The Effect of Bans and Taxes on Passive Smoking

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Abstract

This paper evaluates the effect of smoking bans in public places on the exposure to tobacco smoke of non-smokers and contrasts it with the effect of excise taxes. Exploiting data on cotinine- a metabolite of nicotine- as well as state and time variation in anti-smoking policies across US states, we show that smoking bans in public places can perversely increase the exposure of non-smokers to tobacco smoke by displacing smokers to private places where they contaminate non smokers, and in particular young children. In contrast, we find that higher taxes are an efficient way to decrease exposure to tobacco smoke, especially in those most exposed.

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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. 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.

In this paper, we evaluate the effect of these policies on non-smokers by measuring directly cotinine levels (a metabolite of nicotine) in body fluids. We contrast the effect of smoking bans in different public places with the effect of higher excise taxes. We develop a simple model of passive smoking and show that smoking bans can have two distinct effects on exposure to tobacco smoke: they decrease exposure in public places but can lead to a perverse increase in exposure by displacing smoking towards private areas.

We show that during the nineties, bans in bars, restaurants and recreational areas have lead to a relative increase in the exposure of young children. We hypothesise that such bans displace smoking to places where non-smokers are more exposed. We test this hypothesis by

[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/).

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.

We also show that bans on smoking in other public places have been successful in limiting exposure, so that on average bans have had *no* effects on non-smokers, once one controls for state and year effects. In contrast, 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.

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 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 prices on smokers is quite large, there is hardly any evidence on the effectiveness of these measures nor on the effectiveness of restricting smoking on reducing smoking exposure for *non-smoker*.[5] [6] Yet, the debate in public circles

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

[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] 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

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[7]. There is to our knowledge no study evaluating the response of passive smoking to the growing set of regulation and clean air acts passed in the last decade or to changes in excise taxes[8]. One of the main reasons 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.

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 changes in smoking bans and in tax on passive smoking. In particular, we define our measure of passive smoking and describe our identification strategy.

2.1 A Model of Passive Smoking

To fix ideas, we present a simple model of the effect of smoking regulation on passive smoking. Suppose that a smoker derives utility from the number of cigarettes in two places, c_H (smoked at home) and c_B (smoked in bars). The smokers also derive utility from a composite good, *q*. Denote by *p*, the relative price of tobacco and by *y* total income. The problem of the smoker is:

$$(1) \qquad \max_{c_H, c_B, q} u(c_H, c_B, q) \qquad \text{subject to} \qquad y = p(c_H + c_B) + q$$

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).

[7] See for instance ASH (2005) for a summary of the case for smoke free public places.

[8] A search in EconLit for the key words "passive smoking" generates only 4 hits that are unrelated to the issue discussed here.

Suppose that the utility function takes the following form, $u(c_H, c_B, q) = c_H^{\alpha_H} c_B^{\alpha_B} q^{\alpha_q}$. The optimal number of cigarettes for a smoker in both places is:

(2) $$c_H^* = \frac{\alpha_H}{\alpha_H + \alpha_B + \alpha_Q} \frac{y}{p} \qquad \text{and} \qquad c_B^* = \frac{\alpha_B}{\alpha_H + \alpha_B + \alpha_Q} \frac{y}{p}$$

The government can impose a smoking ban in bars but not within a home. If such a smoking ban is imposed, the smoker is allowed to smoke only in a fraction $\rho \in [0,1]$ of all bars. We discuss later the case when this policy changes the preferences over cigarettes or location. Assuming constant preferences and no change in locations, the optimal number of cigarettes smoked at home is then:

(3) $$\tilde{c}_H = \frac{\alpha_H}{\alpha_H + \alpha_Q} \frac{y}{p} \left(1 - \rho \frac{\alpha_B}{\alpha_H + \alpha_B + \alpha_Q} \right) \geq c_H^*$$

When a ban is in place, a smoker will compensate by increasing the number of cigarettes smoked at home.

First, consider a non-smoker who does not live with a smoker. Exposure comes solely from outside home. A ban on smoking in public places will unambiguously benefit that individual. Second, consider a non-smoker living with a smoker and who spends a fraction λ of his time at home and the rest in bars. In each of these places he is exposed to a fraction δ of each cigarette smoked by the smoker. Hence exposure is equal to:

$$Expo = \delta \lambda c_H + \delta (1 - \lambda) c_B$$

The effect of the ban on non-smokers is ambiguous and depends on the time spent at home and in bars. After some simple algebra, the change in exposure resulting from a ban can be expressed as:

(4) $$\Delta Expo = \frac{\delta \alpha_B (1 - \rho)}{\alpha_H + \alpha_B + \alpha_Q} \frac{y}{p} \left(\lambda - \lambda^* \right) \quad \text{and} \quad \lambda^* = \frac{\alpha_H + \alpha_Q}{2\alpha_H + \alpha_Q}$$

Non-smokers spending all their time in bars (λ=0) have a decrease in their exposure, while those spending most of their time away from bars (e.g. children) have an increase in exposure as $\lambda^* < 1$.

