Intergenerational transmission of nicotine within families: have e-cigarettes influenced passive smoking?[*]

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Abstract

Using an objective biomarker of active and passive smoking, we estimate Galtonian regressions of nicotine transmission and test whether the use of new nicotine delivery products (NDP) by parents had an influence on the transmission to children through passive smoking. We find evidence of a strong intergenerational transmission through passive smoking and that this is around four times larger for mothers compared to fathers. Moreover, we estimate an intention to treat difference-in-differences (DiD) model using parental cotinine as a continuous measure of exposure to the treatment and we find that the level of transmission of cotinine from parents was reduced to 51 per cent of the previous level just after the spread in the use of e-cigarettes in England and to 77 per cent when considering transmission from mothers. This is confirmed also by a DiD model which considers interaction between cotinine levels and self-reported use of NDP by parents and suggests that lower taxation of these devices may be justified on externality grounds.

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). A high intergenerational correlation is found also for what concerns health conditions. For instance, Dolton and Xiao (2017) estimate an intergenerational BMI elasticity of around 0.2 per parent. Classen and Thompson (2016) further indicate that this transmission occurs primarily in biological parent-child pairs thus suggesting an important role of genetic factors. 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; Currie and Stabile, 2004; Case, Fertig and Paxson, 2005; Currie and Madrian, 1999; Currie and Hyson, 1999; Yi et al., 2015; Smith, 2015). 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) and "have quantitatively large impacts on virtually all key adult indicators of socioeconomic status that economists use" (Smith, 2009).

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 from 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 children's welfare in particular. Exposure to passive smoking is immediately dangerous for children's health, such as the development of respiratory tract infections and cases of aggravated asthma among children (Environmental Protection Agency, 1994). This is a relevant topic as cigarettes cause fully a third of deaths in later life. They are the leading cause of lung cancer and chronic obstructive pulmonary disease mortality, as well as a major cause of cardiovascular death, chronic disability (Bengtsson and Nilsson, 2018) 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 health damage to children is likely to generate costs that spill-over into broader society

(Chaloupka and Warner, 2000) and that taxation of these products might have important consequences on the intergenerational well-being. Using 1989–1992 census of births data from the US, Evans and Ringel (1999) find that taxes alter the smoking behaviour of pregnant women and that increased cigarette taxes have a beneficial impact on infant birth weight. Similarly, Simon (2016) estimates that the impact of a dollar increase in the state cigarette tax on in utero exposure causes a 10 percent decrease in sick days from school, a 4.7 percent decrease in having two or more doctor visits and decreases in hospitalizations and asthma.

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 in natural units that can be replicated and compared across different settings. 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[1].

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[1] Estimates of intergenerational elasticity derived by regressing children's outcomes on parental outcomes are also known as Galtonian regressions. Galtonian regression is the workhorse of the research into intergenerational mobility. It takes the name from the well-known study by Sir. Francis Galton looking at the correlation between the height of individuals and that of their parents. To the best of our knowledge, the use of cotinine as both a parental outcome, measuring active smoking, and children's outcome, measuring exposure to passive smoking, is new in the literature. Thus, in our study, Galtonian regression coefficients allow us to quantify the scale of intergenerational transmission of nicotine from parents to children through exposure to passive smoking.

Electronic cigarettes (e-cigs) and other novel nicotine delivery products (NDP) are 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 novel nicotine delivery system. They do not 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).[2] Indeed, lab studies suggest that both toxic chemical concentrations (Goniewicz et al., 2014) and airborne nicotine levels in second-hand smoke are lower in e-cigarettes than in traditional cigarettes (Czogala et al. 2014). 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 small sample of 54 individuals living in homes with cigarette 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 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-

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[2] E-cigs have been found as effective, though not more so, as 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 longer than cessation methods. In addition, cartridges have been found to contain hazards, such as cytotoxic heavy metal and silicate particles (Williams and Talbot, 2011).

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 waves of the Health Survey for England (HSE) spanning between 2002 and 2014. To assess the effect of NDP on nicotine transmission, we follow two 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).[3] This allows us to assess the influence of NDP on the intergenerational transmission of nicotine in an intention to treat difference-in-differences (DiD) framework using parental cotinine as a continuous measure of exposure to the treatment. As a second sharper test for the effect of e-cig on the intergenerational transmission, we exploit information on the self-reported use of e-cig and other NDP by parents that is available in waves 2013 and 2014 of the HSE. In this case the focus is on the actual exposure to e-cigs, rather than an intention to treat analysis. The coefficient of DiD interaction terms indicates whether the transmission of nicotine inhaled through NDP is lower than conventional smoking, other things being equal.

We find evidence of substantial transmission of nicotine from parents to children and that transmission is around four times as large for mothers than for fathers. Moreover, both DiD type strategies lead us to conclude that nicotine transmission to children is lower when it is delivered through NDP. This has 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 each year 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[4]. 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

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[3] This coincided with favourable guidance on the use of e-cigs by active cigarette smokers by Action on Smoking and Health (a public health charity established by the Royal College of Physicians) in October 2009.

