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Estimating the equity impacts of the smoking ban in England on cotinine levels: a regression discontinuity design

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ABSTRACT

Objective To estimate the equity impacts of the 2007 smoking ban in England, for both smokers and non-smokers.

Design Doubly robust regression discontinuity analysis of salivary cotinine levels. Conditional average treatment effects were used to estimate differential impacts of the ban by socioeconomic deprivation (based on the Index of Multiple Deprivation). Distributional impacts were further assessed using conditional quantile treatment effects and inequality treatment effects.

Setting In 2007, England introduced a ban on smoking in public places. This had little impact on tobacco consumption by smokers but was associated with decreases in environmental tobacco smoke exposure for non-smokers. However, the impact of the ban on socioeconomic inequalities in exposure is unclear.

Participants 766 smokers and 2952 non-smokers responding to the Health Survey for England in 2007.

Outcome measure Levels of salivary cotinine.

Results Before the ban, socioeconomic deprivation was associated with higher cotinine levels for non-smokers but not for smokers. The ban caused a significant reduction in average cotinine levels for non-smokers (p=0.043) but had no effect for smokers (p=0.817). Reductions for non-smokers were greater for more deprived groups with higher levels of exposure, and there was a significant reduction in socioeconomic-related inequality in cotinine. Across the whole population (both smokers and non-smokers), there was no significant increase in the concentration of cotinine levels among the socioeconomically deprived.

Conclusion The 2007 ban on smoking in public places had little impact on smokers, but was, as intended, associated with reductions in both (1) average levels of environmental tobacco smoke exposure and (2) deprivation-related inequality in exposure among non-smokers.

INTRODUCTION

Successive UK governments have pursued a range of public health policies to reduce population harms from tobacco smoke exposure since links to lung cancer, and other common fatal conditions were confirmed in the 1950s.[1] With public education, increased taxation and restrictions on sales and advertising, smoking prevalence fell from 70% in

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ We used a doubly robust regression discontinuity design to derive causal estimates of the equity impacts of the smoking ban for both smokers and non-smokers.
⇒ Data were derived from a rich, individual-level data set with an objective measure of tobacco smoke exposure to avoid social desirability bias in reported variables.
⇒ Causal effects of the ban were estimated across the entire distribution of the outcome, identifying conditional distributional effects and the impact on socioeconomic-related inequality.
⇒ We focused only on the short-term effects of the ban and rely on an area-based measure of socioeconomic status.

the 1950s to under 25% by the turn of the century.[2] More coercive policies began to be considered in the 1990s as studies emerged identifying harmful impacts on non-smokers exposed to environmental smoke,[3–12] including exposure at home[7 8] and in the workplace.[13] Estimates of the population impact of environmental exposure suggest that over 10% of smoking-related deaths may occur in non-smokers,[14] and the risks to smokers themselves are further increased by environmental smoke. The impacts of smoking are also unevenly distributed; while rates of smoking have fallen in all socioeconomic groups since 1950s, they fell fastest in the most affluent groups. At the end of the century, over half of the difference in mortality between men in high (professional, managerial and technical) and low (unskilled manual) socioeconomic groups was attributable to tobacco exposure.[15]

The growing weight of evidence on environmental tobacco smoke eventually led to the implementation of national bans in Scotland (March 2006), Wales and Northern Ireland (April 2007) and England (July 2007). For England and Wales, the Health Act 2006 effectively prohibited tobacco smoking in enclosed

public places, including workplaces, public transport and private clubs. The ban was found to have little impact on tobacco consumption in the short run,[16–20] but smokers were found to smoke substantially less in enclosed public places, including at work and in pubs.[17]

Evidence on the equity impacts of the smoking ban, particularly for non-smokers, is less clear. In England, smoking prevalence continued to fall at similar rates across different socioeconomic status (SES) groups after the ban, preserving the gaps between them; by 2015 smoking, prevalence remained above 25% in the most deprived fourth and fifth of areas, compared with 10% in the least deprived fifth.[19] In terms of secondhand exposure, Semple *et al*[21] noted that pubs in more deprived areas, with higher baseline particulate (PM2.5) concentrations, tended to have greater reductions after the ban. In contrast, King *et al*[22] found that higher SES smokers were more likely than low status smokers to prohibit smoking in their own home following the ban.

Obtaining more direct evidence on the equity impacts of smoking bans on non-smokers is subject to two key limitations: a reliance on before-and-after study designs measuring association, rather than strong econometric designs identifying causal effects and a focus on average effects for the whole population, rather than distributional effects relating to baseline exposure to tobacco smoke and socioeconomic circumstances. The latter limitation is particularly important, as national policies of this kind are frequently less effective for less affluent socioeconomic groups with higher baseline levels of exposure, and this has the unintended consequence of widening existing health inequalities. In this paper, we assess the impact of the national smoking ban on cotinine levels, a biomarker of tobacco smoke exposure, for both smokers and non-smokers. We use approaches that assess the full range of effects in relation to socioeconomic characteristics across the entire distribution of exposure levels, to more accurately determine the equity impacts of the smoking ban.

METHODS
Intervention
The national smoking ban in England was implemented on 1 July 2007. Smoking in enclosed public places became an offence, subject to fines of up to £200 for individuals and £2500 for businesses. Exemptions were included, at that time, for psychiatric units, nursing homes, prisons and other specified public places.

Data
Data were derived from the Health Survey for England (HSE), an annual cross-sectional survey, which records health and lifestyle changes over time in England at the individual and household level. The data set includes variables covering tobacco smoking and exposure as well as a range of individual characteristics recorded by an interview and, if consent is given, there is a follow-up nurse's

visit (on average 18.4 days later). Our main analysis uses data from the 2007 wave of the survey, from January 2007 to January 2008, covering the period of the introduction of the public smoking ban in England.

Patient and public involvement
As we conduct a secondary analysis of survey data, there was no direct patient and public involvement.

