DISCUSSION PAPER SERIES

IZA DP No. 6688

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June 2012

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zur Zukunft der Arbeit
Institute for the Study
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ABSTRACT

Taxes, Cigarette Consumption, and Smoking Intensity: Reply*

This paper shows that smoking intensity, i.e. the amount of nicotine extracted per cigarette smoked, responds to changes in excise taxes and tobacco prices. We exploit data covering the period 1988 to 2006 across many US states. Moreover, we provide new evidence on the importance of cotinine measures in explaining long-run smoking behavior and we investigate the sensitivity of smoking cessation to changes in excise taxes and their interaction with smoking intensity.

JEL Classification: D12, H25, I12

Keywords: tobacco, public health, compensatory behavior, excise taxes

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* We are grateful to David Jacobs and Jared Reid for comments.

In this reply, we make a number of contributions to the literature on smoking behavior and tobacco control. We show that the intensity of smoking, defined as the ratio of cotinine levels to the number of cigarettes smoked, does respond to changes in excise taxes as previously found by Adda and Cornaglia (2006) (AC hereafter). We do so by using a data set that spans from 1988 to 2006, allowing for more variations in taxes than in AC (2006), and in Abevaya and Puzzello 2010 (AP hereafter) who considered the period 1988-1994.

We also show that smoking intensity responds to price changes over this period, and that consistent estimates require the use of instrumental variables because of endogeneity issues. We show that OLS estimates are biased towards finding no effects. We find that the tax elasticity of smoking intensity is significantly different from zero and equal to 0.07 and that the price elasticity is higher, at around one. We also find considerable heterogeneity in the response to tax increases across different groups, and notably across different race groups.

We then investigate whether biomarkers such as cotinine measures are informative of long-run outcomes. We provide supporting evidence using novel panel data which follows smokers over 15 years. We show that cotinine levels are a strong predictor of smoking cessation, over and above the number of cigarette smoked. We finally use this data to shed further light on dynamic selection, and its potential to bias OLS regressions of smoking intensity on changes in prices and taxes.

We first present in Section 1 new evidence of compensatory behavior in response to tax changes. In Section 2, we present effects of prices on smoking intensity. Section 3 shows that cotinine measures are significant predictors of long-run outcomes. Finally, Section 4 investigates the potential for dynamic bias.

I. Smoking Intensity and Taxes

AP (2010) expand the original data set used by AC (2006) to cover more states during the same period covered by the NHANES III data set (1988-1994). A main difference between the two

datasets is the inclusion of tobacco states. These states are characterized by higher cigarette consumption, lower taxes, and little variation in excise taxes over that period. Essentially, when including these states, the effect of taxes become much less precise and a number of estimated elasticities become insignificant.

AP (2010) rightly point out that the standard errors should be clustered at state level only, rather than at state times year level. While AC (2006) do not cluster at state level, Adda and Cornaglia (2010) – AC (2010) hereafter - rely on such inference.

AP (2010) observe that one needs sufficient variation in taxes across state and time to be able to identify the parameter of interest. This is certainly the case, but in a context of regressions which include state fixed effects, it is important to expand the data to add more time variation. The NHANES data have expanded over the years and more waves are available for analysis, for a period which has seen more variation in prices and excise taxes than the early nineties. We supplement the NHANES III data set with later waves between 1999 and 2006. This data have been used by AC (2010) to investigate the effect of smoking bans and excise taxes on non-smokers. We use this data set in this reply to analyse the effect of taxes on smoking intensity. For a description of the data set, we refer the reader to AC (2010). Adding these additional years improves the analysis greatly as there has been lots of variation in taxes between 1988 and 2006, and allows, as pointed out by AP (2010) to better identify the parameter of interst.

AC (2006) provide results for various specifications, including conditioning on onset of smoking or cotinine levels, to address the issue of dynamic selection. Here we present the regressions of the baseline specification, for the subset of smokers who started smoking before age 17. We return to the dynamic selection issue in Section IV.