[4] 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).

are collected during nurse visits and include saliva samples that are used for the measurement of cotinine levels (see Section 2.1 for more details). It is important to note that only children aged 4 or above are eligible for cotinine measurement. During the nurse visits, the nurse asks the respondent for permission to carry out various types of measurements and respondents are informed about the purpose and relevance 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 DiD analysis 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 7,666 non-missing observations (6430 for the 2002-2012 sample and 1236 for the 2013-2014 sample). This sample includes only children aged 4-14 years old and excludes those whose cotinine scores indicate that they are active smokers themselves (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 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 Richard, 1988)[5]. Consistent with these recommendations, we identify passive smoking with cotinine values below 15 ng/ml

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[5] 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).

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 that lead to qualitatively comparable results (see Section 4.1).

We use a parsimonious set of controls for our baseline regressions which includes demographic variables for the children (age and gender) and equivalised household income of the family. A larger set of controls and a different specification of the age variable are presented in Section 4.1. 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 DiD 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 0.91 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 49% of children in our sample have been exposed to passive smoking during the period 2013-2014.

[**Table 1 around here**]

Table 1 shows a clear decline over time in cotinine levels of both parents. Average cotinine in fathers dropped from around 35.1 in 2002-2012 to 18.94 in 2013-2014, a reduction of around 46%. A slightly larger drop is found for mothers (51%) and, consequently for the sum of cotinine for both parents (49%). These numbers are consistent with the reduction in the prevalence of smoking previously discussed. 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 both represent 6% of our sample (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 54%. This implies that more than half of parents who are 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 2007-2014 data included in the report "Smoking in England 2007-2014" from the Smoking Toolkit Study (STS) which involves monthly household surveys of nationally representative samples of approximately 1800 adults (aged 16+ years) per month in England, with questions covering key

performance indicators in smoking[6]. Figure 1 shows that e-cig uptake as a smoking cessation method started to be measurable in 2010 and then increased very rapidly becoming the most used smoking cessation method in 2014, i.e. used by around the 32% of smokers trying to quit. This partially crowded out in particular the use of the other NDP and - after 2012 - also the use of other smoking cessation methods, including drugs such as Champix and Zyban and behavioural support.

[**Figure 1 around here**]

3.   Empirical Methodology

3.1   Galtonian Regressions

To quantify the scale of intergenerational transmission of cotinine we follow the standard approach that is more commonly used to measure intergenerational income mobility. This is based on the estimates of a Galtonian regression:

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

Where cotinine levels of children *j=1…K.* depend on the cotinine levels of their mother and father (*M, F*), respectively[7]. The baseline specification includes equivalized household income and children's demographics. Parental 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 that may indirectly increase the degree of exposure. We also include a large set of additional controls following a step-wise approach to take into account family composition, parental education, weight of the children and a different age specification (see Section 4.1 for more details). β_1 and β_2 represent our parameters of interest, namely the scale of transmission of cotinine from parents to children[8]. Estimates are based only the sample of children with cotinine values that are below 15 ng/ml in order consider exclusively non-smoker children exposed to passive smoking and to exclude those who are active smokers themselves. 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).

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[6] 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.

[7] Correlation between the cotinine of mother and father is rather modest in our sample (0.16). This rules out concerns around multicollinearity of the two variables.

[8] In our specification the scale of transmission is measured in natural units of cotinine.

Given the different scaling of childhood cotinine levels between HSE waves (see Section 2.1) we opted to run two separate estimates of equation (1) on the 2002-2012 HSE sample and on the 2013-2014 sample[9]. 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 assess the influence of the introduction of e-cigarettes on intergenerational transmission of nicotine we follow two routes. First, we exploit the spread in the uptake of e-cigs in England. As shown in Figure 1 and discussed in Section 2.1, 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 with other kinds of generally available innovation, the introduction of e-cigs does not allow to have a "natural" control group, i.e. individuals not exposed to the availability of this new nicotine delivery product. However, insofar as the intergenerational transmission is the focus of the analysis, as in our case, a useful source of variation is provided by parental levels of nicotine consumption as reflected in their cotinine values. We exploit this variation to estimate the impact of e-cig on the intergenerational transmission in a Differences-in-Difference (DiD) type framework, with a continuous measure of parental exposure to nicotine (*Cotinine*) interacted with a discrete indicator of the general availability of e-cig post 2010 (*Post*), conceived as follows:

$$Cotinine_j = \beta_0 + \beta_1 Cotinine_j^M + \beta_2 Cotinine_j^F + \beta_3 Post + \beta_{13} Cotinine_j^M * Post + \beta_{14} Cotinine_j^F * Post + \beta_4 Controls_j + \beta_5 Trend + \beta_6 Trend^2 + \varepsilon_j \quad (2)$$

Where cotinine levels of children *j=1...K.* depend on the cotinine levels of their mother and father (*M, F*), respectively. *Post* refers to post e-cig introduction (i.e. year 2010), while the control variables are the same as equation (1). The inclusion of a linear and a quadratic trend as additional controls is useful to take into account variations over time in the exposure to passive smoking. 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 post 2010 period.

