical literature has documented the higher price elasticity for poorer individuals (Chaloupka (1991), Farrelly et al (1998)). However, controlling for income does not change the results.

The next row of Table 6 decomposes the effect of regulations by age groups. In places like restaurants, bars and other recreational places ("going-out"), a one standard deviation change in regulations in such places increase the exposure of children by about 0.65 ng/ml. This is also the case for the next age group, 8 to 12 years old. The effect is smaller for teen-agers and beneficial for adults, although this effect is not statistically different from zero. This can be

[22] The data set contains about 8698 children of age eight or less, 2816 children of age 8 to 12, 4649 individuals of age 12 to 20 and 13504 adults of age 20 or more.

interpreted as the existence of a substitution effect for adults between leisure activities in public places, where regulation can be enforced, and in private places, where no restriction to smoking can be enforced. This effect would lead to a displacement of smoking towards places where children and adults interact.

It is worth putting this increase into context. On average a smoker gets 12 ng/ml per cigarette (see Adda and Cornaglia, 2006). The increase in cotinine following a tightening of smoking bans in places where people go out amounts to smoking $1/20^{th}$ of a cigarette. Even if the increase in exposure is sizable for children, it is consistent with a displacement where adults smoke a few cigarettes more at home.

Tighter regulations in public places other than recreational places have on average negative coefficients, especially for young children. The effect of a ban in schools has the expected sign, and is significantly different from zero, for children of age 8 to 12. A one standard deviation increase results in a decrease in exposure of about 0.10 ng/ml, a 15 % decrease. Tighter regulations in shopping malls have an impact only on the exposure of children. In particular, a one standard deviation increase leads to a decreased exposure of about 0.60 ng/ml in small children and of 0.45 ng/ml in children aged 8 to 12. In general, smoking regulations have a larger impact, either beneficial or detrimental, on young children. For adults, we cannot find evidence of an effect of smoking regulations, wherever they are enforced. This is consistent with a displacement of smoking, where non-smokers accompany smokers to places where smoking is allowed.

4.4.2. - Policy Impact by Household Smoking Status in children

The previous analysis shows that the group of individuals that is the most affected by changes in taxes and regulations are children. In Table 6 we separate children by family smoking status. The assumptions required for the consistency of the differential effect of taxes and anti-smoking regulations are less likely to hold when we consider household smoking status. This has to be taken into account when interpreting the results.

Table 7 reports the effect of one standard deviation in taxes and regulation on children, by place of enforcement and household smoking status. Column 1 refers to children that live in non-smoking households; column 2 refers to children living in smoking households.

[Table 7]

The observed effects of changes in regulations are considerably larger in children living in smoking households than in children living in non-smoking households. Children in smoking households benefit from an increase in excise taxes (a one standard deviation in taxes leads to a reduction in the cotinine level observed in children of about -0.3 ng/ml (row 1 of table 6). The effect is larger in magnitude to the one for all children irrespective of smoking status presented in Table 6. Children in non-smoking household are not affected by changes in taxations, as the coefficient is close to zero and is not statistically significant from zero.

The effect of tighter regulations on children in smoking households differs according to where the regulations are enforced. In bars, restaurants and other recreational places (row 2 of table 7) the coefficient of regulation is positive and significant (a one standard deviation in regulation leads to an increase in the cotinine level in children of more than 1 ng/ml). On the other hand, the effect of regulations on children living in non smoking families is not significant and the point estimate is very close to zero. These results are in accordance with a displacement effect of adults (smokers) toward home.[23] Note that the argument about the endogeneity of smoking bans that we discussed in section 2 becomes more contrived when we analyze the effect of bans in recreational places on children. This is the case for instance of smoking regulations in bars, restaurants. These are in fact often introduced to protect employee from exposure to tobacco smoke and do not have the welfare of children in mind, a group of the population that hardly goes into such places.

Regarding the effect of regulation in other public places we observe that tighter regulations in shopping malls (row 4 of table 7) leads to a statistically significant reduction in the cotinine

[23] An alternative explanation is that the introduction of tighter bans induces light smokers to quit proportionally more than heavy smokers. This would lead to a selected sample of heavy smoking parents within the smoking household group and would lead to a positive bias in the effect of regulations.

levels observed in children living in smoking households. Again this does not have a sizable and significant effect on children in non smoking households.

4.4.3. - Policy Impact by Season

To substantiate further the displacement effect due to tougher regulations in bars, restaurants and recreational places, we investigate the differential effect of these measures during winter and summer. In colder months it is more likely that smokers will light up cigarettes in indoors places, exposing therefore non-smokers to a higher level of environmental tobacco smoke than when they have the option to be outdoor. More particularly, we are interested on how seasonal pattern interact with anti-smoking policies.

Coming back to the assumptions listed in Section 2.4, season is an ideal variable to use as it is likely to be exogenous. Taxes and anti-smoking regulations do not change with season and if sampling is random across seasons the three requirements are met. The differential effect of taxes and smoking regulations is therefore consistently estimated even if their overall effect may be biased due to endogeneity. This will also be true if we condition the regressions on household smoking status.

NHANES 1988-1994 reports the month of the interview and we categorize the months as winter (October to April) or summer. We interact taxes and smoking regulation with an indicator of being examined during the winter months. We concentrate on children given that this group appears to be more subject to displacement effects. The results are presented in Table 8.[24]

[Table 8]

The first row of table 8 indicates that children in smoking household have higher levels of cotinine during winter periods. We find no seasonal effects for children living in non smoking households. For this group, we do not find significant differential seasonal effects of

[24] A caveat is that the time span is limited to 1991-1994, as we do not have information on smoking regulations prior to 1991, nor information on month of interview after 1994. With limited time variations, we cannot control for state effects, but rely instead on regional effects. We use four regional dummies, North East, Mid West, South and West. We also group together bans imposed in other places than “going out” as their effect is more homogenous.

taxes or regulations either. In contrast, when we look at children in smoking families, we find strong seasonal effects. Taxes appear to have a stronger effect in winter which is consistent with adults and children being in-doors. Similarly, the effect of smoking restrictions in places where individuals go out is more pronounced in winter than in summer. Finally, we find that for children in smoking households ‘other regulations’ are more efficient in winter than in summer.

