(Growing) Up in Smoke? The impact of prenatal and postnatal maternal smoking on child development

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This paper estimates the causal impact of maternal pre- and post-natal smoking on child development using a joint model of maternal smoking and child outcomes at birth and age 12 months. Our empirical strategy exploits panel data and instrumental variables and uses data on children of young, disadvantaged first-time mothers in England, amongst whom the likelihood of smoking is almost twice as large as that of the average mother. We show prenatal smoking reduces birth weight and gestational age at birth and adversely affects motor and cognitive development at age 1. Maternal smoking one year after birth negatively affects socio-emotional and personal social development, language and fine motor development, as well as problem solving abilities (over and above the effect of maternal smoking during pregnancy). Failing to account for the endogeneity of maternal smoking severely underestimates the negative impacts of maternal smoking on child development, which is consistent with mothers reducing their smoking to compensate for their children's frailer endowment.

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1 Introduction

Maternal smoking constitutes a major public health concern globally (Murthy and Mishra, 2017). Evidence from a variety of disciplines suggests that exposure to tobacco both in utero and post-natally through second-hand smoke pose serious risks to children's development. Although maternal smoking has decreased over time, it remains high in many countries and exhibits a strong socio-economic gradient. In England, where the data we use in this study is collected, one in ten pregnant women still smoke at the time of delivery (NHS Digital, 2016), and this figure is at least three times as high amongst low-income and young mothers (McAndrew et al., 2012). Unsurprisingly, the public cost of smoking in pregnancy could be very high, possibly reaching £80m per annum (Godfrey et al., 2010).

In this context, designing effective policies to reduce maternal smoking could go a long way in saving public money and reducing socio-economic health inequalities. Because maternal smoking is highly persistent over time however, it is crucial that we understand the separate – and potentially compounding – impacts of prenatal and postnatal smoking on children's outcomes. Indeed, the full benefits of prenatal smoking interventions for children will depend not only on their impact on smoking during pregnancy, but also on their impact on smoking after pregnancy and the effect of postnatal smoking on children's outcomes.

The effect of fetal exposure to tobacco on birth outcomes has been established across a variety of disciplines. A number of bio-medical studies also suggest that it could also lead to developmental deficits in childhood, but most of these studies do not control for postnatal smoking (see Clifford et al. (2012) for a review). This makes it difficult to assess whether these effects capture the impact of prenatal smoking versus that of postnatal smoking (or their interactions). Moreover, while findings of this type are consistent with the possibility that maternal smoking causally affects child development, they could also reflect the impact of genetic and environmental factors that simultaneously influence maternal smoking and child development. To our knowledge, only a few studies attempt to distinguish the causal

impact of maternal smoking from these common influences, and the ones that do solely focus on identifying the effect of prenatal smoking on children's outcomes (see, for example, Del Bono et al. (2012); Lien and Evans (2005); Li and Poirier (2003); Wehby et al. (2011)).

The contribution of this paper is to estimate the impact of maternal smoking during and after pregnancy on a broad set of child development indicators at birth and age 12 months, spanning across health, cognitive, motor and socio-emotional dimensions of development. We address the problem of common unobserved influences by exploiting panel data and instrumental variables for identification. We estimate a joint model of maternal smoking and child outcomes at birth and at age 12 months, where we model the serial persistence in smoking during and after pregnancy with both state dependence and unobserved heterogeneity. We estimate the model using previously unexploited data on the participants to the Family Nurse Partnership (FNP) in England, a home visiting program that targets first time, teenage mothers amongst whom the likelihood of smoking is almost 40%. While our sample is not representative of the English population, it focuses on a group highly targeted by public intervention, thus making our analysis particularly relevant for policy.

Our results show that maternal smoking during pregnancy has adverse effects on birth weight and gestational age at birth of magnitudes that are in line with existing studies. We show that maternal smoking in utero has large and significant adverse impacts on a child's gross and fine motor as well as problem-solving skills measured at age 1. Postnatal smoking also negatively affects fine motor skills and leads to poorer socio-emotional, personal social, problem-solving and communication skills at age 1. We find no evidence that the impact of prenatal and postnatal smoking negatively reinforce each other.

We examine whether effects of prenatal and postnatal smoking depend on the intensity of smoking (rather than only on the extensive margin). To do so, we estimate a variant of our model where smoking is a three-category variable corresponding to no smoking, smoking less than a pack and smoking more than a pack in the past 48 hours. Our results confirm our hypothesis that, the more the mother smokes, the worse the effects of prenatal and postnatal

smoking are on children's development. Finally, we present a series of additional specification checks, which suggest that the innovations of our model – in terms of both modeling smoking dynamics and allowing for rich specification of unobserved heterogeneity – are important for our understanding of the impact of maternal smoking on child development.

The rest of this paper is organized as follows. Section 2 discussed the medical and economics literature on the impact of prenatal and postnatal smoking on child development and outlines the contribution of the paper to these literatures. Section 3 describes our data. Section 3 presents our econometric framework and identification strategy. Section 5 reports our results. Section 6 concludes.

2 Literature review

This section briefly reviews the bio-medical and economics literatures on the impact of maternal smoking during and after pregnancy on child development, paying particular attention to potential biological mechanisms and methodological issues related to the identification of the causal impact of maternal smoking on children's outcomes.

A vast number of bio-medical studies consistently find a link between maternal smoking in utero and children's development at birth and later in life. Specifically, pre-natal maternal smoking has been consistently associated with lower birth weight, shorter gestational age at birth, cleft lips and other health impairments at birth (Wang et al. (1997); DiFranza et al. (2004); Jaddoe et al. (2008); Mund et al. (2013)), which are known to hamper subsequent development of children (Hack et al. (1995); Case et al. (2005)). Moreover, several studies have linked pre-natal smoking to lower cognitive and socio-emotional abilities (Batstra et al. (2003); Julvez et al. (2007)) over and beyond birth outcomes [1], and a few studies report evidence suggesting that pre-natal smoking could lead to worse adult outcomes, such as reduced employment (Case et al., 2005).

[1] Batstra et al. (2003) control for birth weight and gestational length in some of their specifications. Julvez et al. (2007) control for birth weight.

Few of the studies linking pre-natal maternal smoking to children's outcomes after birth are able to control for whether the mother smokes after the child's birth [2]. Because mothers who smoke during pregnancy tend to smoke after their child's birth, part of the associations between pre-natal smoking and child outcomes after birth could reflect an adverse effect of children's exposure to tobacco smoke (ETS). Indeed, a few medical studies find that children exposed to Environmental Tobacco Smoke (ETS) have large cognitive impairments and higher rates of Attention Deficit and Hyperactivity Disorder (ADHD) and conduct disorders (Yolton et al. (2005); Yolton et al. (2008); Kabir et al. (2011)).

The medical literature is pretty clear on the biological mechanisms through which fetal exposure to tobacco constituents adversely impact fetal development. In a nutshell, prenatal smoking introduces toxins in the placenta, which in turn restrict the fetus' access to oxygen and nutrient-rich blood (Maritz (2008); Matta et al. (2007)). Less seems to be known about the mechanisms through exposure to ETS could lead to later developmental problems. Bauman et al. (1991) posit that the adverse effects of exposure to ETS could be caused by a reduction of oxygen in the brain, or by respiratory illnesses related to ETS that may in turn influence other dimensions of child development.

