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Abstract
This study explores the impact of mother's smoking during pregnancy on child bodyweight outcomes, from birth through age five. Using a nationally representative sample of children surveyed in the NLSY79 Children and Young Adults, we exploit 'within mother and across pregnancies' variation in smoking behavior for identification. Consistent with prior literature, our estimates suggest that children of smokers weigh 0.25 pounds less at birth than children of non-smokers. Our examination of the evolutionary impact through pre-school years reveals that by age three there is no significant difference, in any of the several body weight measures we consider, between children of smokers and non-smokers. This lack of difference between the weight outcomes of two groups of children continues through the of age five. The latter findings, regarding the effects during pre-school years, are contrary to the hypothesis that children with fetal exposure to nicotine are associated with a higher risk of being overweight or obese.

Keywords: Maternal smoking; child weight; child BMI; child obesity

JEL codes: I10, I12, I18

1. INTRODUCTION

The effects of maternal smoking during pregnancy on child outcomes have been widely explored across several academic disciplines, ranging from epidemiology, economics, and medicine, to sociology and psychology. However, most of the relevant literature in this space is descriptive in nature, providing correlational rather than causal interpretations. This study extends the literature by exploiting a robust empirical strategy, which controls for mother-specific unobserved time-invariant effects, to investigate the presence of a causal link between maternal smoking during pregnancy and children's bodyweight indicators during the preschool years (from birth through age five). Our contribution is two-fold. First, we confirm the negative effects of maternal smoking during pregnancy on child birthweight documented in prior literature. Second, and more importantly, we study the evolution of the effects of maternal smoking on children's bodyweight through the pre-school years. In exploring the long-term effects of maternal smoking during pregnancy, we test the so called 'catch-up growth', alluded to in the prior literature (see Ong et al. 2000; Von Kries et al. 2002). These studies suggest that children of mothers who smoked during pregnancy rapidly grow during early childhood to catch-up with the children of nonsmokers and are in fact more likely to be overweight or obese in later childhood.[1]

Existing studies have linked maternal smoking during pregnancy with a wide array of both short-term and long-term child health consequences. Examples of short-term consequences include premature childbirth, fetal growth restriction, lower birthweight, and infant mortality (Comstock et al., 1971; Meyer & Tonascia, 1977; Cnattingius, 2004). The longer-term health implications for children include higher blood pressure levels, and respiratory and pulmonary

[1] Ong et al. (2000) focus on likelihood of obesity at the age five whereas Von Kries et al. (2002) focus on the likelihood of being overweight or obese during the age of five to seven years.

disorders (Hanrahan et al., 1992; Stick et al., 1996; Blake et al., 2000; Li et al., 2016); psychological and behavioral problems - such as attention deficit hyperactivity disorder, neurological problems, and poor cognitive functioning (Weitzman, Gortmaker, & Sobol, 1992; Milberger et al., 1996; Wakschlag et al., 1997; Thapar et al., 2003; Huizink & Mulder, 2006; Key et al., 2007; Gilman, Gardener, & Buka, 2008); and increased risks of childhood obesity (Vik et al.,1996; Fried, Watkinson, & Gray, 1999; Von Kries et al., 2002; Oken, Levitan, & Gillman, 2008). Our study focuses on child weight outcomes in particular and examines both short and longer-term impacts of maternal smoking during pregnancy. In particular, we investigate the impact on bodyweight outcomes from birth through age five. We do not study the health effects beyond the age of five, as there are likely to be several confounders such as peer and school effects (see Gaviria & Raphael, 2001) that we would not be able to control for using our data.

In examining the related literature for this study, there are two strands of work to draw on. The first is well-established and illustrates that prenatal exposure to maternal smoking results in lower birthweight (Brooke et al. 1989; Rosenzweig & Wolpin 1991; Blake et al 2000; Ward et al. 2007). The second set of studies signals that the risk of obesity during childhood, mainly during the pre-school period, is much higher among children whose mothers smoke during pregnancy.[2] Upon closer examination, the extant literature suggests that while prenatal exposure to maternal smoking results in fetal growth retardation (Miller et al. 1976; Cnattingius, 2004; Ward et al., 2007), the same is associated with stunted growth (in terms of height), higher risk of adiposity (skin thickness), and obesogenic growth during later childhood (Conter et. al 1995; Oken et al., 2008; Ino, 2010; Howe et al., 2012; Li et al., 2016). Taken together, the above-mentioned literature on maternal smoking during pregnancy indicates that

[2] See Ino (2010) for a meta-analysis of studies exploring the association between maternal smoking and child obesity.

the evolving effects likely prompt smokers' children to not only 'catch-up' with non-smokers' children in terms of weight and Body Mass Index (BMI) but also experience a greater risk of being overweight (or obese) as they grow up. We explore this catch-up phenomenon following a group of children born to a US-based nationally representative sample of mothers. We also explore gender-related differences as the limited evidence on this front is contradictory in nature. For example, while Suzuki et al. (2011; Japanese sample) find that maternal smoking during pregnancy puts boys at a higher likelihood of childhood obesity relative to girls, studies by Durmus et al. (2014; Dutch sample) and Li et al. (2016; Portuguese sample) observe that girls are more likely to have excess weight during later childhood. Our study provides new evidence that speaks to these contradictions.

For our analysis, we link mothers' data from the National Longitudinal Survey of Youth 1979 (NLSY79) with their biological children's information from the National Longitudinal Surveys of Children and Young Adults (NLS-CYA). Per our research objective, we classify our analysis into two parts. First, we look at the aggregate and the gender-specific effects of maternal smoking during pregnancy on children's birthweight. Second, we analyze the impact of maternal smoking during pregnancy on the risk of having excess bodyweight among children aged 2 to 5.[3] We construct indicators for being overweight and obese by using information on a child's percentile rank in World Health Organization's (WHO) Body Mass Index (BMI) distribution. The construction of the BMI-for-age (BFA) distribution-based measures incorporates age- and gender-specific heterogeneities in children's bodyweight. Moreover, we

[3] The prevalence of childhood obesity in the U.S. using BFA distribution is usually analysed from the age of 2. For children, under the age of 2, researchers generally focus on weight-for-recumbent-length distribution. For consistency, since we use BFA distribution for indicators of excess weight, we exclude children aged under 2 (Ogden et al. 2010). However, in a later analysis, controlling for child height, we incorporate age- and gender-specific analyses using weight as well as BMI measures for all children aged 1-5.

also create similar indicators based on percentile rank in WHO's Weight-for-age (WFA) distribution.