We first address the issue of using sample weights in regression involving NHANES data. AP (2010) follow DuMouchel and Duncan (1983) who show that differences between coefficients in weighted and unweighted regressions is a sign of mispecification. As pointed out in DuMouchel and Duncan (1983), this is however only true when weights are derived from exogenous variables. The weighting scheme in NHANES is more complex. The weights are a function of demographic variables, because the survey oversampled certain categories such as age groups or

racial groups. In addition, the weights were constructed to take into account non-participation, especially for the medical exam from where the cotinine measure is taken. Hence, the weights are also a function of the endogenous variable. This is why the NCHS strongly recommends to use weights in the analysis (we refer the reader to the NHANES guidelines (NCHS, 1996) for a detailed description). AC (2006) overlooked this issue, but AC (2010) use them in their regressions.

Before analyzing the behavioral effect of taxes on smoking, we show empirical evidence that the sample weights are indeed correlated with the outcome variable, over and above demographic characteristics. Table 1 displays the correlation between the outcome variable of our regressions and the weights (with p-values in parenthesis). We distinguish between three outcome variables, the log number of cigarettes smoked per day, log cotinine concentration, and the log smoking intensity. The correlation between these three outcome variables and the sample weights varies between 0.1 and 0.27 in the sample 1988-1994, and between 0.1and 0.22 in the extended sample (1988-2006). Once we control for a set of observable characteristics, which include age, sex, race, education, region of residence and year of examination, the correlation is closer to zero but statistically different from zero. The corelation ranges between 0.05 to 0.15 in the sample 1988-1994 and from -0.02 to 0.07 in the extended sample. The results show that the sample weights are indeed correlated with the endogenous variable, even when a set of demographic controls are included. The problem is particularly severe in the sample used by AP, but less so with the extended data set we use in this note. In the presence of endogenous stratification, the assumptions in DuMouchel and Duncam (1983) are violated. As discussed in Maddala (1983), the use of weights is then recommended. When using data from NHANES, we present the results with and without weights for comparison with previous results.

We now turn to the behavioral effect of taxes. We first note that there is a clear difference in focus between AC (2006) and AP (2010). AC are interested in the existence of compensatory behavior, which amounts to testing whether the ratio of cotinine to cigarettes is significantly related to taxes, in other words, whether the tax elasticity of cigarettes is larger than the tax

elasticity of cotinine. The fact that smoking intensity responds to public policies is an important finding for the design of health policies. As argued in AC, this has also consequences on the estimation of popular models such as the rational addiction model. AP, on the other hand, mainly test whether the elasticity of cotinine or cigarettes with respect to taxes or prices is significantly different from zero. It is of course possible that both the elasticities of cotinine and of cigarettes smoked are insignificant - perhaps because of lack of variability in the data - and that the elasticity of the intensity of smoking is significantly different from zero.

Table 2 displays the results of OLS regressions of smoking intensity (defined as the ratio of cotinine and the number of cigarettes smoked), the number of cigarettes smoked and cotinine levels, on log state taxes as well as demographic variables, state indicators and year indicators. All left hand side variables are log transformations for ease of interpretation. We first present regressions as in AP (2010), using the sample between 1988 and 1994, and our extended sample to 2006. The table has two panels, where we use unweighted and weighted regressions. NHANES III and the subsequent NHANES surveys differ in the amount of demographic variables which were recorded. NHANES III has a wider set of explanatory variables. To make our results consistent across the table, we control for a set of variables which are present throught out all waves. These controls are age, sex, race and education levels. AC (2006) controlled also for occupation, household size and passive smoking, and in some cases for the time of examination and height. However, there is little reason why these additional controls should be correlated with state taxes, especially when the regressions include state and year fixed effects. Indeed, the results in AC (2006) show that the coefficients of interest are not significantly different when including a fuller list of controls.

