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Maternal Smoking During Pregnancy and Early Child Outcomes

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Abstract
We estimate the harm from smoking during pregnancy upon child birth outcomes, using a rich dataset on a cohort of mothers and their births. We exploit a fixed effects approach to disentangle the correlation between smoking and birth weight from the causal effect. We find that, despite a detailed set of controls for maternal traits, around one-third of the harm from smoking is explained by unobservable traits of the mother. Smoking tends to reduce birth weight by 1.7%, but has no significant effect on the probability of having a low birth weight child, pre-term gestation or weeks of gestation. Exploring heterogeneity in the effect on birth weight, it is mothers who smoke for the 9 months of gestation that suffer the harm, whereas there is an insignificant effect for mothers who chose to quit by month 5. Additionally, there is evidence of potential complementarity in investment of human capital, as the impact on birth weight of smoking is much greater for low educated mothers, even controlling for the quantity of cigarettes they smoke. We suggest policy should target the low educated mothers, offering a more holistic approach to improving child health, as quitting smoking is only half of the battle.

Keywords: Smoking, Pregnancy, Child Health, Birth Weight.
JEL Classifications: I12, I18, J13, J24

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Introduction

Maternal smoking during pregnancy remains prevalent in many countries, despite decades of research testifying to the harm it imposes upon the unborn children[2]. Since the 1998 "Smoking Kills" White Paper identified maternal smoking cessation as a target of future policy in the UK. A National Helpline telephone service was launched which helps pregnant mothers to devise a plan to quit smoking. However, as one in five mothers still smoke whilst pregnant in the UK, the current support does not seem to be having a strong impact on pregnant mothers. To better understand how policy can be targeted to pregnant mothers, we aim to paint a picture of exactly how smoking during pregnancy lowers child health at birth. We go further than estimating the causal impact, by additionally exploring the heterogeneity and dynamics of this effect across SES and across the duration of the pregnancy.

We focus on the harm from smoking upon four pregnancy outcomes: child birth weight, the incidence of having a low birth weight (LBW) child[3], weeks of gestation and whether the birth was pre-term[4]. As in many studies of this kind, smoking may harm these outcomes not just through the scientific (or causal) route, but through endogeneity inherent in the choice to smoke during pregnancy. Consequently, identification requires techniques to separate the confounding influence of the mother's traits from the causal parameter of interest. One of the contributions of this research is in the unique methodology adopted to confront this issue, by adopting a mother fixed effects approach.

There exists a large epidemiological literature examining the harm from smoking upon a range of child heath outcomes, adjusting for a range of characteristics of the mother such as her education and age at birth[5]. A snapshot of some of the results is that smoking lowers birth weight by 5.7% (Carter *et al* 2006), that this harm accumulates throughout the months of gestation (Hebal *et al* 1988), but that the effect on weeks of gestation is generally of much smaller magnitude than on birth weight (Kramer 1987).

The link between smoking during pregnancy and early child health has also been investigated in the economic literature, in which study designs use econometric techniques which explicitly incorporate smoking as an endogenous choice of the pregnant mother. Evans & Ringel (1999) exploit within state variation in taxes on cigarettes in the US between 1989-1992 as an instrumental variable (IV), finding that smoking during pregnancy lowers birth weight by 356-594 grams, (or 10.6-17.6% of average birth weight in their dataset). The

[2] For a review of the literature, see for example the US Surgeon General's report 1977-78.
[3] LBW babies, weighing less than 2500g, are susceptible to infant death, short- and long-term health problems and raise large hospital costs (Almond, Chay, Lee (2005).
[4] classified as <38 weeks gestation.
[5] See Kramer (1987) and Floyd *et al* (1993) for reviews.

fact that the IV estimate which is higher than their OLS estimates and far higher than many of the epidemiological estimates suggests that the parameter identified may be a Local Average Treatment Effect (LATE). In this case, the variation in behaviour is identified from marginal smokers enticed to change their behaviour as a result of the small tax changes across time and states. Compared to the average, these mothers will have a higher marginal benefit from smoking. This parameter is interesting to policy makers considering an increase in taxes on cigarettes, but less so for a general policy to improve child health outcomes.

Lien & Evans (2005) avoid the problem of estimating LATE by exploiting the introduction of one-off tax hikes in four US states between 1992-1994, which were sizeable enough to change the behaviour of a relatively large group of individuals. The authors estimate that smoking reduces birth weight of the child by 189 grams (or 5.6% of the average birth weight across the 4 states), which was of a similar magnitude to the OLS estimation.

Unfortunately, tax changes may not be a strong instrument for measuring the dosage of cigarette smoked inhaled by the mother. Adda & Cornaglia (2006) explain that, whilst individuals may respond to the higher taxes by cutting down the number of cigarettes they consume, they can top up the level of nicotine by inhaling with a greater intensity and smoking the cigarette right down to the filter. Therefore the instrument used in the above papers is hard to interpret, if there is not a clear cut change in the dosage. This would explain, for example, why the OLS estimates in Lien and Evan's paper were of similar magnitude to IV estimates.

Almond, Chay & Lee (2005) adopt a propensity score matching approach to investigate the effect of smoking during pregnancy upon the probability of having a LBW baby and the duration of gestation. They use birth records from Pennsylvania between 1989-1991, and find that smoking during pregnancy increases the incidence of LBW by 3-4%, but has little effect on gestation. The problem with a propensity score matching technique is that it addresses selection into smoking based upon observable, not unobservable traits of the mother. Therefore, it is possible that the estimate of the harm from smoking is not identified in this study.

The first dimension along which we contribute to the literature is in our identification methodology, which is unique to this question. Utilizing the UK National Child Development Study, we exploit a dataset with detailed information on the mother, allowing us to control for the potential source of endogeneity through variables including grandparent smoking habits during adolescence, maternal birth weight and paternal smoking habits.[6] Further, to eradicate any remaining unobservable elements, we adopt a fixed effects

[6] To our knowledge, no former study has controlled for paternal smoking habits

approach, differencing out any mother level component which is fixed across time, whilst controlling additionally for time-varying traits to allow the mother's behaviour to adapt to health trends. This is likely to make a difference as, for example, Currie et al (2007) find in the UK that much of the variation in child health stems from unobserved family effects.

The second contribution of our paper is that this is the first study in the economics literature to evaluate the harm from smoking within the UK. This is important, as for example Banks *et al.* (2006) find evidence that the effect of poor health accumulates differentially in the USA and the UK. Specifically, they show that health gradients across socio-economic status are stronger in the US, with those at the bottom of the income distribution suffering more in the US than in the UK. For this reason, this study is necessary to gain a full picture of the harm from smoking.

Finally, we are able to provide evidence not just on the harm from smoking, but to explore heterogeneity in the harm from smoking upon early child health. We examine whether the mother can undo some of the harm from smoking if she quits smoking during pregnancy. We categorize mothers in the study into those who never smoked during pregnancy, who smoked for the first 5 months and quit and those who smoked consistently for the entire pregnancy. It is often thought that the first few months, in which the foetus develops its skeleton and organs, are the most important in terms of maternal behaviour. However, using birth weight as the outcome, we may observe a different effect, as it is during the final 20 weeks that the baby gains most of its body weight, which may lead us to expect the final months of gestation to be the most important, with respect to smoking behaviour. This is a useful contribution to the policy debate of maternal smoking, as there is often a time lag between the mother becoming pregnant and learning of the pregnancy.