Coefficients β_{13} and β_{14} represent the effect of the availability of e-cigs on intergenerational transmission of nicotine and can be given an intention to treat interpretation as they reflect the impact of the general availability of e-cigs post-2010 on the intensity of transmission of cotinine from parents to children. Note that a standard

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[9] We might also re-classify cotinine levels in three groups in the 2002-2012 sample and estimate a pooled regression from 2002 to 2014. However, this would not allow us to estimate the scale of intergenerational transmission of nicotine in natural units, which is one of the main aims of our analysis.

DiD specification would interact the post dummy with a binary indicator of treated versus control (e.g. high versus low cotinine levels for the parents). Instead we have exploited the continuous variation that is observed for parental cotinine and interacted that with the post dummy. The identifying assumption of the intention to treat DiD model (equation (2)) is the standard common trend assumption. In our case, this requires that, without the availability of the new nicotine delivery product, the trend in transmission of nicotine from parents to children would remain constant. A potential threat to this strategy might be represented by the existence of a shift in the intergenerational transmission of nicotine around 2010 - other than the one caused by the spread of e-cigarettes - which may bias the effect of e-cig on nicotine transmission within families.

It is important to observe that the existence of a trend in the exposure to passive smoking which did not exhibit a structural break in 2010 is unlikely to be a threat to our identification since we include both linear and quadratic trends as controls in equation (2). A careful search of smoking-related policies in England did not reveal any significant new action directly aimed at exposure to passive smoking that began or was active around 2010. An emphasis on the risks of third-hand smoke exposure, particularly for young children, was contained in a NHS report published in February 2010[10]. This followed the publication of studies on persistent tobacco smoke contamination on nearby surfaces after a cigarette is extinguished and the related risks. However, this is unlikely to drive our results, because exposure to third-hand smoking, i.e. tobacco smoke contamination on nearby surfaces, is relevant especially among infants who spend much time on the floor, while we measure nicotine transmission only among children aged 4 and over.

As a second sharper test for the effect of e-cig on the intergenerational transmission, we exploit information on the self-reported use of e-cig and other NDP by parents that is available in waves 2013 and 2014 of the HSE. In this case the focus is on the actual exposure to e-cigs, rather than an intention to treat analysis. This leads to a DiD model very close to the one reported in equation (2) with parental cotinine as a continuous measure of intensity of exposure to the treatment interacted with self-reported use of e-cigs as the measure of actual treatment:

$$Cotinine_j = \beta_0 + \beta_1 Cotinine_j^M + \beta_2 Cotinine_j^F + \beta_3 Ecig_j^M + \beta_4 Ecig_j^F +$$
$$\beta_{13} Cotinine_j^M * Ecig_j^M + \beta_{24} Cotinine_j^F * Ecig_j^F + \beta_5 Controls_j + \varepsilon_j + (3)$$

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 [11]. All the other terms are the same as discussed in equation (2). In order to consider potential multiplicative effects, we present

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[10] More details can be found at https://www.nhs.uk/news/pregnancy-and-child/concern-over-third-hand-smoke/

[11] Since the use of e-cigs 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 low fraction of e-cig and NDP users and thus is not reported in Section 4.

the 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).

It is important to highlight a key feature of our strategy which is common to both models presented in equations (2) and (3). This requires first to discuss more carefully the specific type of endogeneity issue that may arise in our setting. A key point here is that since the children are not active smokers, their observed cotinine levels can only be explained due to passive exposure to nicotine. In this setting, the interpretation of the interacted cotinine terms (coefficients β_{13} and β_{14} in equation (2) and β_{13} and β_{24} in equation (3)) as causal effects does not require us to assume that the decision to adopt e-cigs is unaffected by unobserved factors. Indeed, parent's decisions on how much to smoke and on whether to become an e-cig user might be due to unobserved preferences, for instance, i.e. risk aversion or time preferences, or to unmeasured peer-effects. However, the endogeneity of parental cotinine levels does not represent a threat unless they are correlated with unobserved factors that lead to higher passive exposure for children through sources *other* than the parents themselves (as we control for parental cotinine in our models). More importantly, and this represents the key of our identification strategy, this would be a threat for the interpretation of the *un-interacted cotinine levels* (β_1 and β_2 in equations (2) and (3)) in causal terms but this need not represent a threat to identification of the interaction effect between parental cotinine and use of e-cigs in the DiD specifications if the assumption of common trends holds. In our case unobservables will only be an issue if children of e-cig adopters *vs* traditional smokers are differently exposed to second-hand smoke through sources *other* than their parents (whose cotinine we are controlling for) or if parents' selection into using e-cigs is influenced by the intensity of the relationship between their own level of nicotine consumption and transmission of nicotine to their children. To help check the robustness of our identification strategy, we present a number of additional analyses in Section 4.3 including placebo regressions with fake e-cig introduction periods and augmented specifications of models (2) and (3) including interaction effects between the control variables and post and e-cig dummies.

