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Intergenerational transmission of nicotine within families: have e-cigarettes had an impact?

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Abstract

Using an objective biomarker of both active and passive smoking (saliva cotinine), we estimate a Galtonian regression of nicotine transmission and test whether the use of new nicotine delivery products (i.e. e-cigarettes and other NDP) by parents reduces nicotine transmission to children through passive smoking. To test the latter effect, we use a variety of strategies encompassing the inclusion of interaction terms between cotinine levels and NDP utilization in the Galtonian regression, an IV strategy to deal with potential endogeneity of NDP utilization and a before-after analysis which exploits the spread in the use of e-cigarettes in England from 2010. Using matched parent-child data from the Health Survey for England from 2002 to 2014, we find evidence of a strong intergenerational transmission of nicotine and that transmission is twice the size for mothers compared to fathers. Moreover, all of our empirical strategies lead us to conclude that the introduction of NDP has lowered intergenerational transmission of nicotine to 70-80% of the level without NDP. Following the externality argument, these results suggest that lower taxation of these devices is justified.

Keywords: Nicotine; passive smoking; intergenerational mobility; Galtonian regression; electronic cigarettes; tobacco taxes.

JEL codes: I12; D62

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1. Introduction

A large body of literature in the social sciences has provided evidence that many outcomes are strongly related to those of an individual's parents. This has been shown in a variety of ways and for a variety of outcomes, that includes family income, individual earnings, social class, occupational status and education (see Blanden, 2013 for a recent review). This high intergenerational correlation is likely to be detrimental for society, both on equity grounds, for the achievement of equality of opportunity, and on efficiency grounds, as a high intergenerational correlation might dampen the incentives for offspring to exert effort to improve their own outcomes.

Somewhat related, there is extensive evidence that early life conditions with reference to childhood health and general circumstances contribute to shape later life opportunities for a wide range of outcomes, such as education, health, labour market outcomes and social status. Case, Lubotsky and Paxson (2002) found that children born into poorer families in the US arrive into adulthood not only in poorer health but also with lower educational attainment, in part, attributable to their poorer health. A similar result is found by Currie and Stabile (2004) with Canadian data. Case, Fertig and Paxson (2005) found a lasting effect of childhood health and economic circumstances on adult health, employment and socioeconomic status. A number of studies have found a strong link between childhood health and labour market outcomes (see Currie and Madrian (1999) for a review). A strand of this literature shows a significant impact of low birthweight on educational attainment and labour market outcomes (see Currie and Hyson (1999) for a review). Some authors have even argued that the early life conditions are the leading explanation of the well-known socio-economic gradient in health observed in adulthood (Adler et al., 1994).

In this paper, we focus on one aspect of the transmission from parents to children which contributes to define early life conditions in a significant way but which has received less attention by economists: the intergenerational transmission of nicotine within families through exposure to passive smoking. This aspect merits attention in the economic debate on at least two grounds. First, it has implications for social welfare and childrens' welfare in particular. Exposure to passive smoking is immediately dangerous for children's health, both with respect to in-utero exposure which may lead to long-term developmental disabilities (Hay, 1991) but also for the development of respiratory tract infections and cases of aggravated asthma among children (Environmental Protection Agency, 1994). Moreover, the exposure to passive smoking during childhood might lead to a future of nicotine addiction, with its associated health risks (Warner et al., 1995). This is a relevant topic as cigarettes cause fully a third of deaths during middle age. They are the leading cause of lung cancer and chronic obstructive pulmonary disease mortality, as well as a major cause of cardiovascular death and they lead all other causes of death in virtually all industrialized nations (Chaloupka and Warner, 2000). A second ground for economic relevance is the fact that exposure to passive smoking among children is a clear example of an externality which is an important rationale for taxation of cigarettes and other tobacco products. While there is some debate on whether to include family members in the computation of the external costs of smoking, it is clear that both health damage to children and their possible future of nicotine addiction are likely to generate costs that spill over into the broader society (Chaloupka and Warner, 2000).

To the best of our knowledge, only a few papers in the economics literature deal with passive smoking. Adda and Cornaglia (2006, 2010) use cotinine levels as a measure of passive smoking for a sample of US adults to explore the effect of tobacco taxes and smoking bans in public places. They

find that taxes lead adults to extract more nicotine per cigarette (Adda and Cornaglia, 2006) and that smoking bans in recreational public places may lead to increased exposure to passive smoke for non-smokers in private places such as the home (Adda and Cornaglia, 2010). More directly relevant to this study is the paper by Frijters et al. (2011) which uses the Health Survey for England from 1997 to 2006 to document the main risk factors that determine children's exposure to passive smoke measured through saliva cotinine and provides estimates of the effect of this exposure on child health. They find that both parental and child carer smoking behaviour are major risk factors in determining children's exposure to passive smoke.

In this paper, we build on Frijters et al. (2011) and contribute to this topic in two ways. First, we quantify the scale of transmission of nicotine from parents to children in England using saliva cotinine (the major metabolite of nicotine) as an objective biomarker for both active and passive smoking. The key advantage of using this marker is that of having a measurement of smoking which is objective, and much less prone to the measurement errors often seen with self-reported smoking behaviour. In contrast to Frijters et al. (2011) who rely on self-reported smoking behaviour by parents, we use cotinine to quantify both exposure to passive smoking and to measure objective nicotine consumption by parents. This is consistent with the idea of measuring the intergenerational transmission of nicotine and it allows us to estimate a Galtonian style regression of nicotine transmission which has the advantage of providing a measure of intergenerational correlation which is directly suitable for comparisons across time and space. Second, we test whether the use of novel nicotine delivery products (i.e. e-cigarettes and other NDP) by parents reduces the nicotine transmission to children.

