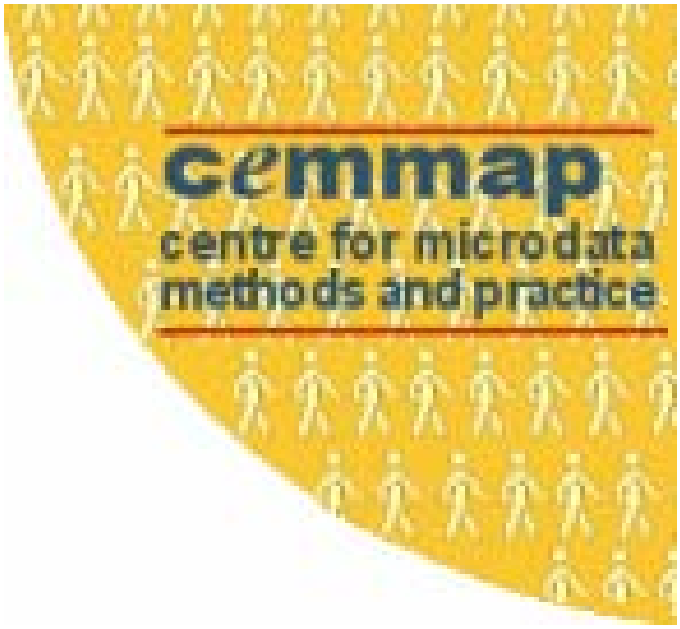

THE EFFECTS OF TAXES AND BANS ON PASSIVE SMOKING

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The Effect of Taxes and Bans on Passive Smoking

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Abstract

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 reach two important conclusions. First, excise taxes have a significant effect on passive smoking. Second, smoking bans have on average *no* effects on non smokers. While bans in public transportation or in schools decrease the exposure of non smokers, bans in recreational public places can in fact perversely increase their exposure by displacing smokers to private places where they contaminate non smokers, and in particular young children. Bans affect socioeconomic groups differently: we find that smoking bans increase the exposure of poorer individuals, while it decreases the exposure of richer individuals, leading to widening health disparities.

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1 Introduction

In the US, 15% of the population smokes regularly. Yet, detectable levels of nicotine in body fluids can be found 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] The government 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 (2005) show that although taxes affect the number of cigarette smoked, smokers compensate by smoking more intensively a given cigarette. 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.

[1] See descriptive evidence in section 3.1
[2] See for instance Law et al (1997), Hackshaw (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).

While the literature on regulations, either through taxes or bans, on smokers is quite large, there is hardly any evidence of 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 state interventions. Cotinine is therefore a straightforward and precise measure of passive smoking especially when evaluating public policies.

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

[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).

passive smoking in the US. We evaluate the effect of tobacco tax increases 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. A 10% increase in the state excise tax reduces the cotinine concentration in non smokers by about 3%. The effect is particularly sizable for children who are exposed to their parents' smoke.

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 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. The reason is that such bans displace the smoking to places where non-smokers are more exposed, especially young children. 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.

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 Conceptual Framework and 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 Effects of Tax Changes and Smoking Bans

Public interventions can have both a direct and an indirect effect on non-smokers. 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 analyse fully 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.

Smokers

The literature has shown the negative effect of taxes (and prices) on the demand for cigarettes. Moreover it has been pointed out that taxes operate both on the intensive and the extensive margin. [9] 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, and not those consumed in company. In this case, non smokers may not benefit at all from a rise in excise taxes.

Regarding smoking regulations, there is evidence that bans in the workplace decrease the prevalence of smoking (Evans et al, 1999). However, bans may also lead to a *displacement* of smoking: smokers may shift their consumption within the day, or decide to avoid spending time in places where bans are effective. For instance, a ban in bars and restaurants may induce smokers to spend more time at home, and therefore contaminate more other members of the household, especially children.

Non Smokers

Contamination from ETS is often termed passive smoking. Passive may, however, be in this context a misleading terminology. Non-smokers may in fact choose whom to socialize with and where, and the introduction of a smoking ban may change their choice of workplace, transportation mode or social venues. In particular, adults who live with a smoking partner represent a selected group of individuals who may care less about exposure to smoke. They may therefore spend relatively more time in public places where smoking is permitted. On the one hand, they may benefit more from tighter regulations, but on the other hand, they may be more likely to switch to other places where smoking is permitted, accompanying their smoking partner. The net effect depends on the relative importance of these two factors.

For some non-smokers, avoiding smokers may not be possible. Young children may have little choice but to stay with their parents or carer.

[9] See section 1 for references.

The effect of state interventions depends on the interaction between smokers and non-smokers. 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. Next section describes the measure of smoking we use.

2.2 Cotinine as a Proxy for Smoking Intake

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.[10] 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. 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 (Figure 1). 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, in fact, highly correlated with the level of tar and carbon monoxide, which causes cancer and asphyxiation.[11,12] 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

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

[11] 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.

[12] 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.

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.

2.3 Methodology

In this section we outline the empirical methodology used for the analysis. 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*:

$$\mathrm{Cot}_{ist} = \alpha_0 + \alpha_1 X_{it} + \alpha_2 \log tax_{st} + \alpha_3 R_{st} + \delta_s + \lambda_t + u_{ist} \qquad (1)$$

where Cot_{ist} is the cotinine concentration (expressed in ng/ml), X_{it} is a vector of individual characteristics that affect exposure such as age, sex, occupation or race; 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; δ_s is a set of state of residence dummies, while λ_t is a set of year dummies. 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 account for by state dummies. Our identification relies on the exogeneity of the timing of changes in taxes and regulation within states. The timing depends in part on the electoral cycle which can be thought of exogenous to exposure to passive smoking.
The coefficients of interest are the effect of taxes and the effect of restrictions on cotinine measures. We relate exposure to excise taxes as this is the relevant policy variable from a public health point of view.