Table 2, column 1, confirms the results obtained by AP (2010). Unweighted regressions for the period 1988-1994 show that a one percent increase in taxes leads to a 6 percent increase in smoking intensity, but this elasticity is not statistically significant. However, using the extended sample (column 2), taxes are significantly associated with smoking intensity. In particular, a one percent increase in taxes leads to an increase in smoking intensity of 7 percent. This number is

smaller than the one obtained in the NHANES sample in AC (2006), but in line with the results in the same paper when using data from NHANES 1999-2000. The second panel uses the weights provided in the NHANES data set. The use of weights appears to be of particular importance in NHANES III, as the elasticity of smoking intensity is larger than the unweighted one, equal to 0.23 and significantly different from zero. Finally, on the larger sample, the use of weights appears to be less important, as we find a very similar tax elasticity of smoking intensity, equal to about 7 percent. Note that we also find a significant tax elasticity for the number of cigarettes smoked (-9 percent), but cotinine levels do not respond much to changes in taxes.

As discussed in AC (2006), an OLS regression could overstate the effect of taxes on smoking intensity because of a dynamic selection bias, where light smokers could be more responsive to changes in taxes and quit smoking at a higher rate. As a robustness check AC (2006) used a sub-sample of smokers who started early (before age 17) and who are therefore less likely to quit. We repeat this analysis in Table 3. Column 1 uses the sample used in AP (2010). None of the elasticities using unweighted regressions are significantly different from zero. With weights, we find evidence of a significant effect of taxes on smoking intensity. With the larger data set (column 2), the results are in line with Table 1 and the original results of AC (2006). A 1 percent increase in excise taxes increases smoking intensity by about 0.1 percent (this is the case in both the weighted and unweighted regressions). The increase in smoking intensity is the outcome of a significant decrease in the number of cigarette smoked, and no effect of taxes on cotinine concentration. These results suggest that the findings on the whole sample of smokers is not due to a change in the composition of smokers. We present further evidence on this issue in Section 4.

Table 4 presents evidence of heterogenous effects. We estimate the tax elasticity of smoking intensity for various subgroups of smokers. We find evidence that compensatory behaviors are more important for men than women, and in particular for african-american than for whites. AC (2006) found evidence that this racial group smoke cigarettes more intensively than other racial groups. It appears that they are also more reactive to tax changes. There is not much evidence of

a change in the elasticity by age groups. None are significantly different from zero at the 5 percentage confidence level. One reason is that sample sizes are becoming smaller when one stratifies the sample in such a way. It therefore shows the limits of such an exercise.
Finally, smokers with income below the median income (defined as $ 26,500 annual income in 2000 dollars) have a higher elasticity than those above the median. The elasticities vary from 0.1 to 0.05. This result is in line with the predictions of the model in AC (2006).

II. Smoking Intensity and Prices

AC (2006) did not provide price elasticities for two reasons. First, there is an issue with endogeneity to which we come back below. Second, from a public health point of view, states and governement can only manipulate prices through taxes, so the tax elasticities are important to shape policy.

AP (2010) write that an OLS regression can recover consistent estimates of price elasticities, arguing that endogeneity is not a problem with micro data. We dispute this fact. First, the regression is indeed using micro data, but the real variation is at the state times year level, as argued by AP (2010) in their discussion about standard errors. Second, in the presence of aggregate (state) shock to demand, it is likely that tobacco companies change their prices to respond to such shocks. The fact that the influence of individuals is too small to affect prices is not the issue. In the presence of endogeneity, an OLS regression would tend to produce coefficients which are biased towards zero. A positive demand shock would induce an endogenous increase in prices, which would counteract the causal effect of prices on demand. To solve the issue of endogeneity, we instrument prices with arguably exogenous tax shocks. Taxes are often changed to raise revenue and not to counter demand shocks.

In Table 5 we report estimates of price elasticities. The first column displays OLS results, without sample weights in the first panel, and with weights in the second. The OLS results are similar to those found in AP (2010), and consistent with the intuition detailed above. None of the

elasticities are significantly different from zero. The second column displays instrumental variable estimates. We also report the F-statistic for the first stage, which has a value of 57. This indicates that state excise taxes are a significant predictor of prices, over and above state and year indicators. The instrumental variable estimates show that smoking intensity responds to price changes. Without sample weights, we find that a one percent increase in prices leads to a 0.76 percent increase in smoking intensity, significant at the 6 percent level. When sample weights are used, we find a price elastiticity of smoking intensity of 1.05, significantly different from zero at the 5 percent confidence level. The table also displays an F test for the endogeneity of prices, which we carried on for the regressions involving smoking intensity. They show that the null of no endogeneity is strongly rejected, at a confidence level of 2 percent.