Electronic cigarettes (e-cigs) and other novel nicotine delivery products (NDP) represent one of the most important recent innovations in the tobacco market. E-cigs are battery-operated devices that aim to simulate combustible cigarettes, while other NDP encompass alternative methods to administer nicotine to the brain without the harms of combustion (i.e. chewing gum, nicotine patches). E-cigs are the newest and the most used nicotine delivery system. They don't contain tobacco but operate by heating nicotine and other chemicals into a vapour that is inhaled. Despite some side effects and some debate on their effectiveness to aid quitting, e-cigs are generally evaluated as much safer than smoking, a valid aid for quitting and able to reduce the risk of second-hand exposure (Public Health England, 2015).[1] Despite that, there is scarcity of evidence on the effects of NDP on intergenerational transmission of nicotine within families through passive smoking. Only recently Ballbè et al. (2014) found no substantial difference in nicotine transmission between traditional cigarettes and e-cigs on a sample of 54 individuals living in homes with smokers and e-cig smokers.

Intergenerational transmission of nicotine deserves further exploration as it is extremely relevant for the evaluation of the externalities deriving from NDP consumption and, thus, for the design of taxes on these devices. E-cigs and other NDP are currently taxed by 20 per cent Value Added Tax in Europe while the average taxation of cigarettes (including VAT and ad valorem excises at 1st July

[1] E-cigs have been found as effective, though not more, than nicotine patches for short-term cigarette cessation (Dockrell *et al.*, 2013; Etter and Bullen, 2011; Bullen *et al.*, 2013), and cartridge analyses find fewer toxins than are found in traditional cigarettes (Goniewicz et al., 2014). However, in a randomized trial 29% of e-cig users continued e-cigs at 6-months compared to only 8% of patch users (Bullen *et al.*, 2013), suggesting e-cig use might persist after other cessation methods. In addition, cartridges have been found to contain hazards, such as cytotoxic heavy metal and silicate particles (Williams and Talbot, 2011).

2016) is around 79 percent of their average retail price and close to 84-86 percent in many EU countries, i.e. Belgium, Estonia Finland, Ireland and the UK (European Commission, 2016). However, there is an ongoing debate around the taxation of e-cigs. In March 2016, European Finance Ministers meeting in Brussels agreed that this should be reconsidered and some EU country members explicitly "asked the European Commission to decide by 2017 whether to propose increasing taxation on e-cigarettes to achieve a closer convergence to tobacco taxes" (Council of the European Union, 2016). The taxation of e-cigs is a relevant issue for public finance as the constant increase in e-cig users opens important opportunities to raise tax revenues: in the UK, there are an estimated 2.6 million e-cigs users (ASH, 2016), while, in 2014, 12.6% of adults had ever tried an e-cig at least one time in the USA (Schoenborn and Gindi, 2015). Insights firm Nielsen found that the e-cigarette industry has become one of the fastest-growing supermarket products by volume and value in the UK, with a 50 per cent year on year increase to around 17.3 million units in 2015 (Forbes, 2016).

We estimate Galtonian style regressions of nicotine transmission by matching parent-child data on cotinine and socio-economic variables from the Health Survey for England (HSE) spanning between 2002 and 2014. To assess the effect of NDP on nicotine transmission, we follow several routes. First, we exploit the spread in the use of e-cigs in England from the beginning of 2010 (as illustrated in Figure 1 in Section 2.1). This coincided with the first official pronunciation in favour of its use among cigarette smokers by Action on Smoking and Health (a public health charity established by the Royal College of Physicians) in October 2009. This provides a natural experiment to assess the effect of NDP on the intergenerational transmission of nicotine in a before-after framework with controls for confounding variables. As a second test, we interact cotinine levels with self-reported NDP use (available only in the 2013 and 2014 waves of the HSE) in the Galtonian regression. The coefficient of this interaction terms indicates whether the transmission of nicotine inhaled through NDP is lower, other things being equal. Last, we deal with the potential endogeneity of NDP utilization by adopting a Lewbel-IV strategy and using medical advice to stop smoking received by parents as an instrument.

We find evidence of substantial transmission of nicotine from parents to children and that transmission is more than twice as large for mothers than for fathers. Moreover, all our empirical strategies lead us to conclude that nicotine transmission to children is lower when it is delivered through NDP. This has direct implications for the taxation of these new devices.

The rest of the paper is organized as follows. The next section presents the data and descriptive statistics. Section 3 discusses the empirical methodology. Section 4 presents the results of our empirical analysis. The final section summarizes and concludes.

2. Data

Our data come from the Health Survey for England (HSE). HSE is a repeated cross-sectional health interview survey of around 15,000 to 20,000 respondents conducted in England by the National Centre for Social Research. The survey started in 1991 and has been carried out annually since then. HSE includes adults aged 16 and over, and since 1995 has also included children aged 2-15. An interview with each eligible person in the household is followed by a nurse visit for those who agree to take part[2]. The interview includes a set of core questions, asked each year, on general health and psycho-social indicators, smoking, alcohol, demographic and socio-economic indicators, questions about use of health services and prescribed medicines. Biomarkers and health assessments are collected during nurse visits and include saliva samples which are used for the measurement of cotinine levels (see Section 2.1 for more details). During the nurse visits, the nurse asks the respondent for permission to carry out various types of measurements. Respondents are informed about the purpose and value of each test.