As discussed above, 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 and year levels. This correction accounts for the presence of a common random

effect at the year-state level. We also check the robustness of our results to serial correlation in the error term.

Taxes and regulation may affect non-smokers differently, according to their age and occupation. For instance, children are probably unaffected by smoking bans in the workplace because they do not spend any time there. The same may be true for individuals out of the labor force. We therefore investigate the specific effect of state intervention on different socio-economic groups. In addition, we also split the analysis according to the smoking status of other members of the household. [13]

After having considered a global measure of restrictions on smoking (R_{st}), we separate cigarette smoking regulations according to the place where these regulations are enforced. Among the different places where smoking bans may be enforced we consider: public transport, shopping malls, workplaces, schools, and recreational places. We consider the following econometric model for a non smoker indexed by *i*, in state *s* and in period *t*:

$$\text{Cot}_{ist} = \alpha_0 + \alpha_1 X_{it} + \alpha_2 \log tax_{st} + \alpha_3 GO_{st} + \alpha_4 PT_{st} + \alpha_5 SM_{st} + \alpha_6 WP_{st} + \alpha_7 S_{st} + \delta_s + \lambda_t + u_{ist} \qquad (2)$$

where X_{it} is a vector of individual characteristics that affect exposure such as age, sex, occupation or race; tax_{st} is the tax of tobacco (adjusted for inflation) in a given state and period; GO_{st} is a measure of restrictions on smoking in recreational places ("going out"); PT_{st} restrictions in public transport; SM_{st} restrictions in shopping malls; WP_{st} restrictions in the work place; S_{st} restriction at school; 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 variation across states and time, and the coefficients of interest are the effect of taxes and the effect of restrictions in the different public places on cotinine measures (Cot_{ist}).

[13] To the extent that the smoking status of other members of the household depends on taxes and regulations, we expect the OLS estimates of the effect of these variables to be biased. 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. This will be further discussed in section 4.2.

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 51835 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, from 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.[14] It represents about 5% of the declared non smokers. In total, we observe 29667 non-smokers with a valid measure of cotinine concentration (Table 1).[15]

[Table 1]

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

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

more likely than non smokers living in non smoking households to have a concentration of cotinine above 1ng/ml.

3.2 Health Effects of Passive Smoking

We briefly present some evidence of the effect of passive smoking on health for children and adults. The purpose of this analysis is not to reproduce results established in the medical literature, but to provide some light on the risks of passive smoking, evaluated within our sample, with measures of exposure that are similar to the one we use to evaluate the effect of state interventions.

We exploit the information on health outcomes contained in the NHANES III. We consider four categories of passive exposure based on cotinine concentrations: 0 to 0.1 ng/ml, 0.1 to 0.2 ng/ml, 0.2 to 1.0 ng/ml and 1 to 10 ng/ml. We select different health outcomes for children and adults. For adults we consider strokes and chronic bronchitis. As the incidence of strokes in children is very low, we consider symptoms of respiratory diseases such as asthma and chest wheezing. We run logistic regressions and control also for age, sex and race. The results are presented in Table 3 in the form of odds-ratios. The omitted category is the lowest exposure group.

[Table 3]

The first two columns displays the results for children. Exposures resulting in cotinine concentrations lower than 1 ng/ml do not lead to higher risks of developing asthma or chest wheezing. For the highest exposure group, the odds of having asthma or chest wheezing are respectively 1.62 and 1.41, both statistically significantly different from one.
For adults, we find a similar pattern, low exposures up to 0.2 ng/ml do not lead to higher risks of developing strokes or chronic bronchitis. For the highest exposure group, the odds are 1.70 for strokes and 1.56 for chronic bronchitis.

The effect of passive smoking appears to be non-linear, with no or limited effect for light exposure, and a significant effect when the exposure results in a cotinine concentration higher than 1ng/ml. We shall return to these results below when we discuss the effect of state interventions.

3.3 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 are however differential variations over time 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 law 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.[16] 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, 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.[17] 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

[16] http://www.impacteen.org
[17] See appendix A for a detailed description of the regulations variables.

(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 to a complete restriction on smoking.

3.4 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). These are non smokers more at risk of developing smoking related diseases. Over this period, the proportion of individuals with a cotinine level in excess of 1 ng/ml has decreased from 21% to 11%.[18]

[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 (Figure 6). 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. [19]

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

[19] An alternative interpretation is that of a change in composition in the pool of smokers due to the fact that light smokers are more prone to quit (see section 2.3). We reweighted the sample by matching on a number of observable characteristics (sex, race, age group and income group). We found no substantially different results compared to the analysis presented above.

4 Empirical Results: Passive Smoking and State Intervention

In this section, we estimate the empirical model outlined in section 2.3. We start with the whole sample and then break down the results by age groups, family smoking status and by type of smoking ban.

4.1 Whole Sample of non smokers

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 4.[20] We consider first the effect of taxes on cotinine concentration in non smokers (columns 1 and 2), then of bans (columns 3 and 4), and finally of both taxes and bans together (column 5).

[Table 4]

Column (1) of Table 4 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 through variations through time and state differences. Raising taxes by 100% reduces cotinine levels by about 0.1ng/ml. Note that the average concentration of cotinine is equal to 0.44ng/ml. The doubling of the excise tax would therefore lead to a reduction in exposure to smoke of about 25%. From Table 2, a one standard deviation change in state taxes would lead to a reduction in exposure of 0.27*0.097=0.026ng/ml.