We are also able to look at potential complementarities in investment by considering the harm from smoking separately for low- and high- educated mothers. Complementarity in investments in this framework would mean that later investments are more productive for relatively high earlier investments. In this case, high educated mothers may be able to offset some of the harm.

The impact of maternal smoking during pregnancy on child outcomes is policy relevant because birth weight gestation, have been shown in the literature to drive a range of short- and long- term outcomes. The return to birth weight is estimated for one year mortality, education and wages (Black *et al* (2007)), self-reported health and earnings (Currie & Hyson (1999)) and the input foetal growth[7] estimated to drive educational outcomes (Behrman & Rosenzweig (2004)). If we find that smoking during pregnancy does indeed

[7] Estimated as birth weight divided by gestation

lower pregnancy outcomes, then policy makers could use birth weight and gestation as tools for raising not just health, but economic prospects for individuals.

A brief summary of results is that the effect from smoking during pregnancy is surprisingly uncorrelated with our detailed set of mother level variables. We find in the conditional OLS regressions, that smoking during pregnancy lowers child birth weight and gestation by 5.6% and 0.188 weeks on average, respectively and, increases the probability of a LBW and pre-term birth by 4.2% and 1.7% respectively. However, once we condition for remaining unobservable traits of the mother in the fixed effects estimate, only the birth weight effect remains significant, falling to 1.8%. As unobservable traits are very important in identification of the harm from smoking, quitting smoking is only half of the battle. The results suggest that in order to alleviate inequalities in child health, a more holistic approach is essential, to adapt behaviour not just on smoking but in other dimensions such as nutrition and exercise.

Focusing the remaining analysis on birth weight to understand how the harm accumulates, we find that contrary to beliefs that the greatest harm to the baby will be borne during the first trimester of pregnancy (weeks 1-12), a negligible effect on birth weight to smoking during pregnancy exists if the mother quits smoking by the fifth month of the gestation period. This result is intuitive given our chosen child outcome of birth weight, because it is during the final 20 weeks that 90% of the growth of the child occurs. This is a positive result, suggesting that government policy could be effective if aimed at mothers not just during the first trimester of pregnancy, but into the second. The message is that it is never too late to quit.

Finally, we find evidence of strong complementarities in investment of human capital, as it is the low educated mothers who bear the greatest burden of smoking, even conditioning on the quantity of cigarettes consumed.

The paper is structured as follows. Section 2 and 3 we present our estimation strategy and the dataset. Section 4 and 5 report and discuss the results and a robustness check, section 6 discusses the magnitude of the results. Finally, section 7 concludes.

2 Empirical Methodology

A production function for human capital h of child c, to mother m, is detailed below[8].

[8] Although the specification is linear, we can allow for polynomial and interaction terms, to explore a more flexible relationship between inputs and child outcomes.

$$h_{cm} = \beta_0 + \beta_{1m} S_{cm} + \beta_2 X_{cm} + \delta_m + u_{cm} \quad \textbf{(1)}$$

where outcomes h = {log birth weight, LBW, gestation, pre-term birth}.

S is a dummy variable which takes the value 1 if the mother smokes during pregnancy and 0 otherwise. β_1 is the parameter of interest in this model, the harm from smoking during pregnancy upon child human capital. Notice that β_1 has an m subscript to allow for heterogeneity in the effect. We control additionally for mother specific characteristics which vary across births (X), discussed further below. u denotes the latent unobservable term.

δ is the maternal component of child human capital which is assumed unobservable and fixed across time. The econometric issues with the estimation lies in disentangling β_1 from δ. The problem arises from the existence of two mechanisms through which smoking during pregnancy is likely to lower child health.

Firstly is a causal mechanism. Tuormaa (1995) describes how child birth outcomes are driven by maternal smoking. When a mother smokes during pregnancy, nicotine causes the flow of blood between the uterus and the placenta to slow. Additionally, the intake of carbon monoxide transforms oxygen carried to the foetus, into carboxyhaemoglobin. The result is that smoking during pregnancy causes foetal hypoxia – or low levels of oxygen – which slows foetal growth and reduces gestation. Note however that, as Floyd *et al* (1993) point out, "This lower birth weight results primarily from intrauterine growth retardation (IUGR), observable at all gestation ages" (pp. 381), therefore we may expect the effect on birth weight to be stronger than that for gestation.

Secondly, there may be traits of the mother which drive her to smoke during pregnancy whilst simultaneously choosing other behaviours which lower the health of the child. Smoking habits are easily predicted by socio-economic status (in terms of education and age at birth)[9] and family background. If the selection into smoking is also driven by unobservable traits of the mother which lower birth weight and gestation, then estimates of the harm from smoking during pregnancy will be prone to bias. Consequently, if this were the case, efforts to entice smoking mothers to quit during pregnancy would have less effect on the health of their child than conventional estimates suggest.

Intuitively, we can classify three potential sources of bias. Firstly, it may be the case that mothers who smoke during pregnancy have relatively high discount rates, or favour risky

[9] In the 2000 Millennium Cohort Study, smoking mothers were on average 2.6 years younger and had 1.29 fewer years if education than non-smoking mothers.

behaviour during pregnancy. This creates an omitted variable bias in our estimate of the harm from smoking, if this difference leads to lower child health independently of the smoking habits, for example through inducing other adverse behaviour. Secondly, smoking may be correlated with the health endowment of the mother. Rosenzweig & Schultz (1983) raise the issue of endogeneity of health inputs in the child health production function. They conjecture that individuals have superior information on the expected health of their children, which may cause adverse selection. For example, if the mother has a low endowment, she may increase prenatal care in anticipation of her child's poor endowment. So a potential bias may exist in our estimates of the harm from smoking, through complementarity in investment of human capital. Finally, to the extent that parents assortatively mate by health behaviour, paternal smoking habits will be an important control in the child health production function, to identify the causal effect of maternal smoking. If smoking mothers tend to live with smoking fathers, then we will overestimate the harm from the maternal smoking effect itself, by picking up the harm from passive smoking.

When estimating the harm from maternal smoking upon child human capital, we want to take into account the endogenous decision of the mother to smoke during pregnancy. We adopt two approaches to absorb the endogeneity, δ.

Firstly, using an OLS regression, we proxy for the potential sources of bias discussed above by controlling for a wealth of information about the mother, spanning her lifetime. Using the National Child Development Study, we include variables which aim to capture the discount rate, or attitudes towards healthy behaviour of the mother, such as whether she regularly exercises or drinks over the recommended alcohol limit as well as whether her parents smoked when she was aged 16. We also include a set of measures of the endowment of maternal human capital, which are often unobserved in other studies within this literature. These are the birth weight of the mother and her height at age 16. As mentioned above, we have information additionally on the smoking behaviour of the child's father. To the extent that parents assortatively mate by smoking habits, this acts as an additional control for the endogeneity.

Using this methodology, the estimate β_1 will be unbiased if $E(\partial_m + u_{cm} \mid S_{cm}, X_{cm}) = 0$

We can never guarantee that we have adequately captured the endogenous smoking behaviour of the mother with the above methodology. Therefore we secondly exploit information on sibling births, for all mothers with more than one child, estimating a fixed effects model. A fixed effects model is more appropriate than a random effects model in this context, to allow the possibility of a systematic relationship between maternal smoking during pregnancy and the time constant maternal term - the potential source of endogeneity.