Throughout this section, we track the effect of a ban in recreational places which appeared to increase the exposure of tobacco smoke in non-smokers of all age. While this effect may be surprising in the first place, we show that it is consistent with a displacement effect, where adult smokers chose to smoke at home instead of in bars or restaurants. The results show that the effect is present for the group which is more likely to be affected by displacement: young children whose parents are smoking, especially in periods during which they stay indoor.

4.5 Health and Economic Consequences of Anti-Smoking Policies

The results presented so far are about the effect of anti-smoking policies on the exposure of non-smokers. As we discuss in the introduction, passive smoking has been linked to cardio-vascular diseases, cancers and respiratory diseases, especially in children. To put our results in perspective, we briefly present some evidence of the effect of passive smoking on health. Given the lack of evidence of any large effect of excise taxes or smoking bans on adults[25], we concentrate on children. The purpose of this analysis is not to reproduce results established in the medical literature, but to provide some rough estimates to convert the effects of state interventions uncovered in our previous section into health and economic effects.

We exploit the information on health outcomes contained in the NHANES III. As the incidence of cardio-vascular diseases or tobacco-related cancers in children is very low, we consider symptoms of respiratory diseases such as asthma which is reported in the data set. This respiratory disease is a serious condition which results in hospitalisation and is the most common cause of school absenteeism due to chronic conditions. The prevalence of asthma for children of this age is about 10%.

[25] See paragraph 4.2

We estimate a simple linear probability model of the prevalence of asthma and we control for the cotinine concentration as well as for age, sex and race. We include all children aged four to twelve. We find that an increase of one ng/ml in cotinine concentration leads to a 0.8 percentage point increase in the prevalence of asthma. [26] These estimates obviously do not consider the possibility of confounding by other unmeasured variables which were not included in the regression. With this caveat in mind, we can calculate the effect of anti-smoking interventions on the incidence of asthma in children. To evaluate the economic consequences we use estimates in Wang et al (2005). They estimate the overall cost of asthma at $ 791 per child and that each child with asthma misses 2.48 days of schooling per year. From the NHANES III, we estimate the number of children of age 4 to 12 to be around 36 millions.

From Table 6, a one standard deviation increase in taxes will lead to a 0.16 percentage point decrease in the prevalence of asthma for children aged 4 to 8. For the age group 8 to 12, the reduction would correspond to 0.1 percentage points. Hence a one standard deviation increase in taxes across all states would reduce the number of children suffering from asthma by about 45,000 cases, corresponding to a saving of about $36 millions per year and a reduction of 116,000 days of school missed.

Similarly, a one standard deviation increase in regulation in bars, restaurants and other recreational places leads to an increase of 0.5% in the prevalence of asthma for the youngest age group. This back-of-the-envelope calculation suggests that a tightening of smoking bans across all states would lead to an average increase in the cost due to asthma of about $126 millions and about 400,000 days of school missed every year (out of a total of approximately 7.5 billion days).

[26] The standard error is equal to 0.3.

5 Conclusion

The effect of passive smoking is of increasing public concern. Although the economic literature has evaluated the effect of government intervention on smoking intensity or prevalence, there has been, so far, no direct evaluation of these measures on non-smokers.

In this paper we characterize the extent of exposure to environmental smoke, and evaluate the effect of changes in excise taxes and bans on passive smoking. We use a direct measure of passive smoking which has not been used in the economic literature, the concentration of cotinine, a metabolite of nicotine, in body fluids of non smokers. This allows us to precisely identify the effect of state intervention on non-smokers.

We find that increasing taxes on cigarettes reduces on average exposure to cigarette smoke of non smokers. The effect of state excise taxes also varies across demographic groups. We find that taxes have a strong effect on young children living with smokers but no effect on non smoking adults. This suggests that smokers cut down on the cigarettes they smoke at home but not those in social activities with other adults.

Using information on the implementation of the Clean Air Act across time and different US states, we also find that smoking regulations have on average no effect on exposure. We show that this latter result is not due to a lack of statistical power to detect a precise effect but rather to the fact that regulations have contrasting effects depending on where they are imposed and depending on which group of the population is affected. While bans in public transportation, shopping malls, and schools lead to the desired decrease in exposure of non smokers, we find that bans in recreational public places can perversely increase tobacco exposure of non smokers by displacing smokers to private places where they contaminate non smokers. Children seem to be particularly affected by this displacement. The level of cotinine in small children considerably increases as a result of bans in recreational public places, while decreases if tighter bans are put in place in public transport or shopping malls.

A third and important finding is that smoking regulations increases exposure of poorer individuals, while it is beneficial to individuals in higher socio-economic position. The rise in the number of regulations observed over the nineties is likely to have increased health inequalities related to passive smoking.

Our results question the usefulness of bans in reducing smoking exposure for non smokers. More precisely, we show that policies aimed at reducing exposure to tobacco smoke induce changes in behaviors which can offset these policies. It is therefore of crucial importance to understand how smoking behaviors are affected by regulations. So far, the literature has not gone far enough in studying smoking behavior to be able to evaluate their effect on non smokers. It is not enough to show that smokers react to prices or taxes. Information on which particular cigarette is cut down during the day, where smokers smoke and with whom are also relevant. There are complex interactions at play and considerable heterogeneity in their effects across socio-demographic groups. Using a biomarker such as cotinine concentrations is a very direct way of evaluating the overall effect of interventions and the induced changes in behaviors.

On the policy side, it seems therefore important when designing public policies aimed at reducing tobacco exposure of non smokers to distinguish between the different public places where bans are introduced. Displacing smoking towards places where non-smokers live is particularly inefficient. It may also increase health disparities across socio-economic groups and in particular in children. Therefore, total bans may not be the optimal policy. A better policy may be to allow for alternative places to which smokers can turn to. It would benefit children but harm non smoking adults. There are several reasons why one may want to protect children. They constitute a vulnerable group with little choices to avoid contamination. This age group is particular prone to tobacco related diseases and poor health in childhood has lasting consequences not only for future health but also for the accumulation of human capital (Case et al, 2005).

Governments in many countries are under pressure to limit passive smoking. Some pressure groups can be very vocal about these issues and suggest bold and radical reforms. As often, their point of view is laudable, but too simplistic in the sense that they do not take into account how public policies can generate perverse incentives and effects. Up to know there is little guidance on how to design optimal policies to curb passive smoking. This paper fills this gap.