We matched child-parent data using waves from 2002 to 2014 of HSE. This time window allows us to have an updated and comparable picture across time of the intergenerational transmission of nicotine within families. Moreover, it allows us to have sufficient pre- and post- waves around 2010 to perform the before-and-after analysis of e-cigarettes discussed in the introduction. We discard the 2005, 2006 and 2012 waves as they have too few valid measurements of cotinine for both children and parents within the same family. This leads to a total sample of 8,654 non-missing observations. Children, aged below 15 years old, are our observational unit (see Section 3.1 for more details).

2.1 Variables and Descriptive Statistics

We use cotinine levels among children as the dependent variable for exposure to passive smoking and cotinine levels among parents as the main regressor of interest in the Galtonian regressions (see Section 3.1). Cotinine is the predominant metabolite of nicotine and it is an objective quantitative indicator of both active and passive smoking. Cotinine levels greater than or equal to 15 ng/ml are widely accepted as a marker of objective active smoking, while levels of cotinine below 15 ng/ml identify exposure to passive smoking with high sensitivity (Jarvis et al. 1987). In HSE, cotinine is detected through the analysis of saliva sample by a laboratory. Compared to other methods to detect cotinine (i.e. blood and urine), saliva samples are considered to be the best non-invasive procedure especially for the target of identifying low concentrations of cotinine consistent with exposure to passive smoking (Avila-Tang et al., 2013). Up to the 2013 wave of HSE, cotinine measurements among children (i.e. individuals below 15 years-old) are provided on a continuous scale, while in waves 2013 and 2014 cotinine measurements are released on an ordinal scale with three intervals (0; 0.01-1; 1-12) with a maximum range of 12 ng/ml to identify passive smoking. This is consistent with the revised optimal cotinine cut-points for passive smoking (Jarvis et al., 2008). The change in optimal cut-points (down from 15 to 12) is explained by the reduction in the prevalence of smoking over the last years, and optimal cut-points depend on the prevalence of smoking in the population under study in order to minimize the false positive rate (Cummings and

[2] The average agreement rate is quite high (close to 60%) and does not show a systematic pattern across socio-economic groups (see for instance, Carrieri and Jones, 2016a).

Richard, 1988)[3]. Consistent with these recommendations, we identify passive smoking with cotinine values below 15 ng/ml for the first waves (2002-2012) while we rely on the three-level cotinine variable bounded to 12 ng/ml for analysis based on 2013 and 2014 waves. The different scaling of the cotinine variable involves estimates of two separate regressions which lead to qualitatively comparable results (see Section 4.1).

We use demographic variables for the children (age and gender), equivalised household income of the family and the smoking status of grandparents (whether at least one grandparent smoked) reported by the parents as the main controls in our regressions. Household income includes total income of a household from all sources, after tax and other deductions, divided by the number of household members converted into equivalised adults. Self-reported current, past or intermittent use of e-cig and other NDP is used in the interaction model presented in Section 3.2.

A summary of both the dependent and independent variables used in our analysis along with the descriptive statistics are provided in Table 1. Table 1 shows that average cotinine scores in children were 0.89 over the years 2002-2012, consistent with some exposure to passive smoking. A direct comparison with the values arising in 2013-2014 is not possible as cotinine is expressed in three levels in these two waves. However, we find that around 40% of children in our sample have been exposed to passive smoking during the period 2013-2014.

TABLE 1. Descriptive Statistics

	2002-2012 Sample		**2013-2014 Sample**	
Variables	***Mean***	***St.dev***	***Mean***	***St.dev***
Cotinine Children	0.89	1.74	0.48[a]	0.68
Cotinine Father	41.86	143.66	18.70	81.36
Cotinine Mother	62.36	186.82	27.67	101.26
Cotinine Parents (M+F)	104.22	246.96	46.37	138.82
Household Income	22983.17	21937.46	27220.69	24931.72
Parental GP smoking (y/n)	0.37	0.62	0.22	0.41
Maternal GP smoking (y/n)	0.59	0.76	0.33	0.47
Age	9.31	3.01	9.20	3.12
Male	0.50	0.50	0.52	0.50
E-cig father			0.06	0.24
E-cig mother			0.07	0.25
Number of E-cig parents (M+F)			0.13[b]	0.38
Observations	7273		1381	

[a]Cotinine measured in three levels. 0= 62.35%; 0.01-1: 26.79%; 1-12:10.86%
[b] Number of e-cig parents: 0: 88.05%; 1: 10.50% 2: 1.45%