While many of the studies mentioned above control for a large range of parental and child characteristics, one cannot rule out that the reported effects may capture the impact of common unobserved factors. Mothers of young children who smoked during and/or after their pregnancy tend to be poorer, less educated and more likely to be single than mothers who don't. It is therefore plausible that they also systematically differ in ways that are harder to observe and measure in datasets and that matters for the child's development. For example, smoking mothers could differ from non-smoking mothers in terms of their preferences and information about child-rearing, which could in turn affect their parenting

[2]One such study is Batstra et al. (2003) who control for smoking after delivery in some of their specifications. They find that smoking during pregnancy significantly affects children's externalizing behavior and attention deficit in the early years, and this result is robust to controlling for mother's smoking after delivery. They also find that maternal smoking in pregnancy is linked to worse mathematics and spelling scores, however without controlling for post-natal smoking.

behavior.

A few studies, mostly in economics, have attempted to deal with the bias arising from mothers' selection into smoking based on unobservable characteristics. These studies have essentially used one of three main approaches. Firstly, a few studies have used instrumental variables, such as cigarette prices, tobacco taxes, and smoking bans, to exogenously vary maternal smoking behavior (Rosenzweig and Schultz (1983); Evans and Ringel (1999); Lien and Evans (2005)). On the other hand, a few other studies have used within-family estimators exploiting within-mother variation in smoking behavior between two pregnancies (Abrevaya (2006); Tominey (2007); Arellano and Bonhomme (2011)). Thirdly, Li and Poirier (2003) employ Bayesian techniques to identify the causal effect of maternal smoking in pregnancy on child outcomes at birth. To our knowledge, most papers in the economics literature have focused on the impact of maternal smoking in utero on child's birth outcomes. One exception is Wehby et al. (2011) who evaluate the effects of prenatal smoking on child development between 3 and 24 months old using the prevalence of smoking during pregnancy at the clinics, excluding the individual's smoking status as IV for maternal smoking.[3]

Our paper adds to the existing literature on the impact of maternal smoking, and more broadly on the effect of parental investments, on early childhood development in at least two distinct ways. First, our paper estimates, in the same framework, the impact of pre-natal and post-natal smoking on early childhood development and examines their potential interactions. The importance of the prenatal period in affecting a variety of outcomes, since birth and across life, is now documented in a vast interdisciplinary literature, to which economics has provided several contributions in recent years (see Almond and Currie (2011), Royer and Witman (2013), and Corman et al. (2018) for reviews). At the same time, investments occurring in the immediate postnatal period have also been shown to significantly improve

[3]While this study attempts to address the issues that are typical of bio-medical studies, such as small sample size and the inability to account for unobserved confounders, the identification strategy heavily relies on the assumption that mothers do not self-select into areas based on preferences that correlate with child neurodevelopment and that are relevant for the smoking choice. Moreover, Wehby et al. are not able to control for maternal smoke post-natally in their regressions, meaning their estimates could reflect the effect of pre-natal smoking as well as post-natal smoking and changes in behavior between the periods.

child, adolescent, and adult socio-economic and health outcomes (Attanasio et al. (2015); Attanasio et al. (2017); Campbell et al. (2014)). However, so far little work has been done to join these two literature strands and to understand the relative importance of pre- and post-natal factors in improving well-being over the lifecourse. Our paper is a first attempt to bridge this gap, by focusing on an important maternal behavior - smoking in pregnancy and post-natally.

The second contribution of our paper is to propose a new methodological approach, which circumvents a number of important identification issues in the previous literature looking at the impact of maternal smoking on children's outcomes. First, our approach relaxes the assumption imposed by within-family estimators that mothers make smoking choices independently of their knowledge (or at least partial knowledge) of their child's unobserved endowment.[4] Indeed, we allow smoking choices to be correlated with the child's unobserved endowment at birth and in early childhood so as to allow for the more realistic possibility that the mother has some (even imperfect) information about the development of her child (e.g. through scans during pregnancy and direct observation after birth) and adjust her smoking behavior accordingly.

Second, although we use cigarette prices as a source of exogenous variation for maternal smoking in the initial conditions, our identification strategy does not only rely on this source of variation, which has been often been found to be too weak in related studies to generate precisely estimated IV estimates of the impact of maternal smoking. Instead, we impose further structure on the joint distribution of unobserved heterogeneity that drives the endogeneity of smoking and exploit the availability of panel data and multiple measures of child development at birth and age 1 to identify our model, along with instrumental variables.

Economists have long recognized the importance of modeling smoking as an addictive

[4]Del Bono et al. (2012) relaxes somewhat this assumption by allowing the mother's smoking behavior during her second pregnancy to depend on her first child's birth weight. Still the mother's smoking behavior during her first pregnancy is not allowed to depend on her first child's unobserved endowment and the the mother's smoking behavior during her second pregnancy is not allowed to depend on her second child's unobserved endowment.

behavior. Consistently with the seminal model proposed in Becker and Murphy (1988), we allow for the smoking decision in a given period to depend on the addictive stock of smoking capital, which we proxy using the number of cigarettes smoked in the previous period. Only few papers in economics have modeled smoking and human capital outcomes in a joint fashion (see, for example, Adda and Lechene (2013) and Darden (2017)). Most of the empirical literature on smoking demand has focused on testing the rational addiction assumption by analyzing the effect of contemporaneus, lead and lagged prices on the demand for cigarettes (see Chaloupka and Warner (2000) for an extensive review). However, provided individuals derive utility from their (or their offspring's) human capital , they may also consider the potentially harmful impact of smoking on their (or their offspring's) health when choosing cigarette consumption. For example, Darden (2017) provides empirical evidence showing that individuals react to information from biomarkers on their own health status by changing their smoking behavior. More generally, Hai and Heckman (2015) show the importance of allowing for endogeneity in health production when modeling addictive behaviors. Our empirical application explicitly captures the potential trade-off mothers face between addiction and taste for smoking, and the detrimental effects that her smoking choice could have on her child's human capital. In particular, we explicitly allow unobserved heterogeneity affecting the mother's smoking decision to be correlated with unobserved child endowment. Thus, we allow the mother's smoking decision to change in response to what she observes about her child's development.

3 Data

3.1 The FNP programme and data

Our empirical analysis uses anonymized administrative data on participants to the Family Nurse Partnership (FNP). The FNP is a large-scale home visiting program for first-time mothers beginning during pregnancy and continuing through the child's second birthday

with three main goals: to improve the outcomes of pregnancy by helping women improve their prenatal health; to improve children's health and development by enabling parents to provide more competent care for their children; and to improve women's life course by planning subsequent pregnancies, finishing their education and finding employment.[5].

To be eligible for the FNP, mothers have to be aged 19 or under at last menstrual period, have a first pregnancy confirmed by health services (including those expecting multiple births) unless previous pregnancy ended in miscarriage/termination/stillbirth, be at less than 28 weeks of gestation, live within the catchment area covered by the local FNP team, and have no planned adoption at enrollment. Non-English speaking clients and clients with learning disabilities are also eligible. Although mothers can self-refer into the FNP program, the most common way eligible mothers enter the program is through a referral to the local FNP team from the midwife at the hospital or from their general practitioner (GP).

The data we use for the empirical analysis is drawn from data routinely collected by the family nurse during her home visits and uploaded onto the FNP Information Systems (FNP IS). These data are very appropriate to conduct our analysis, as they include questions about maternal smoking both on the extensive and intensive margins at various points in the program, as well as a rich set of assessments of child health, cognitive and socio-emotional development from birth to the age of 2. Finally, it includes rich information about the mother's socio-demographic characteristics, physical and mental health, which are potentially important determinants of both maternal smoking and child development and thus important to control in the analysis.