The within-group estimator will eliminate δ_m which is common across different births to the same mother. The regression below shows the difference equation which is estimated for each mother in the fixed effects approach.

$$h_{2m} - h_{1m} = \beta_{1m}(S_{2m} - S_{1m}) + \beta_2(X_{2m} - X_{1m}) + (u_{2m} - u_{1m}) \quad \textbf{(2)}$$

To allow causal interpretation of the parameter β_1 we assume $E(u_{2m} - u_{1m} \mid S, X) = 0)$.

Notice that this assumption is violated if the mother adapts her smoking behaviour along with other behaviours, which will independently drive child human capital. This potential problem is exacerbated in our dataset where the mothers are a birth cohort. Across time, health trends have determined that fewer women smoke during pregnancy. Therefore mothers may be less likely to smoke for later births. Simultaneously, the cohort nature of the dataset ensures that for later births, the mothers are older than in earlier births, therefore may cope better with the pregnancy, independently of their smoking habits. This is a scenario in which the assumption above may fail. However, we control for time-varying health behaviours, such as whether the mother drinks over the alcohol limit at different points in time, to pick up such health trends. More rigorously, in Section 5.2, we use a dataset of Norwegian births, where within each year mothers are a cross section of the population, enabling us to avoid this problem. By including interaction dummies between the mother's age and the year of birth, we test the extent to which health trends and maternal age drive our results. We find no significant difference in our estimates from omission of this information.

Another potential problem is that identification using the fixed effects model stems from mothers whose smoking behaviour changes during pregnancy. This means that outcomes for mothers who smoke during pregnancy for all births, or for no births do not create the variation which generates the estimates. It is possible that these group of mothers (called 'non-changers' for the remainder of the paper) will look different to the "changers". To understand that extent to which this is true, we estimate the OLS additionally for the group of changers, which we compare to the fixed effects estimate to understand the true extent of endogeneity in the smoking parameter.

3 Data

We use two datasets in this paper. For the bulk of the analysis, the UK National Child Development Study (NCDS) and for some robustness checks, a Norwegian administrative dataset.

The NCDS is a longitudinal panel dataset, whose participants are the cohort of children born in the UK between 3-9 March 1958. The most recent period of observation we use from the study was in 2001, which gives over 40 years of information on the cohort members. It is the information on children of the cohort members which we exploit for this study, using the in-depth pregnancy information on siblings. The children in our sample are born between 1973-2000.

Our sample includes 3368 female cohort member mothers and 6860 children[10]. 2799 of these mothers (and 6291 children) had more than one child, which is necessary for the fixed effects approach, and therefore will be our chosen sample.

An advantage of the panel data nature of the NCDS over other data sets with pregnancy information, is that it allows us to observe in-depth smoking habits of pregnant mothers. We observe the incidence of smoking during pregnancy and whether the mother stopped smoking by the fifth month of gestation.

The birth outcomes of the children of the cohort members are very detailed. Our measures of child human capital are log birth weight, whether the child was low birth weight (LBW), weeks of gestation and whether the birth was a pre-term birth (<38 weeks). Note that gestation is believed to be measured with lower accuracy than birth weight, due to errors in identifying the exact date of conception. Therefore we focus on the birth weight results We observe additionally child gender, which will be an important control as boys tend to be heavier than girls at birth.

Given that the cohort members have been tracked in the NCDS since birth, we are able to incorporate a wealth of information on the mothers which will drive their smoking behaviour and form important inputs into the child human capital production function.

To proxy for the mother's inherent discount rates, or attitudes towards health, we control for the grandfather's social class at the date of birth of the mother, as well as her parents' smoking habits when she was aged 16. Further to this, it is important to allow for health traits of the mother which are time-varying as, across births, the mother may change her behaviour according to health trends, or experience. Therefore, we control additionally

[10] we restrict the sample to families with fewer than six children, to eliminate outliers

for whether the mothers exercise regularly and drink over the recommended allowance of alcohol, at the period of observation most recent to the birth of the child.

As explained in our model, the endowment of the child is partly driven by maternal health endowments. We proxy for the endowment, using information on the mother's birth weight observed directly in the first wave of the NCDS and additionally the mother's height at age 16. To my knowledge, no other study looking at the impact of maternal smoking upon child birth outcomes has controlled directly for such health inputs of the mother. However, it is a very important control in the child production function for three reasons. Firstly, Conley & Bennet (2000) report the impact of maternal birth weight upon the probability of having a LBW child. Using PSID sibling data between 1968-92, the authors find LBW of the mother increases the probability of the child being LBW by a factor of four. Secondly, it is important to control for endowments in child human capital as for example, low birth weight may not be a negative outcome, rather a genetic trait. Finally, if mothers adapt their behaviour in response to their own health endowments, the result of insufficient control along this dimension will produce a bias in our estimates of the birth weight effect of maternal smoking.

We take into account whether the mother lives with a smoker. We may expect living with a smoker to drive child birth weight, through a direct and an indirect channel. Firstly, through the indirect channels, to the extent that partners do assortatively mate by "healthy behaviour", controlling for the mother living with a smoker will in part absorb the endogeneity inherent in the mother's smoking status during pregnancy. Further, the event of living with a smoker may drive motivation for the mother to quit smoking, therefore the control will allow a more accurate description of the mechanisms through which mothers smoke during pregnancy. The direct channel is through passive smoking. We will be able to partly disentangle these two effects as the fixed effect should eradicate the indirect mechanism, leaving only the passive smoking effect.

Black *et al.* (2005) find that birth order is an important determinant of child outcomes, thus this will be a control in our data. It is additionally important however, given our identification as, if for example mothers decide to smoke for the first child but not for the second, then we may attribute the change in health to smoking behaviour when really, it is the birth order effect that is being picked up. We control additionally for marital status, ethnicity education of the mother and the age of mother. The descriptive statistics for this dataset are reported in the section below.

Additionally, we use a unique dataset with merged administrative and birth records in Norway, between 1999-2003. We create our dataset by selecting all births in the period, which gives us precise information on birth weight plus other birth inputs such as the age of

the mother at birth and smoking habits. Further, for all births during this period, we use unique identifying codes and merge onto this information the education of the parents and the birth weight of parents additionally. The details of the Norwegian dataset are discussed in section 5.1.

4 Results

4.1 Descriptive Statistics

Summary statistics are reported in Table 1. The total number of children born to female cohort members in our sample is 6291. Of these, 68% were born to non-smoking mothers, 6% born to mothers who smoked for the first start, but quit by month five and 26% of the mothers smoked consistently for the duration of the pregnancy, with a noticeable difference for children born to mothers who smoked for the full gestation period. Child birth weight tends to fall with the duration of the smoking habit during pregnancy. The mean birth weight for non-smoking births is 239g heavier than the mean for consistently smoking births and the probability of having a low birth weight child is 0.6 percentage points higher. There is no large difference in the mean gestation period, however pre-term births (<38 weeks) are more likely for the smoking mothers. Smoking during pregnancy is not random across observable mother characteristics, as smoking mothers give birth at an earlier age, have more children, leave school at a younger age and are three times more likely to live with a smoker and have parents who smoked when the mothers were aged 16. All in all, these statistics signify that mothers smoking during pregnancy tend to have less advantageous outcomes.

It is important to understand mothers' smoking behaviour across births, as our identification exploits variation within mothers, across siblings. Table 2 shows the transition matrix for three child families. Within each cell, the top and bottom values represent the column and total percentages respectively. For each child, we calculate whether the mother smoked during pregnancy (=1) or not (=0). Looking at behaviour across the first and second births, we can see that smoking habits are very persistent. Of the mothers smoking (not smoking) for the first birth, 78% (89%) smoke (do not smoke) for the second. Similarly, of the mothers refraining from smoking for the first, only one in ten choose to smoke for the second birth. The numbers are similar for comparisons in the remaining cells and suggest that mothers tend to stick to their smoking habits across births. However, there is variation in

habits and, if they do change behaviour, they are more likely to quit smoking than to start smoking on average.