Appendix A: Proof of Proposition (1)

Consider the following model:

$$Cot_i = \alpha_0 + \alpha_1 x_i + \alpha_2 D_i + \alpha_3 x_i D_i + u_i \qquad i = 1, \ldots N$$

Assume that $E(x_i \mid D_i) = E(x_i) = \overline{x}$, where upper bar variables denote variable means. Let $Z = [1,\ x_i,\ D_i, x_i D_i]$ be N by 4 matrix and let σ_X^2 denote the variance of x_i, σ_{Xu} and σ_{Du} the covariance between x_i and u_i and D_i and u_i. The expression for the asymptotic bias can be expressed (after some straightforward algebra):

$$E\left(\frac{Z'Z}{N}\right)^{-1}\frac{Z'u}{N} = \begin{bmatrix} \dfrac{-\overline{x}\sigma_{Xu}}{\sigma_X^2} \\ \dfrac{\sigma_{Xu}}{\sigma_X^2} \\ 0 \\ 0 \end{bmatrix}$$

Appendix B: Smoke free regulations data

The information contained in this appendix, are drawn from the codebook for the "Tobacco Control Policy and PrevalenceData: 1991-2001" [27] , compiled by researchers in the Department of Health Behavior at the Roswell Park Cancer Institute (RPCI) in Buffalo, New York, in conjunction with researchers at the MayaTech Corporation in Washington, DC.[28]
Eleven different locations were regulations were enacted were identified: Government worksites, Private worksites, Child care centers, Health care facilities, Restaurants, Recreational facilities, Cultural facilities, Public transit, Shopping malls, Public schools, and Private schools. And for each of these locations it has been measured the degree of restrictions enforced in the various years (1991-2001).

General Location Restriction Decisions

The following "standard coding scheme" was employed for the majority of locations of interest (including: government worksites, private worksites, health care facilities, restaurants, public transit, and shopping malls).

[27] http://www.impacteen.org/tobaccodata.htm

[28] Coding for public schools, private schools and cultural facilities were developed by MayaTech from the state smoke-free air law coding provided by RPCI.

0	*No provision/not meet a restriction*
1	*Restrict smoking to designated areas or require separate ventilation with exemptions for locations of a certain size (e.g. restaurants with a seating capacity of less than 50)*
2	*Restrict smoking to separately ventilated areas or a ban with exemptions for certain locations where only a restriction applies*
3	*Ban at all times*

For locations other than those mentioned in the table above, different coding schemes were used. In the following we report the coding schemes that were used for each of them.

1. Child Care Centers coding scheme

0	*No provision/not meet a restriction*
1	*Restrict smoking to designated areas*
2	*Restrict smoking to separately ventilated areas or a ban when children are present with exemptions*
3	*Ban when children are present (commercial daycare)*
4	*Ban at all times when children are present (explicitly including home-based)*
5	*Ban at all times (explicitly including home-based)*

2. Recreational Facilities coding scheme

0	*No restriction*
1	*Restricts smoking to DSAs in gyms or arenas*
2	*Restricts smoking to DSAs in both gyms and arenas*
3	*Restricts smoking to DSAs in all recreational facilities*
4	*Bans smoking in gyms or arenas and restricts to DSA(s) in other recreational area(s)*
5	*Bans smoking at all recreational locations*

3. Cultural Facilities coding scheme

0	*No restriction*
1	*Restricts smoking to DSAs in fewer than 3 cultural areas*
2	*Restricts smoking to DSAs in 3-5 cultural areas*
3	*Restricts smoking to DSAs in more than 5 cultural areas*
4	*Restricts smoking to DSAs in all cultural facilities*
5	*Bans smoking at all cultural locations*

4. Schools coding scheme

0	*No provision/not meet a restriction*
1	*Restrict smoking to designated areas*
2	*Restrict smoking to separately ventilated areas or a ban when children are present with exemptions*
3	*Ban when children are present (school buildings)*

4	*Ban at all times when children are present (buildings and grounds)*
5	*Ban at all times (buildings and grounds)*

For details about the choices made in interpreting the language of the laws and regulations case by case in the different States we refer to the official codebook drawn by Impacteen (http://www.impacteen.org).

We have aggregated these different locations in a number of ways. First, we have constructed a general measure of restriction, considering an average of all the locations. In a second time, we have aggregated the different public locations in: 1. recreational activities ("going out") which includes restaurants, cultural and other recreational public places; 2. public transport; 3. shopping malls; 4. workplaces, which includes both governmental and private workplaces; 5. school, which includes childcare centres, and both public and private schools.

REFERENCES

Adda J and F Cornaglia (2006) “Taxes, Cigarette Consumption and Smoking Intensity“, forthcoming, *American Economic Review*.

American Cancer Society. *Cancer Facts and Figures 2003.* Atlanta, GA.

ASH (2005) “Going Smokefree: the case for all pubs and clubs”. http://www.ash.org.uk/

Becker G and K Murphy (1988) “A Theory of Rational Addiction”, *Journal of Political Econom*y, 96, 4, 675-699.

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

Bertrand, M, Duflo E and Mullainathan S (2004) “How Much Should We Trust Difference in Differences Estimates?”, *Quarterly Journal of Economics* 119(1), pp. 249-275.

Case, A Fertig A and C Paxson (2005) “The Lasting Impact of Childhood Health and Circumstance”.*Journal of Health Economics,* 2005, v. 24, pp. 365-389.

Chaloupka F (1991), “Rational Addictive Behavior and Cigarette Smoking”, *Journal of Political Econom*y, 99, 4, 722-742.

Chaloupka, F., and Warner, K., (2000). “The Economics of Smoking”, in *The Handbook of Health Economics*, edited by Newhouse, J., and Cuyler, A., New York: North-Holland, Elsevier Science B.V., 1539-1627.

DeCicca P, Kenkel D, and A Mathios (2002), “Putting Our the Fires: Will Higher Taxes Reduce the Onset of Youth Smoking?”, *Journal of Political Economy* , 110, 1, 144-69.

DiNardo, J Fortin N, and T Lemieux (1996), "Labour Market Institutions and the Distribution of Wages, 1973-1992: a Semiparametric Approach", *Econometrica*, 64, 1001-1046

Environmental Protection Agency –US (1992), “Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders”, Washington, DC: EPA, Office of Air and Radiation, EPA publication 600/6-90/006F.