We use data on maternal smoking at three points in time, namely at intake (on average at 18 weeks gestation), 36 weeks of gestation, and when the child is about 12 months old. We present estimates of our model with two types of measures: one is an indicator for

[5]The program is the English adaptation of the well-known US Nurse Family Partnership (NFP), which has been shown to have consistent and enduring effects on maternal and child health in three randomized controlled trials with long-term follow-up (Olds et al., 1999). The FNP was first introduced in England in 2007 and has been progressively rolled out across the whole United Kingdom. Its impacts have been evaluated by means of a randomized controlled trial (Robling et al., 2015) and are still being widely discussed (Olds, 2015).

whether the mother has smoked at least once in the past 48 hours; the second one is a categorical variable we created based on variables asking the mother how many cigarettes she has smoked in the past 48 hours. We create three categories: 0, less than a pack (i.e. less or equal than 18 cigarettes) and more than a pack.

With respect to child outcomes, our analysis makes use of three widely used measures of child health at birth, namely birth weight (in grams), gestational age at birth (in weeks) and an indicator for whether the child required neo-natal intensive care at birth (NICU). All of these three indicators have been shown to be highly predictive of later outcomes (Almond et al., 2005; Figlio et al., 2005).

Our child's outcomes in the postnatal period are scores on the well-known Ages and Stages Questionnaire (ASQ-3) and the Ages and Stages Questionnaire - Social-Emotional (ASQ-SE) when the child is 14 and 12 months old, respectively. The ASQ-3 and ASQ-SE are widely used and validated developmental screeners for children between the ages of one month and 66 months (72 for the ASQ-SE) (Squires et al., 2015, 2009). The properties of both scales have been widely analysed and both are reported to have high concurrent and predictive validity. For example, Schonhaut et al. (2017) show that the ASQ-3 amongst children at 8, 18 and 30 months of age is highly predictive of their intelligence between 6 and 9 years of age, as measured by the Wechsler Intelligence Scale for Children-third edition (WISC-III). Since 2015, they are collected on all children in England by health visitors.

The ASQ-3 includes 6 questions for each of the five domains it covers: communication, gross motor, fine motor, problem solving and personal-social. The ASQ-SE includes 30 questions covering various domains, includign self-regulation, compliance, social-communication, adaptive functioning, autonomy, affect, and interaction with people. The FNP IS data does not report the child's score on each individual questions, but rather the child's total score on each of the 5 domains in the ASQ-3 and on the whole of the ASQ-SE. Both the ASQ-3 and ASQ-SE are completed by the mother, who is asked whether the child is able to perform particular tasks (e.g. stacking a small block or toy on top of each other, turning the pages

of a book) or how she behaves in particular situations (i.e. whether the child tries to hurt other children, adults or animals by biting or kicking). Each test takes about 10-15 minutes to complete and is then scored by the family nurse.

3.2 Descriptive statistics

While our sample is far from being representative of the English population, it focuses on a group of economically and socially disadvantaged women that is often the target of early intervention in England (and elsewhere). To build our sample, we start with all mothers who enroll in the FNP during 2010 and 2014. This is because in our analysis we employ data collected for individuals who have children that have reached at least age 1 by the time we observe them. The FNP participants can drop out from the program at any stage however, and so our analytical sample includes all mother-child pairs who have non-missing information on all variables we use in the model (and that are reported in Table 1).This results in a sample of 3,745 observations.

As is evident from the descriptive statistics reported in Table 1, mothers in our sample are highly socially and economically disadvantaged. At intake they are on average 18 years old and have been pregnant for about 18 weeks. Less than half have completed Level 2 education, which is broadly the equivalent of a high school diploma in the US, 22% of them work for pay, and around 60% report having an annual income so low that they are eligible for job seeker allowance. While 80% of them report having a partner at intake, more than half of them still live with their own mother.

Smoking rates during and after pregnancy are very high in our sample with 31 % and 45% reporting smoking at 36 weeks gestation and one year after the child's birth, respectively. This compares with rates of 23% and 30% in the Millennium Cohort Study (MCS), a representative sample of children born between 2000 and 2001 in the UK.

At birth, the children of mothers in our sample weigh 3.26 kgs on average and are born after 39.3 weeks of gestation, which is in line with the average population, as reported in the

Table 1: Descriptive statistics of the sample

	Mean	SD	Min	Max
Intake characteristics				
Age at enrolment	18.0	1.23	13.2	21.7
Child is female	0.49	0.50	0	1
Black/Asian/Mixed	0.12	0.32	0	1
Multiple birth	0.0099	0.099	0	1
Gestational age at intake	17.7	4.73	5	42
Income below 1,600 pounds per year	0.30	0.46	0	1
Income between 1,600 and 3,099 pounds per year	0.29	0.45	0	1
All income from benefits	0.32	0.46	0	1
Works	0.22	0.41	0	1
Educ level 2+	0.47	0.50	0	1
Has partner	0.79	0.41	0	1
Lives with own mother	0.53	0.50	0	1
BMI	0.40	1.23	-2.37	3.16
Mental health concerns	0.16	0.37	0	1
Physical health concerns	0.31	0.46	0	1
Positive affect score	-0.0087	1.07	-1.83	4.37
Smoking behavior				
Smoked at intake	0.35	0.48	0	1
Smoked at 36 weeks pregnant	0.31	0.46	0	1
Smoked at 12 months	0.45	0.50	0	1
Nr of cigarettes smoked in past 48h intake	4.21	8.00	0	80
Nr of cigarettes smoked in past 48h at 36 weeks pregnant	3.48	7.00	0	70
Nr of cigarettes smoked in past 48h at 12 months	6.68	10.4	0	100
Smoked one pack or less at intake	0.27	0.45	0	1
Smoked more than one pack at intake	0.079	0.27	0	1
Smoked one pack or less at 36 weeks pregnant	0.25	0.43	0	1
Smoked more than one pack at 36 weeks pregnant	0.062	0.24	0	1
Smoked one pack or less at 12 months	0.29	0.45	0	1
Smoked more than one pack at 12 months	0.16	0.37	0	1
Other behaviors				
Child was breastfed	0.52	0.50	0	1
Child received early antenatal care	0.88	0.33	0	1
Works at 12 months	0.13	0.34	0	1
Child outcomes at birth				
Birth weight	3255.3	547.2	1054	4980
Gestational age at birth	39.3	1.91	26	45
NICU	0.090	0.29	0	1
Child outcomes at age 1				
ASQ socio-emotional	13.8	11.7	0	85
ASQ communication	52.7	9.97	0	60
ASQ gross motor	54.9	11.3	0	60
ASQ fine motor	52.0	9.67	0	60
ASQ problem solving	50.5	10.5	0	60
ASQ personal social	55.0	7.91	0	60
Prices				
Lagged monthly cigarette price in pounds at intake	6.45	0.57	5.11	7.67

MCS data. Figure 1 shows the distribution of ASQ scores at age 1. As we discussed earlier,