In addition to the excess weight indicators, we utilize children's weight and BMI measures as dependent variables to perform additional analysis to gain an in-depth understanding of the catch-up phenomenon conjectured in the existing literature. Unlike the binary BFA and WFA age and gender dependent indicators, we address the age and gender-specific differences in weight and BMI by analyzing separate samples of boys and girls at each child age ranging from one through five.

Our empirical strategy is to model children's bodyweight outcomes as a function of their own characteristics, mother's characteristics, and family information. We exploit 'within-mother and across pregnancies' variation in smoking behavior to identify any causal effects of maternal smoking on children's weight outcomes. Our estimates are, therefore, based on differences in weight outcomes of siblings whose mother smoked during certain pregnancies and did not smoke during others. Although we rely on fixed effects (FE) estimates for the interpretation of our key findings, we present estimates obtained from alternative modelling techniques to ensure robustness of our results. This includes Ordinary Least Squares (OLS), Probit, and Correlated Random Effects (CRE) models.

In line with the findings in the previous literature, our estimates suggest a significant reduction in children's birthweight because of maternal smoking during pregnancy. Specifically, we find that, on average, children of mothers who smoked during pregnancy weigh 0.252 pounds less than children of mothers who did not smoke while pregnant. We also find that this negative impact is stronger for boys compared to girls. Not surprisingly, the negative effects are also larger for smoking a greater quantity of cigarettes.

With respect to the BFA distribution-based indicators for children aged 2 to 5, our pooled Probit regression estimates indicate, as suggested in prior literature, that there is a statistically significant and positive association between maternal smoking during pregnancy and the likelihood of being overweight and obese. However, after accounting for unobserved mother-specific time-invariant characteristics, we do not find any significant evidence in support of a higher prevalence of excess weight due to prenatal exposure to maternal smoking. These findings hold when we consider WFA distribution-based bodyweight indicators.

Exploring the dynamic effects of smoking at an even more granular level, when we examine the effects of maternal smoking during pregnancy on child weight and BMI at each specific age from one through five, results from the preferred FE models suggest that negative effects seen at birth start to diminish as early as one and completely disappear by the age of three. However, there is no evidence to suggest that the children of smokers may be more likely to be obese compared to the children of nonsmokers.

Our findings have important implications for public health policy. Specifically, our results support and encourage public policies targeted at curbing smoking among expecting mothers but question the understanding that maternal smoking during pregnancy may lead to obesity in later childhood.

The remainder of this paper is organized as follows: Section 2 discusses the relevant literature, Section 3 describes the data and variables used in the empirical analysis; Section 4 explains the identification strategy employed; Section 5 discusses the key findings; and Section 6 presents concluding remarks.

2. RELATED LITERATURE

There are two strands of literature relevant to this study: one that investigates the effect of maternal smoking during pregnancy on child birthweight and the other that examines the effect on weight outcomes during early childhood. The evidence pointing to negative effects of maternal smoking during pregnancy on birthweight is overwhelming. Multiple studies have substantiated the causal relationship between maternal smoking during pregnancy and children's birthweight using randomized smoking interventions. For instance, Sexton & Herbal (1984) use a randomized clinical trial in a controlled experiment setting where pregnant smokers (prior to the 18^{th} week of their gestation) were randomly assigned to an intervention that was designed to aid with smoking cessation through provision of counselling and instructional guidance. Evaluating the difference between the treatment and the control group, the study finds that the intervention significantly reduced smoking among pregnant women subjected to the treatment. Additionally, the authors observe that the mean birthweight of children whose mothers stopped smoking during pregnancy exceeded the control group children's mean birthweight by 92 grams (equivalent to 0.20 pounds). In later studies such as Evans & Ringel (1999) and Lien & Evans (2005), researchers have used smoking-related public policy regulation such as state-level cigarette tax increases as instrument to evaluate the impact of maternal smoking during pregnancy on children's birthweight. Their empirical methodology is based on the literature that explores the deterrent effect of macro-level smoking regulations (such as Federal and state-level excise tax) on individuals' smoking behavior (O'Campo et al. 1995; Ringel & Evans 2001; Colman et al. 2003). Lien & Evans (2005) find that smoking during pregnancy leads to a substantial increase in the likelihood of infants having a low birthweight (defined as less than 2500 grams or 5.5 pounds approximately).

Utilizing an alternative empirical strategy, Rosenzweig & Wolpin (1991) employ within-mother FE regression models to control for confounding influences arising from mothers' time-invariant characteristics that may affect their prenatal behavior (including smoking) as well as their children's well-being. The authors' findings support the negative relationship between maternal smoking during pregnancy and children's birthweight. More specifically, Rosenzweig & Wolpin's (1991) regression estimates suggest that on average, birthweight of children of mothers who smoke less than a pack per day is 0.18 pounds (2.92 ounce) less than non-smoker's children's birthweight. The difference increases to 0.35 pounds (5.62 ounce), when birthweights are compared across non-smokers' children and children whose mothers smoked more than a pack a day. The results we obtain in this paper are consistent with the prior findings with respect to the effects of maternal smoking during pregnancy on child birthweight.

Compared to the strong causal evidence with regard to children's birthweight, the empirical findings in terms of impact of maternal smoking during pregnancy on child weight over the pre-school years are mostly descriptive in nature and, at best, represent association. For example, controlling for socio-economic characteristics and mothers' post-natal maternal behavior, the majority of studies including, but not limited to, Von Kries et al. (2002), Wideroe et al. (2003), and Al Mamun et al. (2006) rely on logistic regressions to assess the relationship between maternal smoking during pregnancy and the risk of being overweight and/or obese during childhood. Furthermore, using a child cohort of 848 singletons, Ong et al. (2000) employ a correlational analysis with UK data to investigate the catch-up growth among children subjected to prenatal exposure to maternal smoking. Failure to account for unobserved heterogeneities that are potentially correlated with maternal characteristics and child well-being imposes restrictions on causal interpretation of findings presented in these past studies.