Evans W, Farrelly M and E. Montgomery (1999) “Do Workplace Smoking Bans Reduce Smoking?”, *American Economic Review*, 89, 4, 728-747.

Evans W and J Ringel (1999), “Can Higher Cigarette Taxes Improve Birth Outcomes?”, *Journal of Public Economics*, 72, 1, 135-154.

Farrelly MC, Bray JW, Office on Smoking and Health. “Response to increases in cigarette prices by race/ethnicity, income, and age groups - United States, 1976-1993”. *Morbidity and Mortality Weekly Report* 1998;47(29):605-9.

Gergen P J, Fowler JA, Maurer KR, Davis WW, and M D Overpeck (1998) “The Burden of Environmental Tobacco Smoke Exposure on the Respiratory Health of Children 2 Months Through 5 Years of Age in the United States: Third National Health and Nutrition Examination Survey, 1988 to 1994” *Pediatrics*, February 1, 101, 2, 8e-8.

Hackshaw AK, Law MR, and NJ Wald (1997), “The accumulated evidence on lung cancer and environmental tobacco smoke”, *BMJ*, 315, 980-988.

He J, Vupputuri S, Allen K, Prerost MR, Hughes J, Whelton PK (1999), “Passive smoking and the risk of coronary heart disease—a meta-analysis of epidemiologic studies”, *N Engl J Med* , 340, 920-6.

Hopkins DP, Briss PA, Ricard CJ, Husten CG, Carande-Kulis VG, Fielding JE, Alao MO, McKenna JW, Sharp DJ, Harris JR, Woollery TA, Harris KW (2001), “Reviews of the Evidence Regarding Interventions to reduce Tobacco Use and Exposure to Environmental Tobacco Smoke”, *American Journal of Preventive Medicine*, 20, 16-66.

Howard G, Wagenknecht LE, Burke GL (1998) “Cigarette smoking and progression of atherosclerosis: the Atherosclerosis Risk in Communities (ARIC) study”, *JAMA*, 279, 119-124.

IARC (2004) “Tobacco smoke and involuntary smoking, Monographs on the evaluation of carcinogenic risks to humans”, Vol 83, Lyon, France.

Jarvis M et al. (2000), “Children’s exposure to passive smoking in England since the 1980’s: cotinine evidence from population surveys”, *British Medical Journal,* 321, 343-5.

Jarvis M, Tunstall-Pedoe H, Feyerabend C, Vesey C and Y Salloojee (1984), “Biochemical markers of smoke absorption and self reported exposure to passive smoking”, *Journal of Epidemiology and Community Health*, Vol 38, 335-339

Jarvis M, Tunstall-Pedoe H, Feyerabend C, Vesey C and Y Saloojee (1987), “Comparison of tests used to distinguish smokers from non smokers”, *American Journal of Public Health*, Vol 77, 1435-8.

Jarvis M, Feyerabend C, Bryant A, Hedges B, and P Primatesta (2001), “Passive smoking in the home: plasma cotinine concentrations in non-smokers with smoking partners”, *Tobacco Control* 10(4), 368 – 374.

Kriz P, Bobak M, and B Kriz (2000), “Parental smoking, socio-economic factors, and risk of invasive meningococcal disease in children: a population based case-control study. Archives of Disease in Childhood”, 83, 117-21.

Lam T, Leung GM, and LM Ho (2001), "The effects of environmental tobacco smoke on health services utilization in the first eighteen months of life", *Pediatrics*, 107, 6.

Law MR, Morris JK, and NJ Wald (1997), "Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence", *BMJ*, 315, 973-980.

Levy DT, and KB Friend (2003), "The effects of clean indoor air laws: what do we know and what do we need to know?", *Health Educ Res*, 18(5), 592–609.

Mannino DM, Moorman JE, Kingsley B, Rose D, Repace J., (2001) "Health effects related to environmental tobacco smoke exposure in children in the United States", *Archives of Pediatric Adolescent Medicine*, 155, 36-41.

NHS Scotland (2005), "International Review of the Health and Economic Impact of the Regulation of Smoking in Public Places"
www.healthscotland.researchcentre/pdf/internatioonalreviewfullreport.pdf

Otsuka R (2001), "Acute effects of passive smoking on the coronary circulation in healthy young adults", *JAMA*, 286, 436-441.

Orzechowski and Walker (2001) "The Tax Burden on Tobacco. Historical Compilation", Volume 36.

Pirkle J L, Flegal K M, Bernert J T, Brody D J, Etzel R A, and K R Maurer (1996), "Exposure of the US population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988 to 1991", *JAMA*, 275, 1233-1240.

Rosenzweig, M and P Schultz (1983) "Estimating a Household Production Function: Heterogeneity, the Demand for Health Inputs, and Their Effects on Birth Weight", *Journal of Political Economy*, 91:5 (October), 723-746.

Siegel M, Albers AB, Cheng DM, Biener L, Rigotti NA (2004), "Effect of Local Restaurant Smoking Regulations on Environmental Tobacco Smoke Exposure Among Youths", *American Journal Public Health*, 94, 321-325.

Strachan DP and DG Cook (1997), "Parental smoking and lower respiratory illness in infancy and early childhood", *Thorax*, 52, 905-914.

Travers M et al. (2003), "Indoor air quality in hospitality venues before and after implementation of a clean indoor air law", Western New York, MMWR, 53, 1038-1041.

Wang LY, Zhong Y, Wheeler L. (2005) "Direct and indirect costs of asthma in school-age children". *Preventing Chronic Disease*, 2, 1, 1-10.

Whincup P, Gilg J, Emberson J, Jarvis M, Feyerabend C, Bryant A, Walker M, Cook D (2004), "Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement", *BMJ*.

Whitlock G, S MacMahon, S Vander Hoorn et al. (1998), “Association of environmental tobacco smoke exposure with socioeconomic status in a population of 7725 New Zealanders”, *Tobacco Control*, 7, 276-280.