Tobacco exposure
The primary outcome is exposure to tobacco smoke, estimated by salivary cotinine concentration. Cotinine is a metabolite of nicotine, which diffuses from the bloodstream into saliva, making salivary cotinine concentration a convenient and reliable biomarker of regular tobacco exposure.[23] Salivary cotinine concentrations above 12 ng/mL indicate smoking status with high sensitivity and specificity[24] and scale with the extent of tobacco use. Lower concentrations are also detectable, so cotinine is useful for monitoring changes in both tobacco use by smokers and exposure to secondhand smoke in non-smokers. Distributions of salivary cotinine levels tend to be heavily skewed; for non-smokers, levels are often undetectable, whereas for smokers, a minority of concentrations are extremely high, and these can have a disproportionate impact on results. We therefore used log(cotinine +1) as our primary outcome, but also report results using untransformed values (i.e. cotinine concentration) online supplemental appendix A.1. The subset of respondents to the HSE receiving a nurse visit is asked to provide a saliva sample, which is then analysed for cotinine concentration. For 2007, 4058 valid cotinine measurements were available.[i] Current smoking status (smoker/non-smoker) was based on self-report.[ii]

Socioeconomic deprivation
Our primary measure of socioeconomic deprivation was the 2007 Index of Multiple Deprivation (IMD) quintile.[25] This is an area-based measure linked to postcode, for which we use the quintiles or group into 'deprived' (the lowest two quintiles) and 'non-deprived' (highest three quintiles). IMD was selected over alternative SES variables such as household income and occupation, as it had fewer missing data, it demonstrated the largest differential conditional effect and was more stable during the period between the main survey and nurse follow-up.

Analysis
Regression discontinuity design
We used a sharp regression discontinuity (RD) design to estimate the causal effects of the intervention. The

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[i] In our main analysis 3718 responses were used. Of these, 309 were excluded due to the bandwidth of the regression discontinuity design and a further 31 were excluded because observations for control variables were missing.

[ii] In the pre-intervention sample, 95.7% of self-reported smokers and 4.3% of non-smokers had cotinine concentrations >12 ng/mL. Reporting error on smoking status, therefore, appears low.

design uses 1 July 2007, the date of implementation of the national smoking ban, as a threshold dividing the sample into 'control' (interviewed before the ban) and 'treated' (interviewed after the ban): with a bandwidth of ±190 days. Doubly robust methods were used, with inverse probability of treatment weights in locally weighted regressions with triangular kernels, to control for potential covariate imbalances at the time of the ban. More details are provided in online supplemental appendix A.2.

The model takes the general form:

$$y = \beta_0 + D\beta_1 + Z\beta_2 + (D \times Z)\beta_3 + u \text{ for } |Z| \leq h \quad (1)$$

where y is the outcome variable (log cotinine), D is the treatment dummy and Z is the forcing variable, time from ban, which takes the value zero on the day of the policy change, it is positive if the observation is after the ban and negative before. The error term is u, which is assumed to be independent and identically distributed with an expected value of zero. For the coefficients, β_0 represents the average outcome for the control group at the point of discontinuity where $Z \to 0$ (date of the ban), β_1 is the treatment effect at the discontinuity, β_2 is the time trend before the ban and β_3 indicates the change in the trend after the ban.

Treatment effects

We used the RD approach to estimate the potential outcomes individuals would have had with and without treatment at the threshold.[26] The overall impact of the smoking ban on tobacco exposure was measured as an average treatment effect:

$$ATE = \beta_1 = E\left[Y(1) - Y(0) \mid Z = 0\right]$$

Where Y(0) is the *potential* outcome an individual would have had without treatment and Y(1) is the *potential* outcome with treatment. We then evaluated the distributional impacts of the smoking ban using a series of approaches: (1) conditional average treatment effects (CATEs) (2) quantile treatment effects (QTEs); (3) conditional QTEs (CQTEs) and (4) inequality treatment effects (ITEs).(2)
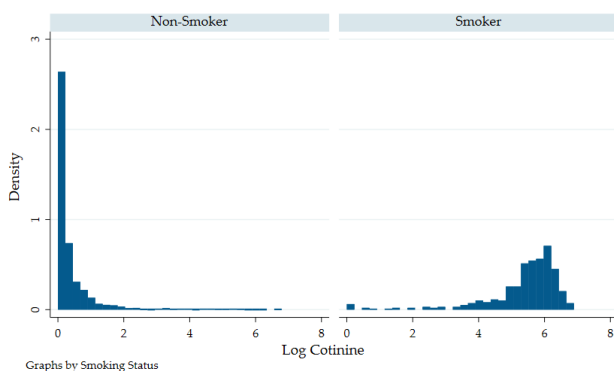

Figure 1 Distribution of log cotinine: non-smokers and smokers.

CATEs estimate the average effect of the treatment (the smoking ban) on y (log cotinine) conditional on x (eg, whether the effect of the smoking ban varied with socioeconomic deprivation):

$$CATE\left(\widetilde{x}\right) = E\left[Y(1) - Y(0) \mid \widetilde{X} = \widetilde{x}, Z = 0\right] \quad (3)$$

QTEs estimate the effect of the treatment on *y* at different quantiles (τ) of *y*:

$$QTE(\tau) = Q^{\tau}_{Y(1)|Z=0} - Q^{\tau}_{Y(0)|z=0} \quad (4)$$

where $Q^{\tau}_{Y(.)|Z=0}$ is the quantile function, which returns the cotinine level Y(.) at the threshold for each quantile, $\tau \in (0,1)$ (conditional rank of the outcome). The differences between these quantile functions give the treatment effect at each quantile. These effects are estimated directly, using quantile regressions,[27] within the RD framework.[28] This allows us to determine how the ban changed the distribution of tobacco exposure, revealing whether levels fell more at the top or bottom of the distribution.

CQTEs combine QTEs and CATEs, estimating the effect of the treatment at the quantile of y (log cotinine), conditional on x (eg, socioeconomic deprivation):

$$CQTE(\tau, \tilde{x}) = Q^{\tau}_{Y(1)|\tilde{X}=\tilde{x}, Z=0} = Q^{\tau}_{Y(0)|\tilde{X}=\tilde{x}, z=0} \quad (5)$$

Finally, ITEs[29] identify the difference in inequality indices between the treated and control:

$$ITE = W(Y(1) \mid Z=0) - W(Y(0) \mid Z=0) \quad (6)$$

where $W(.)$ denotes an inequality index, in this case, the Concentration Index,[30] a *relative* measure of bivariate inequality:

$$W\left(y, \widetilde{x}\right) = \frac{1}{N} \sum_{i=1}^{N} \left(\left(2R\left(\widetilde{x}_i\right) - 1\right) \frac{y_i}{\overline{y}}\right) \quad (7)$$

where $\left(2R\left(\widetilde{x}_i\right) - 1\right)$ is the fractional rank of $\widetilde{x}_i$ and $\overline{y} = \frac{1}{N} \sum_{i}^{N} (y_i)$ is the mean outcome. The concentration index indicates how concentrated health-related outcomes are among those of a higher/lower SES. Where there is no socioeconomic-related inequality, the concentration index is zero, with values further from zero indicating greater inequality. We used methods developed by Heckley *et al*[31] to estimate ITEs within the RD framework using recentered influence function regressions.