4. Results

4.1 Benchmark Galtonian regression results

Table 2 presents estimates of the benchmark Galtonian regression using two specifications. In columns 1 and 2 we consider separately cotinine of the father and mother while in columns 3 and 4 we consider the sum of cotinine for parents. All estimates are presented with clustered standard errors at household level that are robust to measurement error or correlated shocks at household level.

[**Table 2 around here**]

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 (especially the coefficient measuring the impact of maternal cotinine) are substantially similar in magnitude whether controls are included or not. We find that the impact of the mother's nicotine level is around four times 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 (117.51) leads to an increase of around 0.16 in cotinine scores of children, while for the mother the increase for one standard deviation (132.46) is 0.67. To give a sense of these magnitudes, our estimates imply that being a "moderate" smoker, compared to a non-smoker, typically increases the levels of the child's cotinine by 0.40 for fathers and 1.48 for mothers. While being a "heavy" smoker increases the amount by 0.52 for fathers and 1.91 for mothers.[12]

Time spent at home might explain also the negative relationship of children's cotinine 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: children of better-off parents are exposed to less nicotine. This might be due to factors such as housing conditions (dimensions, availability of outdoor space) which may indirectly reduce the degree of exposure.

In order to check the robustness of these findings, we expand baseline specification including a large set of controls in Table 3 following a step-wise approach. As a first set of control, we consider household composition. In particular, the presence of single-parent families would assign zero levels of cotinine for one of the parents and this might bias the parental cotinine effect. Thus, in columns 1-2 of Table 3 we include a dummy for a single-parent family, while in columns 3-4 we include separate controls for single-mother and single-father families. We find that children living in single-parent families have generally higher levels of cotinine and this is especially true for single-father families (compared to single-mother ones). Importantly, this does not affect the coefficients on parental cotinine; neither when they are measured separately for mothers and fathers (columns 1 and 3) nor when the sum of cotinine over both parents is considered (columns 2 and 4).

[**Table 3 around here**]

Next, we consider educational status of the parents as an additional control. Indeed, greater health investment by parents might reduce nicotine transmission to children and this may

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[12] These calculations are based on the average cotinine levels detected in the Health Survey for England among adults reporting consumption of a half-pack of cigarettes per day (average cotinine= 292) and at least a pack of cigarettes per day (average cotinine=378), respectively.

pose a potential omitted variable issue in our main specification. Thus, in columns 5 and 6 in Table 3 we repeat the baseline regressions including a dummy variable for higher educated parents (with a degree) as an additional control. The coefficients of parental education are negative in both specifications. This implies that children with more educated parents are less exposed to nicotine. However, also in this case, the coefficients on parental cotinine are substantially unaffected by the inclusion of this additional control.

As a third set of controls, we include a different specifications of age. A careful control for age might be relevant since the exposure to passive smoking might vary across children of different ages. Indeed, very young children might be more exposed to third hand smoke at home due to time spent on the floor/rugs, where the chemicals settle. Although this concern is likely to be highly limited in our study since we do not observe children aged less than 4 (due to missing saliva measurements, as discussed in Section 2), non-linear differences in the effects on infants versus older children and pre-adolescents might be relevant. For this reason, we replaced continuous age variable with two age categories (4-7, 7-10, and above 10 as the reference category) in our main specifications. Results shown in columns 7 and 8 of Table 3 show that younger children (aged 4-7) present significantly higher levels of cotinine with respect to their older counterparts. However, the coefficients on parental cotinine are substantially unaltered.

Next, we deal with the possibility that the same amount of nicotine exposure may generate different cotinine levels among children of different weights. Although some of this effect could be caught up in the age coefficient, we include the weight of the child (measured by the nurse during the nurse visit) as an additional control. Results of this check, reported in the last two columns of Table 3, suggest that heavier children have higher levels of cotinine but that this does not affect our main effects of interest for exposure to parental cotinine.

As a last check, we test the hypothesis that mother effects are stronger due to the amount of time they spend with their children. Thus, we repeat main regressions in Table 2 including a dummy variable for the activity status of the mother (employed versus unemployed, retired or inactive) as an additional control. Results of this test are reported in Table 4 for specifications (2) and (3) of Table 2. We find that the activity status variable is negative and significant. This implies lower levels of cotinine among children with active mothers; coefficients for cotinine of mother, and the sum of cotinine for parents in the augmented model are reduced compared to the ones shown in Table 2. Moreover, the interaction terms between mother's cotinine and mother's activity status (column 3 of Table 3) is negative and significant, meaning that transmission of cotinine is lower when the mother is active. These results support the idea that time spent with children might be an important explanation of the larger transmission effect found for mothers.

[**Table 4 around here**]

In order to test whether the parental transmission is present also in more recent years in

England, we re-estimate the Galtonian regression using the 2013 and 2014 waves of the HSE with cotinine of children measured at three categorical levels. Results from these regressions are reported in Table 5 and they are qualitatively similar to the ones shown in Table 2. This confirms that the intergenerational transmission of nicotine is present also in more recent years in England. However they do not show the same difference between mothers and fathers that was evident in Table 2. This may be because the variation in cotinine is condensed to the 3-point ordinal scale.