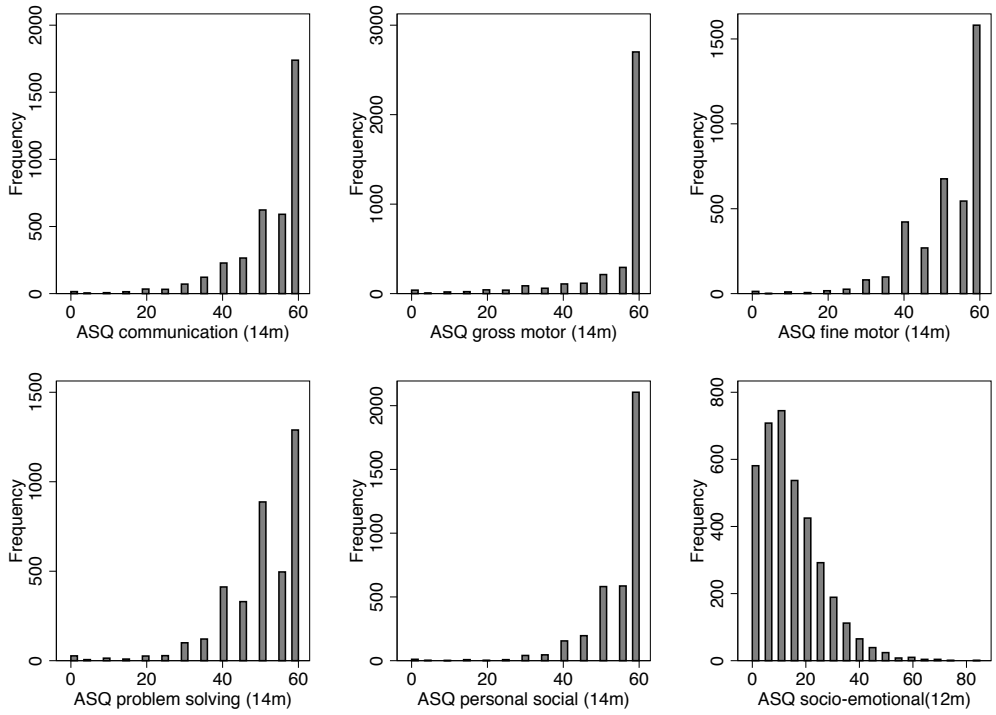

Figure 1: Distribution of ASQ scores

the ASQ is a developmental screener and its distribution is truncated, which reflects the fact that a large proportion of assessed children do not show developmental delays at this age.

4 A joint model of maternal smoking and child development in utero and early childhood

In this section we present the joint dynamic model of maternal smoking and child development that underlies the econometric analysis. We consider a first-time mother and two periods, the prenatal period, which we refer to as period 1, and the postnatal period, which we refer to as period 2. In each period, the mother makes a decision about smoking. For expositional simplicity, we focus here on the case where s is a binary choice, although we will consider smoking on the intensive margin as well in our empirical work, as we detail at the end of this section.

Let U_t be the difference between the perceived benefit and cost of smoking for the mother during pregnancy, including the opportunity costs of foregoing other goods in each period. U_t is determined by:

$$U_t = \pi_t s_{t-1} + X\beta_t + \upsilon_s + \epsilon_t \tag{1}$$

The mother chooses $s_t = 1$ if $U_t > 0$ and $s_t = 0$ otherwise. Thus:

$$s_t = 1(\pi_t s_{t-1} + X\beta_t + \upsilon_s + \epsilon_t > 0) \tag{2}$$

The net benefit of s_t depends on a set of covariates X and an unobserved mother specific component υ_s to capture that mothers have different (observable and unobservable) preferences and different costs of smoking. ϵ_t is a transitory error component for the mother in period t. The net benefit of s_t also depends on s_{t-1}, the mother's smoking behavior in the previous period. The dependence parameter π_t reflects at least two mechanisms. The first one is the effect of habit formation and addiction (consistent with a model such as Becker and Murphy (1988), in which yesterday's smoking decision affects the marginal utility from smoking today). The second one is the effect of the information provided by or actions of the nurses on the mother to influence her decision, which are likely counter to some extent the effect of addiction.

This conditional demand function is consistent with the result of an optimization process where the mother maximizes her utility over these two periods, which is a function of her consumption, smoking behavior and her child's development, subject to a financial constraint and her perception of the production function for her child's human capital at birth and in early childhood.

The second set of equations in our econometric model describes the impact of maternal smoking on the child's birth outcomes. As anticipated in the previous section, we consider three birth outcomes, namely birth weight, gestational age at birth and an indicator for

whether the child went through the hospital's neo-natal intensive care unit (NICU) at birth. We refer to these three birth outcomes as y_1^j where y_1^1 is birth weight, y_1^2 is gestational age and y_1^3 is a binary indicator for NICU. We model each of these outcomes as follows:

$$y_1^j = \lambda_1^j s_1 + X\gamma_1^j + \alpha_1^j \upsilon_1 + \xi_1^j \qquad \text{for j=1,2} \tag{3}$$

$$y_1^3 = 1(\lambda^3 s_1 + X\gamma_1^3 + \alpha_1^3 \upsilon_1 + \xi_1^3 > 0) \tag{4}$$

where the birth outcomes depend on prenatal maternal smoking s_1, a set of covariates X, an unobserved child specific component υ_1 specific to all the prenatal outcomes, and a transitory shock ξ_1^j in the prenatal period. These shocks are outcome specific as they could affect the different birth outcomes differently. The coefficient λ^j captures the causal effect of smoking in the prenatal period on each of the birth outcomes j.

The third set of equations describe the impact of maternal smoking on the child's indicators of postnatal development y_2^j. As mentioned above, we use 6 different indicators, namely the child's score in communication, problem-solving, gross motor, fine motor, and personal-social dimensions of the ASQ-3 and the child's score on the ASQ-SE. We model all of these scores as continuous variables.

$$y_2^l = \lambda_2^j s_1 + \delta_2^j s_2 + X\gamma_2^j + \alpha_2^j \upsilon_2 + \xi_2^j \qquad \text{for l=1,...,L} \tag{5}$$

where λ_2^j and δ_2^j measure the impact of prenatal and postnatal smoking respectively on postnatal outcomes y_2^j. These outcomes depend on a set of covariates related to the mother, an unobserved component υ_2 common to all of the child's postnatal outcomes and transitory shocks ξ_2^l. As with prenatal shocks, postnatal shocks are outcome specific as they could affect the different child outcomes differently.

The unobserved terms υ_1 and υ_2 can be interpreted as capturing the child's unobserved endowments as well as time-invariant unobserved inputs in her development pre- and postnatally. We allow these terms to be correlated with each other and with the mother's

unobserved endowment or preference for smoking υ_s, but we allow them to affect each of the prenatal and postnatal outcomes differently through the inclusion of factor loadings α_1^j $(j = 1, \ldots, J)$ and α_2^l $(l = 1, \ldots, L)$ respectively. For identification however, we need to set their scale by placing a restriction on one of their factor loadings. Without loss of generality, we normalize the factor loading in the birth weight and the ASQ-communication equations to 1. [6]

Since maternal smoking is dynamic and we do not observe mothers' behavior when they initiate smoking, we need to augment equations (2) for s_1 and s_2 with one equation for the initial conditions. We assume that the choice of s_0 in the first period where we observe the mother is determined by:

$$s_0 = 1(X\beta_0 + \upsilon_s + \epsilon_0 > 0) \tag{6}$$

We estimate the model by maximum likelihood. For estimation, we assume that mother specific error component υ_s and the child specific error components υ_1 and υ_2 are jointly normally distributed where the correlation between the three components are freely estimated. The transitory shocks entering the maternal smoking equations ϵ_t $(t = 0, 1, 2)$ are i.i.d and normally distributed as $N(0, 1)$. The transitory shocks entering the continuous child's birth and postnatal outcomes are normally distributed with mean 0 and standard deviation σ_t^j for birth outcomes and σ_t^l for postnatal outcomes. The transitory shocks entering the NICU outcome follow a standard normal distribution $N(0, 1)$. We integrate the residuals numerically using a Gauss-Hermite quadrature with 4 integration points.[7].