RESULTS
Baseline characteristics

Figure 1 shows the distribution of transformed cotinine values (log(cotinine+1)) for non-smokers and smokers in the control group (preintervention). For non-smokers, the distribution was heavily right skewed (31.4% of the sample had zero values) with a mean of 0.43 (actual value of 4.95 ng/mL). For smokers, the distribution was left skewed with a mean of 5.37 (303.24 ng/mL).

Baseline characteristics, by quintile of cotinine, are shown in online supplemental appendix A.3. Higher cotinine levels were associated with younger age, being
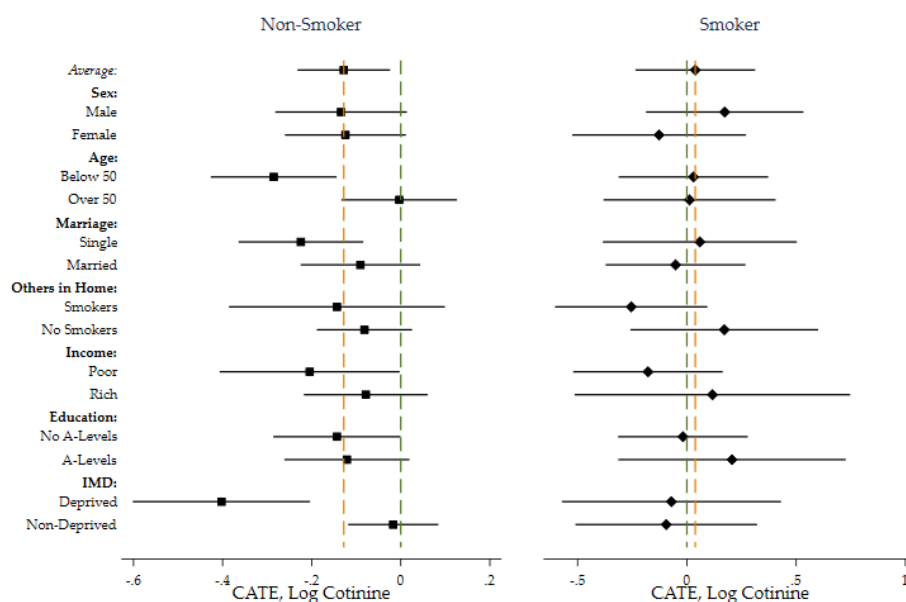
Figure 2 Forest plot of conditional average treatment effects of log cotinine: non-smokers and smokers key: bars denote 95% CIs. CATE, conditional average treatment effect; IMD, Index of Multiple Deprivation.

unmarried, living in an urban environment, lower SES, poorer self-assessed health and higher levels of alcohol consumption, in addition to smoking status and tobacco consumption.

Average treatment effects

At the time of the ban, the expected baseline level of log cotinine (without treatment) was 0.40 for non-smokers and 5.34 for smokers. There was a large and significant reduction in cotinine concentrations for non-smokers (−0.128, p=0.043), but no significant effect of the ban for smokers (0.036, p=0.817). Regression outputs are shown in online supplemental appendix A.4. These results are robust to a set of sensitivity analyses, see online supplemental appendix A.5.

These effects are shown at the top of figure 2, which shows forest plots of average effects of the smoking ban conditional on sex, age, marital status, smoking status of others in the household, highest qualification and IMD (CATEs). For smokers, there were no significant effect conditional on any variable. For non-smokers, there were both significant effects and significant differences between subgroups, most notably larger reductions in cotinine levels for younger, single, income poor or IMD-deprived respondents.

The differences in CATEs for non-smokers are partially explained by different baseline predictions across subgroups. For example, the predicted average level of log cotinine at the time of the intervention was 0.65 for deprived and 0.30 for non-deprived. After the ban, these levels fell to 0.23 and 0.27, respectively (not significantly different), see online supplemental appendix A.6 for more detail.

Distributional analysis

Figure 3 shows QTEs, that is, the effect of the smoking ban on the level of cotinine at different parts of the distribution (quantiles of cotinine). For non-smokers, there was no effect on cotinine levels at the lower quantiles (which were already at 0) but significant, and increasing, reductions appeared at higher quantiles of cotinine. For smokers, there were no significant effects at any quantile.

Figure 4 shows QTEs conditional on IMD deprivation. Significant reductions in cotinine levels are apparent for deprived non-smokers at higher levels of exposure. For non-deprived non-smokers, there are significant small effects at the mid-range of exposure (going from very little to zero exposure) but not at higher levels. For both groups of smokers, there was no significant reduction at any quantile of cotinine.

Changes in inequality

Table 1 shows the causal effect of the ban on bivariate inequality (the concentration index), that is, how the concentration of log cotinine levels among the socioeconomically deprived (IMD quintile) changes.

The negative constants in models (1) and (2) show that higher levels of cotinine were concentrated among more socioeconomically deprived respondents at baseline for the whole sample (indicating higher levels of smoking) and non-smokers (indicating higher levels of second-hand exposure). However, the constant is close to zero for smokers, therefore conditional on being a smoker, deprivation has limited impact on cotinine levels.

The treatment effect shows that the ban caused a significant increase in the concentration index for non-smokers (ie, towards zero), reducing the concentration
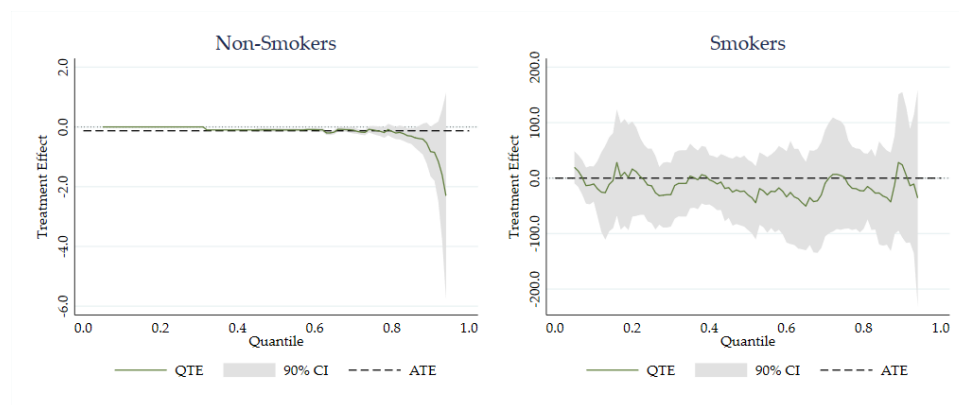
Figure 3 QTEs: log cotinine QTEs for smokers and non-smokers key: solid line denotes QTE; dotted line denotes ATE; grey area denotes 90% CI. ATE, average treatment effect; QTE, quantile treatment effect.

of cotinine levels among deprived non-smokers. No significant effects were found for the whole sample or for smokers. These results indicate that the ban caused a reduction in deprivation-related inequality in cotinine levels among non-smokers but had no significant effect for smokers.