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, with a reduction of the cotinine levels of about 0.14ng/ml, corresponding to about a 30% reduction in exposure to smoke. Column (3) displays the effect of regulations on smoking. As described in section 2.3 we have scaled the regulation variable from 0 (no regulations) to 3 (smoking prohibited in all public places). The result reported in column (3) implies that going from no regulations to a total ban would reduce cotinine by 0.48ng/ml. This means that, given that the average concentration of cotinine is equal to 0.44ng/ml, a total ban would eliminate exposure completely. However, if both taxes and regulation are introduced in the model (column 5 of Table 4) results change. The effects of excise taxes are comparable to the one estimated in

[20] Given that ignoring serial correlation in a differences-in-difference framework can lead to wrong standard errors (Bertrand et al., 2004), we check the robustness of our results by clustering the standard errors at state level only, letting the covariance structure across years to be free. The standard errors do not change much as a result. Our main analysis is therefore robust to serial correlation. Results available on request.

column (2). Regulations, on the other hand, appear to have no overall effect. The 95% confidence interval for the effect of bans ranges from -0.05 to 0.07. Even if the effect is at the lowest part of that interval, the effect of regulations would be small. At most, a two standard deviation change in regulations eliminates about 0.02 ng/ml of exposure.

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 4, column (3)).

As shown in Figure 4, exposure to ETS has dramatically decreased over the period of analysis. However, the results in Table 4 seem to indicate that very little is due to changes in regulations. The effect of taxes, although statistically significant, is not large enough to explain much of the decline in cotinine levels. These results are however not incompatible with the descriptive evidence. The identification of these effects relies in fact on year to year changes and it may be that the effect of state interventions are more pronounced in the long run. Moreover, the average decrease in cotinine could also be due to other factors such as a rise in education levels or in health awareness.

4.2 Policy Impact for Light and Heavy Exposure to Passive Smoking

In the previous paragraph we show the effect of taxes and smoking regulation on the average non-smoker (Table 4). We now present results for non-smokers in other parts of the exposure distribution. Table 5 displays the effect for individuals at 25%, 50%, 75 and 90% percentile of the distribution of cotinine concentration.

[Table 5]

The effect of taxes is important for individuals with light exposure at the 25 percentile, but is not different from zero in a statistical sense for any other points in the distribution. Smoking regulations have no effect except for light exposure, where a total ban in all public places would decrease cotinine levels by 0.21ng/ml. This appears to be a large number given that the average level of cotinine for this group is only 0.035ng/ml. Given the variation in the data, it means that a one standard deviation increase in regulation leads to a reduction in cotinine levels of 0.015ng/ml.

The evidence suggests that regulations and changes in excise taxes failed to have an effect in those who are the most exposed and would gain most from a reduction in ETS, as shown in Section 3.2.

4.3 Policy Impact by Age Group and Family Smoking Status

It may well be that taxes and regulation affect non-smokers differently, according to their age and whether they live with smokers. We therefore investigate the specific effect of state interventions on different age groups and family smoking status (Table 6). [21]

In Table 6 we 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. We therefore pool the two groups together to gain in precision. Finally, given that we do not have enough infants in the sample to run a separate analysis on them, we group children up to eight years of age.

[Table 6]

The first row of the first column of Table 6 displays the results relative to the overall sample already reported in Table 4 for an easier comparison of the results. Column 2 considers the sub-

[21] While Table 6 reports the results from an OLS regression, we do not find significant differences in the coefficients when we re-weight the sample to take into account a possible change in the composition of the sample over time. Our results are also robust to serial correlation (see note 20).

sample of individuals belonging to non-smoking families, and finally column 3 displays the results for non-smokers sharing a household with one or more smokers.

We start by analysing individuals of all ages. Non smokers living in a non-smoking family (column 2) benefit from an increase in excise taxes. A doubling of the excise tax leads to a reduction of about 0.10ng/ml, which represent a third of total exposure for this group. Regulations have, on the other hand, on average no effect on exposure. The exposure to passive smoking of non smokers living with smokers is not significantly affected by tax changes. There is, on the other hand, some evidence that bans on smoking decreases exposure in this group. A total ban would decrease cotinine levels by 0.90ng/ml, a 40% reduction.

We now look at the effect by age groups (Rows 2 to 5, Column 1 of Table 6) We first analyse the effect of taxes on the level of cotinine in non smokers. Children aged eight or younger (row 2, Column 1) benefit from increases in excise taxes. Doubling the excise tax would reduce cotinine concentrations by as much as 0.56ng/ml. Similar results are found for next two age groups, eight to twelve years old (row 3, Column 1 of Table 6), and thirteen to twenty (row 4, Column 1 of Table 6). It is interesting to note that the effect of excise taxes gradually decreases with age: the effect is -0.56 for the first age group (age less than 8), -0.43 for the second (age 8-12), and -0.39 for the third (age 13-20). As we argue in the conceptual framework section, small children are the age group that may have little choice but to stay with their parents or carer, and therefore benefit the most from a reduction of the number of cigarettes smoked. The last group is the adult non smoking population aged 21 and over. We find that on average taxes have no effects.

We now decompose the results by family smoking status (Column 2 and 3, Table 6). As we conjectured above, the observed effect of taxes is mainly due to children living with smokers (row 2, column 3 of the table) and to teen-agers in non smoking families. Doubling the excise tax reduces cotinine levels of children aged less than 8 in smoking households by almost 1 ng/ml. This suggests that excise taxes are effective at reducing smoking within the household.

For teen-agers (age 13-20), the effect of taxes is mainly due to individuals living with non smokers (row 3, column 2 of the table), although we also find a negative (but not significant) coefficient for teen-agers living in smoking families. At this age these children are likely to spend time with smoking friends, who may be more sensitive to changes in excise taxes than adults. Indeed, for the age group 21 and over, we find no effect of taxes for individuals in non smoking

households, and even a positive (though marginally significant) effect for those in smoking households. In this group, excise taxes weakly increase cotinine levels: a one percent increase in excise taxes leads to 0.45 percent increase in cotinine levels. This result is in line with the hypothesis that cigarettes smoked in social moments are likely to be more difficult to be cut. Higher prices induce smokers to cut down on cigarettes at home and not on the cigarettes smoked with friends. This leads on one side to the observed reduction of the exposure of children in smoking families, but does not induce a decrease of the exposure of non smoking adults engaged in social activities with smokers. A tentative explanation is that non smoking adults may spend more time with their smoking partners, when the number of cigarette smoked decreases. The resulting exposure depends then on the relative strength of these two factors.