III. Long Run and Short Run Measures of Smoking

It has been argued (AP, 2010) that the number of cigarettes smoked per day is a long-run measure of smoking whereas cotinine levels are a short-run measure. It is not clear what justifies such a categorisation, especially when only cross-sectional data is at hand in NHANES. Following this stated opinion, AP (2010) dismiss the measure of smoking intensity constructed by AC (2006), that is, the ratio of cotinine extracted per cigarette smoked. We dispute this fact and present two arguments in favor of considering cotinine measures a long term measure of smoking (and health).

The first point is already developed in AC (2006). They present evidence of long-run health outcomes which are linked to the way cigarettes are smoked. We refer the reader to the discussion about lung cancer rates by race in the US, in section II.B in AC (2006).

We provide further evidence using novel data from the Coronary Artery Risk Development in Young Adults (CARDIA) study. These data allow us to follow the same smokers over time, in contrast with the cross-sectional aspect of the NHANES dataset.

The data were collected between 1985 and 2001 in four locations in the US (Alabama, Ilinois, Minesotta and California). A group of 5115 individuals aged 18-30, were followed over 15 years, which provides ample longitudinal variation as opposed to the NHANES. The survey asks a number of questions on smoking behavior at each wave, whether the individual is still smoking at the time, and if not, the age at which smoking cessation took place. We define age at smoking cessation as the age of the first recorded quit, whether the individual relapses after that period or not. We therefore only consider one smoking spell per smoker. In addition, the survey records the number of cigarettes smoked in all waves, cotinine levels in the first wave as well as the age of smoking initiation. Table 6 presents key desciptives of the dataset, and we refer the reader to Friedman et al (1988) for a detailed description of this data set.

We measure the propensity to quit as a function of both the number of cigarettes smoked and the cotinine levels at baseline in 1985. We estimate a duration model, using a Cox proportional form, where we stratify by geographical location, sex, race, education and age at smoking onset. The data provide us with information on 11,073 observations following 1,459 smokers until they quit or are right censored. We normalised both the number of cigarettes and cotinine levels to have mean zero and variance one, so that we can interpret the coefficients in a straightforward way.

Table 7 displays the results. We find that the number of cigarettes smoked per day is not significant at the 5 percent level, but interestingly, cotinine levels are a highly significant predictor of quitting behavior (column 1). Controling for the number of cigarettes, a one standard deviation increase in cotinine levels at baseline decreases the likelihood to quit by 49 percent. Similarly, holding cotinine levels constant, a one standard deviation increase in cigarettes is associated with a decrease of 2 percent in the likelihood of quitting. Hence the statement that cotinine is only a short-run measure does not appear to be grounded in facts as it significantly predicts quitting over a period of 15 years.

We also explored heterogenous effects. Table 7 columns labelled 2 and 3 distinguish the effect of cotinine and the number of cigarettes on quitting behavior by sex. Men and women differ in their propensity to quit. In particular, cotinine levels play a bigger role for men than for women.

IV. Dynamic Selection

An important issue refers to dynamic selection, whereby smokers who quit following an increase in taxes may come disproportionally from a low smoking intensity group. This has been raised by both AC (2006) and by AP (2010). If this is the case, an OLS regression of smoking intensity on excise taxes may find a spurious positive effect due to a change in composition in the pool of smokers. AC (2006) investigate this point in two ways. They first include in their sample individuals who are less likely to quit, for instance individuals who started smoking at a young age to explore the effect of taxes on smoking intensity. Second, they use an econometric technique developed by Manski (1994), to use worst case bounds. However, with only cross-sectional data this point could not be fully addressed as the bounds tend to be large. Using the panel data from CARDIA, we now present new evidence on this issue, which helps to interpret the results in AC (2006), and in Table 1 in this article.