We contribute to the current knowledge base by combining the two aforementioned strands of the literature and exploring the validity of the catch-up phenomenon using a panel of children born to a nationally representative sample of American mothers. We employ 'within-mother across pregnancies' variation in maternal smoking behavior to test if there is a causal influence of prenatal exposure to maternal smoking on the risk of having excess bodyweight during early childhood. The within-mother FE regression models account for unobserved mother-specific heterogeneities and are likely to yield plausibly causal estimates.

3. DATA: THE NATIONAL LONGITUDINAL SURVEYS

We link mothers' information from the original cohort of the NLSY79 with information on their biological children from the NLS-CYA. The NLSY79 is a widely utilized dataset for social and policy-relevant research. The surveys incorporate a wide range of socio-economic and demographic information on a nationally representative sample of 12,686 individuals, who were born between 1957 and 1964. Commencing in 1979, the surveys were administered annually until 1994, and biennially thereafter. The NLS-CYA commenced in 1986 and are conducted biennially. These surveys document health, schooling, behavioral, as well as family-specific information of biological children born to women in the original NLSY79 cohort. Upon linking the two surveys, the total matched sample includes 11,511 children born to 4,931 mothers. However, depending on the availability of the data required to test the relationship of interest, we apply further restrictions to this sample. These restrictions are explained in detail in our following discussions.

Our main analysis has two elements: (i) impact on child birthweight, and (ii) investigation of weight outcomes of pre-school aged (2-5) children. For the first element, we limit our sample to children who were born between 1979 and 2011 (the last year in which a childbirth was recorded in the NLS-CYA). This is done in order to maximize our sample size and

simultaneously control for important mother- and family-level covariates derived from the NLSY79 (which commenced in the year 1979). Further, the NLSY79 were conducted annually until 1994 and biennially thereafter. Therefore, for children born during odd-numbered years i.e. from 1995 onwards until 2011, the covariates derived from the NLSY79 are based on information from survey years immediately succeeding the birth years. At this point, it is important to note that excluding children with odd-numbered birth year post-1994 survey does not affect our key findings in the birthweight analysis. Moreover, these children account for only 5 percent of the birthweight regression sample.

The main outcome used in the birthweight analysis is children's birth weight reported in NLS-CYA's pre- and post-natal information. Table 1 provides descriptive information on this outcome in addition to all other variables used in our analysis. For easy comparison, we present the estimates for samples separated by mothers' smoking behavior during pregnancy. We observe that children of smoking mothers are born with significantly lower weight and height compared to non-smokers' children.

For the analysis of pre-school aged children between 2 and 5 years, we use the children's weight and height estimates recorded in the NLS-CYA to construct binary indicators for extreme bodyweight conditions classified by the BFA distribution (see Table 2 for descriptive statistics). Since the bodyweight indicators are estimated based on children's height and weight estimates reported in the biennial survey years of NLS-CYA, the study period for the post-birth analysis is from 1986 through 2012. In particular, we refer to the World Health Organization's (WHO 2006)[4] growth charts to create our binary indicators of excess weight for the 2-5-year-old age group. The 'overweight' indicator equals 1 when the observed BMI exceeds the BMI

[4] The children's growth charts are available in http://www.who.int/childgrowth/standards/bmi_for_age/en/ and http://www.who.int/childgrowth/standards/weight_for_age/en/; Accessed on July 19, 2018.

value at the 85 percentile of the age- and gender-specific BFA distribution and the 'obesity' indicator equals 1 for BMI values above the 95th percentile of the same distribution (examples of studies using similar measures include Powell & Bao 2009; Wang & Lim 2012; Winter et al. 2017).[5] Additionally, we also create an 'underweight' indicator for having BMI below the 5th percentile of the BFA distribution. This allows us to study the persistence of low weight among children who were born with low birthweight because of maternal smoking during pregnancy. As shown in Table 1, for children aged 2-5, we do not find any significant difference in the prevalence of any of the extreme BFA-based bodyweight measures between the smokers' and non-smokers' children.

As with the BFA-based indicators, we use the WHO's WFA distribution to construct two additional excess bodyweight indicators (above 85th and above 95th percentile) and one underweight indicator for being below the 5th percentile of the same distribution. For these indicators we do find some difference between the samples categorized based on mothers' smoking behavior during pregnancy. Interestingly, a greater (smaller) proportion of children of smokers are underweight (obese), compared to non-smokers, and these differences are statistically significant at conventional levels.

The data on mothers' smoking behavior during pregnancy, our key explanatory variable, are obtained from NLS-CYA's pre- and post-natal information. There are two potential NLSY variables to draw on: (i) a dichotomous indicator of whether someone smoked during the 12 months *prior to her childbirth*, and (ii) a categorical indicator referring to the number of cigarettes smoked per day *during pregnancy* (none, less than a pack, at least one pack but less than two packs, and two packs or more). Since, our research focus is particularly on mothers'

[5] It is important to note that the WHO's BFA distribution is provided in terms of kg/m^2. Given that the NLS-CYA estimates of children's weight and height are recorded in pounds and inches, the BMI formula to convert the survey measures in kg/m^2 is: BMI = [Weight in Pounds / (Height in inches) x (Height in inches)] x 703; See https://www.bcbst.com/providers/MPMTools/BMICalculator.shtm.; Accessed on July 17, 2018.

smoking behavior *during pregnancy*, we draw on the latter variable. Using this categorical indicator, we construct two variables: 'smoked' and 'quantity smoked'. The binary variable 'smoked' turns on if a mother smoked during her pregnancy. The variable 'quantity smoked' takes a value of zero if a mother did not smoke during pregnancy, one if she smoked less than a pack a day, and two if she smoked one or more than a pack a day.

In regression analysis we control for mothers' substance use behavior during pregnancy. We create a dichotomous indicator that equals 1 for mothers who reported to have used either alcohol and/or marijuana and/or cocaine during pregnancy. The child-specific controls include birth order, race, ethnicity and gender, and are all obtained from the NLS-CYA. We control for children's birth order since mothers' smoking behavior may vary with children's birth parities. In the birth regression analysis, we additionally control for children's birth length.