We now analyse the effect of regulation by age and then by family smoking status. Regulations have no significant effect on tobacco exposure in children, except for the first age group (four to eight years old). Bans on smoking in public spaces appear to increase their cotinine levels by 0.6ng/ml, but the effect is only significant at the 10% level.
Decomposing the effect of increasing regulations by family smoking status, the only noticeable effect is for adults in smoking households (Row 5, Column 3, Table 6): going from no regulation to a total ban reduces cotinine levels by about 2.1 ng/ml. Regulations may have no effects on non smokers from non smoking households, because they may less go to public places where they would be exposed to smoke and may therefore be less affected by stricter regulations. Members of smoking households, on the other hand, may have less of an a priori against exposure. Tighter regulations may therefore benefit them more.

In summary, excise taxes have an important effect on reducing the exposure of children living in families where at least one parent smokes. This suggests that smokers are more likely to cut down on cigarettes smoked within the household rather than those smoked in social occasions. Bans on smoking on the other hand seem to be effective only for adults who live with smokers. This group of non-smokers are likely to be exposed to ETS both in private and public places. Tighter regulations in public places will contribute to an overall reduction of nicotine exposure in adults. On the other hand, stricter regulations seem to lead to an increase in the cotinine levels in young children.

4.4 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 7

displays the effect of taxes and regulation on passive smoking considering separately different places where regulation may be enforced. In particular, we have distinguished 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[22].

[Table 7]

The first row of Table 7 reports the coefficient of (log) excise taxes. The other rows of the table report the regression coefficients of regulation in different places. [23] Column (1) of Table 7 refers to the overall sample of non smokers. The other columns of the table refer to different age groups. A number of interesting results emerge from this analysis. Most remarkable is the difference in sign between the coefficient of what we have defined as "going out", meaning the leisure activities carried out outside home, and the regulations coefficients relative to the other public places where regulations can be enforced (column 1, Table 7).

The regression coefficient of regulation in places where individuals spend their free time engaging in social and cultural activities ("Going out") is positive. Regulations in these places significantly increase exposure of non smokers: going from no regulation to a total ban increases on average cotinine levels by about 1.5 ng/ml, whereas a one standard deviation change in state regulation increases cotinine levels by 0.13ng/ml. This can be interpreted as the existence of a *substitution* effect between leisure activities in public places, where regulation can be enforced, and in private places, where no restriction to smoking can be enforced.

The findings about regulation in public transportation, shopping malls, and schools are different. The regression coefficients relative to regulation in these public places are on average negative indicating a negative relationship between a tighter regulation and exposure to environmental tobacco smoke. Going from the absence of regulation to a total ban reduces on average cotinine levels by about 0.4 ng/ml in public transport and in schools, and by about 0.6 ng/ml in shopping malls. In workplaces, the effect of tighter smoking regulations on tobacco exposure of non smokers is not significantly different from zero. It seems therefore that there is no evidence of an effect of bans on non smokers' exposure. However, the precision of the estimates does not exclude the fact that a ban in such places could decrease exposure. This is not necessarily in

[22] See Appendix A for a more detailed description of the regulation data.
[23] Our analysis is robust to serial correlation (see note 20).

contradiction with the association found in the epidemiology literature between bans and a reduction in the number of cigarettes smoked by continuing smokers, the increases in quitting, and the reduction in smoking prevalence. [24] This could in fact be due to our study design, as we control for state and time dummies and rely on differential changes in regulation between states and time period for identification. We therefore eliminate potential confounding by state and year effects. It should also be noted that the lower point of the confidence interval implies a reduction of about 0.16ng/ml for a total ban.

Columns (2) to (5) of Table 7 distinguish between four different age groups. As noted above, children of the first age group considered (4 to 8 years old) are mostly either at home or in school or day-care, and supervised by an adult. Exposure at this age comes therefore either from adults living in the same household, or from public places. The regression coefficient relative to regulation in recreational public places is positive and large: going from no regulation to a total ban increases on average cotinine levels by about 6 ng/ml, whereas an increase of one standard deviation in state regulations would increase cotinine levels by 0.5ng/ml. The substitution between public and private recreational places seems therefore to heavily affect children of this age group. On the other hand, these children are also those who benefit more from tighter regulations in public places other than recreational ones like public transport and shopping malls. Adult individuals that can no longer smoke in public transport or while shopping, will expose the children less to ETS. Going from no regulation to a total ban reduces on average cotinine levels by about 1.5 ng/ml in public transports, and by about 2.5ng/ml in shopping centres. This last result seems to indicate that small children that need constant adult supervision are those that can benefit the most from tighter regulations in such public places. The effect for the second and third age groups (8-12 and 13-20) is of similar sign but of smaller magnitude. For these individuals, the effect of regulations in public recreational places is about half of that observed for younger children (going from no regulation to a total ban increases on average cotinine levels by about 3.6 ng/ml compared to an increase of 6.2 ng/ml for younger children). The substitution between public and private recreational places is therefore observed also for these two age groups but it is smaller than that observed for very young children. Older children may be more independent from their parents and may go out on their own. Finally, the regression coefficient relative to public recreational activities is positive but not significant for individuals aged 21 or above (group 4). A plausible explanation of this is that adult non smokers spend their recreation time with other adults both in public and private places, and are therefore exposed to ETS either in one

[24] See for instance Hopkins et al (2001), and Levy and Friend (2003).

or the other place. What is relevant is the overall amount of ETS. It makes no difference for this age group whether smokers choose to smoke in public or private places.