4.2 Regression Results

Table 3 reports the estimated coefficients and standard errors for the explanatory dummy variable of maternal smoking during pregnancy, for the four pregnancy outcomes. All regressions control for child year of birth dummies, to absorb health trends in smoking habits and we cluster at the level of the mother [11]. Moving from column 1 to 5, we sequentially include sets of controls, in order to examine the independent correlation with smoking during pregnancy and birth weight. The raw harm from smoking during pregnancy is a reduction in child birth weight by 5.8%, an increase in the probability of having a low birth weight child by 4.5%, a reduction in gestation by 0.224 weeks and raises the probability of having a pre-term birth by 2.3%. All of these estimates are significantly different to zero.

We would expect that the inclusion of additional controls will reduce the magnitude of this estimate, if crudely speaking, unhealthy mothers smoke and give birth to lighter children. In column 2, we control firstly for a set of standard child health production function inputs. The coefficients either increase or have no effect on the effects, indicative that on average, omitting these controls led to an upward bias, contrary to our expectations. This result is driven mainly by the control for birth order, which appears positively correlated with child birth weight and also with maternal smoking habits. That is, conditional upon traits such as the mother's age and education, mothers tend to smoke more for later births.

Moving across the table, as we add further controls up to column 5, the estimates for the harm from smoking fall in magnitude, as we would expect. However, each set of controls reduces the effect only slightly. For example, including the set of health and endowment controls, which are usually very difficult to observe, reduces the effect on birth weight by 0.2 percentage points. Information on the partner's human capital and smoking behaviour are important controls for the estimate of maternal smoking however, and have the largest impact on the latter estimate. Smoking during pregnancy now reduces log birth weight and weeks of gestation by 5.6% and 0.188 weeks respectively, and raises the probability of giving birth to a LBW and pre-term births by 4.2% and 1.8% respectively. Controlling for a detailed set of background traits of the mother, including whether her parents smoked during her adolescence, has no significant impact upon the harm from smoking during pregnancy.

[11] The full set of results can be seen in Appendix 1.

Although the first 5 columns of Table 3 control for a particularly detailed set of traits of the mothers in our dataset, we can never be certain that the endogenous decision to smoke during pregnancy has been fully absorbed. Therefore, in column 6, we estimate a mother fixed effect model to difference out any inherent healthiness or endowment of the mother which may have been creating an omitted variable bias in the previous estimates.

We first analyze the estimated coefficients for the dependent variable log birth weight. Row 1 shows the harm on log birth weight has fallen dramatically, from 5.6% to 1.7%, a difference which is statistically significant. This suggests that two thirds of the harm from smoking upon child birth weight is driven by unobservable traits of the mother.

Initially, this may suggest that the OLS estimates are prone to bias from the inability to observe characteristics of the mother which is fixed over time, but clearly very correlated with birth weight and her decision to smoke whilst pregnant. This is somewhat surprising, given the wealth of controls we had incorporated into our final OLS estimate – from the choices of the mother regarding her education and age at birth, measures of her health endowment and also behaviour of her family spanning her lifetime. Therefore, we now seek to understand exactly what the fixed effects estimate is estimating.

The fixed effect estimate will exploit variation in the smoking behaviour of each mother across her multiple births. That is, unless the mother changes habits (we will call her a changer) from one birth to another, she will not influence the estimate of the harm from smoking.

Within a heterogeneous returns model, the harm from smoking for changers may differ than that for mothers whose habits persist (non-changers). The fixed effect estimate, rather than estimating the harm from smoking controlling for unobservables may instead estimate this effect for changers. In column 7 therefore, we estimate the OLS or linear probability regressions (for log birth weight and gestation, then LBW and pre-term births respectively) for a sample of changers, with the full set of controls akin to column 5. The number of observations has fallen accordingly, to 734. As expected, the harm from smoking, excluding those mothers who smoke consistently across births, is lower than the previous estimate in column 5. Comparing the harm from smoking upon log birth weight, in the fixed effects model and the OLS for changers, we see that looking at the appropriate comparison group, one third of the harm is owing to unobservable traits – half the size of the originally estimated bias. However the OLS bias is still large and the fixed effects estimate is significantly different to the OLS estimate for the changers.

Column 6 shows that the fixed effects regressions are insignificantly different to zero for all three remaining outcomes. Comparing these coefficients to column 7, there is small and only just significant impact upon the probability of pre-term gestation, for the group of

changers, increasing the probability by 3%, which compares to 1.8% for the full sample and 1.4% in the fixed effects regression.

The conclusion to draw from these figures is that giving up smoking is only half of the battle. In order to raise child health, a more holistic approach is necessary, to understand other behaviours of the mother which could be improved during pregnancy.

For the remaining analysis, we restrict our attention to the outcome log birth weight, as fixed effect estimates and regressions for the changers produce insignificant results for the other three outcomes. It is interesting to briefly consult the estimates in the birth weight equation, of other inputs into the child production function, in columns 1-3 of Appendix 1. We see that boys are heavier than girls, that the mother's education, age and marital status birth, exercise and drinking habits are all insignificant in these conditional regressions. What is interesting, is the difference between the coefficient for living with a smoker, in the OLS and the fixed effects regression. Section 3 outlined that living with a smoker may have an effect on child birth weight firstly through passive smoking and secondly by changing the behaviour of the mother. Additionally, controlling for a variable for whether the mother lived with a smoker may condition for endogeneity of the mother's choice to smoke, if there exists assortative mating by health behaviour. We can see that whilst in the OLS regression, living with a smoker lowers child birth weight by 2.1%, there is no significant effect once we control for the mother fixed effect, suggesting that passive smoking plays only a small role in the transmission of poor health to the foetus. Indeed, this finding is supported by scientific evidence. For example, Jarvis *et al*. (2001) have found that the cotinine concentration for passive smokers is 0.6-0.7% that of smokers.

4.3 Heterogeneity

4.3.1 Duration

The next stage of our analysis is to explore heterogeneity in the harm from smoking during pregnancy. Results are reported in Table 4. We first estimate an OLS, conditional upon the full set of controls from column 5 of Table 1, then in column 2 run OLS for the group of changers and finally in column 3, estimate a fixed effect model.

In the first regression of Table 4, we examine possible non-linearity in the duration of pregnancy the mother smoked for, as scientific studies suggest that if mothers give up smoking, the month they quit may be important for the health of their child. The detailed

information in the NCDS allows us to change the explanatory variable to dummies for the mother not smoking, smoking during the first 5 months and for the entire 9 months of pregnancy.

In all three specifications, we estimate a negligible birth weight effect of smoking during the first five months, relative to non-smokers during pregnancy. This result is replicated in scientific research where, for example, Hebal *et al*. (1988) find that women quitting smoking during pregnancy bear no harm of the smoking on birth weight.

This result could be due to two factors. If we consider the stages of growth and development of the foetus during the pregnancy, it is in the first trimester of pregnancy (weeks 1-12) that the baby develops facial features, limbs, heart and organs. During the second trimester (weeks 13-28) the baby strengthens and grows. 90% of weight growth occurs from week 20 onwards.