Smoking can also be regulated through higher cigarette prices. If prices are increased by a factor γ>1, the change in exposure for non smokers (living with a smoker) can be written as:

(5) $$\Delta Expo = -\frac{\delta}{\alpha_H + \alpha_B + \alpha_Q}\frac{\gamma - 1}{\gamma}\frac{y}{p}\left(\lambda(\alpha_H - \alpha_B) + \alpha_B\right)$$

Here again, the effect is ambiguous. If $\alpha_H > \alpha_B$, then the change in exposure is larger for those who spend more time at home.

The model predicts an ambiguous effect of smoking bans due to a displacement of smoking from public areas, where smoking is banned, to private areas (home). Non smokers who live with smokers and spend most of their time at home (e.g. infants) may suffer from an increase in exposure. An increase in tobacco prices leads to heterogeneous changes in exposure to tobacco smoke, with a bigger effect for those who spend more time at home.

It is possible that smoking bans affect not only the choices of how many cigarettes to consume, but also preferences towards tobacco and choice of location.[9] Extending the model to allow for an endogenous choice of location would lead to more exposure of non-smokers as smokers turn away from bars. Due to the addictive nature of tobacco, it is also possible that in the longer-run smokers are induced to smoke less as their stock of habit is decreased by a ban.

The model shows that it is 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). In general, the change in passive exposure is not proportional to the change in the number of cigarettes smoked because smokers and non smokers do not spend the same amount of time in each location. Therefore, an empirical analysis should measure passive smoking *directly* in non-smokers.

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

[9] Adda et al (2006) provides evidence that a smoking ban introduced in Scotland decreased the number of customers.

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. 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.[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 such as tobacco related diseases which take time to develop. Finally, there is minimal measurement error, compared with self-declared exposure to cigarettes, which has been used as a measure of passive smoking. Cotinine is therefore a straightforward and precise measure of passive smoking, particularly suited to evaluate policies aimed at reducing smoking.

2.3 Econometric Specification

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*:

[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.

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

where Cot_{ist} is the cotinine concentration (expressed in ng/ml); R_{jst} is a measure of restrictions on smoking in public place *j* in the state at the period of interest; We consider different places: j=*GO* is a measure of restrictions on smoking in bars, restaurant and other recreational places ("going out"); j=*PT* restrictions in public transport; j=*SM* restrictions in shopping malls; j=*WP* restrictions in the work place; j=*Sch* restrictions at school; tax_{st} is the state excise tax on tobacco (adjusted for inflation) in a given state and period; D_i 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. 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 smoking restrictions and the effect of taxes on cotinine measures. We relate exposure to excise taxes as this is the relevant policy variable from a public health point of view. We test for displacement effects by testing whether $\tilde{\beta}$ or $\tilde{\gamma}_j$ are zero. If any of these coefficients are positive, it means that the policy has some unintended consequences on non-smokers as smoking is displaced from public to private places. We discuss in detail which groups we consider in section 4.[13]

The identification of the effect of regulation and taxes 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

[13] Note that under the assumption that $E(x_i \mid D_i) = E(x_i)$, $E(x_i u_i \mid D_i) = E(x_i u_i)$ and $E(u_i D_i) = 0$, where x_i is either bans or taxes, the estimator of $\tilde{\beta}$ or $\tilde{\gamma}_j$ are consistent even if bans or taxes are endogenous. The intuition behind this result is that these coefficients capture the differential impact of x_i across groups characterized by D_i and by assumption, the endogeneity affects both groups in the same way. Proof available upon request.

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 the prevalence of smoking is decreasing 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.

The model is estimated by OLS, and standard errors are adjusted for heteroskedasticity and clustered at state level.[14] 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.

[14] 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.

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.[15] It represents about 5% of the declared non smokers. In total, we observe 29667 non-smokers with a valid measure of cotinine concentration.[16]

[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.