Tighter regulations in public places other than recreational ones (i.e. transportation, shopping malls, and schools) seem on average to lead to a reduction in tobacco smoke exposure of non smokers. This is particularly the case for small children who are most likely spend time in these public places accompanying older individuals. This interpretation is supported by the finding about adults. We observe, in fact, that whenever a private alternative to the public place exists, we do not observe a reduction in nicotine exposure as a consequence of a stricter regulation. A first example is the regression coefficient of regulation in public transport (column 5, Table 7) relative to individuals older than 21. It is positive though not significant. This may be due to the fact that this group of individuals may in fact use the car as a private alternative to the public transportation. We therefore observe for this age group the public/private substitution effect. A second example is the coefficient of the regulation in shopping malls. Though generally negative, it is positive, though not significant, for the age group 8-12, which most likely is the one that spend less time engaging in shopping activities in commercial centres.
Finally, the effect of tighter regulation in school, though on average significant, is not significantly different from zero when we consider separately the different age groups.

4.5 Distributional Effects of Smoking Regulations and Taxes

Finally, we investigate whether state interventions affect differently individuals 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 8.

[Table 8]

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 0.3 ng/ml. 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 (weakly) 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 consequence of strengthening smoking regulations would be a widening in health disparities across socio-economic groups.

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 little 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 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 state interventions through taxes and bans only affect non smokers who have light exposure to tobacco smoke. Given the non-linear effect of exposure on

health, it implies that state interventions, including taxes, have had a moderate impact on health outcomes. Finally, we find 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: Smoke free regulations data

The information contained in this appendix, are drawn from the codebook for the "Tobacco Control Policy and PrevalenceData: 1991-2001"[25], 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.[26]
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 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).

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*

[25] http://www.impacteen.org/tobaccodata.htm
[26] Coding for public schools, private schools and cultural facilities were developed by MayaTech from the state smoke-free air law coding provided by RPCI.

	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.

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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: Health and Exposure to Tobacco Smoke: Odds-Ratio of Poor Health

		Children		Adults	
		Asthma	Wheezing In Chest	Stroke	Chronic Bronchitis
Cotinine Level (ng/ml)	[0-0.1]	1	1	1	1
	[0.1-0.2]	1.06	1.17	1.12	1.02
		(0.17)	(0.14)	(0.22)	(0.13)
	[0.2-1.0]	1.07	1.18*	1.00	1.45**
		(0.14)	(0.12)	(0.19)	(0.16)
	[1.0-10.0]	1.62**	1.41**	1.70**	1.56**
		(0.22)	(0.15)	(0.41)	(0.23)

Robust standard errors in parenthesis. The regression also controls for age, age square, sex and race. ** significant at 5%, * significant at 10%

Table 4 - Effects of Taxes and Regulations on Passive Smoking.

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

	(1)	(2)	(3)	(4)	(5)
Log Tax	-0.097**	-0.14**			-0.14**
	(0.034)	(0.04)			*(0.06)*
Regulations			-0.16**	-0.032	0.01
			(0.028)	(0.032)	(0.03)
Controls:					
Year Dummies		X		X	X
State Dummies		X		X	X
Age, sex, race	X	X	X	X	X

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

Table 5: Effect of Taxes and Smoking Regulations for Different Percentiles in the Cotinine Distribution.

Percentile	Mean	25%	50%	75%	90%
Cotinine level	0.44ng/ml	0.035ng/ml	0.10ng/ml	0.35ng/ml	1 ng/ml
Log Tax	-0.14**	-0.17**	-0.007	-0.02	0.01
	(0.06)	*(0.06)*	*(0.03)*	*(0.03)*	*(0.01)*
Regulations	0.01	-0.07**	0.003	0.02	-0.001
	(0.03)	*(0.03)*	*(0.015)*	*(0.01)*	*(0.008)*
Controls:					
Year Dummies	X	X	X	X	X
State Dummies	X	X	X	X	X
Age, sex, race	X	X	X	X	X

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

Table 6 - Effect of Taxes and Regulation on Passive Smoking. Dependent variable: cotinine

	(1) All Individuals		(2) Individuals in Non smoking families		(3) Individuals in smoking families	
Average Cotinine Level	0.44ng/ml		0.26ng/ml		1.47ng/ml	
	Log Tax	Regulations	Log Tax	Regulations	Log Tax	Regulations
All ages	-0.14**	0.01	-0.098**	-0.004	-0.05	-0.29*
	(0.06)	(0.03)	(0.04)	(0.03)	(0.28)	(0.17)
Age <8	-0.56**	0.21*	0.056	0.057	-0.97**	-0.37
	(0.19)	(0.11)	(0.07)	(0.04)	(0.49)	(0.36)
Age 8-12	-0.43**	0.067	0.05	-0.03	-0.94*	-0.10
	(0.20)	(0.12)	(0.06)	(0.03)	(0.55)	(0.31)
Age 13-20	-0.39**	0.09	-0.41**	0.08*	-0.08	0.14
	(0.12)	(0.06)	(0.09)	(0.05)	(0.34)	(0.18)
Age 20 +	0.02	-0.047	-0.067	-0.02	0.67*	-0.69**
	(0.06)	(0.04)	(0.05)	(0.03)	(0.40)	(0.33)