DISCUSSION

Given the socioeconomic gradient of tobacco smoke exposure, public smoking bans could potentially benefit deprived groups more. Conversely, poorer individuals may respond less well to such policies due to lack of opportunities and resources, contextual factors[32] and attitudes towards antismoking policies.[33] We found that the smoking ban in England significantly reduced exposure for non-smokers with greater reductions for

socioeconomically deprived groups with higher baseline levels of exposure. However, the ban had no significant impact on exposure levels, or on inequalities in exposure, for smokers.

Strengths and limitations of the study

We used a doubly robust RD design to derive causal estimates of the differential impacts of a national smoking ban across the entire distribution of exposure levels, for both smokers and non-smokers. We also used a rich, individual-level data set with an objective measure of tobacco smoke exposure to avoid social desirability bias in reported variables and explored differential effects of the ban both between groups using CATEs and across the entire distribution of cotinine levels using quantile treatment effects. We unified these approaches to examine conditional distributional effects and estimate the causal
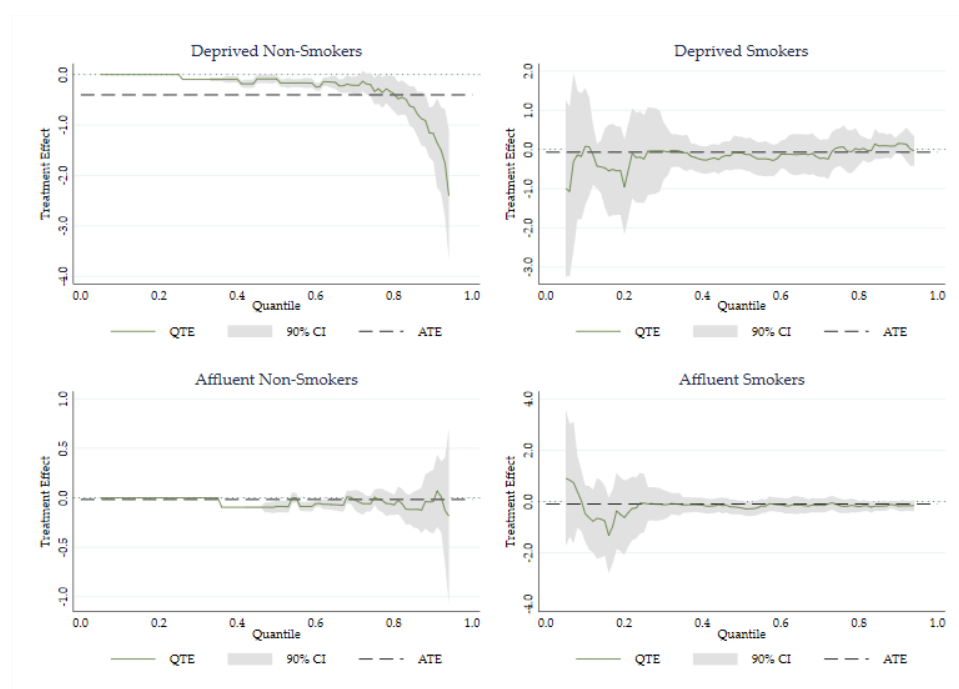

Figure 4 Conditional QTEs: log cotinine key: solid line denotes QTE; dotted line denotes ATE; grey area denotes 90% CI. ATE, average treatment effect; QTE, quantile treatment effect.

Table 1 Log cotinine: concentration index

	(1)	(2)	(3)
	Sample	**Non-smokers**	**Smokers**
	Coefficient/ SE	**Coefficient/ SE**	**Coefficient/ SE**
Treatment	0.0265	0.2097**	−0.0285
	(0.0693)	(0.0910)	(0.0182)
Time	0.0005	−0.0015*	0.0003**
	(0.0005)	(0.0008)	(0.0001)
Treatment × Time	−0.0009	0.0007	−0.0003
	(0.0008)	(0.0011)	(0.0002)
Constant	−0.1501***	−0.2329***	0.0119
	(0.0394)	(0.0559)	(0.0127)
N	3718	2952	766
R^2	0.0009	0.0029	0.0076

*p<0.10, **p<0.05, *** p<0.01.

impact of the ban on socioeconomic-related inequality in cotinine.

The study had several limitations. First, the HSE is a repeated cross-sectional survey and we were, therefore, unable to observe individual's over time; rather, we compared distributions of exposure for different individuals surveyed before and after the ban. Second, we relied on self-report to determine smoking status at the time of the survey. The distributions of cotinine levels for smokers and non-smokers suggest that self-report was accurate for the majority of subjects, but there may have been some misclassification. Third, social deprivation was measured by area deprivation rather than by individual social status and was arbitrarily categorised in quintiles, our analysis of socioeconomic deprivation was, therefore, less fine grained than our analysis of cotinine levels. Finally, we have only measured the short-term effects of the ban, and these may change over the longer term.