We estimate the effect of tax changes on quitting behavior and whether the effects vary with smoking intensity as measured at baseline by the ratio of cigarettes to cotinine levels. Using geographical information on the center of examination, we merge information on excise taxes to the original CARDIA data. We therefore have variation on taxes across years and geographical location, which we exploit to estimate a model of the duration to quitting.

Table 8 presents the effect of taxes on quitting, for the whole sample, as well as for smokers divided into two groups, below or over the median smoking intensity measured at baseline in 1985. The first panel displays the results, controlling for age, sex, race, education, state of residence and a quadratic time trend. We find a significant effect of taxes on quitting behavior. A doubling of the excise tax increases the likelihood of quitting by 63 percent. Interestingly, the point estimate of this effect is larger for individuals with a high smoking intensity than for a low smoking intensity. The former have a likelihood of quitting of 55 percent and the latter of 93 percent. However, the estimation is not precise enough to conclude that the difference is

significant. The second panel includes year fixed effects rather than a quadratic trend. We get qualitatively similar results, although with much less precision.

The results suggests that the dynamic bias may not be of such importance, and that OLS regressions do not overstate the effect of taxes on smoking intensity. Understanding why individuals with higher smoking intensity may be more likely to quit when taxes increase is an interesting question that we leave to future research.

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Table 1: Correlation between Sample Weights and Smoking Behavior

	NHANES 1988-1994		NHANES 1988-2006	
Log no. of cigarettes	0.27**	0.15 **	0.22**	0.07**
	(0.00)	(0.00)	(0.00)	(0.00)
Log cotinine	0.10**	0.11**	0.10**	0.05**
	(0.00)	(0.00)	(0.00)	(0.00)
Log smoking intensity	-0.24**	-0.05**	-0.13**	-0.02*
	(0.00)	(0.00)	(0.00)	(0.07)
No. of observations	3,514	3,514	6,318	6,318
Controls	No	Yes	No	Yes
Notes: p-values in parenthesis. Controls include age, age square, sex, race, education, state of residence and year indicators. *,** significant at the 10^{th}, 5^{th} percentage level.				

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

	(1)	(2)
	AP Sample 1988-1994	Expanded Sample 1988-2006
Unweighted regressions		
Elasticity smoking intensity	0.062	0.074**
	(0.055)	(0.035)
Elasticity no. of cigarettes	-0.001	-0.070
	(0.088)	(0.051)
Elasticity, cotinine	0.060	0.005
	(0.057)	(0.030)
Weighted regressions		
Elasticity smoking intensity	0.227**	0.0691**
	(0.089)	(0.029)
Elasticity no. of cigarettes	-0.059	-0.089**
	(0.132)	(0.044)
Elasticity, cotinine	0.168*	-0.020
	(0.095)	(0.038)
Number of observations	3514	6318

Notes: All regressions control for age, sex, race, education, year and state effects. Robust standard errors clustered at state level. *,** significant at the 10th, 5th percentage level.

Table 3: Tax Elasticity of Smoking Intensity, Number of Cigarettes and of Cotinine. Early Starters.

	(1)	(2)
	AP Sample 1988-1994	Expanded Sample 1988-2006
Unweighted Regressions		
Elasticity smoking intensity	0.081	0.097**
	(0.067)	(0.037)
Elasticity no. of cigarettes	-0.022	-0.105**
	(0.093)	(0.050)
Elasticity, cotinine	0.059	-0.009
	(0.086)	(0.031)
Weighted Regressions		
Elasticity smoking intensity	0.205**	0.109**
	(0.077)	(0.033)
Elasticity no. of cigarettes	0.084	-0.126**
	(0.134)	(0.060)
Elasticity, cotinine	0.280**	-0.016
	(0.118)	(0.046)
Number of observations	2060	3611

Notes: All regressions control for age, sex, race, education, year and state effects. The regressions exclude smokers who started after age 17. Robust standard errors clustered at state level. *,** significant at the 10^{th}, 5^{th} percentage level.