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

Table 7 - Effect 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+
Log Tax	-0.12**	-0.58**	-0.38*	-0.36**	0.01
	(0.06)	(0.20)	(0.21)	(0.12)	(0.07)
Regulation Going out	0.52**	2.08**	1.15**	1.22**	0.30
	(0.17)	(0.51)	(0.59)	(0.33)	(0.23)
Regulation Public Transport	-0.13**	-0.50**	-0.67**	-0.34**	0.008
	(0.05)	(0.18)	(0.16)	(0.10)	(0.06)
Regulation Shopping Mall	-0.21**	-0.85**	0.15	-0.42**	-0.15**
	(0.06)	(0.22)	(0.25)	(0.16)	(0.07)
Regulation Workplace	-0.005	-	-	-	-0.13
	(0.05)				(0.10)
Regulation Schools	-0.13**	0.14	-0.15	-0.10	-
	(0.05)	(0.22)	(0.19)	(0.12)	

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

Table 8: Distributional Effects of Taxes and Bans

	Household Income: [0,33%]	Household Income: [33%,66%]	Household Income: [66%, 100%]
Log Tax	-0.12	-0.24**	-0.07
	(0.09)	(0.10)	(0.05)
Regulations	0.10**	0.07	-0.07*
	(0.05)	(0.05)	(0.04)
Controls:			
Year Dummies	X	X	X
State Dummies	X	X	X
Age, sex, race	X	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

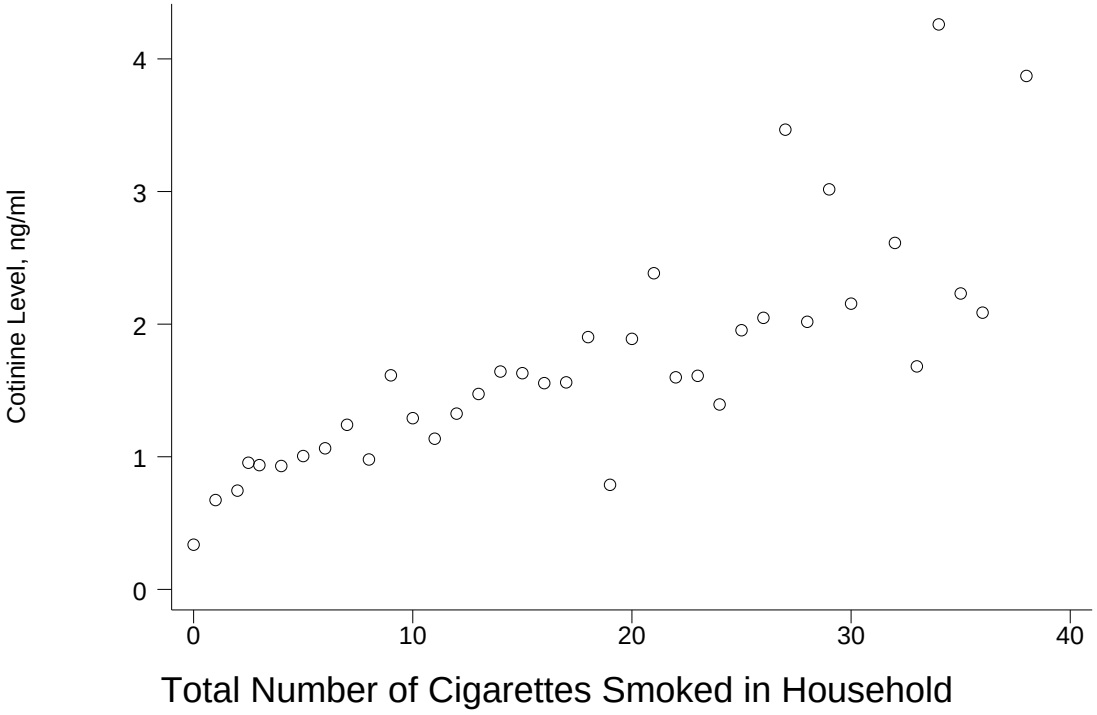


Figure 2: State Excise Taxes, by US State 1988-2002.