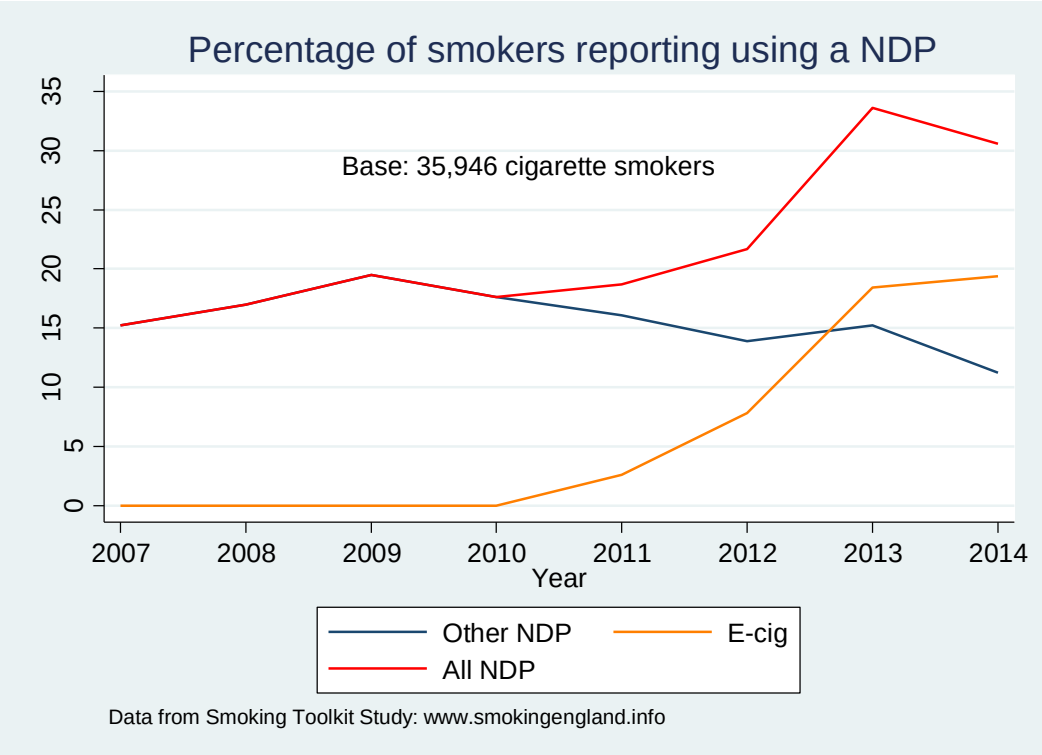
Table 1 shows a clear decline over time in cotinine levels of both parents. Average cotinine in fathers dropped from around 41.8 in 2002-2012 to 18.7 in 2013-2014, a reduction of around 55%. A similar drop is found for mothers (55.6%) and, consequently for the sum of cotinine for both

[3] The suggestion is that when the prevalence of smoking is low, the number of misclassifications will depend primarily on the false positive rate of the test. Thus the optimal cut-point should then be higher to minimize the false positive rate (see Jarvis, 2008 for more details).

parents (55.5%). These numbers are consistent with the reduction in the prevalence of smoking previously discussed. This is observed also in the reduction of grandparents who smoke, especially for maternal grandparents (59% vs 33%). With respect to the other covariates we do not detect significant variations over the two periods and just a slight increase in average household income. Lastly, we find that mother and father e-cig users represent 6% and 7% of our sample, respectively (calculated for the total sample which includes non-smokers). When considering the sample of smokers (i.e. individuals smoking at least one cigarette per day) the share of parents using NDP is around 55%. This implies that more than half of parents current smokers used (even intermittently) e-cigs and other NDP in 2013 and 2014.

The uptake of NDP increased dramatically from 2010 driven by the diffusion of e-cigarettes, as shown in Figure 1. The figure is based on data from the Smoking Toolkit Study (STS) involving monthly household surveys of nationally representative samples of approximately 1800 adults (16+ years) per month in England, with questions covering key performance indicators in smoking[4]. Figure 1 shows that e-cigarette uptake started in 2010 and then increased very rapidly to reach around the 20% of smokers in 2014. This only partially crowded out the use of the other NDP. As a consequence, the total share of smokers currently using any NDP increased from 2010 to decline only after 2013 due to a drop in other NDP users.

Figure 1. Trend in e-cig and other NDP uptake in England



[4] The STS is a large national project funded by Cancer Research UK, the English Department of Health and private partners. Full details can be found at www.smokinginengland.info.

3. Empirical Methodology

3.1 Galtonian Regressions

To quantify the scale of intergenerational transmission of cotinine we follow the standard approach, commonly used to measure intergenerational income mobility, based on the estimates of a Galton regression:

$$Cotinine_j = \beta_0 + \beta_1 Cotinine_j^M + \beta_2 Cotinine_j^F + \beta_3 Controls_j + \varepsilon_j \quad (1)$$

Where cotinine levels of children $j=1...K$. depend on the cotinine levels of their mother and father (M, F), respectively. Controls include equivalized household income, children's demographics and grand-parental smoking status. The income variable is used to measure socio-economic status of the family. Socio-economic status might be correlated with the effort by parents in protecting the children from the exposure to passive smoking and/or with housing conditions which may indirectly increase the degree of exposure. Grandparental smoking status is useful in order to rule-out potential exposure to passive smoking by grandparents. The parameters β_1 and β_2 represent our effects of interest, namely the scale of transmission of cotinine from parents to children. Estimates are based only on children's cotinine values that are below 15 ng/ml in order consider exclusively non-smoker children exposed to passive smoking. In Section 4.1, we present estimates of an alternative specification which replaces $Cotinine_j^M$ and $Cotinine_j^F$ in equation (1) with $Cotinine_j^{M+F}$, the sum of cotinine of both parents, plus the same control variables as equation (1).

Given the different scaling of childhood cotinine levels between HSE waves (see Section 2.1) we present two separate estimates of equation (1) on the 2002-2012 HSE sample and on the 2013-2014 sample. Both regressions are estimated by OLS with the inclusion of year fixed effects. An alternative estimation based on ordered probit models for the three-level cotinine dependent variable is presented in Section 4.3 for waves 2013-2014.