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

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 Smoking Restrictions to Tobacco Exposure and Excise Taxes

We 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.[17] This data set reports the regulation in place by year and by state in different locations. Note that while the data is at state level it combines both state level regulations and an aggregation of county level restrictions. The advantage of aggregating data at state level is to minimize cross-boundary effects. The disadvantage is a lack of precision if there is dispersion around the state mean within state.

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). Eleven different locations where 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. For each of these locations the dataset records the degree of restrictions enforced in each year (1991-2001). 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] http://www.impacteen.org. The data was 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.

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. Our identification strategy relies on within state variation in excise taxes and smoking regulation. 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.

We also 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. Taxes have on average increased by 2 cents per year.

3.3 Trends in Passive Smoking

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 2). 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. 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 2]

Next, we separate non smokers who share their household with smokers, from non smokers who live in "smoke free" households. Figure 3 plots the cotinine concentration in non-smokers living in non smoking households from 1988 to 2000. Figure 4 shows, for the same time period, the cotinine concentration of non smokers sharing the house with smokers.

[Figure 3 and Figure 4]

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

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 4). 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]

4 Empirical Results: Passive Smoking and State Intervention

4.1 Passive Smoking and Anti-Smoking Policies

We first analyse the impact of bans and taxes on passive smoking in the whole sample of non smokers. We first group bans in all locations into a single index. This is formed by taking the average of all regulations across all public places and ranges between 0 (no bans anywhere) to 3 (bans everywhere). The results are presented in Table 2. We consider first the effect of smoking bans on cotinine concentration in non smokers, then of excise taxes, and finally of both bans and taxes together[20].

[Table 2]

Column (1) displays the effect of regulations on smoking and shows that a one standard deviation increase in regulations would decrease the cotinine concentration in non-smokers by 0.03 ng/ml. This result is in accordance with many epidemiological analyses which views bans as a successful tool to limit passive smoking. Controlling for state effects reduces by a

[19] 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 re-weighted 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.

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

third the effect of regulations on passive smoking, but we can still rule out a zero effect (Column 2). As discussed in the previous section, smoking bans have become more prevalent through the eighties and nineties, and exposure decreased during that period. It is therefore important to control for an aggregate time effect. Column 3 shows that when both time and state fixed effects are used, the effect of a ban is further halved and becomes statistically insignificant. Moreover, the standard error of the estimate is low. The evidence in Table 2 shows that the decrease in exposure to tobacco smoke occurred both in states with and without these regulations, so that smoking bans appears to have no role in preventing exposure.

We now turn to the effect of (log) excise taxes on passive smoking without controlling neither for state of residence nor for year of survey (column 4). The effect is identified here through variations through time and state differences. 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 5 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 slightly stronger. A 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%.

Columns 6 and 7 of Table 2 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.[21] 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

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

further into this result below when we break down the effect by age and by family smoking status.

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.

Column 7 of Table 2 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.

4.2 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 3 displays the effect of regulation and taxes 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.

[Table 3]

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 places such as bars and restaurants. We observe a significant increase in the cotinine level in non-smoking individuals when bans are enforced in such places: a one standard deviation increase in bans 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 increase in exposure is through an indirect contamination due to the displacement of smokers towards non-smokers as derived in equation (4). We now investigate this point further.

4.3 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.

4.4.1 - Policy Impact by Age Group

We now proceed in analysing the effect of taxes and regulation on passive smoking across age groups. Table 4 separates non-smokers by age groups. Column (1) refers to the overall sample of non smokers. Columns (2) to (5) of Table 4 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 4]

The first row of Table 4 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 interpreted as the existence of a displacement 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 leading 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

[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.

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.

The last row of Table 4 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 is line with the prediction of equation (5). 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.

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 5 we separate children by family

smoking status and report 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 5]

The observed effects of changes in regulations are considerably larger in children living in smoking households than in children living in non-smoking households.

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 1 of table 5) 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 places such as bars and 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 to such places.

Regarding the effect of regulation in other public places we observe that tighter regulations in shopping malls leads to a statistically significant reduction in the cotinine levels observed in children living in smoking households. Again this does not have a sizable and significant effect on children 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. The effect is larger in magnitude than the one for all children irrespective of smoking

[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. Our attempt to control for this bias using the methodology detailed in footnote 19 was not supportive of this alternative explanation.

status presented in Table 4. 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.

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 indoors, 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.

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 6.[24]

[Table 6]

The first row of Table 6 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 taxes or regulations either. In contrast, when we look at children in smoking families, we find strong seasonal effects. The effect of smoking restrictions in places where individuals go out is more pronounced in winter than in summer. We also find that for children in smoking households 'other regulations' are more efficient in winter than in summer.