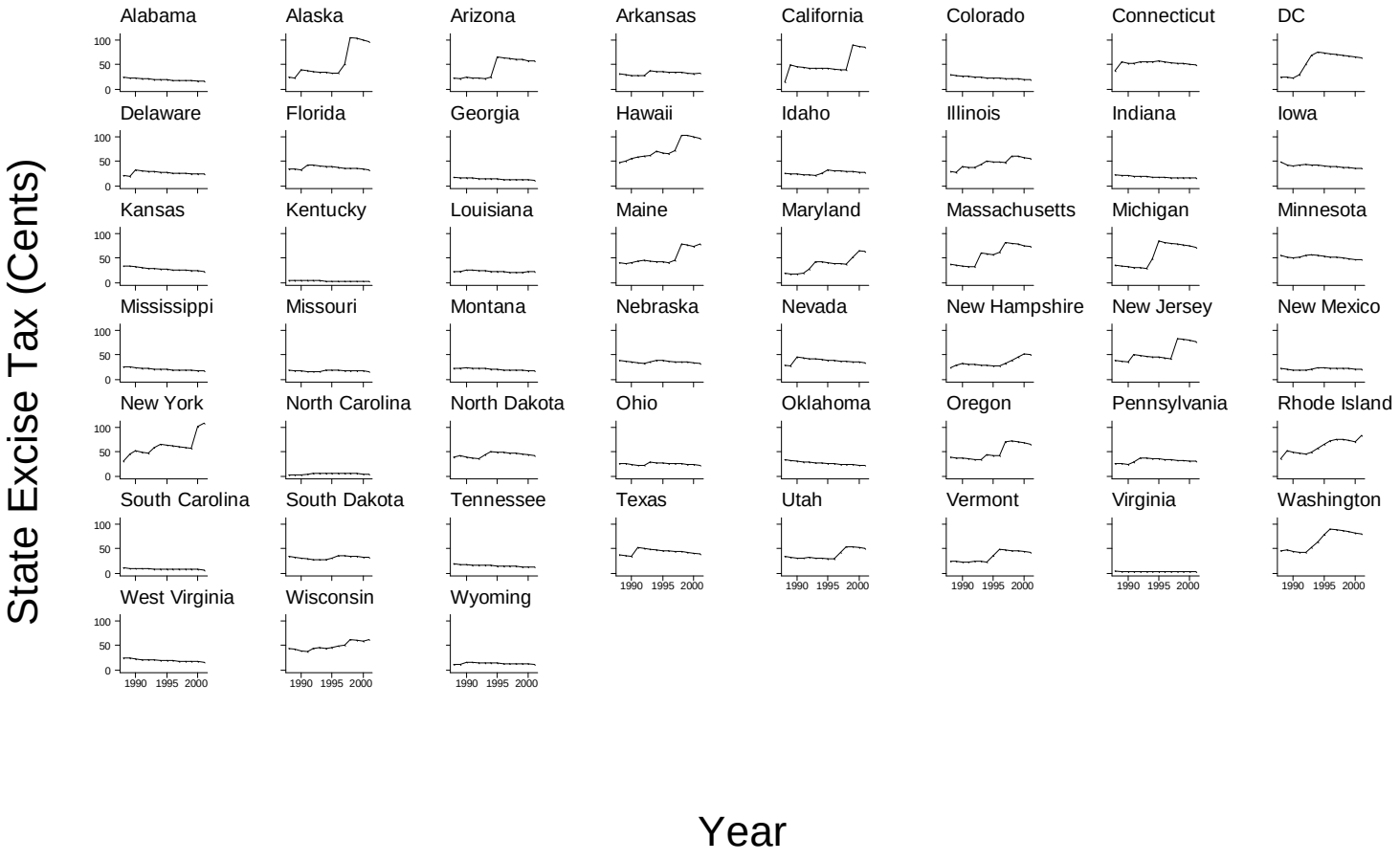


Figure 3: Level of Restrictions on Smoking, by US State, 1991-2001.

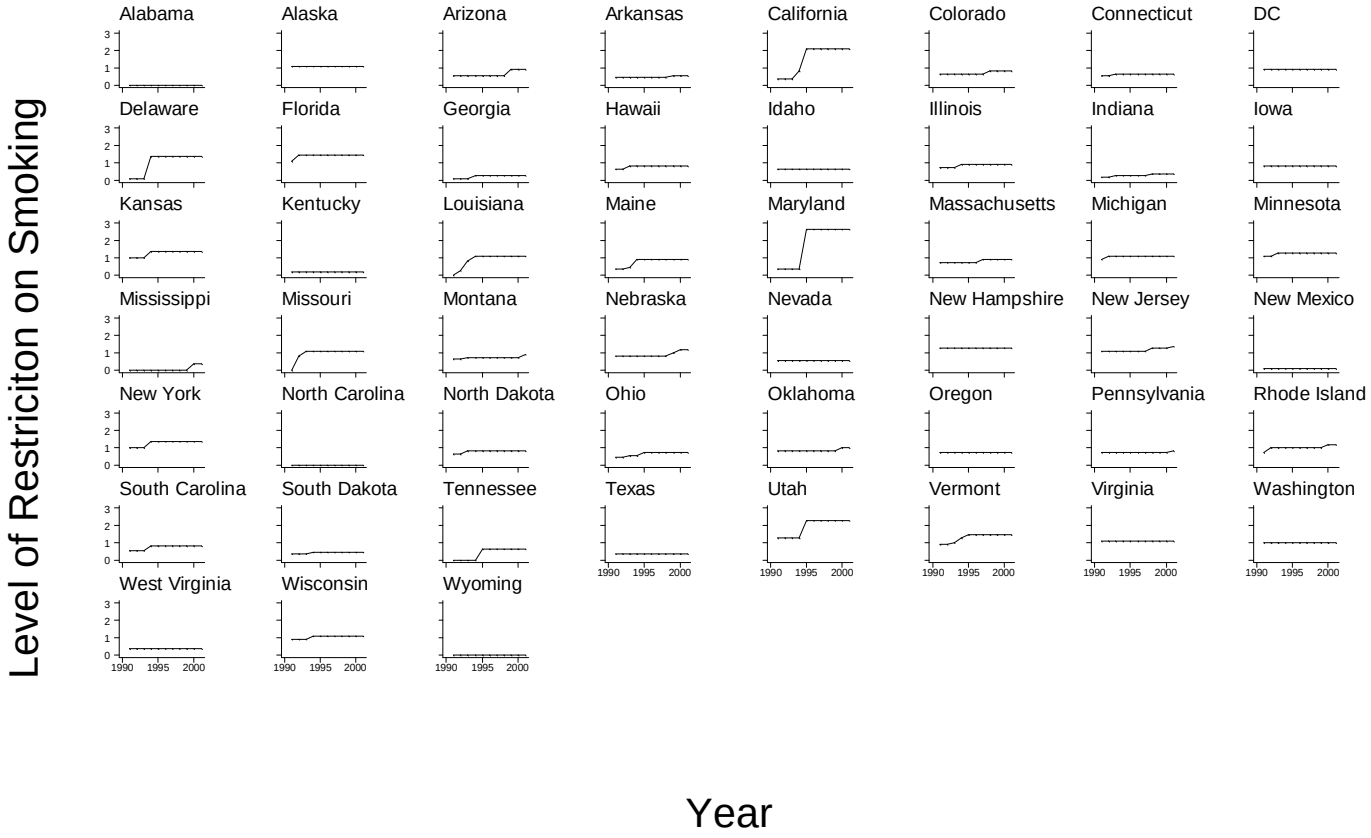


Figure 4: Average Cotinine Concentration in Non-Smokers.