The model generalizes easily to allowing the effect of prenatal and postnatal smoking to depend on other observables, such as mother's education or income level, and to allow

[6]These normalizations set the scale of the unobserved components υ_1 and υ_2 so that the former is measured in grams and the latter is measured in ASQ points. Other normalizations would change the magnitude of the estimates the distribution of unobserved components and of the factor loadings, but they would not affect the magnitude of the causal effects of smoking we are interested in.

[7]We chose 4 integration points because the results are essentially similar if we use 5 or 6 integration points, but the computation time is obviously shorter with 4 integration points

the effect of prenatal and postnatal smoking in the ASQ equations to interact with each other. Given that we would expect the main effect of pre and postnatal smoking to have a negative effect on child postnatal outcomes, a negative interaction between prenatal and postnatal smoking in the ASQ outcomes would reflect a compounding effect of smoking in both periods. We test of all of these hypotheses and report results below.

Finally, while modeling maternal smoking on the extensive margin is a good place to start, it is plausible that children's outcomes are affected by the *intensity* of maternal smoking. Moreover, Altonji et al. (2017) show that state dependence in smoking is also higher amongst heavier smokers in a sample of adolescents in the United States. We capture these possibilities by estimating an alternative specification of our model where maternal smoking is a three category variable corresponding to no smoking, low intensity smoking and high intensity smoking, which we model as an ordered probit model. In all of the equations, we replace our dummies for whether the mother smoked in a particular period by two dummies, one for whether the mother smoked between 1 and 18 cigarettes in the past 48 hours and another for whether the mother smoked more than a pack in the past 48 hours. To our knowledge, our paper is the first to consider the impact of maternal smoking on the intensive margin, as well as on the extensive margin.

5 Results

5.1 Main results

Table 2 reports the estimates of our main specification with maternal smoking modelled as a binary outcome. The first panel of the table reports the coefficient estimates on the smoking variables and factor loadings in all of the equations of the model. The second panel reports the estimates of the trivariate distribution of unobserved heterogeneity terms $(\upsilon_s, \upsilon_1, \upsilon_2)$.

The parameters on lagged smoking in the smoking equations are large and highly significant: 2.6947 (0.0693) in the equation for smoking at 36 weeks gestation and 1.9076 (0.0624)

in the equation for smoking when the child is one year old.[8]. Thus, state dependence capturing the importance of habit formation matters a lot in explaining the serial correlation in smoking amongst these mothers. However, serial correlation in smoking is also driven by unobserved heterogeneity. The standard deviation for the component υ_s is estimated to be 0.0875 (0.0277) and highly significant as well.

Turning to the child's birth outcomes, our results show that maternal smoking during pregnancy (as measured at 36 weeks gestation) decreases birth weight by about 230 grams and gestation by 0.3 weeks. Both of these effects are significant at the 1% and in the range of existing estimates.[9] Maternal smoking in utero does not seem to significantly affect the child's likelihood to need neo-natal intensive care at birth.[10]

The last block of estimates in Panel A of Table 2 reports the impacts of prenatal and postnatal smoking on the ASQ scores. Note that all these scores have been standardized to have mean 0 and standard deviation 1 in the sample and the ASQ-SE score has been reversed so that a higher score indicates a better level of development (or a lack of developmental issues). Our results reveal a series of interesting patterns. Problem solving and motor skills (both gross and fine) are adversely affected by prenatal smoking. Fine motor and problem solving skills are also significantly adversely affected by postnatal smoking. On the other hand, communication and socio-emotional skills (measured both by the ASQ-3 personal-social and ASQ-SE scores) are only worsened by postnatal smoking. The magnitudes of these negative effects are quite large, ranging from effects between 8 and 17% of a standard deviation.

[8]Note that these parameters report the coefficients in the latent index underlying the binary decision of smoking and should not be interpreted in terms of marginal effects. The associated marginal effects are 0.765 and 0.633 respectively.

[9]For example, Del Bono et al. (2012) find that smoking in pregnancy reduces birth weight by about 190 grams. Evans and Ringel (1999) find negative effects of pre-natal smoking on birth weight in the range of 350 and 600 grams. Li and Poirier (2003) estimate the effect of smoking on gestational age to be negative, although their estimate is not statistically significant.

[10]Although Almond et al. (2005) does not use exactly the same outcome variable, they find that maternal smoking in utero decreases birthweight by 203 grams on average, hat it has only a small (but imprecisely estimated) effect on gestational lengths and no effect on other birth outcomes, including the five-minute APGAR score, assisted ventilation use after birth, and infant mortality.

Finally, we turn the estimates of the distribution of unobserved heterogeneity and the factor loadings associated with these terms in the different coefficients. First, the standard deviation of the child-specific unobserved endowment in the prenatal and in the postnatal period are large and significant: 0.4007 (0.0098) and 0.7196 (0.0159), respectively. Second, the three terms are positively correlated with each other. While we did expect that the correlation between the two child-specific components would be positive, we did not have a strong prior with respect to the sign of the correlation between the mother and the child-specific components. The fact that it is positive is consistent with the fact that mothers adjust their smoking down to compensate for their child's frailer endowment. This also suggests the fact that failing to control for this correlation would lead to under-estimating the negative impact of smoking on children's outcomes (which we confirm in Online Appendix Table **??** where we report the OLS estimates of these parameters.) Finally, the estimates of the factor loadings are all significant and of the sign that we would expect with the

child-specific unobserved components proxying for their endowment.

Table 2: Estimates of the main specification

A - Estimates of the impact of smoking and factor loadings					
	Estimated coefficient associated with:			Factor loading on:	
	Smoking at intake	Smoking at 36 weeks	Smoking at 12 months	υ_1	υ_2
Outcome variable:					
Maternal smoking					
Smoking at 36 weeks	2.6947 ***				
	(0.0693)				
Smoking at 12 months		1.9076 ***			
		(0.0624)			
Child's birth outcomes					
Birthweight		-0.2328 ***		1	
		(0.0224)			
Gestational age		-0.0324 ***		0.3382 ***	
		(0.0078)		(0.0150)	
NICU		0.0139		-1.4178 ***	
		(0.0835)		(0.1037)	
Child's age 1 outcomes					
ASQ Communication 14 m		-0.0357	-0.0761 *		1
		(0.0468)	(0.0419)		
ASQ Gross Motor 14 m		-0.1146 **	-0.0168		0.8215 ***
		(0.0500)	(0.0464)		(0.0288)
ASQ Fine Motor 14 m		-0.1112 **	-0.1289 ***		1.0872 ***
		(0.0461)	(0.0430)		(0.0308)
ASQ Problem Solving 14 m		-0.1112 **	-0.1316 ***		1.1310 ***
		(0.0467)	(0.0434)		(0.0292)
ASQ Personal Social 14 m		-0.0626	-0.0953 **		1.0044 ***
		(0.0463)	(0.0427)		(0.0323)
ASQ Socio-emotional 12 m		0.0051	-0.1747 ***		0.3253 ***
		(0.0447)	(0.0422)		(0.0260)
B - Estimates of the distribution of unobserved heterogeneity					
SD mother specific term	0.0875 ***				
	(0.0277)				
SD child birth outcome term	0.4007 ***				
	(0.0098)				
SD child post natal outcome term	0.7196 ***				
	(0.0159)				
Corr(υ_s,υ_1)	0.7775 ***				
	(0.0173)				
Corr(υ_s,υ_2)	0.6305 ***				
	(0.0207)				
Corr (υ_1,υ_2)	0.1860 ***				
	(0.0195)				

Notes: The outcome variable birth weight is measured in kilograms. The outcome variable gestational age is measured in weeks and divided by 10.