With respect to socio-economic and demographic conditions, our controls include variables that capture mother's employment status (derived from number of jobs in the last year), marital status, educational attainment (derived from highest grade completed), mothers' age, and net family income (adjusted for inflation using annual consumer price index estimates considering 2005 as the reference year). Additionally, we include mothers' weight as a proxy for maternal health. All the aforementioned variables are derived from the NLSY79.

The mean/ proportions of all control variables used in our main analysis are presented in Table 1. This descriptive information is based on the largest regression sample used in each analysis pertaining to children's birthweight and children's bodyweight indicators during pre-school ages (2-5). Importantly, with respect to maternal and family-level covariates, we find some noteworthy differences in the sample of smoker mothers versus non-smoker mothers. Focusing on the birth analysis sample, compared to smoker mothers, non-smoker mothers are significantly (at the 1 percent level) less likely to undertake substance use during pregnancy

(27 versus 50 percent) and more likely to be employed (73 versus 68 percent); be married (74 versus 55 percent); have higher family income (US$ 61352 versus 36148); and have college education (41 versus 15 percent). Referring to the demographic characteristics, on average, the sample of smokers includes a higher proportion of Whites and lower proportion of Hispanics. We observe similar differences in the mean values of the variables in the regression sample used to study preschoolers' bodyweight outcomes.

4. IDENTIFICATION STRATEGY

Much of the existing literature that investigates the relationship between maternal smoking and childhood weight outcomes estimates the association between the two variables rather than providing a causal interpretation (Von Kries et al. 2002; Wider et al. 2003; Whitaker 2004; Al Mamun et al. 2006; Oken et al. 2008).[6] This is because most analyses in this research space do not account for the potential confounding influences that may arise from unobserved heterogeneities. As such, exclusion of key variables that are likely to be correlated with mothers' smoking behavior (such as unobserved ability and personality traits) and also affect children's health outcomes may generate biased estimates (Blackburn & Neumark 1993; Nizalova & Murtazashvili 2016).

To address these empirical concerns, we adopt Rosenzweig and Wolpin's (1991) strategy by controlling for mother-specific unobserved time-invariant effects. In the context of our study, as each mother represents a unique family unit, the mother FE regressions further account for sibling- and family-specific fixed effects. Following our discussion from the previous section, our regressions incorporate a wide range of important socio-economic as well as demographic

[6] The findings from previous analyses are generally based either on descriptive analyses (comparing sample means of bodyweight measures across groups classified by mothers' smoking behavior) or simple regressions that exclude a number of potential observed and unobserved confounders.

indicators to minimize omitted variable biases that may hinder the credibility of causal interpretation of the coefficients of interest.

To study the effect of maternal smoking on children's birthweight, we estimate:

$$Y_{cm} = \alpha_1 + \delta.Mother\ Smoking_{cm} + \alpha_2.X_{cm} + \mu_m + \epsilon_{cm} \qquad (1)$$

where Y_{cm} represents birthweight (in pounds) of child c born to mother m. $Mother\ Smoking_{cm}$ is the key indicator of whether a mother m smoked during pregnancy before giving birth to child c. X_{cm} is a vector of child- and family-specific characteristics that vary within each cluster of mothers including variables that vary either by birth year or just across sibling (such as child's birth order). μ_m represents mother-specific time-invariant effects that are likely to be correlated with their smoking behavior and may affect child health outcomes. Because we control for mothers' FE, our identification is based on within-mother and across pregnancies variation in smoking behavior. Finally, ϵ_m is the error term. When standard assumptions of the FE regression are met, the parameter of interest δ in equation (1) measures the impact of maternal smoking on children's birthweight. As a cautionary note, the FE estimates may be biased if there are time-variant unobserved characteristics that are correlated with both smoking behavior and children's health outcomes.

For our analysis on children aged 2 to 5, we estimate a similar model as equation (1). The outcome of interest for the later childhood years is replaced by BFA distribution-based binary indicators of extreme bodyweight (underweight; overweight; and obese) and analogously generated WFA distribution-based indicators.

Although our primary objectives is to empirically test the presence of a causal link between maternal smoking and children's bodyweight outcomes, we estimate pooled regressions to estimate the association between the two variables of interest implied by these models. We use

pooled regression analysis to compare the sensitivity of our results across different specifications and to compare with similar estimates suggested in previous literature. In pooled analysis, we employ OLS models for the continuous measure of children's birthweight, and Probit models for binary bodyweight indicators of children aged 2-5. Finally, we report regression results generated from CRE models, which estimate within effects in a random-effects model setting (Wooldridge 2010; Schunck 2013). First proposed by Mundlak (1978), the CRE models, unlike FE regressions, estimate regression coefficients with respect to variables that vary within mothers while allowing inclusion of variables that vary between mothers only. For variables that vary within as well as between mothers, these models generate similar coefficients as FE regressions.[7] In all our regressions, we adjust the standard errors for clustering around mothers (Bertrand, Duo, & Mullainathan, 2004; Cameron & Miller, 2015).

5. RESULTS

We begin our empirical analysis by simply comparing the average weight and BMI of children across samples of mother separated by smoking behavior. As shown in Table 2, the raw difference at birth between boys born to non-smokers and boys born to smokers is 0.509 pounds. The corresponding difference in weight for girls is 0.531 pounds. Both of these differences are statistically significant at the one percent level. The statistical significance of these raw differences, which do not control for any child/mother/family characteristics, disappear by age one and are indistinguishable from zero throughout the pre-school ages with only one exception for boys at the age of four. We observe similar pattern in terms of BMI. As we cannot make causal inferences from these unadjusted differences between the children of

[7] We further attempt to estimate instrumental variable (IV) regressions using federal excise tax rates as an instrument for maternal smoking during pregnancy. Due to weak IV concerns in the saturated specifications (that control for the full set of covariates used in pooled regressions), we do not include the IV analysis in the main study. The IV estimation results are however qualitatively similar to our main analysis, especially with respect to the excess weight indicators. These results are available from the authors upon request.

smokers and non-smokers, we move next to the regression analysis.