Findings
Our results suggest that the smoking ban in England did little to reduce overall risk or socioeconomic inequalities in smoking-related diseases for the smoking population, but it did, as intended, reduce both overall risk and inequalities in risk for non-smokers, with greater reductions in environmental tobacco smoke exposure for socioeconomically deprived non-smokers with higher baseline levels of exposure. These findings align with systematic reviews showing that compulsory smoking restrictions are generally more equitable than voluntary policies[34] and Semple et al[21]'s findings of greater reductions in environmental smoke in pubs in more deprived areas following the UK ban. A previous study of cotinine levels in non-smokers by Sims et al[35] similarly reported significant reductions in average exposure following the ban but, in contrast to our results, found no significant reductions in the lowest SES households and a significant

reduction for higher SES households (a 37% reduction in the geometric mean of cotinine), implying an increase in bivariate inequality, whereas our study used an RD approach. Sims et al[35] modelled impacts based on preintervention trends that indicated exposure was already falling prior to 2007. However, these models lacked data for the 3 years immediately prior to 2007 and may have underestimated additional reductions resulting from the ban. Sims et al[35] also measured SES using social class of the household head, resulting in a relatively small sample of low status non-smokers; our different results might, therefore, be explained by reduced levels of exposure being achieved in more deprived areas generally, but not in the very lowest status households. This is consistent with our previous study, which found significant reductions in self-reported exposure for non-smokers, which increased with social deprivation, with the exception of individuals with extremely low SES, for whom there was no significant impact.[20]

A major concern at the time of the ban was that smokers would substitute smoking in private areas for smoking in public places, and that this could unintentionally increase risk for vulnerable groups, as happened in the US following similar bans.[36] Our results suggest that such substitution activity did not occur and that there was a net reduction in exposure for adult non-smokers; this is supported by findings of reductions in smoking prevalence inside cars (from 32% to 26%) and the home (from 65% to 55%) reported by smokers following the ban.[17]

CONCLUSIONS
The 2007 smoking ban in England reduced environmental tobacco smoke exposure for non-smokers, with the largest benefits for more deprived non-smokers with high levels of exposure. This group was most at risk from the negative externalities of smoking, and the ban was, therefore, not only an effective but an equitable intervention. Nevertheless, there might still have been a negative equity impact of the ban across the whole population due to an increased gap in exposure between smokers (who are more likely to be deprived) and non-smokers (who are more likely to be affluent). However, our results show that gaps in overall levels of tobacco smoke exposure narrowed between affluent and deprived non-smokers after the ban and bivariate inequality across the whole population did not increase.

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A   Appendix

A.1   Sensitivity: Cotinine

Throughout the paper we use log(cotinine +1) as our outcome variable. We use this transformed variable due to the highly right skewed nature of cotinine and the potential impact of extreme values on results. Here we present the main results using cotinine, rather than log cotinine. Throughout results are robust to the choice of variable, but standard errors tend to increase for cotinine.

As in the main analysis, for the average treatment effect there are large and significant reductions for non-smokers, and no significant reductions for smokers.

Table A1: Cotinine: Regression Discontinuity

	(1) **Non-Smokers** Coef./S.E.	(2) **Smokers** Coef./S.E.
Treatment	-4.9852**	-12.1932
	(2.5121)	(32.1071)
Time	0.0016	-0.0230
	(0.0293)	(0.2154)
Treatment X Time	0.0501	-0.0729
	(0.0335)	(0.3449)
Constant	5.3445**	301.0218***
	(2.4040)	(18.6132)
N	2916	759
R-squared	0.0026	0.0030

$* p < 0.10, ** p < 0.05, *** p < 0.01$

For conditional average treatment effects, we observe no significant effects for smokers conditional on any variable. For non-smokers, we observe both significant effects and differences between subgroups. The difference between age groups here disappears and standard errors do increase in comparison to the main results. When we estimate these effects using negative binomial regressions, however, these standard errors reduce.

Figure A1: Forest Plot of CATEs of Cotinine: Non-Smokers and Smokers
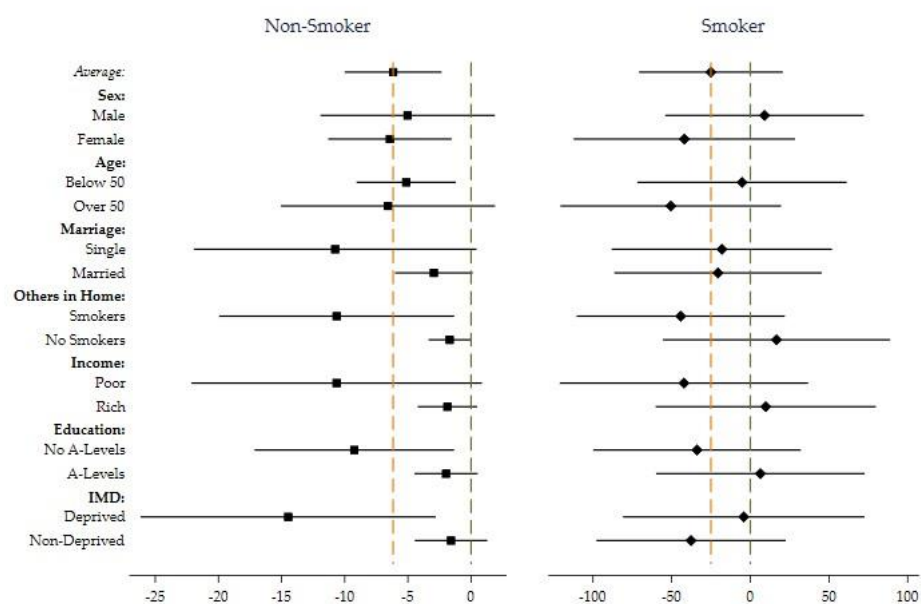
Quantile treatment effects are similar in significance and direction to the main results. There are significant, and increasing, treatment effects for higher quantiles for non-smokers and no significant effects for smokers, at any quantile.

Figure A2: Quantile Treatment Effects: Cotinine
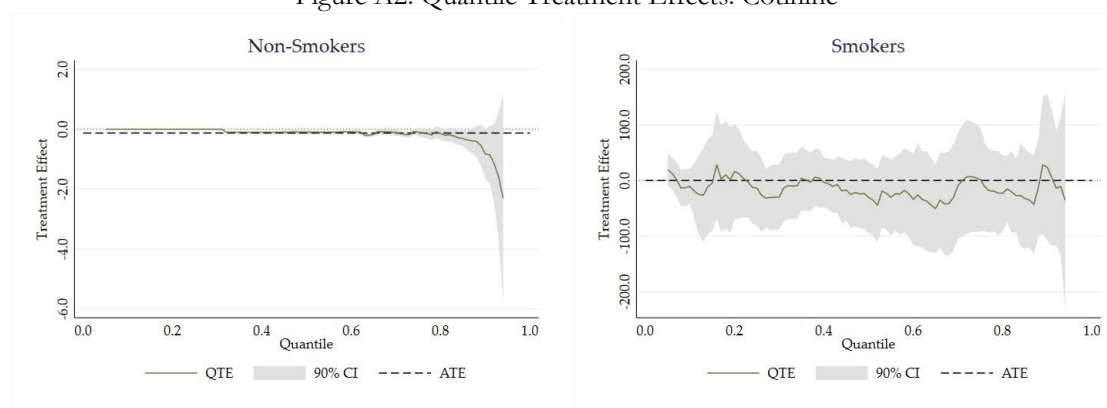
Inequality treatment effects are very similar to the main results. The concentration of cotinine amongst deprived non-smokers was significantly reduced, whilst no significant effects were observed at the sample level or for smokers.