5.2 Specification checks

Having commented at length on the main results, we now turn to the results of the various specification and robustness checks we perform on our model.

Intensive margin Table 3 reports our estimates of the specification of our model where we allow children's outcomes (and state dependence) to depend on the intensity of maternal smoking. In line with the results presented in Altonji et al. (2017), the estimates support the idea that state dependence is stronger amongst heavy smokers. Moreover, the impact of maternal smoking is also worse if the mother smokes more than one pack of cigarettes every two days than if she smokes less than one pack. For example, low intensity smoking in pregnancy decreases birth weight by 220 grams, while high intensity smoking decreases birthweight by 270 grams. With respect to postnatal child outcomes, similar patterns are visible though we can only reject that the coefficients on the two indicators are significantly different from each other for the effect of prenatal smoking on gross motor skills and for the effects of postnatal smoking on communication and socio-emotional skills.

Heterogeneity Although our sample is particularly socio-economically disadvantaged, mothers in our sample do vary in terms of their education, age and income level. To examine whether results are different across these dimensions, we estimate three versions of our model where we interact in turn the coefficients on the smoking variables with maternal age, an indicator for whether the mother has achieved GCSEs or less, and an indicator for whether the mother reports having low income. We don't find evidence of heterogeneity by mother's age or mother's income level, and thus we do not report results for these estimates. Table 4 displays results for the specification estimating heterogeneous effects by mother education level. We find that having GCSEs can compensate for the negative effects of pre-natal smoking on the gross motor domain of the ASQ test. However, we do not find any significant effect of having GCSEs on any other child outcome.

Table 3: Estimates of the model with smoking intensity

A - Estimates of the impact of smoking and factor loadings								
	Estimated coefficient associated with:						Factor loading on:	
	Light smoking at intake	Heavy smoking at intake	Light smoking at 36 weeks	Heavy smoking at 36 weeks	Light smoking at 12 months	Heavy smoking at 12 months	υ_1	υ_2
Outcome variable:								
Maternal smoking								
Smoked at 36 weeks	2.2101 ***	3.0496 ***						
	(0.0724)	(0.0942)						
Smoked at 12 months			1.5284 ***	2.3008 ***				
			(0.0515)	(0.0943)				
Child's birth outcomes								
Birthweight			-0.2152 ***	-0.2708 ***			1	
			(0.0222)	(0.0415)				
Gestational age			-0.0309 ***	-0.0270 *			0.3392 ***	
			(0.0078)	(0.0138)			(0.0150)	
NICU			0.0326	-0.1338			-1.4198 ***	
			(0.0885)	(0.1810)			(0.1042)	
Child's age 1 outcomes								
ASQ Communication 14 m			-0.0089	-0.0465	-0.0527	-0.1234 **		1
			(0.0482)	(0.0863)	(0.0435)	(0.0615)		
ASQ Gross Motor 14 m			-0.0834	-0.1635 **	0.0041	-0.0561		0.8208 ***
			(0.0521)	(0.0817)	(0.0481)	(0.0650)		(0.0290)
ASQ Fine Motor 14 m			-0.0813 *	-0.1644 **	-0.1155 ***	-0.1454 **		1.0876 ***
			(0.0475)	(0.0786)	(0.0433)	(0.0622)		(0.0309)
ASQ Problem Solving 14 m			-0.0770	-0.1605 **	-0.1109 **	-0.1667 ***		1.1309 ***
			(0.0479)	(0.0785)	(0.0436)	(0.0622)		(0.0293)
ASQ Personal Social 14 m			-0.0360	-0.0356	-0.0602	-0.1765 ***		1.0036 ***
			(0.0475)	(0.0857)	(0.0431)	(0.0628)		(0.0324)
ASQ Socio-emotional 12 m			0.0638	-0.1829 ***	-0.1476 ***	-0.2226 ***		0.3231 ***
			(0.0484)	(0.0698)	(0.0440)	(0.0581)		(0.0260)
B - Estimates of the distribution of unobserved heterogeneity								
SD mother specific term	0.0758 ***							
	(0.0237)							
SD child birth outcome term	0.4003 ***							
	(0.0098)							
SD child post natal outcome term	0.7193 ***							
	(0.0159)							
Corr (υ_s, υ_1)	0.7779 ***							
	(0.0174)							
Corr (υ_s, υ_2)	0.6315 ***							
	(0.0207)							
Corr (υ_1, υ_2)	0.1866 ***							
	(0.0196)							

We also test for the presence of gender differences in the impact of maternal pre- and postnatal smoking on child development, by estimating a model in which we interact the coefficients on smoking with the child's gender. We do not find any significant differences of the impact of maternal smoking on child development by gender.

Interaction between pre- and post-natal smoking We explore the possibility that the effect of prenatal smoking is worse if the mother also smokes postnatally. To do so, we add to all child postnatal outcome equations in our main model interactions between our dummies for pre- and postnatal smoking. The results of this specification are reported in Table 5 and show that there is no evidence of such compounding effects in this maternal behavior over time.

Model with one family level unobserved heterogeneity component One of the contributions of our paper is to propose and estimate a model in which we allow the maternal response in terms of her smoking behavior to vary based on what she observes about her child's development. This is captured by the fact that we allow for three distinct levels of unobserved heterogeneity, namely the mother's smoking taste and the child endowments both at birth and postnatally, which are freely correlated. The main idea is that the mother may change her smoking behavior over time based on what she observes about her child's development at different developmental stages. Other papers in the child development literature have instead attempted to tackle the issue of endogeneity of maternal behaviors in the estimation of child development production functions by assuming one single family-level unobserved heterogeneity component which links maternal behaviors to child development (see Liu et al. (2009) and Pavan (2016)). We estimate a specification that employs this assumption to compare the results with those in our main specification. The results from a specification with a unique family-level unobserved heterogeneity component are summarized in Table 6. Our results show that allowing the maternal response in smoking to vary based on different observations of her child's development is important. Failing to do so leads

Table 4: Estimates of the model allowing the effect of maternal smoking to vary with maternal education

A - Estimates of the impact of smoking and factor loadings							
	Estimated coefficient associated with:					Factor loading on:	
	Smoking at intake	Smoking at 36 weeks	Smoking at 36 weeks X high education	Smoking at 12 months	Smoking at 12 month X high education	υ_1	υ_2
Outcome variable:							
Maternal smoking							
Smoking at 36 weeks	2.6940 ***						
	(0.0695)						
Smoking at 12 months		1.9068 ***					
		(0.0626)					
Child's birth outcomes							
Birth weight		-0.2195 ***	-0.0373			1	
		(0.0276)	(0.0371)				
Gestational age		-0.0356 ***	0.0055			0.3379 ***	
		(0.0092)	(0.0129)			(0.0150)	
NICU		0.0381	-0.0481			-1.4169 ***	
		(0.1085)	(0.1607)			(0.1037)	
Child's age 1 outcomes							
ASQ Communication 14 m		-0.0063	-0.0656	-0.1274 **	0.1018		1
		(0.0636)	(0.0917)	(0.0596)	(0.0827)		
ASQ Gross Motor 14 m		-0.2004 ***	0.1993 **	0.0052	-0.0572		0.8197 ***
		(0.0692)	(0.1000)	(0.0686)	(0.0920)		(0.0288)
ASQ Fine Motor 14 m		-0.1579 **	0.1073	-0.1117 *	-0.0484		1.0855 ***
		(0.0639)	(0.0896)	(0.0630)	(0.0846)		(0.0308)
ASQ Problem Solving 14 m		-0.1535 **	0.0967	-0.1127 *	-0.0525		1.1293 ***
		(0.0641)	(0.0907)	(0.0637)	(0.0850)		(0.0293)
ASQ Personal Social 14 m		-0.0873	0.0605	-0.1293 **	0.0634		1.0030 ***
		(0.0623)	(0.0898)	(0.0612)	(0.0841)		(0.0323)
ASQ Socio-emotional 12 m		-0.0014	0.0123	-0.1484 **	-0.0600		0.3250 ***
		(0.0608)	(0.0904)	(0.0580)	(0.0839)		(0.0261)
B - Estimates of the distribution of unobserved heterogeneity							
SD mother specific term	0.0915 ***						
	(0.0279)						
SD child birth outcome term	0.4009 ***						
	(0.0099)						
SD child post natal outcome term	0.7205 ***						
	(0.0160)						
Corr (υ_s, υ_1)	0.7777 ***						
	(0.0185)						
Corr (υ_s, υ_2)	0.6311 ***						
	(0.0212)						
Corr (υ_1, υ_2)	0.1859 ***						
	(0.0197)						