5.1 Birthweight analysis

In Table 3, we present the full set of results obtained by estimating Equation (1). The regression estimates allow us to check if our data yields the birthweight effects in the ballpark of those estimated in prior studies. We examine effects on both a full sample, as well as sub-samples disaggregated by gender. We control for child gender in the full sample analysis. In addition to our preferred FE model depicted in Equation (1), we also present, for each sample, results obtained from the corresponding OLS (pooled analysis) and the CRE models. Coefficients on the variable "smoked" are negative and statistically significant at conventional levels across most specifications and sample types, except for girls. Focusing on the FE estimate on the full sample (column (2)), we find that children of smokers weigh 0.252 pounds less than children of non-smokers. This estimate is statistically significant at the one percent level. Corresponding estimates for the boy sample (column (5)), which is significant at the five percent level, shows that boys born to smokers weigh 0.353 pounds less than boys born to non-smokers. While the FE estimate in the girls only sample (column (8)) is also negative, it is not precise enough for us to reject the null hypothesis of no effect.

That the children of smokers on average weigh 0.252 pounds (114 grams) less than children of non-smokers is in the same vicinity as the 125-136 gram reduction in birthweight estimated by Suzuki, et al (2016) in their Japanese sample. As noted earlier, our estimates are also similar to and within the range of the estimated birthweight effects observed by Rosenzweig and Wolpin (1991).

With respect to the controls other than our key explanatory variable, the estimates in Table 3 illustrate interesting patterns in terms of direction of their relationship with the outcome variable. For example, the OLS model implies that children born to mothers with college

education have higher birth weight (albeit significant at just the 10 percent level), which is not evident in either the FE or CRE models. The OLS model also shows that as the age of the mother increases, child birthweight falls; but the FE model shows no discernible effect of mother's age on child birthweight. Additionally, the OLS model shows a significant positive effect of marriage on child birthweight, but this effect disappears in the FE specification. These differences in results across specifications is likely indicative of the fact that the FE model accounts for unobserved heterogeneities, whereas OLS does not. The former is therefore our preferred specification.

Further in Table 4, we test the effect of smoking different cigarette quantities on child birthweight. As a reminder, the key explanatory variable for these regressions is an indicator variable that takes a value of 0 if not smoking at all, 1 if smoking less than one pack a day, and 2 if smoking one pack or more a day. The FE estimate presented in Column (2) shows that mothers who smoked less than one pack a day had children who weighed 0.234 pounds less than children of non-smokers. The estimate is significant at the five percent level and is close to the effect of the binary smoking indicator shown in Table 3. The effect of smoking more than one pack a day is much larger. Specifically, we find that mothers who smoked more than one pack a day during their pregnancy had children who weighed 0.354 pounds less than those of non-smokers. This estimate is statistically significant at the one percent level. We find similarly larger and statistically significant effects of smoking a greater quantity of cigarettes in the subsamples of boys and girls.

5.2 Evaluating the impact on weight outcomes between 2 and 5 years

Next, we seek to examine the relationship between maternal smoking during pregnancy and weight outcomes during the preschool years. The WHO growth charts present children's BFA and WFA distributions starting from birth through childhood. However traditionally, the health

literature on childhood obesity primarily focuses on children of a minimum age of 2 years (Kuczmarski 2002; Cawley 2010; Wang & Lim 2012). Therefore, for consistency, we restrict our analysis to children aged between 2 and 5.

First, we examine the BFA distribution-specific indicators for this age range. We look at three binary indicators: less than the 5^{th} percentile of WHO's BMI-for-age distribution (underweight), above the 85^{th} percentile (overweight), and above the 95^{th} percentile (obese). In Table 5, the estimates from the FE models, presented in columns (2), indicate that, on average, children of smokers are 5 percent less likely to be among the lowest ventile of the WHO's BFA distribution. This estimate is consistent with previous findings that the weight disadvantage seen in children of smokers at the time of birth disappears during childhood. However, we do not see any evidence to show that smokers' children are more likely to be in upper tail of the distribution. Specifically, we find that mothers' smoking during pregnancy doesn't affect the likelihood of being among the top three or top one ventile of the WHO's BMI-for-age distribution. This finding in particular is in contrast to the positive association found in prior literature between maternal smoking during pregnancy and risk of childhood obesity. We suspect that this discrepancy in findings is because prior studies failed to control for important unobserved heterogeneities which are accounted for in our FE models. This argument is further substantiated by the statistically significant positive effects of being overweight or obese found in our pooled probit models presented in (see columns (4) and (7)) Table 5.

In Table 6, we examine the differential effects of smoking varying quantities of cigarettes on the same set of outcomes based on the BFA distribution. The evidence from thee regressions is consistent with what we find with binary indicator for smoking. Precisely speaking, we find that smoking less than one pack a day makes children 5 percent less likely to be at the lower end of the distribution. FE coefficients, presented in columns (5) and (8), that measure the

effect of smoking on the likelihood of belonging to top three or top one ventile are indistinguishable from zero.

In addition to BFA distribution-based indicators, we examine the link between maternal smoking during pregnancy with child's position in the WFA distribution. These regression estimates are presented in Table 7. The impact of differential quantities of smoking on the same outcome are provided in Table 8. None of the coefficients presented in either table are statistically different from zero.

To check the robustness of our findings in Tables 5 through to 8, we repeat these analyses using only objective measurements of children's height and weight. This check addresses the concern related to potential bias in our estimates resulting from misreporting of child health and height information (Cawley et al. 2015). This additional analysis is presented in Appendix Table A.1 and provides qualitatively similar findings.[8]

5.3 A closer look at the catch-up phenomenon

Ino (2010) suggests two possible mechanisms for the catch-up phenomenon. The first involves nicotine inducing maternal starvation or reduced appetite, and thus creating an altered hypothalamic regulatory process of energy intake and expense. The second relates to fetal exposure to nicotine resulting in abnormalities in fat cells. We further investigate the implied evolutionary impact of maternal smoking while pregnant by examining children's body weight at each specific age from one through five. The use of body weight (in pounds) permits examination right from age one as opposed to our earlier focus on ages two and beyond. Results of this additional analysis, with the preferred FE specification, are provided in Table 9. We

[8] NLS-CYA incorporates information on whether children's height and weight information are measured by scales. Although useful as an additional check, limiting analysis to objective measures substantially decreases the sample size. Hence, we prefer the estimates from the larger sample that also includes self-reported measures.

note that these results must be treated with caution given the relatively smaller sample sizes.