Table A2: Cotinine: Concentration Index

	(1) Sample Coef./S.E.	(2) Non-Smokers Coef./S.E.	(3) Smokers Coef./S.E.
Treatment	-0.0209	0.5571***	-0.0728
	(0.1031)	(0.1647)	(0.0648)
Time	0.0011	-0.0062**	0.0006
	(0.0008)	(0.0024)	(0.0004)
Treatment X Time	-0.0018	0.0045	-0.0007
	(0.0012)	(0.0031)	(0.0007)
Constant	-0.1335**	-0.5714***	0.0356
	(0.0609)	(0.1835)	(0.0379)
N	3675	2916	759
R-squared	0.0010	0.0026	0.0060

$* p < 0.10$, $** p < 0.05$, $*** p < 0.01$

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A.2 Covariate Balance and Weights

A.2.1 Covariate Balance

Central to the estimation of treatment effects is the comparability of characteristics of the control and treated groups. If there are imbalances in covariates the estimated effects may be biased. To test if there are significant differences in covariates, $x_j, \ \forall j \in J$, the covariate of interest can be regressed against the treatment dummy, D:

$$x_j = \beta_0 + D\beta_1 + u \tag{8}$$

Here β_1 identifies the extent and significance of covariate imbalance between control and treated groups. In regression discontinuity designs, however, the crucial assumption is that there are no significant discontinuities in covariates at the *threshold*. Imbalances between control and treated groups *on average* are not important if this assumption holds. In a simple linear framework, this assumption can be tested by regressing the covariates against the treatment dummy, D, (centred) forcing variable, Z and their interaction:

$$x_j = \gamma_0 + D\gamma_1 + Z\gamma_2 + (D_0 \times Z)\gamma_3 + u, \qquad for \ |Z| \leq h \tag{9}$$

If γ_1 is significant it shows there is a discontinuity in x_j at the threshold. This imbalance at the threshold could bias the estimation of the treatment effect.

A.2.2 Inverse Probability of Treatment Weights

To mitigate any potential biases that these imbalances could cause, doubly-robust methods which use propensity weights alongside the regression discontinuity design, can be used. We estimate the inverse probability of treatment to use as weights. This balances covariates across treatment groups, giving lower weight to those characteristics which are predictors of treatment (i.e. via selection into treatment).

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To estimate propensity weights the following regression is run, as a logit:

$$\boldsymbol{D} = \delta_0 + \sum_{j}^{J}\left(\boldsymbol{X_j}\delta_{1j} + \left(\boldsymbol{X'_j} \times |\boldsymbol{Z}|\right)\delta_{2j}\right) + \boldsymbol{u} \qquad for\ |\boldsymbol{Z}| \leq h \qquad (10)$$

This formulation includes both covariates $\boldsymbol{X_j}$ and the interaction between those covariates and the absolute value of the forcing variable $\boldsymbol{Z}$. This particular formulation is used to ensure balance not just in expectation (as in Equation (8)) but also at the discontinuity. By using the absolute value of $\boldsymbol{Z}$ we provide this flexibility, whilst ensuring the overlap assumption is not violated. These weights are estimated separately for each subgroup.

Using this we predict the probability of being treated, given the observable characteristics $Pr(D = 1|X,Z)$. The inverse probability of treatment weights are then calculated as:

$$w_i^{IPT} = \frac{D}{\Pr(D = 1\ |X,Z)} + \frac{1 - D}{1 - \Pr(D = 1\ |X,Z)} \qquad (11)$$

Table A3 shows the consequence of using these weights on covariate balance. Each coefficient (and standard error) are results from separate regressions, with the outcome variable x_j in the first column. Average differences are the β_1 coefficients of Eq.(8), with differences at the discontinuity being the γ_1 coefficients of Eq.(9).

In the unweighted sample we see that the treated group, on average, live in more rural and less deprived areas. At the discontinuity, respondents have significantly fewer children, are more likely to be retired and live in rural areas. These differences could potentially cause bias in the estimates of the treatment effect and need to be controlled for. When the weights are applied, however, we observe no significant differences either on average or at the discontinuity; removing the selection bias (on observables) which could have biased estimates.

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Table A3: Balance

	Unweighted		Weighted	
	(1)	(2)	(3)	(4)
	Average	**Discontinuity**	**Average**	**Discontinuity**
	Coef./Std. err.	Coef./Std. err.	Coef./Std. err.	Coef./Std. err.
Age	0.6179	3.5169***	-0.0543	-0.2464
	(0.5288)	(1.0511)	(0.6107)	(1.1586)
Sex (1 = female)	0.0025	-0.0051	0.0005	0.0019
	(0.0149)	(0.0297)	(0.0175)	(0.0337)
Married Couple	-0.0155	-0.0023	-0.0004	0.0026
	(0.0148)	(0.0296)	(0.0173)	(0.0331)
Number of Children	-0.0248	-0.2123***	-0.0007	0.0145
	(0.0239)	(0.0445)	(0.0298)	(0.0592)
Retired	0.0153	0.0829***	-0.0016	-0.0038
	(0.0132)	(0.0266)	(0.0149)	(0.0283)
Ethnicity (1 = white)	0.0170**	0.0089	-0.0023	-0.0056
	(0.0085)	(0.0166)	(0.0106)	(0.0207)
Urban	-0.0088	-0.1027***	0.0034	0.0069
	(0.0126)	(0.0249)	(0.0141)	(0.0254)
IMD Quintile (1 = deprived)	0.0880**	0.1039	-0.0013	-0.0064
	(0.0418)	(0.0838)	(0.0496)	(0.0960)
Occupation: Manager	0.0065	0.0202	-0.0017	-0.0085
	(0.0142)	(0.0284)	(0.0165)	(0.0316)
Education: A-Level	0.0183	-0.0047	-0.0023	-0.0040
	(0.0149)	(0.0297)	(0.0175)	(0.0336)
Home Owner	0.0419***	-0.0038	0.0024	0.0043
	(0.0130)	(0.0259)	(0.0152)	(0.0289)