Of course, it is important to note that the group of quitters is possibly an endogenous one, with unobserved heterogeneity that not only led to them quitting smoking during pregnancy, but taking other precautions to ensure the foetus is healthy. None of our controls for health behaviour vary within a pregnancy, therefore we cannot adequately capture the endogeneity. For this reason, we give a cautious interpretation to the result and conclude that mothers are able to undo the harm from smoking during pregnancy if they *change their behaviour* during the first two trimesters, which may include quitting smoking.

On the other hand, there is a large impact of smoking consistently for the full 9 months of pregnancy, relative to not smoking. In column 1, the OLS effect on the full sample, shows that mothers smoking for the entire 9 months of pregnancy will give birth to babies 7.2% lighter than non-smoking mothers. This falls to 5% when we exclude mothers who smoke continuously and estimate the effect for changers only. Finally, column 3 shows that differencing out the mother fixed effect reduces the estimate by half, compared to column 2 – indicating that for the full gestation smoker mothers, 50% of the harm from smoking is due to unobservable factors omitted from OLS regressions. Again, this suggests that along with quitting smoking, it is important for mothers to adapt other behaviour to improve the health of their child.

Socio-Economic Status

We are interested in whether the impact of maternal smoking during pregnancy displays heterogeneity across the distribution of education. We may expect this to be the case for two main reasons. Firstly, if there are complementarities in investment of human capital, then high educated mothers are able to extract a higher return to their investment, or a lower harm

from smoking. Additionally, high education may shift the budget constraint faced by a pregnant mother. This means that high educated mothers may be able to reduce the harm, by for example buying a higher quality of prenatal care than low educated mothers.

We define mothers as having a low level of education if they leave school by the compulsory age, which is 16 for the NCDS cohort, and high education otherwise. Comparing across regressions 2 and 3, in column 1 we see that the OLS harm from smoking is nearly double for low educated mothers than high educated mothers. The harm for low educated mothers classified as changers, shown in the second column of regression 2, is 1.3 percentage points lower than for the total sample of smokers, at 5%. For the high educated changers, there is no significant effect of smoking. In the final column we see that over half of the harm from smoking, for low educated mothers is differenced out in the fixed effect model, as compared to the pervious column, and the harm from smoking falls to 1.9%. For the high educated mothers, there is no significant effect of smoking during pregnancy once we include mother fixed effects into the model. This suggests that the harm from smoking is borne by low educated mothers.

This result may be due to the fact that the quantity of cigarettes consumed is inversely related to education. Indeed, low educated mothers on average smoke 3 cigarettes a day more than high educated mothers, hence the above result may be due to a dosage effect, rather than due to complementarities or difference in the budget constraint. Therefore, in regressions 4 and 5, we change the explanatory variable of interest, to the quantity of cigarettes consumed during pregnancy[12]. The results show that the harm from smoking each cigarette is relatively higher for low educated mothers, at 0.2% per cigarette in the fixed effect regression, compared to a result insignificantly different to zero for other mothers. The significance of this result is very relevant for policy targeting. As it is the low educated mothers who are more likely to smoke, who smoke a greater quantity during pregnancy and whose child receives the greatest harm for each cigarette smoked during pregnancy, it seems necessary that the low educated, or more generally low socio-economic status mothers, be the focus of targeted policies to change behaviour during pregnancy.

[12] The cohort members were not directly questioned about the number of cigarettes they smoked during pregnancy, but rather at each wave of observation. Therefore, assigning a value of the quantity of cigarettes smoked requires restricting the sample to mothers who reported not changing their habits during pregnancy.

5 Robustness Checks

5.1 Health Trends

A potential concern raised by our identification is that we will overestimate the harm from smoking, if the incidence of smoking is lower in more recent years due to trends in health behaviour, whilst simultaneously the mothers from the cohort are older. In this case, we will assign a positive birth weight effect to the non-smoking mother when really the age of the mother is driving the result.

We formally check the extent of the bias using a unique data set of birth records from Norway, between 1999-2003. During this period, the incidence of smoking during pregnancy fell in Norway from 47% to 36%, suggesting that within the time period, trends in smoking behaviour had changed. The advantage of the Norwegian dataset, as compared to NCDS, is that in each year of birth, we observe a cross section of mothers giving birth, whereas in the NCDS, the age of the mother is constant within the child's birth year. Therefore, we are able to separate the effects of the mother's age at birth (which ties in with time trends in the NCDS) and the change of habits. We will estimate the birth weight regressions as above, but controlling for additional dummies of the year of birth of the mother interacted with the year of birth of the child. The Norwegian data lacks information on some of the previous control variables, such as sporting activity and alcohol consumption, but a large set of controls are still comparable across the two data sets. Our regressors are the mother's smoking habits, age at birth, age squared, marital status, education, gender of child, birth order and maternal birth weight.

The summary statistics for the Norwegian data set are reported in Table 5, which compares to Table 1 for the NCDS. Excluding families smaller than one child and greater than six, there are nearly 65,000 children observations in the data set. A larger number of mothers smoke during pregnancy in the Norwegian data set, than in the NCDS data set: 37% compared to 32%. Birth weight is slightly higher in Norway and on average, smoking mothers in the UK are younger than the sample of smoking mothers in Norway. The education variable in the Norwegian dataset refers to the years of schooling, which is 13 if the mothers left school at age 17. Comparing the results, we find no large difference in the education of mothers within Norway and the UK.

The results are reported in Table 6. It is important firstly that the estimates for the UK and Norway are similar, if we are to generalize any results drawn from the Norwegian results to our analysis. Thus, we include a column for the NCDS results under the restricted set of covariates. Columns 2 and 3 detail the Norwegian results with the full set of controls and

additionally with dummy variable interactions between the mother's year of birth and the child's year of birth, respectively.

There are large differences in estimated coefficients in the UK and Norway, comparing the first 2 columns of the table. The harm is much larger in the UK, at 6.2% in regression 1[13], the OLS for the total sample, but half in Norway at 2.7%. The same is true of OLS results for the changers only and fixed effects estimates, in regressions 2 and 3. The coefficients reported in columns 2 and 3 are statistically from the same distribution, which suggests that in Norway, health trends are not driving the harm from smoking during pregnancy. However, we cannot at this stage say anything about the relevant bias in our analysis for the UK, as the experience for smoking mothers in Norway seems so different to that of UK mothers.

On average, mothers who smoke during pregnancy in the UK, tend to smoke 16 cigarettes per day, whereas in Norway the figure is much lower, at 7.3 per day, which could explain the difference in the cross-country estimates. Therefore, we extend the analysis by examining specifically how dosage of cigarettes harms child health[14]. Results are shown in regressions 4-6 of Table 6[15]. Per cigarette smoked, child birth weight falls by 0.04% and 0.05% for UK and Norwegian regressions respectively. Restricting the sample to mothers who change their habits across births in regression 5, the figures are no longer significant in the UK. However in the fixed effect regressions, we find that each cigarette smoked causes birth weight to fall significantly, independently of the unobservable traits of the mother, by 0.2% and 0.4% in the UK and Norway and again, these numbers are not statistically different to each other at the 95% confidence interval.

The alignment of results analyzing the harm from smoking in the UK and Norway is important as it suggests that our robustness check is valid, the data sets seem comparable. In Section 5.2.1 below, we investigate further the extent to which we can generalize the results of Norway to the UK estimates.