3.2 Effect of e-cigarettes on intergenerational transmission

To evaluate the effect of the introduction of e-cigarettes on intergenerational transmission of nicotine we follow several routes. First, we exploit the spread in the uptake of e-cigs in England. Indeed, while e-cigs were in principle available in the European market since April 2006, the uptake among English smokers started essentially from the beginning of 2010 (as shown in Figure 1) coinciding with a publication by Action on Smoking and Health, a public health charity established by the Royal College of Physicians, which concluded "that e-cigarettes, which deliver nicotine without the harmful toxins found in tobacco smoke, are likely to be a safer alternative to smoking" (ASH, 2009). However, this does not provide us with a control group (people not exposed to the informational shock). We can exploit this quasi-experimental setting in a before-after design with controls for confounding variables. This leads to the following regression:

$$Cotinine_j = \beta_0 + \beta_1 Cotinine_j^M + \beta_2 Cotinine_j^F + \beta_3 Controls_j + \beta_4 Post + \beta_{14} Cotinine_j^M * Post + \beta_{15} Cotinine_j^F * Post + \varepsilon_j \quad (2)$$

Where Post refers to post 2010, while the other variables are the same of equation (1). Coefficients β_{14} and β_{15} represent the effect of e-cigs on intergenerational transmission of nicotine in a before-and-after framework.

As a second route, we exploit information on the use of e-cig and other NDP by parents that is available in waves 2013 and 2014 of the HSE. We thus interact NDP utilization in the Galtonian equation (1). This leads to the following equation:

$$Cotinine_j = \beta_0 + \beta_1 Cotinine_j^M + \beta_2 Cotinine_j^F + \beta_3 Controls_j + \beta_4 Cotinine_j^M * Ecig_j^M + \beta_5 Cotinine_j^F * Ecig_j^F + \varepsilon_j + (3)$$

Where $Ecig_i^{M,F}$ indicates the self-reported use of e-cigs or other NDPs by the mother and father (*M, F*), of children *j=1...K.* , respectively [5]. In order to consider potential multiplicative effects, we also present estimates of an alternative specification which considers the sum of cotinine of both parents ($Cotinine_j^{M+F}$) interacted with the number of NDP users among parents ($Ecig_j^{M,+F}$). We use an OLS estimator with year fixed effects and employ an ordered probit estimator as robustness check which confirms the sign of interaction effects estimated by OLS (see Section 4.3 for more details).

As a third route, we deal with potential endogeneity of e-cig and other NDP utilization. Indeed, one potential concern with equation (3) is that the choice of consuming nicotine through e-cigs instead of traditional cigarettes might potentially be correlated with unobserved factors which also influence the degree of children's exposure to passive smoking. To deal with this, we adopt an Instrumental Variable (IV) strategy using information on whether parents received medical advice to stop smoking as an instrument for their e-cig and other NDP utilization. The advice received from physicians is likely to influence the parents' decisions to move towards safer smoking methods but should not have any direct effect on the exposure to passive smoking for children.

In order to improve the efficiency of the IV estimator, we use the method proposed by Lewbel (2012) which supplements the external instruments with generated instruments constructed from the auxiliary equations' residuals, multiplied by each of the included exogenous variables in mean-centered form. The method is particularly appropriate in situations where available instruments might not be sufficiently strong[6]. Moreover, one nice feature of this method in exactly identified models – as in our case - is that additional generated instruments allows for Sargan–Hansen tests of the orthogonality conditions to check the validity of the instruments. On the other hand, a precondition of using this method is the presence of heteroscedasticity in the first-stage regression and this seems to be largely verified in our case (details are provided in Section 4.2). Successful applications of the Lewbel IV method are increasingly common both within health economics (e.g. Stutzer et al. 2016; Brown, 2014; Block, 2007) and outside of the health economics literature (e.g. Sabia, 2007; Denny and Oppedisano, 2013).

[5] Since the share of use of e-cig and NDP is often intermittent, we use the current or intermittent self-reported use variable in our main specification. Analysis based only on current use of e-cig leads to qualitatively similar results (not shown but available upon request) but it is based on a very low fraction of e-cig and NDP users and thus is not reported in Section 4.

[6] We also performed standard IV estimates which lead to similar results of the Lewbel IV estimates shown in Section 4.2. However, coefficients of the interaction terms in standard IV are weakly significant. This is likely to due to fact that albeit correlated with the endogenous variables instruments are not sufficiently strong. This is also confirmed by the values of the first stage F-test generally smaller than the common cut-off of ten in standard IV estimates (results not shown but available upon request).

4. Results

4.1 Galtonian regression results

Table 2 presents the estimates of the Galtonian regression using two specifications. In columns 1 and 2 we consider separately cotinine of father and mother while in columns 3 and 6 we consider the sum of cotinine for parents. Estimates in column 1 are also replicated with the inclusion of control variables (columns 2). Moreover, we present estimates based on clustered standard errors at household level (columns 4-6) which are robust to measurement error or correlated shocks at household level.