We find that negative effects on child weight may linger up to the age of two but not beyond. There is also no evidence of a positive impact on child weight beyond the age of three. When the child sample is classified by gender, we find that negative effects of smoking on weight up to age two are only present for the subsample of boys, and not for the girls.

There is some evidence to show that boys born to smoking mothers may have higher weight in later pre-school ages but the same cannot be said for girls. While this gender disparity is consistent with evidence from Suzuki et al. (2011), it is important to recognize that this result is only statistically significant at the 10% level. Further, our finding of no impact on weight gain in the girl sub-sample is in contrast to findings by Durmus et al. (2014) and Li et al. (2016), based on Dutch and Portuguese samples respectively.

We also present the estimates obtained from these age specific FE regressions along with the 95% confidence intervals in Figures 1 (full sample) and 2 (gender-specific samples). These figures reinforce our findings of a lack of significant difference in weight outcomes between children of smokers and non-smokers. Thus, there is only a partial support for the catch-up phenomenon outlined by Ino (2010). [9] More specifically, we find that the weight disadvantage of smokers' children disappears in later ages but there is no evidence to show that smokers' children have a higher risk of being overweight and obese in early childhood.

5.4 Potential mechanisms

A number of prior studies have sought to shed light on the mechanisms by which the maternal smoking affects child weight outcomes at birth and beyond. One of the suggested pathways for

[9] A similar analysis using children's BMI measures is also performed. The results are presented in Appendix Table A.2 and Figures A.1 and A.2.

reduced birthweight is a shorter gestation period. In Table 11, we do not find any significant association between smoking behavior and gestation period (measured in number of weeks). This finding suggests that regardless of a premature childbirth, maternal smoking during pregnancy is likely to result in lower birthweight among children.

Other pathways that could potentially affect children's health may include mothers' child-rearing behavior. Studying the associations between mother's smoking behavior and indicators of maternal care may provide some useful insights into explaining why the pooled regressions, which fail to account for mothers' unobserved characteristics, signal towards a positive and statistically significant link between maternal smoking during pregnancy and risk of excess weight among children. As an indicator of prenatal care, we consider NLS-CYA's information on whether a mother took vitamins during pregnancy, which likely promotes child health at birth (Hjertholm et al. 2018). For the later childhood measures of maternal care, we consider NLS-CYA's Home Observation Measurement of the Environment-Short Form (HOME-SF) score. HOME-SF is a measure of children's home environment quality and incorporates a wide range of information including maternal and family-level inputs provided to children.[10] We use standardized scores (ensures comparability across all child ages) as well as child age-group-specific raw scores (classified into 0-2 years and 3-5 years) of HOME-SF.

We present these tests in In Table 11. We observe that smoking behavior is strongly and negatively associated with home environment quality and has some bearing on antenatal vitamin intake. This indicates that the associational evidence presented in the prior literature with respect to maternal smoking during pregnancy and greater risk of child obesity may be driven by prevalence of lower socio-economic and health-related attributes among mothers

[10] For more details on the measure, see https://www.nlsinfo.org/content/cohorts/nlsy79-children/topical-guide/assessments/home-home-observation-measurement; Accessed June 14, 2018.

who smoke during pregnancy.

6. CONCLUSION

The numerous health hazards associated with smoking demonstrate its severity on the public health front. In fact, cigarette smoking is known to harm almost every organ of the human body, cause several diseases, and reduce the health of smokers in general, making it the leading preventable cause of death in the United States (Centers for Disease Control and Prevention; CDC 2002). Moreover, the health effects of cigarette smoking reach far beyond the smokers themselves. This study provides new and policy-relevant evidence on the child health impacts of maternal smoking during pregnancy. Our contribution to the literature in this space is a focus on the causal link between prenatal exposure to maternal smoking and future child weight outcomes.

In summary, while maternal smoking is found to have a significant negative impact on children's birth weight, the same is not related to a higher risk of obesity during later childhood. Our analysis indicates that the associational evidence on the positive link between maternal smoking and child obesity may be mediated by poor socio-economic, health, and behavioral characteristics that can commonly be attributed to mothers who smoke during pregnancy. Assuming that these characteristics are driven by mothers' time-invariant traits, our empirical models account for the potential confounders and produce plausibly more reliable estimates.

This study provides enhanced motivation for implementing policies to effectively address smoking behavior among pregnant mothers. In addition to continuing with traditional ways to curb smoking such as through information campaigns, smoke-free policies, age restrictions, etc., there is a need to implement broader and more effective methods to motivate quitting. For example, a recent paper by Islam, Folland and Kaarboe (2017) provides evidence on how investing in social capital variables such as community trust and participation in organization

activities could lower the incidence of smoking.[11] Finally, our results underscore the role of socioeconomic causes of childhood obesity and indicate the need for effective intervention in this area of public health.

[11] There has been some evidence that quitting smoking may be associated with increased risk of obesity (see Chou et al, 2004 and Liu et al, 2010). However, recent evidence by Pieroni & Salmasi (2016) maintain the importance of policies aimed at reducing smoking, as they find limited consequences in terms of a rise in obesity risk.