Table 1 - Descriptive Statistics

	Whole sample	Individuals in smoking families	Individuals in Non smoking families
# of observations	29687	5770	23897
Average level of cotinine (ng/ml)	0.44	1.47	0.26
	(1.02)	*(1.59)*	*(0.75)*
Proportion with detectable cotinine measure (>0.035ng/ml)	84%	99%	79%
Proportion with cotinine>1ng/ml	14%	46%	5%
Proportion with cotinine>5ng/ml	1%	4%	0.5%
Average age	33.5	22.7	35.7
Age range	4-90	4-90	4-90
sex (% male)	46	46.8	45.8
% white	74	72	74
% black	12	18	11

Note: Standard deviations in parenthesis. The whole sample consists of all non-smoking individuals who have a valid cotinine measure lower than 10ng/ml.

Table 2 - Descriptive Statistics. Excise Taxes and Regulations

	Average Level	Range	Within State Standard dev.
Log tax	3.43	0.97-4.62	0.27
Average Regulation	0.79	0-2.63	0.22
Bans Going-out	0.76	0-2.67	0.25
Bans public transportation	1.24	0-3	0.31
Bans shopping mall	0.27	0-3	0.31
Bans workplace	0.70	0-3	0.28
Bans schools	0.85	0-2	0.27

Table 3 - Effects of One Standard Deviation in Taxes and Regulations on Passive Smoking

Dependent variable: cotinine (ng/ml). Average Cotinine Level: 0.44ng/ml

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log Tax	-0.02*	-0.03**				-0.04**	-0.05**
	(0.012)	(0.012)				(0.015)	(0.019)
Regulations			-0.032**	-0.012**	-0.006	0.005	0.004
			(0.009)	(0.002)	(0.008)	(0.006)	(0.005)
Controls[#]:							
Year Dummies		X			X	X	X
State Dummies		X		X	X	X	X
Age, sex, race, state GDP	X	X	X	X	X	X	X
State smoking prevalence							X

Robust standard errors adjusted for clustering at state level in parenthesis. ** significant at 5%, * significant at 10%

[#] Controlling in addition for income level does not substantially change the results.

Table 4 - Distributional Effects of Taxes and Bans (One Standard Deviation Effect)

	(1)	(2)	(3)
	Household Income: [0,33%]	Household Income: [33%,66%]	Household Income: [66%, 100%]
Log Tax	-0.02	-0.09**	-0.02
	(0.03)	(0.03)	(0.03)
Regulations	0.02**	0.02	-0.01**
	(0.01)	(0.02)	(0.01)
Controls:			
Year Dummies	X	X	X
State Dummies	X	X	X
Age, sex, race	X	X	X
State Smoking Prevalence	X	X	X

Robust standard errors adjusted for clustering at state and year level in parenthesis. ** significant at 5%, * significant at 10%

Table 5 - Effects of One Standard Deviation of Taxes and Regulation on Passive Smoking, by place of enforcement. Dependent variable: cotinine.

	All ages
Average Cotinine Level	0.44ng/ml
(Standard Deviation)	(1.00)
Log Tax	-0.04**
	(0.02)
Regulation Going out	0.21**
	(0.07)
Regulation Public Transport	0.05
	(0.04)
Regulation Shopping Mall	-0.28**
	(0.10)
Regulation Workplace	-0.001
	(0.01)
Regulation Schools	-0.04**
	(0.015)
Controls:	
Year Dummies	X
State Dummies	X
Age, sex, race, state GDP	X
State smoking prevalence	X

Regressions controls for age, sex, race, state GDP, state of residence and year of survey. Robust standard errors adjusted for clustering at state level in parenthesis. ** significant at 5%, * significant at 10%.

Table 6 - Effect of One Standard Deviation of Taxes and Regulation on Passive Smoking, by place of enforcement. Dependent variable: cotinine.

	(1) All ages	(2) Age<8	(3) Age 8-12	(4) Age 13-20	(5) Age 20+
Average Cotinine Level	0.44ng/ml	0.94 ng/ml	0.63 ng/ml	0.74 ng/ml	0.43 ng/ml
(Standard Deviation)	(1.00)	(1.47)	(1.03)	(1.26)	(0.84)
Log Tax	-0.04**	-0.20**	-0.12**	-0.01	-0.01
	(0.02)	(0.06)	(0.03)	(0.05)	(0.02)
Regulation Going out	0.21**	0.65**	0.46**	0.07	-0.03
	(0.07)	(0.14)	(0.10)	(0.11)	(0.14)
Regulation Public Transport	0.05	-0.04	-0.01	-0.03	0.04
	(0.04)	(0.10)	(0.06)	(0.09)	(0.04)
Regulation Shopping Mall	-0.28**	-0.60**	-0.45**	-0.01	-0.19
	(0.10)	(0.22)	(0.17)	(0.15)	(0.11)
Regulation Workplace	-0.001				0.07
	(0.01)				(0.08)
Regulation Schools	-0.04**	0.06	-0.10**	-0.04	
	(0.015)	(0.06)	(0.05)	(0.03)	

Regressions controls for age, sex, race, state GDP, state of residence and year of survey. Robust standard errors adjusted for clustering at state level in parenthesis. ** significant at 5%, * significant at 10%.

Table 7: Effect of One Standard Deviation in Taxes and Regulation on *Children*, by Place of Enforcement and Household Smoking Status

	(1)	(2)
	Non Smoking Households	Smoking Households
Average Cotinine Level	0.27 ng/ml	1.97 ng/ml
(Standard Deviation)	(0.44)	(1.85)
Log Tax	0.012	-0.30**
	(0.02)	(0.06)
Regulation Going Out	0.03	1.08**
	(0.04)	(0.15)
Regulation Public	0.03	-0.03
Transport	(0.02)	(0.13)
Regulation Shopping Mall	0.01	-1.05**
	(0.07)	(0.23)
Regulation Schools	0.008	-0.09
	(0.01)	(0.07)
Controls:		
Year Dummies	X	X
State Dummies	X	X
Age, sex, race, state GDP	X	X
State smoking prevalence	X	X

Regressions controls for age, sex, race, state GDP, state of residence and year of survey. Robust standard errors adjusted for clustering at state level in parenthesis.** significant at 5%, * significant at 10%.