TABLE 2. Galtonian regression estimates - pooled sample 2002-2012[a]

	(1) Children Cotinine (Simple)	(2) Children Cotinine (With Controls)	(3) Children Cotinine (M+F)	(4) Children Cotinine[b] (Simple)	(5) Children Cotinine[b] (With Controls)	(6) Children Cotinine[b] (M+F)
Cotinine F	0.00149***	0.00164***		0.00149***	0.00164***	
Cotinine M	0.00301***	0.00302***		0.00301***	0.00302***	
Hhold Income		-0.00001***	-0.00001***		-0.00001***	-0.00001***
Parental GP smoking		-0.17471***	-0.24427***		-0.17471***	-0.24427***
Maternal GP smoking		-0.12338***	-0.08862***		-0.12338***	-0.08862**
Age		-0.02119***	-0.02182***		-0.02119***	-0.02182***
Male		-0.06107*	-0.06386*		-0.06107*	-0.06386*
Cotinine M+F			0.00251***			0.00251***
Year FE	YES	YES	YES	YES	YES	YES
Observations	7273	7273	7273	7273	7273	7273

Children Cotinine >0 and < 15 in all regressions (objective passive smoking).
***, **, * indicate significance at 1%, 5% and 10%, respectively.
[a]Pooled Estimates 2002-2012. Waves 2005, 2006 and 2012 are not used, since matching of parental and children cotinine leads to few available observations.
[b] S.E. Clustered at Household Level

All the specifications show that there is a significant effect of parental nicotine (both maternal and paternal cotinine) on children's exposure to nicotine. Results are similar in magnitude whether controls are included or not. Moreover, inference based on clustered standard errors leads to similar conclusions. We find that the impact of the mother's nicotine level is twice the size of father's nicotine. This result is in line with Frijters et al. (2011) and is likely due to the fact that mothers typically spend more time with their children. Our estimates (according to the specification with controls in Column 2) are that one standard deviation increase in cotinine level of the father (143.66) leads to an increase of around 0.24 in cotinine scores of children, while for the mother the increase is 0.56.

Time spent at home might explain the negative relation of children's nicotine with their age, as older children usually spend less time at home. Each additional year of age for children is associated with a reduction in cotinine of around 0.02. We also find a significant impact of household income: better off parents, at the same level of nicotine, transfer less nicotine to their children. This might

be due to factors such as housing conditions (dimensions, availability of outdoor space) which may indirectly reduce the degree of exposure.

In Table 3, we present results of the Galtonian regression using 2013 and 2014 waves of the HSE with cotinine of children measured at three categorical levels. Results from these regressions are qualitatively similar to the ones shown in Table 2 and show that the intergenerational transmission of nicotine is present also in more recent years in England.

TABLE 3. Galtonian regression estimates - pooled sample 2013-2014[a]

	(1) Children Cotinine 3 groups (Simple)	(2) Children Cotinine 3 groups (With Controls)	(3) Children Cotinine 3 groups (M+F)	(4) Children Cotinine[b] 3 groups (Simple)	(5) Children Cotinine[b] 3 groups (With Controls)	(6) Children Cotinine[b] 3 groups (M+F)
Cotinine F	0.00206***	0.00203***		0.00206***	0.00203***	
Cotinine M	0.00194***	0.00190***		0.00194***	0.00190***	
HH Income		-0.00001***	-0.00001***		-0.00001***	-0.00001***
Parental GP smoking		-0.07963*	-0.07429*		-0.07963	-0.07429
Maternal GP smoking		0.04766	0.04441		0.04766	0.04441
Age		-0.03105***	-0.03115***		-0.03105***	-0.03115***
Male		0.02024	0.02039		0.02024	0.02039
Cotinine M+F			0.00195***			0.00195***
Year FE	YES	YES	YES	YES	YES	YES
Observations	1381	1381	1381	1381	1381	1381

[a] Cotinine measured in three levels: 0; 0.01-1; 1-12
[b] S.E. Clustered at Household Level
***, **, * indicate significance at 1%, 5% and 10%, respectively

4.2 Estimates of the effect of e-cigarettes

Before-and-after results

Before-and-after estimates of the effect of e-cigarettes on intergenerational transmission of nicotine are reported in Table 4. We find a lower transmission of parental nicotine to children after the spread in the use of e-cigs in 2010. The effect is significant when total nicotine consumed by parents is considered (column 2). The effect is guided especially by mothers but it goes in the same direction also when considering fathers (column 1). The coefficient of total quantity of nicotine consumed by parents (0.00256) indicates that one standard deviation increase in these levels (246.96) leads to an increase of 0.63 in the nicotine of children. According to our specification, the effect of the introduction of NDP in a before-and-after framework implies that nicotine transmission is reduced to 82% of the level without NDP (0.00256-0.00046)/0.00256).

TABLE 4. Before-and-after estimates

	(1) Children Cotinine	(2) Children Cotinine
Cotinine F	0.00166***	
Cotinine M	0.00309***	
Cotinine M+F		0.00256***
Post 2010	-0.36902***	-0.36472***
Post*Cotinine Father	-0.00019	
Post*Cotinine Mother	-0.00055**	
Post*Cotinine M+F		-0.00046***
HH Income	-0.00001***	-0.00001***
Parental GP smoke	-0.23472***	-0.30398***
Maternal GP smoke	-0.18830***	-0.154437***
Age	-0.01947***	-0.02012***
Male	-0.07407**	-0.07748***
Observations	7273	7273

***, **, * indicate significance at 1%, 5% and 10%, respectively

Interaction model results

Table 5 presents a sharper test on the effect of NDP on the adult-child transmission of nicotine. We interact nicotine consumption with self-reported NDP utilization by one parent separately (columns 1 and 2) and by the sum of NDP users in the family (i.e. 0-1-2) (column 3). Interestingly, we find that when parental nicotine is consumed through NDP it has smaller impact on children's passive smoking. We are not able to separate nicotine consumed from NDP from that consumed from traditional cigarettes because virtually all NDP users are also current smokers, as documented in Carrieri and Jones (2016b). However, we find evidence that NDP use reduced the transmission of nicotine from adults to children. The effect is mainly guided by the mother (the interaction between the nicotine of the father and NDP use is not statistically significant), but it is significant also when the total nicotine consumed by parents is interacted with the number of NDP users among parents. According to our estimates, the transmission of nicotine to children is reduced to around 68% of the level for cigarettes when only mother use NDP ((0.00246-0.00079)/(0.00246)), while it is reduced to 83% when the sum of parents using NDP is considered ((0.00226-0.00039)/0.00226). The magnitude of this impact is very similar to the one found in the before-and-after analysis.