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Table 1
Descriptive information of variables

Variables	Birth analysis			Aged 2-5		
	Overall	Did not smoke	Smoked	Overall	Did not smoke	Smoked
Child-level characteristics						
Birth weight (pounds)	7.324	7.474	6.955***			
< 5th percentile (BMI distribution)				0.093	0.093	0.090
< 5th percentile (WFA distribution)				0.061	0.057	0.071**
> 85th percentile (BMI distribution)				0.322	0.320	0.327
> 85th percentile (WFA distribution)				0.193	0.196	0.181
> 95th percentile (BMI distribution)				0.200	0.201	0.197
> 95th percentile (WFA distribution)				0.087	0.090	0.076*
Height (inches)	20.043	20.141	19.800***	38.493	38.528	38.391
Girl	0.488	0.489	0.486	0.492	0.495	0.484
White	0.589	0.549	0.688***	0.527	0.486	0.646*
Hispanic	0.180	0.215	0.095***	0.211	0.247	0.106**
Mother-level characteristics						
Smoked	0.288			0.255		
Used substance	0.338	0.273	0.498***	0.312	0.253	0.484***
Weight	167.449	168.756	164.221***	146.854	148.158	143.038***
Employed	0.717	0.734	0.675***	0.684	0.701	0.637**
Married	0.686	0.739	0.553***	0.674	0.715	0.552***
Age	26.199	26.683	25.003	29.811	30.219	28.615***
Family income	54087.200	61352.34	36148.09***	52886.22	57941.39	38091.38***
Less than high school	0.215	0.165	0.340***	0.178	0.140	0.289***
Completed high school	0.450	0.428	0.507***	0.473	0.456	0.525***
College education	0.334	0.406	0.152***	0.348	0.404	0.185***
Observations	6,477	4,610	1,867	5,784	4,311	1,473

Notes: The descriptive information in the above table are based on the largest samples used in the respective analyses of birthweight and later childhood bodyweight outcomes (ages 2-5 years). Except for information on weight, height, and family income, all variables are dichotomous in nature. Family income is adjusted for inflation using annual consumer price index measures with 2005 considered to be the reference base year. ***, **, * denote that the difference between the means of samples related to smokers and non-smokers is significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 2
Weight and BMI, by smoking behaviour of mothers during pregnancy

	Age	Average weight (pounds)	Weight - children of non-smokers	Weight - children of smokers	Difference (1) – (2)	Average BMI (kg/m^2)	BMI - children of non-smokers	BMI - children of smokers	Difference (6) – (7)
		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Boys	Birth	7.451	7.598	7.089	0.509***	12.996	13.106	12.725	0.381***
	1 year	22.155	22.037	22.509	-0.472	19.496	19.417	19.728	-0.309
	2 years	28.279	28.392	27.951	0.440	18.221	17.986	18.901	-0.914
	3 years	32.624	32.658	32.527	0.131	22.617	24.156	18.286	5.869
	4 years	37.441	37.797	36.524	1.272**	16.482	16.381	16.744	-0.363
	5 years	42.345	42.473	41.980	0.493	16.160	16.256	15.883	0.373
Girls	Birth	7.192	7.345	6.814	0.531***	12.915	13.113	12.425	0.688***
	1 year	20.439	20.382	20.623	-0.241	18.571	18.534	18.689	-0.155
	2 years	26.669	26.811	26.254	0.557	17.745	17.533	18.365	-0.832
	3 years	31.186	31.035	31.643	-0.607	16.908	16.653	17.682	-1.028
	4 years	36.146	36.345	35.593	0.751	16.235	16.190	16.362	-0.172
	5 years	41.287	41.245	41.398	-0.152	17.462	18.128	15.700	2.426

Notes: The body mass index (BMI) estimates are reported by age and gender for greater comparability in weight outcomes, as indicated by the CDC 2000 growth charts. The above estimates are based on regression samples used for corresponding age- and gender-specific regressions. ***, **, * denote that the difference between the means is significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 3

Estimation of relationship between maternal smoking during pregnancy and birth weight

	All sample			Boys only			Girls only		
	OLS	Mother FE	Correlated RE	OLS	Mother FE	Correlated RE	OLS	Mother FE	Correlated RE
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Sample mean		7.324			7.451			7.192	
Mother (& family) characteristics									
Smoked	-0.377***	-0.252***	-0.252***	-0.354***	-0.353**	-0.356**	-0.401***	-0.206	-0.201
	(0.037)	(0.091)	(0.091)	(0.054)	(0.169)	(0.169)	(0.047)	(0.154)	(0.154)
	[-0.450; -0.304]	[-0.430; -0.075]	[-0.430; -0.074]	[-0.459; -0.248]	[-0.684; -0.022]	[-0.687; -0.025]	[-0.494; -0.308]	[-0.508; 0.095]	[-0.502; 0.100]
Used substance	0.027	0.017	0.017	0.046	0.067	0.065	0.002	-0.074	-0.084
	(0.032)	(0.051)	(0.051)	(0.045)	(0.086)	(0.087)	(0.043)	(0.112)	(0.117)
Employed	-0.048	-0.050	-0.049	-0.061	-0.128	-0.141	-0.034	0.017	0.028
	(0.036)	(0.054)	(0.054)	(0.051)	(0.094)	(0.094)	(0.048)	(0.107)	(0.108)
Married	0.151***	0.005	0.005	0.107*	-0.096	-0.101	0.194***	0.153	0.148
	(0.038)	(0.063)	(0.063)	(0.055)	(0.113)	(0.113)	(0.049)	(0.124)	(0.122)
High school education	0.076*	-0.084	-0.102	0.032	-0.390	-0.087	0.132**	0.073	-0.120
	(0.045)	(0.110)	(0.092)	(0.065)	(0.243)	(0.175)	(0.058)	(0.190)	(0.173)
College education	0.097*	-0.282	-0.273	0.080	-0.128	-0.172	0.117*	-0.498	-0.408
	(0.053)	(0.194)	(0.183)	(0.077)	(0.346)	(0.344)	(0.067)	(0.375)	(0.348)
Weight (at delivery)	0.008***	0.009***	0.009***	0.008***	0.010**	0.010***	0.008***	0.009***	0.009***
	(0.001)	(0.002)	(0.002)	(0.001)	(0.004)	(0.004)	(0.001)	(0.003)	(0.003)
Age (at delivery)	-0.014***	-0.007	-0.007	-0.007	-0.038*	-0.041**	-0.022***	0.010	0.009
	(0.004)	(0.010)	(0.010)	(0.006)	(0.019)	(0.020)	(0.005)	(0.017)	(0.017)
Family income✓	-0.000	-0.000*	-0.000*	-0.000	-0.000***	-0.000***	0.000	-0.000*	-0.000*
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Child characteristics									
Female child	-0.148***	-0.170***	-0.170***						
	(0.028)	(0.035)	(0.035)						
Birth order	0.082***	0.056	0.056	0.077***	0.110	0.116	0.087***	-0.020	-0.015
	(0.017)	(0.037)	(0.036)	(0.027)	(0.071)	(0.072)	(0.021)	(0.060)	(0.060)
Birth length	0.329***	0.254***	0.254***	0.318***	0.210***	0.212***	0.344***	0.242***	0.244***
	(0.023)	(0.031)	(0.032)	(0.032)	(0.059)	(0.059)	(0.025)	(0.045)	(0.045)
White	0.317***		0.299***	0.407***		0.397***	0.224***		0.226***
	(0.051)		(0.053)	(0.071)		(0.071)	(0.062)		(0.063)