Table 8 – Seasonality Effect in Children, by Household Smoking Status (One Standard Deviation Effect)

	Children Non Smoking Households	Children Smoking Households
Winter	0.001	0.59**
	(0.04)	(0.21)
Log Tax	-0.13	0.04
	(0.09)	(0.08)
Tax*Winter	0.12	-0.27**
	(0.09)	(0.12)
Going out	0.07	0.08
	(0.05)	(0.11)
Going out*Winter	0.002	0.70**
	(0.16)	(0.32)
Other regulation	-0.05	-0.02
	(0.04)	(0.13)
Other regulation*Winter	-0.02	-0.95**
	(0.13)	(0.31)
Controls:		
Year Dummies	X	X
Regional Dummies	X	X
Age, sex, race	X	X

Robust standard errors adjusted for clustering at state and year level in parenthesis. ** significant at 5%, * significant at 10%

Figure 1: Cotinine Level by Number of Cigarettes Smoked in the Household

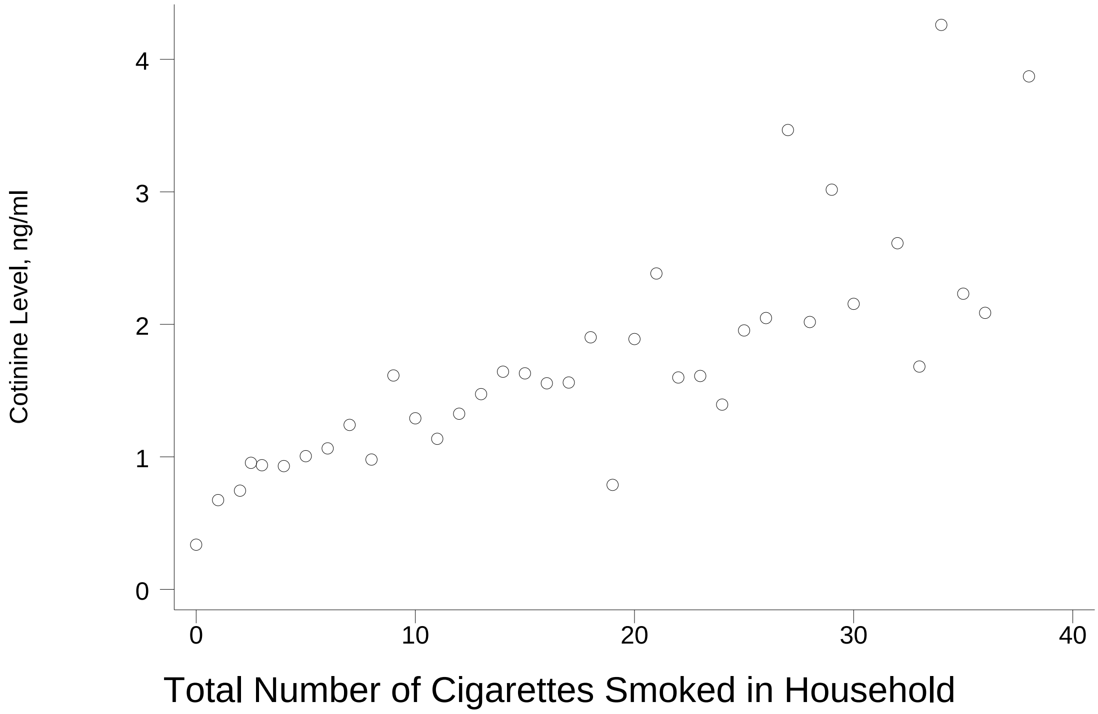

Figure 2: State Excise Taxes, by US State 1988-2002.