Similarly, taxes appear to have a stronger effect in winter which is consistent with adults and children being in-doors.[25]

[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.

[25] Taxes and anti-smoking regulations do not change with season and if sampling is random across seasons, the requirement listed in footnote 12 are met. The differential effect of taxes and smoking regulations is

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. This is consistent with the simple model we derived in Section 2.

4.4 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, we concentrate on the exposure of 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%.

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

therefore consistently estimated even if their overall effect may be biased due to endogeneity of anti-tobacco regulations. This will also be true if we condition the regressions on household smoking status.

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 4, 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).

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

[26] The standard error is equal to 0.3.

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.

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. 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. 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. This paper provides insights on how to design optimal policies to curb passive smoking.

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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 - Effects of One Standard Deviation in Smoking Bans and Taxes on Passive Smoking

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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Smoking Bans	-0.032**	-0.012**	-0.006			0.005	0.004
	(0.009)	(0.002)	(0.008)			(0.006)	(0.005)
Log Tax				-0.02**	-0.03*	-0.04**	-0.05**
				(0.012)	(0.012)	(0.015)	(0.019)
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 3 - 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)
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)
Log Tax	-0.04**
	(0.02)
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 4 - 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)
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)	
Log Tax	-0.04**	-0.20**	-0.12**	-0.01	-0.01
	(0.02)	(0.06)	(0.03)	(0.05)	(0.02)

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 5: 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)
Regulation Going Out	0.03	1.08**
	(0.04)	(0.15)
Regulation Public Transport	0.03	-0.03
	(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)
Log Tax	0.012	-0.30**
	(0.02)	(0.06)
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 6 – 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)
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)
Log Tax	-0.13	0.04
	(0.09)	(0.08)
Tax*Winter	0.12	-0.27**
	(0.09)	(0.12)
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

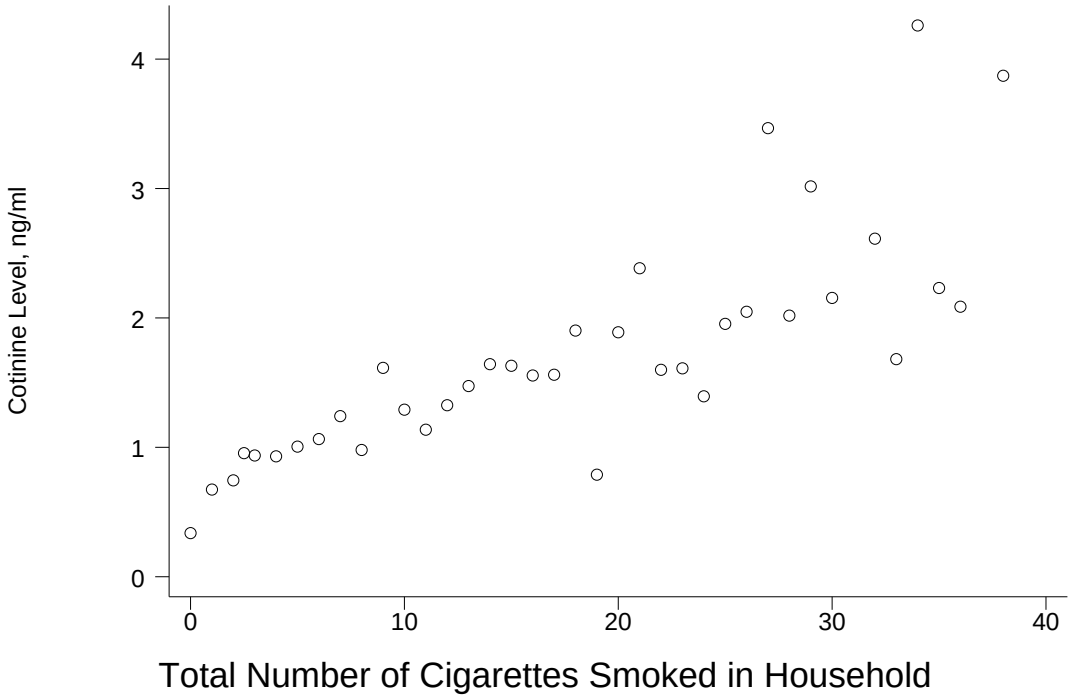

Figure 2: Average Cotinine Concentration in Non-Smokers.

Figure 3: Average Cotinine Concentration in Non-Smokers – Non Smoking Households

Figure 4: Average Cotinine Concentration in Non-Smokers – Smoking Households