TABLE 5. Galtonian regression with interactions – Sample 2013-2014

	(1) Children Cotinine 3 groups (Simple)	(2) Children Cotinine 3 groups (with Controls)	(3) Children Cotinine 3 groups (M+F)
Cotinine F	0.00204***	0.00199***	
Cotinine M	0.00246***	0.00237***	
CotinineF*Father e-cig user	0.00003	0.00010	
CotinineM*Mother e-cig user	-0.00079**	-0.00070**	
HH Income		-0.00001***	-0.00001***
Parental GP smoke		-0.08230*	-0.06768
Maternal GP smoke		0.04424	0.05125
Age		-0.03089***	-0.03103***
Male		0.02216	0.02298
Cotinine M+F			0.00226***
Cotinine M+F*Total Adults e-cig users			-0.00039**
Observations	1381	1381	1381

***, **, * indicate significance at 1%, 5% and 10%, respectively

IV results

Table 6 reports IV estimates based on Lewbel's method for the main specifications used so far. In column 1 we consider separately nicotine of father and mother using the advice to stop smoking received by each of the parents as external instruments, while in columns 2 and 3 we consider the sum of nicotine consumed by parents using advice to stop smoking received by each of the parents (column 2) or the sum of advice received by the parents (column 3) as external instruments, respectively.

TABLE 6. IV estimates – Sample 2013-2014

	(1) IV Lewbel	(2) IV Lewbel	(3) IV Lewbel
Cotinine F	0.00198***		
Cotinine M	0.00263***		
Cotinine F*e-cig F	-0.00008		
Cotinine M* e-cig M	-0.00116***		
Cotinine Parents		0.00247***	0.00246***
Cotinine M+F* Total e-cig users		-0.00074***	-0.00073***
Observations	1373	1373	1373
First Stage F-test	1407.0	1810.6	2770.6
Sargan-Hansen's statistics	7.500	0.00925	0.601
Degree of over identification (L-K)	4	2	1
Sargan-Hansen p-value	0.112	0.995	0.438
Breush-Pagan First stage p-value	0.0000 (Father) 0.0000(Mother)	0.0000	0.0000

(1) External instruments: Medical advice to stop F* Cotinine ; Medical advice to stop Mother* Cotinine M
(2) External instruments: Medical advice to stop F* Cotinine ; Medical advice to stop Mother* Cotinine M
(3) External instruments: Medical advice to stop to M or F * Cotinine M+F
***, **, * indicate significance at 1%, 5% and 10%, respectively

Our main results are substantially confirmed using this IV strategy. The scale of transmission of cotinine is now reduced to around 56% when only mothers use NDP ((0.00263-0.001169)/(0.00263)) and to around 70% when the sum of parents using NDP is considered ((0.00247-0.00074)/(0.00247)). Compared to the interaction and before-and-after model estimates, these estimates suggest that the estimated reduction in nicotine transmission due to NDP remains large when endogeneity of NDP utilization is taken into account and the estimated impact for mothers is greater when endogeneity is taken into account.

Table 6 also reports a number of tests to assess both the strength and the validity of the instruments. To assess the first, we report the Breusch-Pagan test of the first-stage regression. Indeed, the presence of heteroscedasticity is a precondition of using the Lewbel IV method as the scale heteroscedasticity determines the correlation between additional generated instruments and the endogenous variables (Lewbel, 2012). The last row of Table 6 show that the residuals are strongly heteroscedastic in each of the first-stage equations as the null hypothesis of constant variance is largely rejected in all the specifications. Moreover, the first stage F-test is substantially greater than the common cut-off of 10 in all the specifications suggesting that the estimates do not suffer from the weak instruments problem. Second, we report standard Sargan-Hansen overidentification test to assess the validity of the set of instruments used, and, thus, to test the set of the identifying assumptions of the Lewbel method. As discussed in Section 3.2 this is possible as external instruments (medical advice to stop smoking) are supplemented by the additional generated instruments, as documented by the degrees of overidentification reported also in Table 6. The p-values of the Sargan-Hansen tests indicates that our set of instruments are valid and that the assumptions of the Lewbel IV model are not rejected in all the specifications adopted.

4.3 Robustness checks

To test the reliability of our before-and-after identification strategy, we performed some placebo regressions by dating the starting of e-cig uptake in the England up to three years before 2010. Results of this check are reported in Table 7 and include both the Galtonian regression with cotinine levels for father and mother alone (columns 1, 3 and 5) and the sum of cotinine of both parents (columns 2, 4 and 6). The interaction terms are not significant for 2008 and 2009 and they are only weakly significant when 2007 is used as "placebo" starting year. However, the coefficient for this year is positive which implies an increase in the transmission of nicotine from 2007 onwards. This suggests that there was effectively a negative and significant break in the parent-child transmission of nicotine only after 2010 in England.

As an additional check, we test whether our results are confirmed using an ordered probit estimator for both Galtonian regression and interaction model when cotinine is measured in three levels, i.e. in waves 2013 and 2014. Results of this additional check are reported in Table 8 and show that our main conclusions are substantially unchanged: both the intergenerational transmission of nicotine (columns 1 and 2) and the reduction in its transmission to children from parents using NDP (columns 3 and 4) are confirmed.