Table 4: Smoking Intensity - Tax Elasticities for different Subsamples

	Coeff.	Std. Err.	Sample size
Full sample, 1988-2006	0.069**	(0.029)	6,318
Men	0.105**	(0.039)	3,423
Women	0.049	(0.045)	2,895
White	0.053	(0.039)	3,690
Black	0.193**	(0.075)	1,943
Ages 17-29	0.085	(0.06)	1,464
Ages 30-44	0.075*	(0.041)	2,167
Ages 45+	0.062	(0.043)	2,687
Below median income	0.110**	(0.055)	2,919
Above median income	0.058*	(0.034)	3,399

Notes: Regressions control for age, sex, race, education, state of residence and year of interview. All regressions are weighted using NHANES sample weights. Standard errors in parenthesis are clustered by state. Median 2000 income is $ 26,500. . *,** significant at the 10^{th}, 5^{th} percentage level.

Table 5: Price Elasticity of Smoking Intensity, Number of Cigarettes and Cotinine

	(1)	(2)
	OLS	IV
Unweighted regressions		
Elasticity smoking intensity	0.062	0.761*
	(0.336)	(0.418)
Elasticity no. of cigarettes	0.101	-0.245
	(0.501)	(0.577)
Elasticity, cotinine	0.164	0.517
	(0.377)	(0.418)
F statistic first stage; (pval)	-	57.12; (0.00)
F test of endogeneity; (pval)	-	10.99; (0.02)
Weighted regressions		
Elasticity smoking intensity	0.393	1.056**
	(0.397)	(0.455)
Elasticity no. of cigarettes	-0.514	-0.891*
	(0.313)	(0.492)
Elasticity, cotinine	-0.121	0.165
	(0.365)	(0.407)
Number of observations	4870	4870
F statistic first stage; (pval)	-	57.12; (0.00)
F test of endogeneity; (pval)	-	10.99; (0.002)

Notes: Sample: NHANES 1988-2006. All regressions control for age, sex, race, education, year and state effects. Prices are instrumented with state tax levels. Robust standard errors clustered at state level. *,** significant at the 10^{th}, 5^{th} percentage level.

Table 6: Descriptive Statistics, CARDIA Sample

	(1)	(2)	(3)
	All individuals at baseline (year 0)	Year 15	Year 15 individuals still smoking
Number of observations	1546	923	633
Smoking prevalence, %	100	68.5	100
Mean number of cigarettes	13.1 (9.1)	9.2 (10.0)	13.4 (9.5)
Mean number cigarettes at baseline	13.1 (9.1)	13.4 (9.5)	13.8 (8.9)
Mean cotinine level at baseline ng/ml	224.4 (158.4)	222.2 (153.8)	241.1 (152.1)
Male, %	47.2	45.3	47.0
White, %	57.1	52.9	60.5
African-American, %	42.8	47.1	39.5
Mean age	25.0 (3.6)	40.0 (3.6)	40.0 (3.6)
Mean years of schooling	12.8 (2.0)	13.0 (2.0)	12.8 (1.9)
Notes: Standard deviations in parenthesis where appropriate.			

Table 7: Hazard Rates for Quitting Smoking

	(1)	(2)	(3)
	All	Men	Women
Number of cigarettes	-0.021	0.183	-0.153
	(0.077)	(0.115)	(0.11)
Cotinine levels	-0.493**	-0.598**	-0.445**
	(0.093)	(0.162)	(0.114)
Number of observations	1459	691	768
Time at risk	11073	5418	5655

Notes: Cox proportional regression. Age is the analysis time. Stratified by sex (first column), race, education, state, year and age at smoking onset. CARDIA data. Number of cigarettes and cotininces levels have been normalised to mean zero and variance one for ease of interpretation.

Table 8: Effect of State Tax on Quitting, by Level of Smoking Intensity

	(1)	(2)	(3)
	All	Intensity<median	Intensity>median
Controlling for year and year square			
Log tax	0.629**	0.557**	0.930**
	(0.230)	(0.295)	(0.409)
Controlling for year fixed effects			
Log tax	0.380*	0.231	0.693
	(0.239)	(0.311)	(0.434)
Number of observations	1477	857	616
Time at risk	11185	6475	4698

Notes: Cox proportional regression, stratified by sex, race, education, state and age at smoking onset. Regressions include year either through a quadratic specification or through year fixed effects. CARDIA data.