[**Table 5 around here**]

4.2 Estimates of the effect of e-cigarettes

Intention to treat DiD analysis

Difference-in-Differences estimates of model in equation (2) are reported in Table 6. We find a lower transmission of parental nicotine to children after the spread in the use of e-cigs in 2010. The effect is statistically significant when the total cotinine score for both parents is considered (columns 3 and 4). The effect is driven especially by mothers, for whom the interaction term is statistically significant, but it goes in the same direction also when considering fathers (column 1 and 2). The results are robust to the inclusion of both a linear (columns 1 and 3) and a quadratic trend (columns 2 and 4).

The estimates from the intention to treat DiD model imply that the introduction of NDP has reduced the relative impact of the parents' cotinine level on their child's cotinine score and hence reduced the transmission of nicotine. The results in columns 1 and 2 imply that for mothers the relative reduction in the impact of an extra unit of cotinine is 77 per cent (100*(0.00510-0.00114)/0.00510) and for fathers it is 74 per cent (100*(0.00124-0.00032)/0.00124). For the combined model, in columns 2 and 4, the relative impact of both mother and father using NDPs is a reduction to 51 per cent of the level of transmission without NDPs (100*(0.00337-2*0.00082)/ 0.00337).

[**Table 6 around here**]

Difference-in-Differences analysis

Table 7 presents estimates of the DiD model using actual exposure to the treatment as defined in equation (3). We interact cotinine scores 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). This is also confirmed by the coefficients related to the e-cig use by parents (and mother in particular) which are positive and significant. However, all specifications 1-3 in Table 7 suggest that the use of NDP reduces the transmission of nicotine from adults to children. The effect is mainly attributable to mothers (the interaction between the cotinine score of the father and NDP use is not statistically significant) and it is statistically significant when the total nicotine consumed by both parents is interacted with the number of NDP users among parents.

According to our estimates, the transmission of cotinine to children by mothers is reduced to around 40 per cent of the level of transmission for conventional cigarettes when the mother uses NDP (100*(0.00263-0.00157)/(0.00263)). In the combined specification having both parents use NDP reduces transmission to 42 per cent of the level without NDP (100*(0.00231-2*0.00066)/(0.00231)).

[**Table 7 around here**]

4.3 Robustness checks

In this section we present some additional empirical analyses to check the robustness our results. As a first check, we focus on the plausibility of the common trend assumption underlying the intention to treat DiD model depicted in equation (2). We thus perform some placebo regressions by dating the start of e-cig uptake in the England one year before 2010. Results of this check are reported in Table 8 and include both the DiD regression with cotinine levels for father and mother alone (columns 1 and 2) and the sum of cotinine of both parents (columns 3 and 4). Both specifications are compared with (in columns 2 and 4) and without (columns 1 and 3) the set of control variables used in the main specification of equation (2). The interaction terms are not significant in any specification included in Table 8. This suggests that there was effectively a negative and significant break in the parent-child transmission of nicotine only after 2010 in England.

[**Table 8 around here**]

As a second check, we test whether DiD results are confirmed using an augmented specification including the full set of control variables interacted with the treatment dummy[13]. This might be useful in order to check the stability of the DiD estimates to the inclusion of confounding factors which might drive selection into the use of e-cig and to improve the precision of the estimated parameters. Results of this check are reported in

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[13] For the intention to treat analysis we include the full set of control variables reported in the stepwise approach in Table 3 (demographic variables for the children (age and gender), equivalised household income of the family, family composition, parental education, activity status of the mother) with the exception of the weight of the children. The inclusion of weight variable would generate a large drop in the sample size since weight measurement is available only for a sub-sample of the children. Some of the aspects potentially captured by the weight are anyway taken into account in the model by the inclusion of age and gender among controls. For the DiD model using reported e-cig, we use the set of baseline controls that includes the demographic variables for the children (age and gender) and equivalised household income of the family.

Table 9 and include both the intention to treat DiD regression and the DiD regression using the actual treatment with cotinine levels for father and mother alone (columns 1 and 2) and the sum of cotinine of both parents (column 3 and 4). Both the sign and the magnitude of our main effects are practically unchanged. The results for the intention to treat DiD model in column 1 imply that for mothers the relative reduction in the impact of an extra unit of cotinine is 79 per cent (now statistically significant at 10%) an for the combined model (in column 3) is 49 per cent. In the non-augmented model, this was 77 and 51 per cent, respectively. The results for the DiD model in column 2 imply a reduction of 42 per cent for mothers and 37 per cent for both parents using e-cig (column 4). These are also very close to the estimates for the non-augmented model, i.e. 40 and 42 per cent, respectively.

[**Table 9 around here**]

As a last check, we test whether our results are confirmed using an ordered probit estimator for both the benchmark Galtonian regression and the DiD model with actual treatment when cotinine is measured in three levels, as available in waves 2013 and 2014. Results of this check are reported in Table 10 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 10 around here**]

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 had an influence on the 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 had an influence on 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 (aged 4-14) 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 assess the influence of NDP on nicotine transmission, we adopt two difference-in-differences strategies using parental cotinine as measure of continuous exposure to treatment. In a first specification, we rely on an intention to treat analysis exploiting the general availability of e-cigarettes in England from 2010 following the publication of favourable information about their use. Moreover, we

exploit information on the self-reported use of e-cig and other NDP by parents that is available in waves 2013 and 2014 of the HSE. In this case the focus is on the actual exposure to e-cigs, rather than an intention to treat analysis.