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

A.2.3 Triangular Kernels

We run each regression discontinuity design as a locally-weighed regression, using triangular kernels to give higher weights to observations closer to the threshold. These weights are calculated as:

$$w_i^{TRI} = h - |Z_i|$$

The highest weights are at the threshold ($Z = 0$) and decrease linearly as the bound of the bandwidth, h is reached. These are multiplied by the inverse probability of treatment weights to provide the final weights ($w_i = w_i^{IPT} . w_i^{TRI}$) for the weighted regressions run:

$$\sqrt{w_i}\, \boldsymbol{y} = \sqrt{w_i}\, (\beta_0 + \boldsymbol{D}\beta_1 + \boldsymbol{Z}\beta_2 + (\boldsymbol{D} \times \boldsymbol{Z})\beta_3 + \boldsymbol{u}) \qquad for\ |\boldsymbol{Z}| \le h \qquad (12)$$

A.3   Baseline Characteristics

Table A4 summarises the baseline characteristics of the control group, by cotinine quintile. Smoking variables are strongly associated with cotinine, with higher numbers of smokers, higher intensity of smoking and higher exposure to smoke at higher cotinine quintiles. Those with lower levels of cotinine are older, more likely to be female, married and retired, to live in rural areas and have more children. Higher socioeconomic status individuals have lower levels of cotinine, for a range of variables: IMD, income, occupation, education and home ownership. Additionally, those with higher cotinine levels have lower self-assessed health, more limiting longstanding illnesses and they drink more heavily.

Table A4: Descriptive Statistics by Cotinine Quintile

| | Cotinine Quintile | | | | | Summary |
| | Q1 | Q2 | Q3 | Q4 | Q5 | Mean |
	Mean	Mean	Mean	Mean	Mean	Mean
Cotinine Levels						
Cotinine	0.00	0.14	0.38	7.61	338.18	69.13
Log Cotinine	0.00	0.13	0.32	1.37	5.68	1.47
Smoking						
Smoker	0.01	0.00	0.00	0.12	0.93	0.21
Packs Smoked Per Day	0.00	0.00	0.00	0.14	2.40	0.51
Exposed to Smoke	0.35	0.33	0.44	0.60	0.72	0.48
Hours Exposed to Smoke	1.22	1.18	2.95	7.65	12.37	4.89
Ever Smoked	0.45	0.49	0.54	0.61	0.99	0.61
Smoke in Pubs	0.00	0.00	0.00	0.06	0.28	0.07
Exposed in Pubs	0.16	0.16	0.25	0.38	0.38	0.26
Demographics						
Age	52.18	52.35	50.11	48.23	44.80	49.70
Sex (1 = female)	0.54	0.56	0.54	0.52	0.50	0.53
Married Couple	0.67	0.65	0.65	0.54	0.42	0.59
Number of Children	0.46	0.37	0.47	0.40	0.54	0.44
Retired	0.29	0.31	0.28	0.25	0.11	0.25
Ethnicity (1 = white)	0.90	0.91	0.89	0.90	0.92	0.91
Urban	0.76	0.72	0.71	0.80	0.84	0.77
Socioeconomic Status						
IMD Quintile (1 = deprived)	3.47	3.44	3.25	2.94	2.62	3.16
Log Equivalised Income	10.14	10.19	10.17	10.01	9.79	10.06
Occupation: Manager	0.42	0.41	0.38	0.31	0.22	0.35
Education: A-Level	0.50	0.49	0.49	0.44	0.33	0.45
Home Owner	0.84	0.78	0.78	0.73	0.54	0.74
Health Variables						
Self Assessed Health (5 = v. good)	4.09	4.18	4.08	3.89	3.68	3.99
Limiting Longstanding Illness	0.27	0.23	0.25	0.26	0.33	0.27
Alcohol Units: Heaviest Day	5.34	5.42	6.88	7.81	9.49	6.89

A.4 Average Treatment Effects

Table A5 shows the results from the regression discontinuity design. The average treatment effect of the ban on non-smokers is large and significant, at -0.128, whilst there is no significant effect for smokers. The constant shows the expected level of log cotinine exposure at the time of the ban, for the control group: 0.399 for non-smokers and 5.343 for smokers.

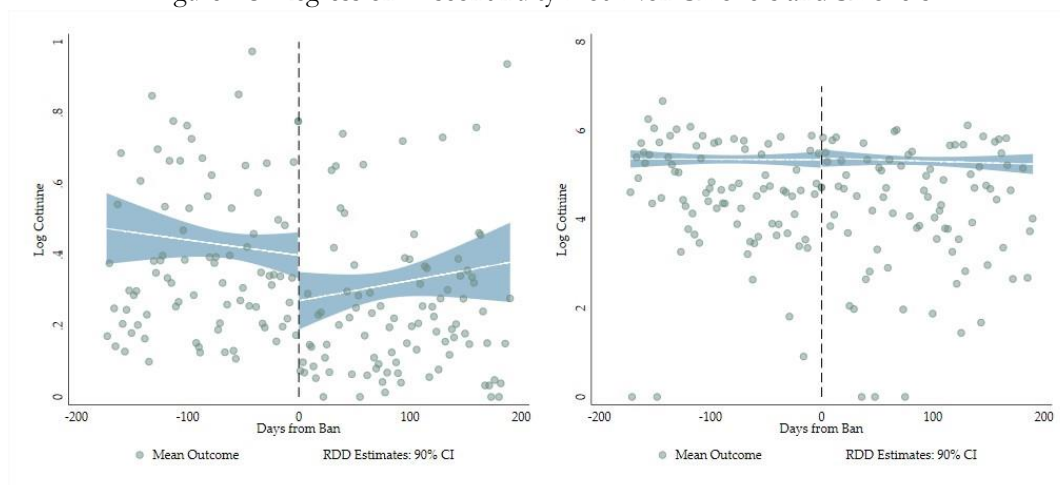Table A5: Log Cotinine: Regression Discontinuity

	(1)	(2)
	Non-Smokers	**Smokers**
	Coef./S.E.	Coef./S.E.
Treatment	-0.1279**	0.0385
	(0.0632)	(0.1662)
Time	-0.0004	-0.0001
	(0.0005)	(0.0012)
Treatment X Time	0.0010	-0.0006
	(0.0008)	(0.0018)
Constant	0.3987***	5.3434***
	(0.0395)	(0.1089)
N	2952	766
R-Squared	0.0062	0.005

[*] $p < 0.10$, [**] $p < 0.05$, [***] $p < 0.01$

Figure A3 shows the regression discontinuity plot for smokers and non-smokers. This shows the (weighted) mean log cotinine (averaged across two days) plotted against the number of days from the ban. The line plot shows the predicted point estimates from the regression of Table A5, whilst the area plot shows the 90% confidence intervals. We observe a discontinuity at the time of the ban for non-smokers, but not smokers. These are the average treatment effects observed in Table A5.