Now, taking the next step to analyze the potential bias in the analysis from the bulk of the paper, we can see that comparing coefficients in columns 2 and 3, under every specification, the controls for dummy variables of the mother's age at birth interacted with the child's year of birth have no effect on the estimated harm from smoking during pregnancy. Therefore, we conclude that our estimates are robust along this dimension.

[13] The difference from Table 3 is due to different controls.

[14] Recall that for the NCDS, we cut the sample in order to measure dosage, to mothers who do not change their smoking habits during pregnancy compared to the observation period preceding the pregnancy.

[15] Note that the number of observations falls in the Norwegian data, as not all mothers responded to quantity consumed.

5.2 Exclusion of Paternal birth weight

In Section 2, a child birth weight production function was described in terms of maternal inputs, including the "endowment" or genetic input into child birth weight. Unfortunately in our data set, we do not have access to the birth weight of partners of the cohort members.

The lack of information on paternal birth weight in the NCDS may have consequences for the estimates of maternal smoking during pregnancy, if partners assortatively mate according to characteristics which are correlated with the decision of the mother to smoke during pregnancy. For example, Burdett & Coles (1999) describe individuals as endowed with "charm", which is observable to the opposite sex. This charm can represent a plethora of characteristics, such as looks, wealth, attitudes towards risk etc. In their model, partnerships are formed according to this endowment. It may be that BMI is an element of charm upon which partners assortatively mate. Smoking habits and BMI may well be correlated, as both are at least partly driven by discount rates, or attitudes towards healthy living.

We are able to directly test the extent to which exclusion of the father's birth weight creates a bias in our estimates of maternal inputs in the child production function, by again utilizing the Norwegian birth registry dataset, which provides birth weight information not just for the mother, but for the father.

The extent to which the NCDS estimates of the elasticity between maternal and child birth weight are biased by omitting paternal birth weight, is determined partly by the correlation between paternal and maternal birth weight. This raw correlation in the Norwegian data set is close to zero, at 0.0145. The parents in Norway do not seem to assortatively mate by birth weight. We can confirm this finding using regression analysis. Columns 1-3 of Table 7 report the raw regression results[16] of the harm of smoking upon child log birth weight in Norway. From column 1, the raw elasticity between maternal and child birth weight in Norway is 0.146. Looking across to column 2, the elasticity between paternal and child birth weight is much lower at 0.028. Both of these results are statistically significant. In column 3, including both terms in the regression both elasticities are unchanged. Mothers and fathers do not seem to assortatively mate on birth weight.

We now turn to the question of possible correlation between paternal birth weight and maternal smoking status during pregnancy, by running regressions of smoking during pregnancy upon child log birth weight, with the relevant controls, in columns 4 and 5 of

[16] conditional upon the full set of controls

Table 7. The harm from smoking, conditional upon all controls excluding paternal birth weight is 2.6%. In the final column, we see that controlling additionally for paternal birth weight has no effect on the harm from smoking. Therefore, we can conclude that in Norway, we find no evidence of a bias in the estimate of the harm from smoking during pregnancy upon child birth weight from omission of paternal birth weight. We now ask whether we can conclude from this table that the same can be said about the UK, by addressing the comparability of the Norwegian and the UK results.

5.2.1 Generalising Results

The conclusion that the estimates of the harm from smoking upon child health are unbiased from omission of paternal birth weight, will apply to the UK as well as to Norway if:

i) $E\left(h_f^i, S_m^i\right) = E\left(h_f^j, S_m^j\right)$ ii) $E\left(h_f^i, h_c^i\right) = E\left(h_f^j, h_c^j\right)$

where *i* and *j* denote Norway and UK respectively, *f*, *m* and *c* denote the father, mother and child respectively and *h* denotes human capital, or in this example birth weight.

We can assess i) by taking a sample of male cohort members from the NCDS, and the smoking behaviour of their wives. The correlation between paternal birth weight and maternal smoking for this sample is -0.0160 in the UK, and very similar at -0.0185 in Norway. This suggests that the bias from omitting paternal birth weight, for the estimate of the harm from smoking during pregnancy in the UK is negligible.

We cannot assess the correlation between father and mother birth weight in ii) directly without observation of both maternal and paternal birth weight in the NCDS. Instead, we calculate raw correlations between educational attainment of partners in the NCDS and compare this to the Norwegian data set. The correlation between father and mother's years of schooling in Norway is 0.5 and in UK is 0.55, which again is very similar. Of course, we can not conclude from this that assortative mating by birth weight is the same in the UK as in Norway. However the finding that assortative mating by educational background is similar in the two countries suggests assumption ii) is possible.

In conclusion, there exists no bias in Norway from the exclusion of paternal birth weight. Our evidence suggests no correlation between birth weight of the mother and father in Norway and more importantly, no correlation between paternal birth weight and maternal smoking during pregnancy in Norway. We believe that the latter result can be generalized to the UK.

5.3 Other effects of smoking

Our paper has analyzed the harm from smoking upon child birth weight and gestation. It can be argued however that smoking during pregnancy may drive other outcomes of the child, which drive child health independently to the smoking. For example, Tuormaa (1995) lists some of the other effects from maternal smoking as placenta previa and placental abruption[17]. These two conditions may lead to pre-term births, which will affect birth weight. Our estimate has identified the total effect of smoking on birth weight, which we believe is the most interesting. However, we conduct a robustness test, by controlling for gestation in the birth weight equation and also for a dummy variable which equals 1 if there were complications during pregnancy and 0 otherwise. The problems cited by Tuormaa were mentioned by mothers, as well as others such as heart problems.

Table 8 shows the estimate of smoking upon log child birth weight under the three specifications, with a full set of controls including complications during pregnancy and gestation. Compared to Table 4, the estimated "direct" impact of smoking during pregnancy is lower, as expected, once we factor out these two pregnancy outcomes. The OLS for the total sample and for changers respectively, has fallen to 4.6% and 3.1% respectively, and fixed effects estimate is 0.3 percentage points lower, at 1.4%. Complications during pregnancy reduce child birth weight by around 5%-6% and an increase of gestation by one week will raise the child birth weight by between 4.6%-5.5%. This table suggests that the channels through which smoking does lower early child human capital are partly explained by gestation and the incidence of complications during pregnancy.

6 Magnitude of the Effect

We have estimated the harm from a mother smoking during pregnancy upon the weight of her child at birth in the fixed effect model, as 1.7%. We now aim to give some magnitude to the estimates on birth weight, to understand the implications of maternal smoking. We calculate how many babies could have been born in a healthy state, had their mothers not smoked during pregnancy. There exists a birth weight threshold, below which the child is defined as unhealthy at birth. Low birth weight (LBW) babies are classified as such, if born weighing less than 2500g. There are implications for the health and development of the baby, and also for hospital costs of LBW status (see Almond, Chay, Lee (2005)).

[17] See Appendix 2 for precise details of other outcomes

In our sample of births, altogether 462 babies are LBW (6.57%). Of these, about half of the mothers smoked during pregnancy (216 mothers). This is a far greater proportion than mothers smoking in the total sample (32%).

We interpret our estimates from the analysis above, as a causal effect of smoking, which shifts the child's birth weight. The number of children born to smoking mothers and classified as LBW, who would otherwise have been born above the 2500g threshold had their mothers refrained from smoking, is our measure of the magnitude of smoking during pregnancy.