Table 5: Estimates of the model with an interaction between prenatal and postnatal smoking

A - Estimates of the impact of smoking and factor loadings						
	Estimated coefficient associated with:				Factor loading on:	
	Smoking at intake	Smoking at 36 weeks	Smoking at 12 months	Smoking at 36 weeks X Smoking at 12 months	υ_1	υ_2
Outcome variable:						
Maternal smoking						
Smoking at 36 weeks	2.6947 ***					
	(0.0696)					
Smoking at 12 months		1.9081 ***				
		(0.0625)				
Child's birth outcomes						
Birth weight		-0.2331 ***			1	
		(0.0225)				
Gestational age		-0.0324 ***			0.3373 ***	
		(0.0078)			(0.0150)	
NICU		0.0145			-1.4143 ***	
		(0.0837)			(0.1036)	
Child's age 1 outcomes						
ASQ Communication 14 m		0.0273	-0.0616	-0.0804		1
		(0.1093)	(0.0451)	(0.1191)		
ASQ Gross Motor 14 m		-0.0143	0.0069	-0.1285		0.8212 ***
		(0.1066)	(0.0515)	(0.1196)		(0.0289)
ASQ Fine Motor 14 m		-0.0545	-0.1160 **	-0.0722		1.0868 ***
		(0.1116)	(0.0460)	(0.1210)		(0.0308)
ASQ Problem Solving 14 m		-0.0503	-0.1177 **	-0.0775		1.1307 ***
		(0.1079)	(0.0464)	(0.1173)		(0.0293)
ASQ Personal Social 14 m		0.0486	-0.0691	-0.1424		1.0042 ***
		(0.0993)	(0.0465)	(0.1100)		(0.0323)
ASQ Socio-emotional 12 m		0.0996	-0.1521 ***	-0.1215		0.3254 ***
		(0.0980)	(0.0465)	(0.1099)		(0.0262)
B - Estimates of the distribution of unobserved heterogeneity						
SD mother specific term	0.0877 ***					
	(0.0277)					
SD child birth outcome term	0.4013 ***					
	(0.0099)					
SD child post natal outcome term	0.7199 ***					
	(0.0159)					
Corr (υ_s, υ_1)	0.7777 ***					
	(0.0174)					
Corr (υ_s, υ_2)	0.6296 ***					
	(0.0207)					
Corr (υ_1, υ_2)	0.1855 ***					
	(0.0196)					

to understating the detrimental effects of maternal pre- and postnatal smoking both on the child's birth outcomes as well as on the outcomes measured at age 1. The effect of prenatal smoking on birth weight, gestational age and NICU is not significantly different from what is estimated in the OLS version of the model. We do not find any significant effect of prenatal smoking on the ASQ scores. In addition to this, the effects of post-natal smoke on the ASQ scores are significantly smaller than those estimated in our main specification.

Model with no persistence in smoking We also estimate a version of the model in which we ignore the state dependence in the smoking equations, to again compare the implications of this with our main specification. The results are presented in Table 8. Failing to account for the presence of state dependence in smoking behavior leads to overstating the negative effects of smoking on child development by several orders of magnitude, compared to our main specification.

Model with prenatal smoking only Refer to Wehby et al. (2011) that does not control for the effects of postnatal smoke on child development after birth compared to our main spec.

Model with other investments One issue that we cannot directly address by means of our identification strategy is related to the possibility of omitted investments that, if correlated with maternal smoking, would bias our estimates of the effects of the latter on child development. While our strategy does not explicitly tackle the issue of omitted investments, we provide estimates of a model in which we control for several relevant maternal investments in her child's development that are observed in our data set. Namely, we augment our main specification by including a dummy for whether the child got early antenatal care (i.e. if an antenatal care visit occurred in the first 12 weeks of gestation), whether the mother worked at intake and when the child is aged 1, and whether the child was breastfed. The results are presented in Table 9. When comparing these results with those in our main specification, we

do not find any significant changes in the estimated coefficients on smoking. This suggests that, at least in terms of observed maternal investments, the omission of the latter does not bias our main results.

6 Conclusion

This paper models for the first time the production of child development as function of both pre- and post-natal investment. We have used a factor structure approach to obtain causal estimates of the effect of maternal smoking in pregnancy and at 12 months of age on child development outcomes. We have shown that failing to account for unobserved heterogeneity leads to severely biased results. The approach we have adopted has also enabled to uncover the behavioural mechanisms underlying our results. We have found that, consistently with results of prior studies, mothers tend to compensate for their children's disadvantageous conditions by selecting out of smoking when their child is frail or less developed to begin with. We have shown that maternal smoking in pregnancy has long lasting effects on child development that go beyond their impacts on birth outcomes such as birth weight. In particular, we have shown that, controlling for birth weight, smoking during pregnancy leads to substantially lower scores on the ASQ-3 and ASQ-SE tests measuring cognitive and socio-emotional skills respectively. Moreover, we have found that child exposure to smoking at 12 months can have detrimental effects on different domains capturing cognitive and non-cognitive skills. All in all, the results highlight the importance of policies that assist pregnant mothers in the process of smoking cessation. Not only does smoking in pregnancy harm child health at birth as measured by birth weight, it also has substantial negative effects on other domains of child human capital later in life. Our results also highlight the fact that inequalities and gaps in children's traits can arise as early as during the pregnancy period as function of parental prenatal behaviours.

Table 6: Estimates of model with one family-level unobserved heterogeneity factor

A - Estimates of the impact of smoking and factor loadings				
	Estimated coefficient associated with:			Factor loading on:
	Smoking at intake	Smoking at 36 weeks	Smoking at 12 months	v_f
Dependent variable:				
Maternal smoking				
Smoking at 36 weeks	2.7019 ***			
	(0.0687)			
Smoking at 12 months		1.9213 ***		
		(0.0615)		
Child's birth outcomes				
Birth weight		-0.1813 ***		1
		(0.0237)		
Gestational age		-0.0150 *		0.4861 ***
		(0.0085)		(0.1185)
NICU		-0.0377		-2.4273 ***
		(0.0759)		(0.6878)
Child's age 1 outcomes				
ASQ Communication 14 m		0.0395	-0.0297	9.6463 ***
		(0.0391)	(0.0358)	(1.4981)
ASQ Gross Motor 14 m		-0.0526	0.0208	8.0667 ***
		(0.0447)	(0.0416)	(1.2549)
ASQ Fine Motor 14 m		-0.0290	-0.0793 **	10.7348 ***
		(0.0360)	(0.0340)	(1.6571)
ASQ Problem Solving 14 m		-0.0264	-0.0785 **	10.6932 ***
		(0.0362)	(0.0343)	(1.6454)
ASQ Personal Social 14 m		0.0129	-0.0486	9.6699 ***
		(0.0386)	(0.0364)	(1.4916)
ASQ Socio-emotional 12 m		0.0294	-0.1593 ***	3.0376 ***
		(0.0436)	(0.0411)	(0.5358)
B - Estimates of the distribution of unobserved heterogeneity				
SD family level factor	0.0732 ***			
	(0.0112)			