TABLE 7. Before-and-after placebo regressions

	Children Cotinine Post>=2007		Children Cotinine Post>=2008		Children Cotinine Post>=2009	
	(1)	(2)	(3)	(4)	(5)	(6)
Post*Cotinine Father	0.00004		-0.00029		-0.00026	
Post*Cotinine Mother	0.00064**		0.00043		0.00011	
Post*Cotinine M+F		0.00028*		0.00001		-0.00016
Observations	7273	7273	7273	7273	7273	7273

Note: Estimates are based on the same specification of equations (2) but with fake before-after groups.
***, **, * indicate significance at 1%, 5% and 10%, respectively

TABLE 8. Galtonian regression - ordered probit estimates, sample 2013-2014

	(1) Children Cotinine 3 groups (with Controls)	(2) Children Cotinine 3 groups (M+F)	(3) Interaction Cotinine 3 groups*ecig	(4) Interaction cotinine 3 groups*ecig (M+F)
Cotinine F	0.00330***		0.00330***	
Cotinine M	0.00309***		0.00384***	
Cotinine M+F		0.00317***		0.00380***
CotinineF*Father e-cig user			0.000008	
CotinineM*Mother e-cig user			-0.00112**	
Cotinine M+F* Total Adults e-cig users				-0.00075**
Controls	YES	YES	YES	YES
Observations	1381	1381	1381	1381

***, **, * indicate significance at 1%, 5% and 10%, respectively

5. Conclusions

In this paper we study the intergenerational transmission of nicotine within families through exposure to passive smoking and we test whether the use of novel nicotine delivery products (e-cigarettes and other NDP) by parents reduces nicotine transmission to children. Both aspects have been relatively unexplored in the economic literature but pose important economic concerns as intergenerational transmission of nicotine has relevant implications for childrens' welfare and testing whether e-cigs have reduced this transmission is relevant for the evaluation of the externalities deriving from NDP consumption and, thus, for design of taxes on these devices.

We quantify the scale of transmission of nicotine from parents to children in England by estimating a Galtonian style regression and using saliva cotinine (the major metabolite of nicotine) to objectively measure both active smoking by parents and exposure to passive smoking by children. To test the effect of NDP on nicotine transmission, we adopt several strategies encompassing the inclusion of interaction terms between parental cotinine levels and their NDP utilization status in the Galtonian regression, an IV strategy to deal with potential endogeneity of NDP utilization and a before-and-after analysis which exploits the spread in the use of e-cigarettes in England from 2010 following the publication of favourable information about their use.

We find evidence of substantial transmission of nicotine from parents to children and that transmission is more than twice as large for mothers than for fathers. The latter result confirms the finding of Frijters et al. (2011) and is likely due to the fact that mothers usually spend more time with their children. Our estimates allow a precise quantification of this transmission: one standard deviation increase in cotinine for fathers leads to an increase of around 0.24 in cotinine scores of children, while for mothers the increase is 0.56 . These numbers are not negligible considering that cotinine scores denoting passive smoking are bounded mainly between 0 and 15ng/ml.

With respect to NDP, we find a lower transmission of parental nicotine on children after the spread in the use of e-cig in 2010. According to our before-and-after specification, the effect of NDP in a implies a reduction to 82% in the level of nicotine transferred when inhaled by NDP compared to cigarettes. This result is corroborated with a similar magnitude also when nicotine consumption is interacted with self-reported NDP utilization in the Galtonian regression. The IV estimates of the effect are 70% for both parents and a larger reduction to 56% for transmission from the mother. A number of checks concerning the specification and the identification strategies support the robustness of these conclusions.

These results have two important policy implications. First, they show that exposure to passive smoking within families is high in England and that more interventions could protect children from this exposure. The potential benefits of such interventions are likely to be very high given the substantial costs that nicotine transmission to children may generate. Only considering the immediate health damage to children, Frijters et al. (2011) calculate that the income equivalence of exposure to passive smoking is £16,000 per year. The possible future of nicotine addiction and the future health risks associated are likely to further increase the societal costs of children's exposure to passive smoking. However, the identification of effective interventions to reduce exposure to passive smoking is less straightforward. A further increase of taxes on cigarettes is an option while the presence of smoking bans in recreational public places may be not appropriate for the specific target of reducing exposure of children. The US experience is that this might have the perverse effect of increasing exposure to passive smoke in private places such as at home (Adda and Cornaglia, 2010). Perhaps, any kind of intervention needs to be coupled with health information campaigns that highlight to adults the risks of passive smoking for their children, the benefits of quitting and the availability of NDP. Such kind of interventions are likely to be especially useful if aimed at mothers whose smoking appears to have a greater impact than fathers on nicotine transmission to children as mothers spend more time on their care.

Somewhat related, a second implication of our results is that e-cigs and other NDP have to be considered as a preferable alternative to smoking for the purpose of reducing the nicotine transmission to children. This has direct implications for the taxation of these new nicotine delivery products. This topic is at the centre of an ongoing debate in Europe and there are many proposals

to increase taxation on these devices (currently taxed by 20% VAT) to reach a closer convergence with taxation on tobacco products (currently taxed by around 80%). Our results show that this may not be justified on economic grounds. Following the externality argument for nicotine taxation, our findings instead suggest that a tax differential is justified because nicotine transmission to children is lower when it is delivered by NDP rather than traditional cigarettes.

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