On average, smoking during pregnancy was estimated to reduce child birth weight by 1.7%. This means that children born to smoking mothers between the weight of 2457.5 and 2500 (1.7% below the LBW threshold) were classified as LBW and, according to our results could have been healthy children had their mothers not smoked. 61 babies were born within this margin, to smoking mothers, which is only 3% of smoking mothers, but 13.2% of the sample of LBW babies born to smoking mothers. Thus, more than one in ten babies born LBW could have been born healthy, had their mothers refrained from smoking. This number is quite small, as we would expect from the small size of the coefficient. This emphasizes once more the need for appropriate policy to reach the mother traits which are unobservable to an econometrician, in order to improve the health of their children.

7 Conclusion

We have used a data set very rich in information about mothers and children, in order to estimate the harm on child human capital from smoking during pregnancy. We estimated the OLS conditional effect on birth weight to be 5.6% for the total sample of mothers, 3.3% for the sample of changers and the fixed effect 1.7%. This suggests that other behaviours of the mother play a large role in child human capital over and above her smoking habits, and policies targeting child human capital should aim to educate the pregnant mother on health behaviour generally during pregnancy. There needs to be more understood about exactly what the unobservable traits are, to get inside this black box of child health outcomes.

We found a negligible effect of maternal smoking during the pregnancy, if the mother quits by month five, which suggests that there is a cumulative effect of smoking. This is reassuring, as cessation or education programs do not have to just target pregnant mothers early in the gestation, but can have some effect later in the pregnancy. The harm from smoking was found to be non-linear and decreasing across the education of the mothers,

results which are robust to controls for the quantity of cigarettes smoked during pregnancy. Giving some magnitude to these results, we find that up to 13% of children classified as low birth weight, born to smoking mothers could have been classified as healthy, had their mothers not smoked.

There are important implications of this paper, which suggest that smoking cessation policies are not enough to address inequalities in child health. Further, not only is it the low SES mothers who choose to smoke, but they are also the mothers bearing the greatest burden from the smoking. Therefore, any potential solution must offer help to these mothers, to target those with the worst habits and poorest records of child health.

8 Tables of Results

Table 1: NCDS Sample Statistics.

Smoking during pregnancy	Never	At the start	Consistently	Total Sample
Number of observations	4266	378	1647	6291
% of sample	67.8	6.0	26.2	100
Birth weight (grams)	3412.17	3411.32	3173.49	3349.63
	(546.29)	(571.03)	(588.40)	(568.77)
LBW	0.05	0.05	0.11	0.06
	(0.22)	(0.22)	(0.31)	(0.25)
Gestation (weeks)	39.73	39.76	39.53	39.68
	(2.05)	(2.41)	(2.44)	(2.18)
Pre-term Gestation	0.10	0.08	0.13	0.10
	(0.30)	(0.28)	(0.34)	(0.31)
Mother age	28.95	25.88	25.36	27.82
	(5.36)	(4.08)	(4.92)	(5.43)
Age left school	17.40	16.70	16.32	17.08
	(2.24)	(1.50)	(1.18)	(2.03)
Mother Height (inches)	63.94	64.00	63.39	63.80
	(2.70)	(2.53)	(2.47)	(2.64)
Live with a smoker	0.23	0.49	0.62	0.34
	(0.42)	(0.50)	(0.49)	(0.48)
Grandmother smoked, mum age 16	0.32	0.39	0.46	0.36
	(0.47)	(0.49)	(0.50)	(0.48)
Grandfather smoked, mum age 16	0.39	0.45	0.50	0.43
	(0.49)	(0.50)	(0.50)	(0.49)

Standard deviation is shown in parentheses.
Note: sample of mothers with at least 2 children and fewer than 6.

Table 2: Transition matrix of smoking habits, for 3 child families.

		1st child		2nd child	
		0	1	0	1
2nd child	0	0.89	0.22		
		0.549	0.083		
	1	0.11	0.78		
		0.069	0.300		
3rd child	0	0.92	0.33	0.95	0.19
		0.576	0.122	0.623	0.067
	1	0.08	0.67	0.05	0.81
		0.050	0.252	0.030	0.280

Key: Row 1: Column percentage, Row 2: Total percentage

Table 3: Regression of dummy variable for mother smoking during pregnancy, upon four pregnancy outcomes.

		1	2	3	4	5	6	7
Regression		Raw	+ standard controls	+ health, endowment	+ partner information	+ real background	Fixed effect	OLS / LP (changers)
1	Log birth weight	-0.058 (0.007)***	-0.064 (0.007)***	-0.062 (0.007)***	-0.056 (0.008)***	-0.056 (0.008)***	-0.017 (0.009)*	-0.033 (0.012)***
2	LBW	0.045 (0.009)***	0.047 (0.010)***	0.046 (0.010)***	0.042 (0.010)***	0.042 (0.010)***	0.008 (0.012)	0.021 (0.013)
3	Gestation (weeks)	-0.224 (0.075)***	-0.212 (0.077)***	-0.206 (0.078)***	-0.188 (0.081)**	-0.188 (0.082)**	-0.080 (0.118)	-0.035 (0.123)
4	Pre-term gestation	0.023 (0.010)**	0.023 (0.010)**	0.022 (0.010)**	0.017 (0.011)	0.018 (0.011)	0.014 (0.017)	0.030 (0.018)*
N		6291	6291	6291	6291	6291	6291	734

Standard errors in parentheses. All regressions control for child year of birth dummies. Standard controls: age mother at birth, age mother squared, education mother, married at birth, child sex, birth order, ethnicity. Health endowment: maternal birth weight and height, exercise regularly, drink over recommended units of alcohol. Father information: age left school, live with smoker. Real background information: region where mother born, region at 11, grandfather's social class at birth, grandmother and grandfather smoked when mother 16. LP denotes the linear probability model.

Table 4: Heterogeneity in the harm from smoking during pregnancy.
Dependent variable is log child birth weight.

		1	2	3
Regression	Sample	OLS (all)	OLS (changers)	FEF
1	Quit by month 5	0.002	-0.022	-0.009
		(0.011)	(0.015)	(0.012)
	Smoke 9 months	-0.072	-0.050	-0.024
		(0.008)***	(0.014)***	(0.012)**
		N=6291	N=734	N=6291
2	Low education	-0.062	-0.049	-0.019
		(0.009)***	(0.014)***	(0.011)*
		N=3980	N=509	N=3980
3	High education	-0.035	0.016	-0.017
		(0.014)**	(0.023)	(0.018)
		N=2311	N=225	N=2311
4	Low education, quantity	-0.004	-0.003	-0.002
		(0.001)***	(0.002)*	(0.001)*
		N=3320	N=145	N=3320
5	High education, quantity	-0.002	0.001	-0.001
		(0.001)	(0.003)	(0.002)
		N=2049	N=54	N=2049

Standard errors in parentheses. All regressions control for child year of birth dummies. Standard controls: age mother at birth, age mother squared, education mother, married at birth, child sex, birth order, ethnicity. Health endowment: maternal birth weight and height, exercise regularly, drink over recommended units of alcohol. Father information: age left school, live with smoker. Real background information: region where mother born, region at 11, grandfather's social class at birth, grandmother and grandfather smoked when mother 16.

Table 5: Time Trends. Norway Registry Data Sample Statistics.

Smoking during pregnancy	Never	Smoked	Total Sample
Number of observations	40 661	24 171	64 832
% of sample	63%	37%	100
Birth weight (grams)	3509.71	3404.72	3470.57
	(579.77)	(619.73)	(597.14)
Mother age	28.77	27.90	28.45
	(3.55)	(3.90)	(3.70)
Years of Education	13.51	12.49	13.13
	(2.23)	(2.28)	(2.30)

Standard deviation is shown in parentheses
Note: sample of mothers with at least 2 children and fewer than 6.