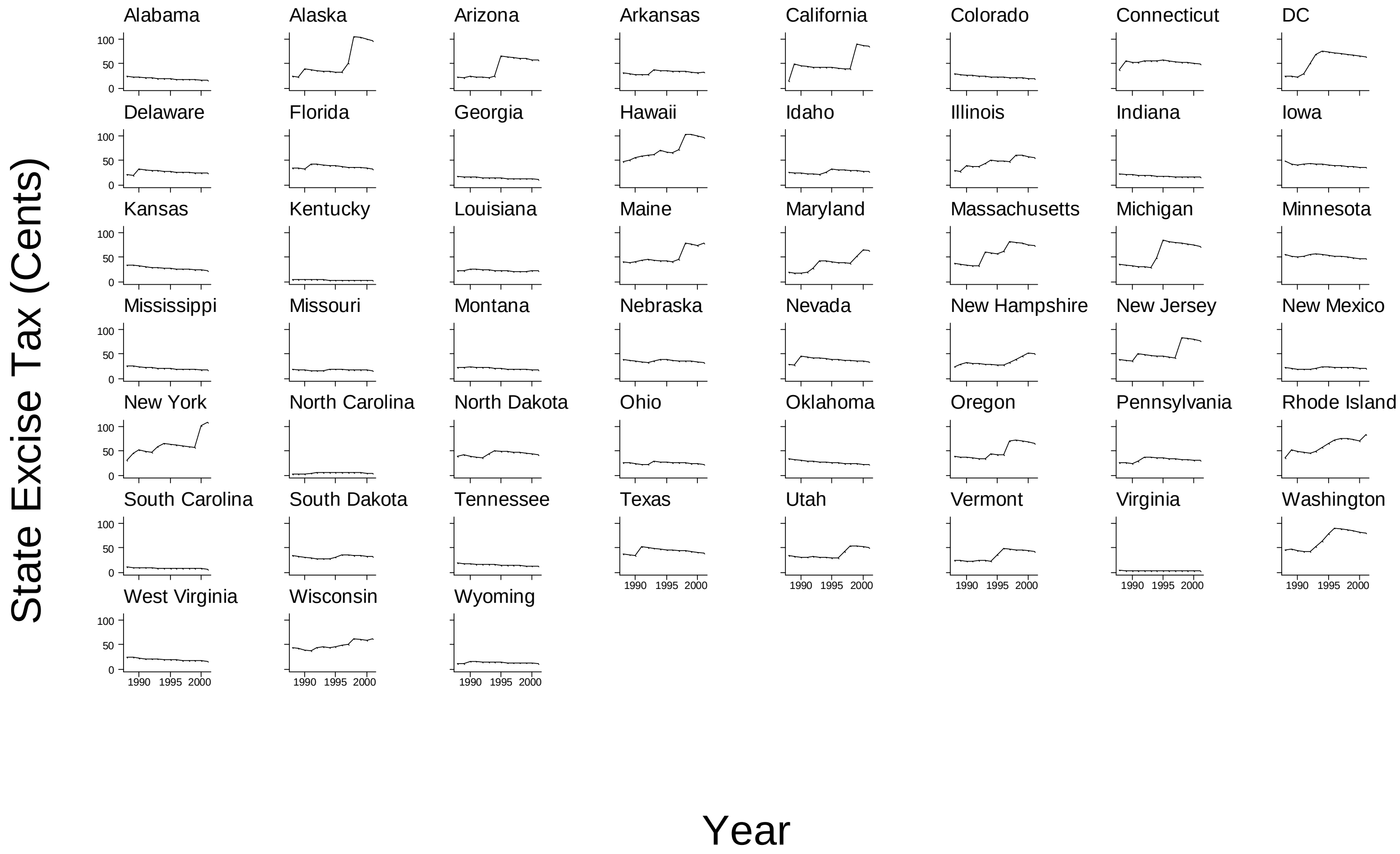

Figure 3: Level of Restrictions on Smoking, by US State, 1991-2001.