We find evidence of substantial transmission of nicotine from parents to children and that transmission is four times larger for mothers than for fathers. The latter result confirms the finding of Frijters et al. (2011) and is most likely due to the fact that mothers usually spend more time with their children. This is confirmed by additional estimates that control for the activity status of the mother. Our estimates allow a precise quantification of this transmission: one standard deviation increase in cotinine for fathers leads to an increase of around 0.16 in cotinine scores of children, while for mothers the increase is 0.67. These numbers are not negligible considering that cotinine scores denoting passive smoking are bounded mainly between 0 and 15ng/ml. Importantly, both the magnitude and the sign of these effects are stable to the inclusion of a large set of additional controls which takes into account household composition, parental education and a careful control for the age and the weight of the children measured by a professional nurse.

With respect to NDP, we find a lower transmission of parental nicotine to children after the spread in the use of e-cig in 2010. According to the intention to treat DiD analysis, the level of transmission of cotinine from mothers was reduced to 77 per cent of the previous level and to 51 per cent if both parents use NDP. In the DiD analysis which uses self-reported NDP, the transmission of cotinine to children by mothers is reduced to around 40 per cent of the level of transmission for conventional cigarettes and to 42 per cent when considering both parents using NDP. 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. This issue is likely to be even more important in recession times, being the probability of become a smoker much higher during these periods (Kaiser et al., 2018). 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 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. It is important to specify that that our conclusions may not apply to pregnant women, newborns and infants (who are excluded from our analysis) because nicotine, which is in most e-cigs and NDP, is a threat to the developing fetus. Pesko and Currie (2016) indeed find that e-cigarette minimum legal sale age laws while increasing pregnant teenagers' smoking by 2.1 percentage points have also modestly improved selected birth outcomes, perhaps by reducing overall nicotine exposure from vaping and smoking combined. That said, our results for children aged 4-14 may be generalizable to the adult population. On the other hand, the availability of objective and accurate measurements of cotinine levels among parents and children on a long time span along with details on self-reported use of e-cig represents a unique opportunity to explore these issues on a representative sample of a population. Keeping in mind these features, our findings may have 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 indicate 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 likely to be justified because nicotine transmission is lower when delivered by NDP rather than traditional cigarettes.

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Tables and Figures

TABLE 1. Descriptive Statistics

Variables	2002-2012 Sample		2013-2014 Sample	
	Mean	*St.dev*	*Mean*	*St.dev*
Cotinine Children	0.91	1.74	0.49[a]	0.68
Cotinine Father	35.10	117.51	18.94	82.92
Cotinine Mother	51.34	132.46	24.67	95.84
Cotinine Parents (M+F)	86.45	190.56	43.62	136.47
Household Income	25602	21671	30414	24440
Age	9.30	3.00	9.24	3.09
Male	0.50	0.50	0.51	0.50
E-cig father			0.06	0.24
E-cig mother			0.06	0.25
Number of E-cig parents (M+F)			0.12[b]	0.37
Observations	6430		1236	

[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: 88.70%; 1: 10% 2: 1.30%

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

	(1) Simple	(2) With Controls	(3) M+F	(4) M+F With controls
Cotinine F	0.00166***	0.00137***		
	0.00022	*0.00022*		
Cotinine M	0.00510***	0.00507***		
	0.00031	*0.00028*		
HH Income		-0.00001***		-0.00001***
		0.00000		*0.00000*
Age		-0.02310***		-0.02392***
		0.00731		*0.00747*
Male		-0.06168		-0.06814*
		0.03767		*0.03834*
Cotinine M+F			0.00353***	0.00339***
			0.00018	*0.00019*
Year FE	YES	YES	YES	YES
Observations	7162	6430	7162	6430

Children Cotinine >=0 and < 15 in all regressions (objective passive smoking).
 ***, **, * indicate significance at 1%, 5% and 10%, respectively. Standard Errors clustered at household level in *Italics*
[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.

TABLE 3. Galtonian regression - pooled sample 2002-2012[a]

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Cotinine F	0.00159***		0.00178***		0.00138***		0.00137***		0.00130***	
Cotinine M	0.00483***		0.00517***		0.00497***		0.00508***		0.00509***	
Single Parent	0.49658***	0.66181***								
Cotinine M+F		0.00335***		0.00365***		0.00334***		0.00340***		0.00336***
Single Father			0.46859***	0.32952***						
Single Mother			0.31551***	0.53399***						
Degree M					-0.23016***	-0.30997***				
Degree F					-0.13670***	-0.12155***				
Age 4-7							0.20673***	0.20324***		
Age 7-10							0.00087	0.00647		
Weight									0.00581**	0.00654**
Observations	6430	6430	6430	6430	6430	6430	6430	6430	6016	6016

Children Cotinine >=0 and < 15 in all regressions (objective passive smoking). ***, **, * indicate significance at 1%, 5% and 10%, respectively. Standard Errors clustered at household level.