Table 6: Time Trends. Regression Analysis.
Dependent variable is log child birth weight.

		1	2	3
Regression	Sample	NCDS	Norway	Norway + cohort * birth year
1	OLS (all)	-0.062	-0.027	-0.027
		(0.007)***	(0.002)***	(0.002)***
		N=6291	N=64832	N=64832
2	OLS (changers)	-0.033	-0.006	-0.006
		(0.011)***	(0.003)**	(0.003)**
		N=734	N=19224	N=19224
3	FEF	-0.017	-0.006	-0.006
		(0.009)*	(0.002)***	(0.002)***
		N=6291	N=64832	N=64832
Quantity smoked				
4	OLS (all)	-0.004	-0.005	-0.005
		(0.00047)***	(0.000)***	(0.000)***
		N=5369	N=49113	N=49113
5	OLS (changers)	-0.002	-0.006	-0.006
		(0.00197)	(0.001)***	(0.001)***
		N=199	N=11019	N=11019
6	FEF	-0.002	-0.004	-0.004
		(0.00090)*	(0.001)***	(0.001)***
		N=5369	N=49113	N=49113

Standard errors in parentheses. All regressions control for child year of birth dummies, plus age mother at birth, age mother squared, education mother, married at birth, child sex, birth order, maternal birth weight.

Table 7: Exclusion of father endowment using Norwegian Dataset.
Dependent variable is log child birth weight.

	1	2	3	4	5
Mother log birth weight	0.146		0.146	0.145	0.145
	(0.007)***		(0.007)***	(0.007)***	(0.007)***
Father log birth weight		0.028	0.028		0.029
		(0.006)***	(0.006)***		(0.006)***
Mother smoked				-0.026	-0.026
				(0.002)***	(0.002)***
Obs	64299	64299	64299	64299	64299
R-squared	0.01	0.00	0.01	0.03	0.03

Standard errors in parentheses. All regressions control for child year of birth dummies, plus age mother at birth, age mother squared, education mother, married at birth, child sex, birth order.
Hausman: coefficient of maternal log birth weight. 1=3: 4.59820418

Table 8: Additional controls for birth outcomes.

	(1) OLS (all)	(3) OLS (changers)	(2) FEF
did CM smoke during pregnancy	-0.046	-0.031	-0.014
	(0.005)***	(0.010)***	(0.008)*
Complications during birth	-0.049	-0.062	-0.051
	(0.008)***	(0.030)**	(0.009)***
Child gestation	0.054	0.055	0.047
	(0.002)***	(0.011)***	(0.003)***
Obs	6291	734	6291
R-squared	0.45	0.46	0.42

Standard errors in parentheses. All regressions control for child year of birth dummies. Standard controls: age mother at birth, age mother squared, education mother, married at birth, child sex, birth order, ethnicity. Health endowment: maternal birth weight and height, exercise regularly, drink over recommended units of alcohol. Father information: age left school, live with smoker. Real background information: region where mother born, region at 11, grandfather's social class at birth, grandmother and grandfather smoked when mother 16.

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Appendix 1a

Full regression results

	Log Birth Weight	Log Birth Weight	Log Birth Weight	LBW	Gestation (weeks)	Pre-term Gestation
	OLS (all)	OLS (changers)	FEF			
did CM smoke during pregnancy	-0.056	-0.033	-0.017	0.042	-0.188	0.018
	(0.008)***	(0.012)***	(0.009)*	(0.010)***	(0.082)**	(0.011)
Child sex	0.031	0.001	0.031	-0.010	-0.093	0.002
	(0.005)***	(0.014)	(0.005)***	(0.006)	(0.060)	(0.008)
age parent left school	0.000	-0.004		-0.002	0.016	-0.002
	(0.002)	(0.006)		(0.002)	(0.019)	(0.003)
age when mother had child	0.005	-0.126	0.057	0.005	-0.505	0.064
	(0.057)	(0.182)	(0.055)	(0.062)	(0.690)	(0.080)
mother age child squared	-0.000	0.003	-0.001	0.000	0.008	-0.001
	(0.001)	(0.004)	(0.001)	(0.001)	(0.012)	(0.001)
married when had baby	0.000	-0.030	0.009	-0.009	-0.018	-0.002
	(0.007)	(0.024)	(0.017)	(0.008)	(0.077)	(0.010)
birth order	0.021	0.019	0.024	-0.007	-0.034	0.003
	(0.003)***	(0.008)**	(0.006)***	(0.004)*	(0.037)	(0.005)
Black	-0.106	-0.103		0.008	-0.512	0.084
	(0.040)***	(0.042)**		(0.043)	(0.483)	(0.082)
India/Pakistan	-0.095	0.000		0	0.258	0
	(0.027)***	(0.000)		0	(0.273)	0
Other Asian	0.000	0.000		0	0.000	0
	(0.000)	(0.000)		0	(0.000)	0
Mixed Race	-0.090	0.000		0	-1.783	0.229
	(0.199)	(0.000)		0	(1.497)	(0.178)
Mother's height aged 16 inches	0.008	0.002		-0.004	-0.005	-0.000
	(0.001)***	(0.004)		(0.001)***	(0.013)	(0.002)
log birth weight parent	0.147	0.143		-0.051	0.365	-0.040
	(0.020)***	(0.071)**		(0.022)**	(0.211)*	(0.028)
exercise regularly	0.005	0.005	0.004	-0.016	0.094	-0.011
	(0.007)	(0.018)	(0.012)	(0.008)*	(0.079)	(0.011)
over alcohol recommended limit per week	0.004	0.008	-0.004	-0.011	0.046	0.002

	(0.008)	(0.022)	(0.010)	(0.008)	(0.087)	(0.011)
Partner's age left school	-0.001	-0.003	-0.008	0.003	-0.016	0.003
	(0.002)	(0.008)	(0.008)	(0.002)	(0.024)	(0.003)
live with smoker	-0.021	0.007	-0.015	0.015	-0.081	0.018
	(0.008)***	(0.023)	(0.018)	(0.009)*	(0.083)	(0.011)
Region mum's birth	-0.001	-0.002		0.001	0.008	-0.001
	(0.002)	(0.008)		(0.002)	(0.016)	(0.002)
Region mum aged 11	0.000	0.004		-0.001	-0.005	0.000
	(0.001)	(0.008)		(0.001)	(0.014)	(0.002)
Grandfather's social class, mum's birth	-0.005	0.026		0.016	-0.117	0.025
	(0.009)	(0.043)		(0.011)	(0.092)	(0.014)*
SES dad when cm born	-0.001	-0.001		-0.001	0.006	-0.001
	(0.001)	(0.004)		(0.001)	(0.010)	(0.001)
Father smoked, mum 16	0.009	0.004		-0.003	0.032	-0.007
	(0.006)	(0.021)		(0.007)	(0.071)	(0.010)
Mother smoked, mum 16	-0.004	-0.016		-0.003	-0.031	-0.001
	(0.007)	(0.020)		(0.007)	(0.072)	(0.010)
Observations	6291	734	6291		6291	6281

Appendix 2

Smoking during pregnancy: other effects

Smoking during pregnancy may reduce child birth weight. Additionally, there may be other health problems. Tuormaa (1995) lists the other effects as:

reduced gestation and prematurity, increase miscarriages, placenta previa, placental abruption, premature rupture of membranes, sudden infant death syndrome.

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