This figure is indicative of the quantile treatment effects we observe in Figure 3: many observations at the lower values of log cotinine move the small distance towards zero, whilst those higher values of log cotinine become much less frequent following the ban.

Figure A3: Regression Discontinuity Plot: Non-Smokers and Smokers

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A.5   Sensitivity Analysis

Figure A4 shows a forest plot of sensitivity analysis for the estimated average treatment effects. The baseline point estimate of Table 3 is shown at the top, with 90% confidence intervals, for non-smokers and smokers. The estimates below show how sensitive these results are to changes in the functional form of the regression discontinuity, the size of the bandwidth, the weights used and to the addition of controls.

Figure A4: Sensitivity Forest Plot: Log Cotinine, Non-Smokers and Smokers
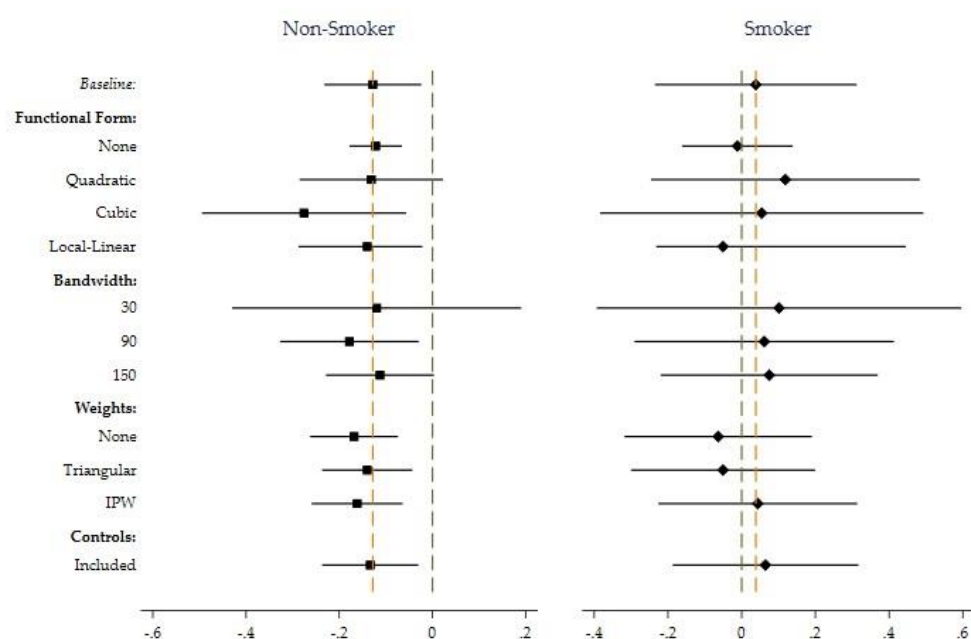

Results show that the analysis appears robust to each of these sensitivity checks. For non-smokers, the effects are never significantly different from the baseline results (dotted orange line) and (with exception to the use of a small bandwidth and quadratic functional form) the estimated effects are always significant, at the 90% level. For smokers, the effects are never significantly different from the baseline effect nor are they significantly different from zero, and indeed appear to hover around a zero effect.

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A.6 Predicted Conditional Potential Outcomes

Figure A5 plots the predicted potential outcomes conditional on observable characteristics. This shows the expected levels of log cotinine at the threshold, for control (red) and treated (blue) groups. These results should be used to supplement the estimated treatment effects in Figure 2; the treatment effects are the differences between these potential outcomes.

Figure A5: Forest Plot of Potential Outcomes of Log Cotinine: Non-Smokers and Smokers
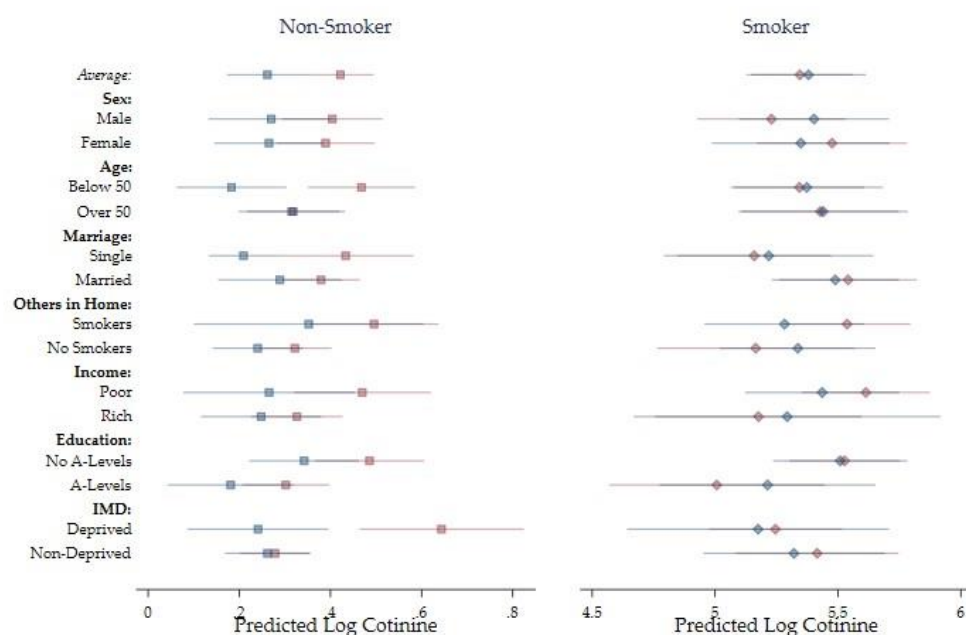
Non-smokers are found to have both significant differences between control and treated potential outcomes (i.e. significant treatment effects) and are found to have differences in exposure levels between subgroups. After the ban the differences between those subgroups that are different before, is much reduced. For smokers, there are no significant differences between potential outcomes; neither between control and treated, nor between subgroups.

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