[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.

TABLE 4. Galtonian regression (control for activity status of the mother) - pooled sample 2002-2012

	(1)	(2)	(3)
	With Controls	**M+F**	**Interaction**
Cotinine F	0.00133***		0.00135***
	0.00022		*0.00023*
Cotinine M	0.00511***		0.00556***
	0.00028		*0.00043*
Activity Status Mother	-0.35362***	-0.32686***	-0.29560***
	0.04475	*0.04583*	*0.03986*
Activity Status M*Cotinine M			-0.00094*
			0.00054
Cotinine M+F		0.00340***	
		0.00019	
Controls	YES	YES	YES
Year FE	YES	YES	YES
Observations	6430	6430	6430

***, **, * indicate significance at 1%, 5% and 10%, respectively. Standard Errors clustered at household level in *Italics*

TABLE 5. Galtonian regression estimates - pooled sample 2013-2014[a]

	(1)	(2)	(3)	(4)
	Simple	With Controls	M+F	M+F with controls
Cotinine F	0.00210***	0.00183***		
	0.00028	*0.00028*		
Cotinine M	0.00208***	0.00212***		
	0.00030	*0.00032*		
HH Income		-0.00001***		-0.00001***
		0.00000		*0.00000*
Age		-0.03107***		-0.03080***
		0.00554		*0.00555*
Male		0.03776		0.03776
		0.03275		*0.03279*
Cotinine M+F			0.00209***	0.00199***
			0.00022	*0.00023*
Year FE	YES	YES	YES	YES
Observations	1381	1236	1381	1236

[a] Cotinine measured in three levels: 0; 0.01-1; 1-12. Standard Errors clustered at household level in *Italics*
***, **, * indicate significance at 1%, 5% and 10%, respectively.

TABLE 6. DiD intention to treat estimates - sample 2002-2012

	(1)	(2)	(3) M+F	(4) M+F
	Linear Trend	Quadratic Trend	Linear Trend	Quadratic Trend
Cotinine F	0.00124***	0.00124***		
	0.00026	*0.00026*		
Cotinine M	0.00510***	0.00510***		
	0.00032	*0.00032*		
Post 2010	0.27868***	0.28318**	0.23955***	0.21485*
	0.07109	*0.12127*	*0.07326*	*0.12612*
Post* Cotinine F	-0.00032	-0.00032		
	0.00045	*0.00045*		
Post* Cotinine M	-0.00114**	-0.00114**		
	0.00056	*0.00056*		
HH Income	-0.00001***	-0.00001***	-0.00001***	-0.00001***
	0.00000	*0.00000*	*0.00000*	*0.00000*
Age	-0.02202***	-0.02202***	-0.02258***	-0.02258***
	0.00731	*0.00731*	*0.00746*	*0.00745*
Male	-0.07149*	-0.07152*	-0.07840**	-0.07828**
	0.03792	*0.03789*	*0.03857*	*0.03854*
Trend	-0.15524***	-0.15299***	-0.14715***	-0.15951***
	0.01512	*0.05445*	*0.01546*	*0.05661*
Quadratic Trend		-0.00033		0.00179
		0.00741		*0.00772*
Cotinine M+F			0.00337***	0.00337***
			0.00022	*0.00022*
Post* Cotinine M+F			-0.00082**	-0.00082**
			0.00036	*0.00036*
Observations	6430	6430	6430	6430

***, **, * indicate significance at 1%, 5% and 10%, respectively. Standard Errors clustered at household level in *Italics*

TABLE 7. DiD estimates – sample 2013-2014

	(1)	(2)	(3)
	Simple	**With Controls**	**M+F**
Cotinine F	0.00207***	0.00179***	
	0.00036	*0.00037*	
Cotinine M	0.00263***	0.00261***	
	0.00033	*0.00037*	
Father e-cig user	-0.07960	-0.11698	
	0.08367	*0.08538*	
Mother e-cig user	0.36815***	0.38545***	
	0.13475	*0.12972*	
Cotinine F*Father e-cig user	0.00023	0.00038	
	0.00059	*0.00058*	
Cotinine M*Mother e-cig user	-0.00157***	-0.00151**	
	0.00061	*0.00064*	
HH Income		-0.00001***	-0.00001***
		0.00000	*0.00000*
Age		-0.03255***	-0.03080***
		0.00556	*0.00558*
Male		0.03811	0.03763
		0.03242	*0.03264*
Cotinine M+F			0.00231***
			0.00021
Total Adults e-cig users			0.17391**
			0.07825
Cotinine M+F*Total Adults e-cig users			-0.00066**
			0.00027
Observations	1381	1236	1236

***, **, * indicate significance at 1%, 5% and 10%, respectively. Standard Errors clustered at household level in *Italics*

TABLE 8. Placebo regressions

	Post=One year before		Post=One year before M+F	
	(1)	(2)	(3)	(4)
	Simple	With Controls	Simple	With Controls
Post*Cotinine Father	0.0003	-0.0003		
	0.0005	*0.0004*		
Post*Cotinine Mother	-0.0006	-0.0008		
	0.0006	*0.0006*		
Post*Cotinine M+F			-0.0002	-0.0005
			0.0003	*0.0003*
Linear and quadratic trend	YES	YES	YES	YES
Observations	7162	6430	7162	6430

***, **, * indicate significance at 1%, 5% and 10%, respectively. Standard Errors clustered at household level in *Italics*.