Table 7: Estimates of the model with prenatal smoking only

A - Estimates of the impact of smoking and factor loadings				
	Estimated coefficient associated with:		Factor loading on:	
	Smoking at intake	Smoking at 36 weeks	υ_1	υ_2
Outcome variable:				
Maternal smoking				
Smoking at 36 weeks	2.7025 ***			
	(0.0692)			
Smoking at 12 months		1.9171 ***		
		(0.0620)		
Child's birth outcomes				
Birth weight		-0.2012 ***	1	
		(0.0223)		
Gestational age		-0.0217 ***	0.3371 ***	
		(0.0077)	(0.0150)	
NICU		-0.0323	-1.4132 ***	
		(0.0833)	(0.1033)	
Child's age 1 outcomes				
ASQ Communication 14 m		-0.0351		1
		(0.0396)		
ASQ Gross Motor 14 m		-0.0853 **		0.8219 ***
		(0.0402)		(0.0288)
ASQ Fine Motor 14 m		-0.1399 ***		1.0894 ***
		(0.0385)		(0.0309)
ASQ Problem Solving 14 m		-0.1394 ***		1.1318 ***
		(0.0389)		(0.0294)
ASQ Personal Social 14 m		-0.0739 *		1.0048 ***
		(0.0379)		(0.0324)
ASQ Socio-emotional 12 m		-0.0896 **		0.3274 ***
		(0.0370)		(0.0259)
B - Estimates of the distribution of unobserved heterogeneity				
SD mother specific term	0.0271			
	(0.0261)			
SD child birth outcome term	0.4007 ***			
	(0.0098)			
SD child post natal outcome term	0.7155 ***			
	(0.0158)			
Corr (υ_s, υ_1)	0.7798 ***			
	(0.0175)			
Corr (υ_s, υ_2)	0.6273 ***			
	(0.0211)			
Corr (υ_1, υ_2)	0.1864 ***			
	(0.0195)			

Table 8: Estimates of model without persistence

A - Estimates of the impact of smoking and factor loadings				
	Estimated coefficient associated with:		Factor loading on:	
	Smoking at 36 weeks	Smoking at 12 months	υ_1	υ_2
Dependent variable:				
Child's birth outcomes				
Birthweight	-0.3563 ***		1	
	(0.0289)			
Gestational age	-0.0719 ***		0.3253 ***	
	(0.0099)		(0.0151)	
NICU	0.1997 **		-1.4706 ***	
	(0.0901)		(0.1097)	
Child's age 1 outcomes				
ASQ Communication 14 m	-1.0222 ***	-0.3575 ***		1
	(0.0472)	(0.0369)		
ASQ Gross Motor 14 m	-0.9200 ***	-0.2463 ***		0.8167 ***
	(0.0523)	(0.0442)		(0.0277)
ASQ Fine Motor 14 m	-1.1903 ***	-0.4370 ***		1.0934 ***
	(0.0451)	(0.0362)		(0.0298)
ASQ Problem Solving 14 m	-1.2136 ***	-0.4453 ***		1.1183 ***
	(0.0444)	(0.0364)		(0.0276)
ASQ Personal Social 14 m	-1.0670 ***	-0.3826 ***		1.0174 ***
	(0.0441)	(0.0372)		(0.0311)
ASQ Socio-emotional 12 m	-0.3060 ***	-0.2628 ***		0.3159 ***
	(0.0505)	(0.0426)		(0.0249)
B - Estimates of the distribution of unobserved heterogeneity				
SD mother specific term	1.7850 ***			
	(0.0285)			
SD child birth outcome term	0.4022 ***			
	(0.0106)			
SD child post natal outcome term	1.0123 ***			
	(0.0221)			
Corr (υ_s, υ_1)	0.2746 ***			
	(0.0343)			
Corr (υ_s, υ_2)	0.8301 ***			
	(0.0067)			
Corr (υ_1, υ_2)	0.2744 ***			
	(0.0274)			

Table 9: Estimates of model with other behaviors

A - Estimates of the impact of smoking and factor loadings									
	Estimated coefficient associated with:								
Factor loading on:	Smoking at intake	Smoking at 36 weeks	Smoking at 12 months	Early ante-natal care	Works at intake	Child was breastfed	Works at 12 months	υ_1	υ_2
Dependent variable:									
Maternal smoking									
Smoking at 36 weeks		2.6943 ***							
		(0.0697)							
Smoking at 12 months			1.9072 ***						
			(0.0627)						
Birth outcomes									
Birth weight			-0.2314 ***		-0.0083	0.0481 *		1	
			(0.0228)		(0.0271)	(0.0248)			
Gestational age			-0.0322 ***		0.0026	0.0108		0.3385 ***	
			(0.0079)		(0.0093)	(0.0091)		(0.0152)	
NICU			0.0194		-0.0100	0.0542		-1.4243 ***	
			(0.0850)		(0.1171)	(0.1055)		(0.1047)	
Age 1 outcomes									
ASQ Communication 14 m			-0.0393	-0.0784 *	0.0137	0.0064	-0.0281	-0.0318	1
			(0.0477)	(0.0436)	(0.0508)	(0.0508)	(0.0359)	(0.0501)	
ASQ Gross Motor 14 m			-0.1169 **	-0.0183	0.0556	-0.0776	-0.0121	0.0820	0.8216 ***
			(0.0515)	(0.0482)	(0.0546)	(0.0590)	(0.0375)	(0.0640)	(0.0290)
ASQ Fine Motor 14 m			-0.1072 **	-0.1306 ***	0.0043	-0.0594	0.0741 **	0.0241	1.0870 ***
			(0.0472)	(0.0451)	(0.0505)	(0.0533)	(0.0359)	(0.0545)	(0.0313)
ASQ Problem Solving 14 m			-0.1070 **	-0.1332 ***	0.0165	0.0131	0.0352	0.0284	1.1286 ***
			(0.0482)	(0.0456)	(0.0501)	(0.0535)	(0.0356)	(0.0536)	(0.0295)
ASQ Personal Social 14 m			-0.0628	-0.0970 **	0.0271	-0.0516	0.0169	0.0340	1.0035 ***
			(0.0479)	(0.0450)	(0.0488)	(0.0521)	(0.0361)	(0.0520)	(0.0328)
ASQ Socio-emotional 12 m			0.0095	-0.1743 ***	0.0090	0.0348	0.0154	0.0555	0.3232 ***
			(0.0450)	(0.0425)	(0.0516)	(0.0504)	(0.0345)	(0.0571)	(0.0263)
B - Estimates of the distribution of unobserved heterogeneity									
SD mother specific term		0.0894 ***							
		(0.0282)							
SD child birth outcome term		0.4002 ***							
		(0.0100)							
SD child post natal outcome term		0.7204 ***							
		(0.0162)							
Corr (υ_s, υ_1)		0.7775 ***							
		(0.0175)							
Corr (υ_s, υ_2)		0.6306 ***							
		(0.0210)							
Corr (υ_1, υ_2)		0.1861 ***							
		(0.0198)							

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