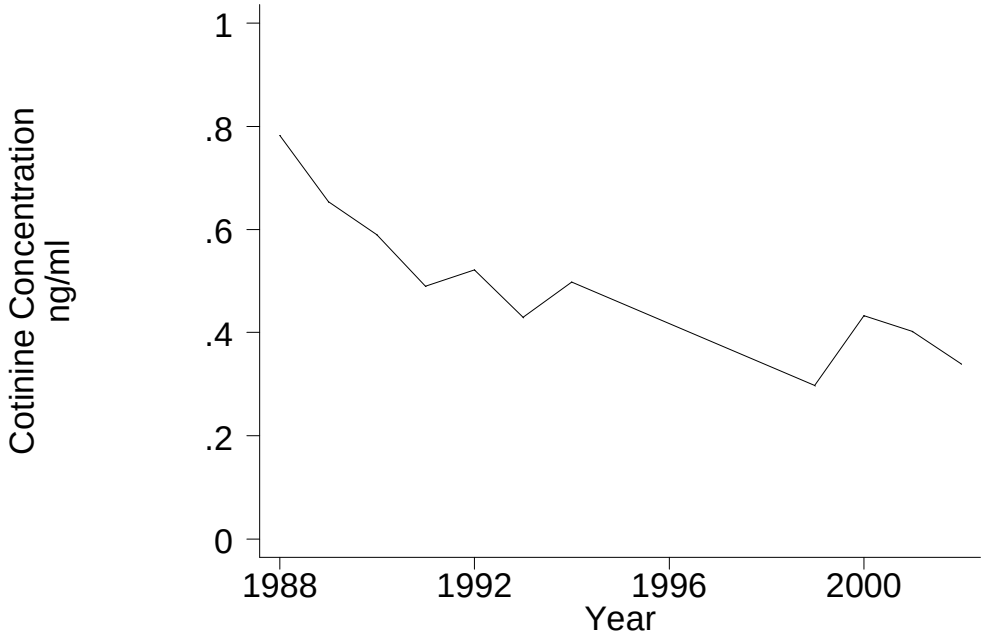


Figure 5: Fraction Individuals with Cotinine larger than 1ng/ml

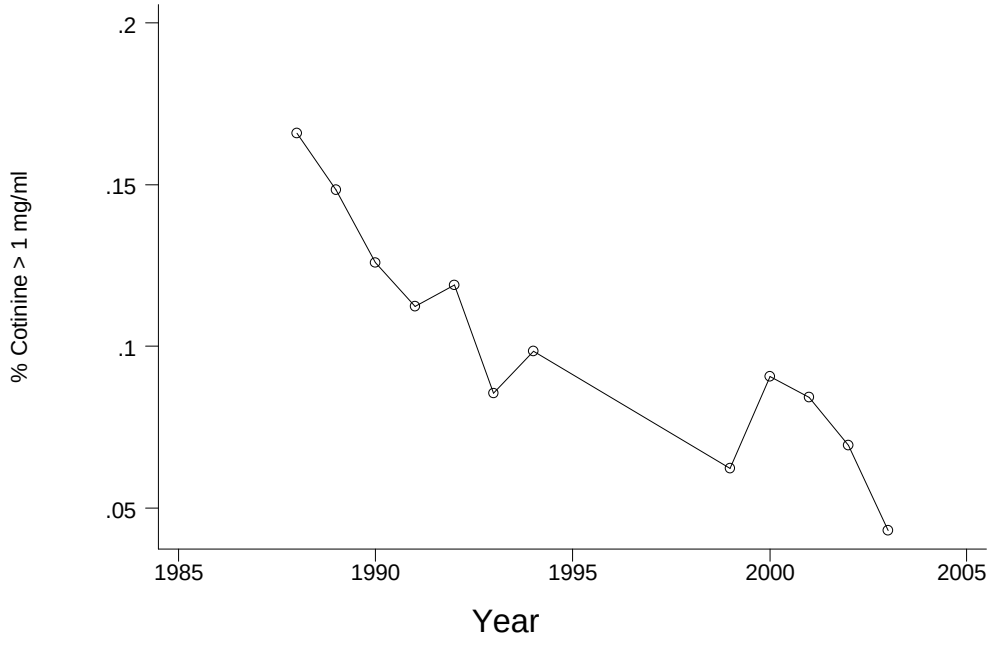
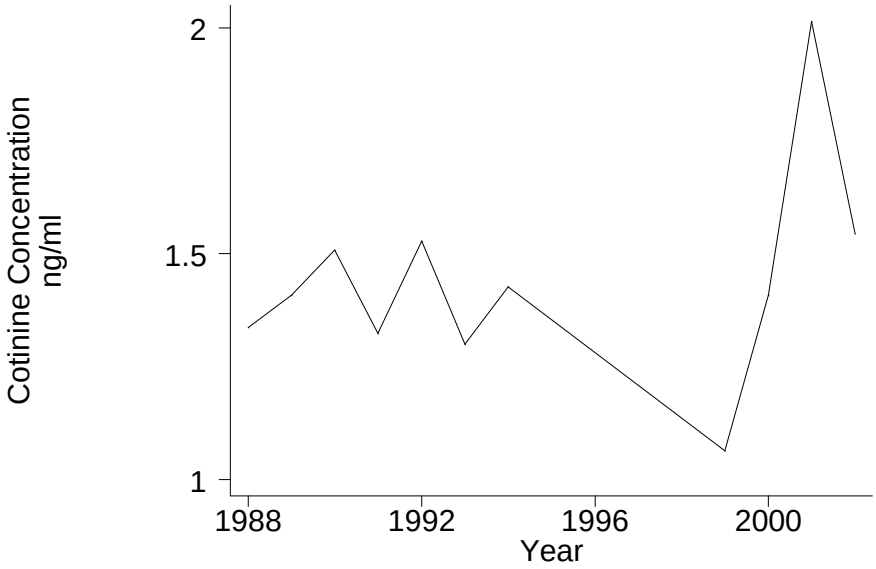


Figure 6: Average Cotinine Concentration in Non-Smokers – Non Smoking Households

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



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