TABLE 9. DiD estimates- Augmented specifications

|	(1) ITT	(2) Actual treatment	(3) ITT (M+F)	(4) Actual Treatment (M+F)
Cotinine F	0.00157***	0.00180***		
	0.00026	0.00036		
Cotinine M	0.00490***	0.00262***		
	0.00032	0.00037		
Post	-0.22094		-0.30762	
	0.23433		0.24010	
Post* Cotinine F	-0.00055			
	0.00045			
Post* Cotinine M	-0.00101*			
	0.00057			
Cotinine M+F			0.00340***	0.00233***
			0.00022	0.00021
Post* Cotinine M+F			-0.00086**	
			0.00035	
Father e-cig user		-0.06938		
		0.27412		
Mother e-cig user		0.43867		
		0.41135		
Cotinine F*Father e-cig user		0.00044		
		0.00059		
Cotinine M*Mother e-cig user		-0.00150**		
		0.00064		
Total Adults e-cig users				0.07050
				0.23324
Cotinine M+F*Total Adults e-cig users				-0.00073***
				0.00028
Control variables	YES	YES	YES	YES
Control variables * Post	YES	-	YES	-
Control variables* E-cig use	-	YES	-	YES
Observations	6430	1236	1236	6430

***, **, * indicate significance at 1%, 5% and 10%, respectively. Standard Errors clustered at household level in *Italics*

TABLE 10. Galtonian regression and DiD - ordered probit estimates, sample 2013-2014

	(1) Galton	(2) Galton (M+F)	(3) DiD	(4) DiD (M+F)
Cotinine F	0.00333***		0.00342***	
	0.00040		*0.00072*	
Cotinine M	0.00322***		0.00425***	
	0.00032		*0.00061*	
Cotinine M+F		0.00326***		0.00403***
		0.00025		*0.00047*
Father e-cig user			-0.13042	
			0.17950	
Mother e-cig user			0.65489***	
			0.21521	
Cotinine F*F e-cig user			0.00020	
			0.00113	
Cotinine M*M e-cig user			-0.00279***	
			0.00098	
Total Adults e-cig users				0.36099**
				0.14395
Cotinine M+F* Adults e-cig users				-0.00134***
				0.00046
Observations	1381	1381	1381	1381

***, **, * indicate significance at 1%, 5% and 10%, respectively. Standard Errors clustered at household level in *Italics*.

Figure 1. Trend in e-cig and other smoking cessation methods in England

Data from Smoking Toolkit Study, Smoking in England 2007-2014
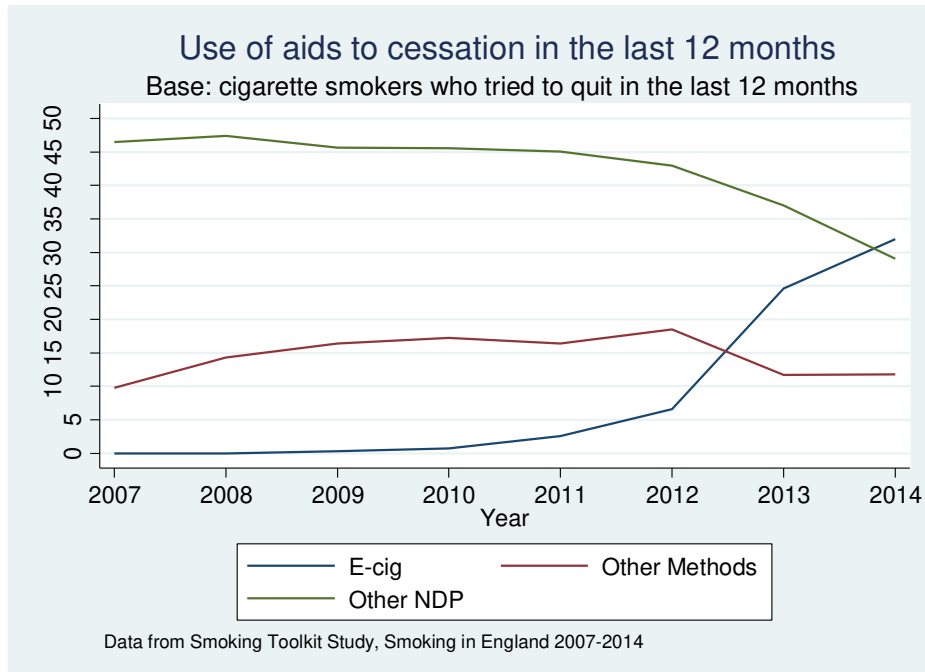
Compliance with Ethical Standards

Conflict of Interest: The authors declare that they have no conflict of interest.