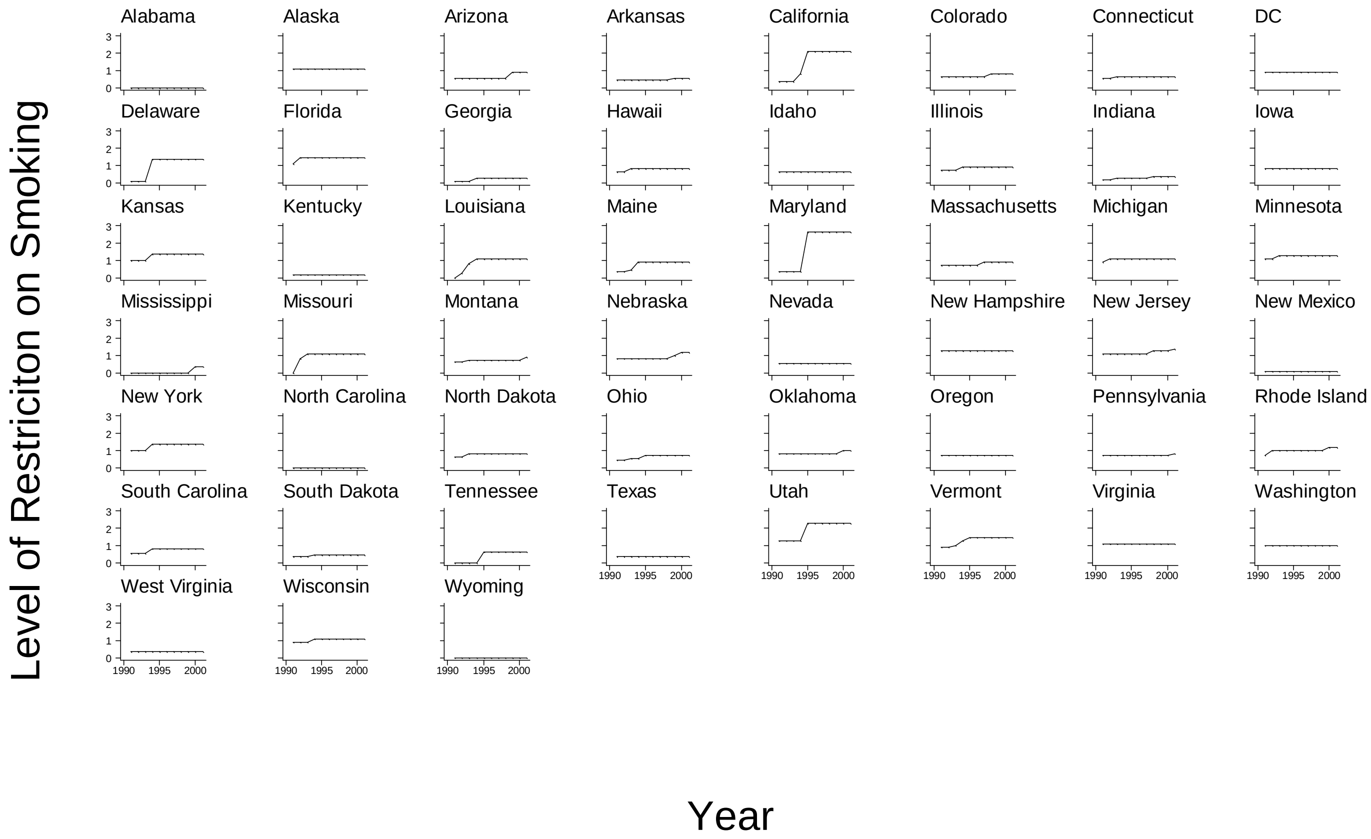

Figure 6: Average Cotinine Concentration in Non-Smokers – Non Smoking Households

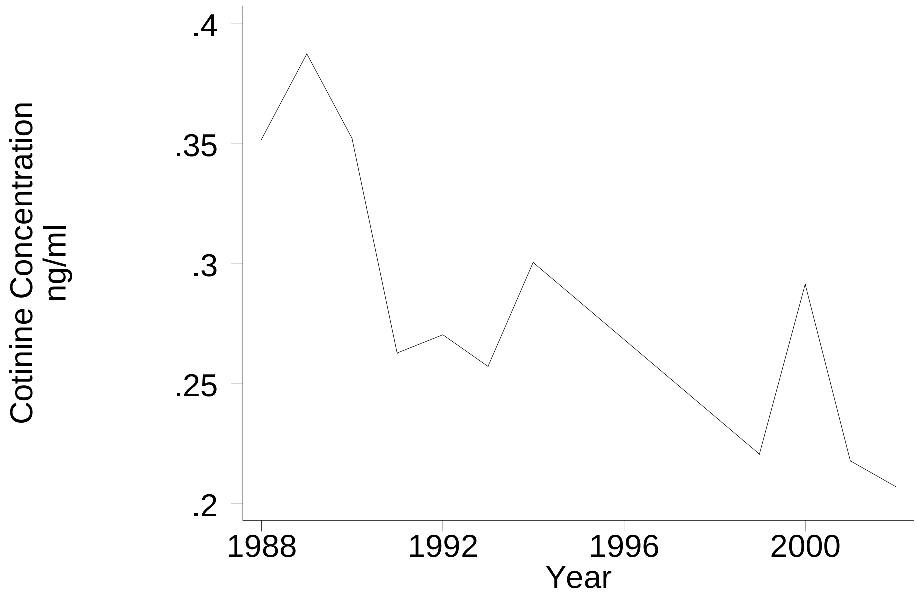

Figure 7: Average Cotinine Concentration in Non-Smokers – Smoking Households

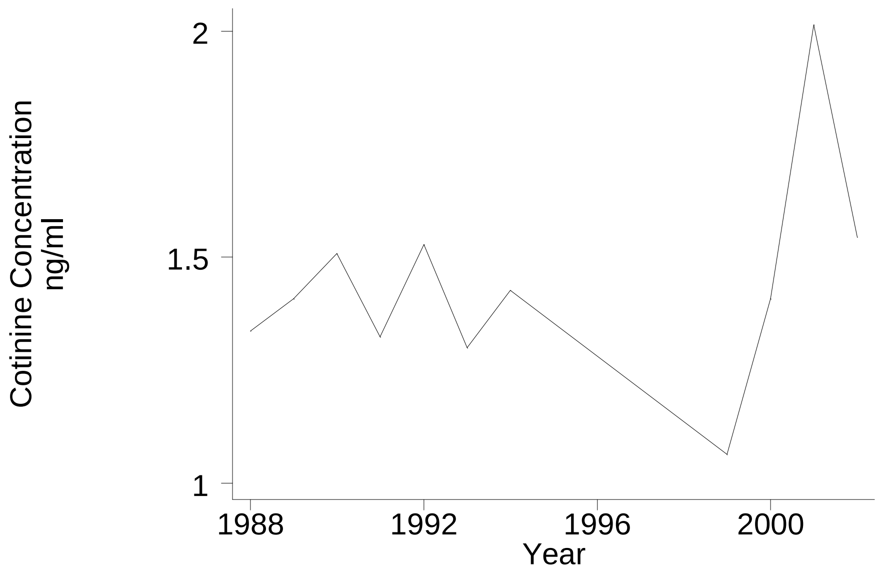

Figure 8: Cotinine Concentration and Excise Tax

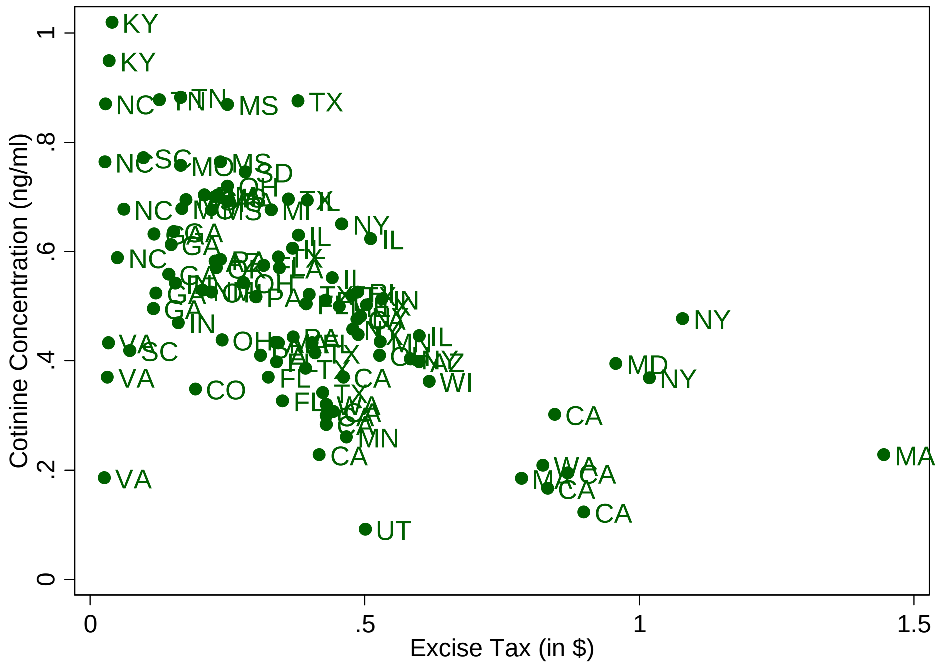

Figure 9: Cotinine Concentration and Smoking Bans

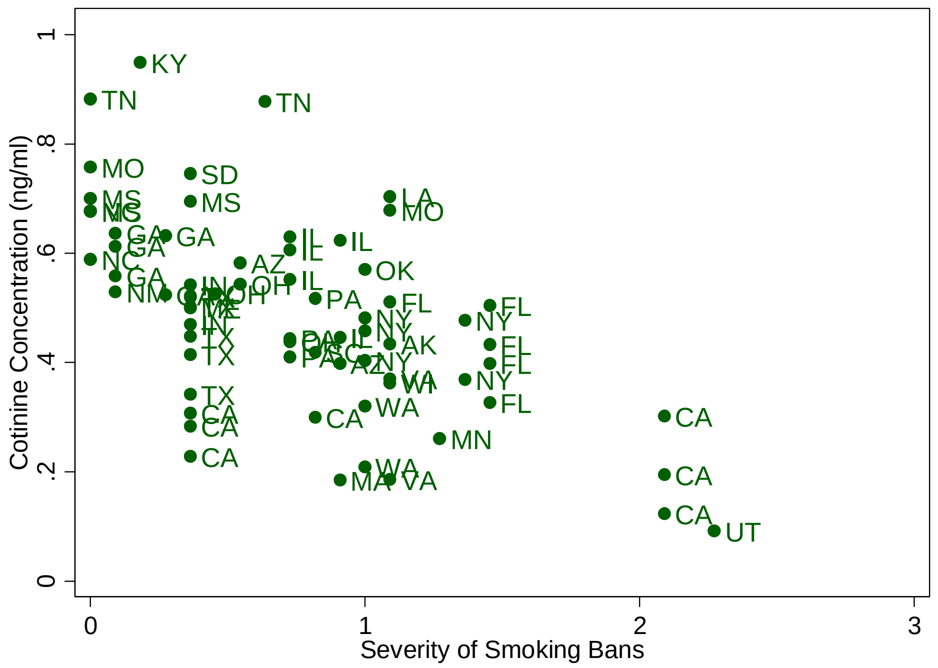

Figure 10: Cotinine Concentration, Excise Tax and Smoking Bans in Selected States.