Hispanic	0.291***	0.267***	0.256***	0.246***	0.333***	0.331***
	(0.057)	(0.058)	(0.078)	(0.078)	(0.069)	(0.071)
Cluster-specific means of variables						
Smoked		-0.119		0.020		-0.209
		(0.099)		(0.178)		(0.163)
Used substance		0.006		-0.028		0.089
		(0.063)		(0.101)		(0.123)
Employed		0.013		0.101		-0.068
		(0.069)		(0.110)		(0.119)
Married		0.164**		0.220*		0.048
		(0.077)		(0.128)		(0.134)
Education		0.184*		0.111		0.275
		(0.097)		(0.176)		(0.180)
Weight (at delivery)		-0.001		-0.002		-0.001
		(0.002)		(0.004)		(0.003)
Age (at delivery)		-0.008		0.038*		-0.033*
		(0.011)		(0.021)		(0.018)
Family income		0.000**		0.000*		0.000*
		(0.000)		(0.000)		(0.000)
Female child		0.028				
		(0.052)				
Birth order		0.050		-0.018		0.116*
		(0.043)		(0.075)		(0.064)
Birth length		0.100***		0.131**		0.115**
		(0.037)		(0.059)		(0.050)
Observations	6,477		3,313		3,164	
Number of mothers	3,832		2,507		2,464	

Notes: The above table presents regression coefficients obtained from ordinary least squares (OLS), mother fixed effects (FE), and correlated random effects (CRE) models for all children, boys only, and girls only samples. Robust standard errors are corrected for clustering on the mothers and are presented in parenthesis. For the regression coefficients of maternal smoking, we also present the 95% confidence interval values of the coefficients in squared parentheses. The above table also presents regression coefficients of the full set of covariates included in each model. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 4

Estimation of relationship between quantity smoked during pregnancy and birth weight

	All sample			Boys only			Girls only		
	OLS	Mother FE	Correlated RE	OLS	Mother FE	Correlated RE	OLS	Mother FE	Correlated RE
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Sample mean		7.324			7.451			7.192	
Quantity smoked									
Smoked (< a pack)	-0.327***	-0.234**	-0.234**	-0.301***	-0.340*	-0.338*	-0.350***	-0.137	-0.135
	(0.040)	(0.092)	(0.092)	(0.060)	(0.176)	(0.176)	(0.051)	(0.153)	(0.153)
	[-0.406 - -0.248]	[-0.415 - -0.054]	[-0.415; -0.053]	[-0.418 - -0.184]	[-0.686 - 0.005]	[-0.682; 0.007]	[-0.450 - -0.251]	[-0.437 - 0.164]	[-0.436; 0.165]
Smoked (≥ a pack)	-0.514***	-0.354***	-0.351***	-0.491***	-0.408*	-0.437**	-0.543***	-0.632***	-0.437**
	(0.059)	(0.127)	(0.127)	(0.082)	(0.214)	(0.212)	(0.077)	(0.217)	(0.212)
	[-0.630; -0.397]	[-0.604; -0.104]	[-0.600; -0.102]	[-0.652; -0.329]	[-0.827; 0.010]	[-0.852; -0.022]	[-0.694; -0.391]	[-1.057; -0.206]	[-0.852; -0.022]
Cluster-specific means of variables									
Smoked (less than a pack)			-0.086			0.061			-0.239
			(0.102)			(0.186)			(0.164)
Smoked (a pack or more)			-0.151			-0.046			0.086
			(0.142)			(0.235)			(0.230)
Observations		6,477			3,313			3,164	
Number of mothers		3,832			2,507			2,464	

Notes: The above table presents estimated impact of varying quantities of cigarettes smoked during pregnancy on children's birthweight. The estimates are obtained from OLS, mother FE, and CRE models for all children, boys only, and girls only samples. Each specification includes similar controls used in the models estimated in Table 3. Robust standard errors are corrected for clustering on the mothers and are presented in parenthesis. For the regression coefficients of interest, we also present the 95% confidence interval values of the coefficients in squared parentheses. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 5

Estimation of relationship between maternal smoking during pregnancy and the risk of having extreme bodyweight using BMI-for-age distribution for children aged 2-5

	< 5th percentile of WHO distribution			> 85th percentile of WHO distribution			> 95th percentile of WHO distribution		
	Pooled probit	Mother FE	Correlated random effects	Pooled probit	Mother FE	Correlated random effects	Pooled probit	Mother FE	Correlated random effects
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Sample mean		0.093			0.322			0.201	
Smoked	-0.007	-0.053*	-0.053*	0.041**	0.006	0.006	0.023*	-0.024	-0.024
	(0.010)	(0.028)	(0.028)	(0.016)	(0.041)	(0.041)	(0.014)	(0.035)	(0.036)
	[-0.026; 0.013]	[-0.108; 0.001]	[-0.108; 0.001]	[0.008; 0.073]	[-0.074; 0.085]	[-0.074; 0.086]	[-0.004; 0.051]	[-0.094; 0.046]	[-0.094; 0.046]
Cluster-specific means of variables									
$\overline{\text{Smoked}}$			0.071**			0.046			0.047
			(0.033)			(0.050)			(0.042)
Observations		5,328			5328			5328	
Number of mothers		2,878			2,878			2,878	

Notes: The above table presents regression estimates of the relationship between maternal smoking during pregnancy and BFA distribution based bodyweight indicators for preschoolers. For each binary indicator of extreme bodyweight indicators (underweight, overweight, and obese), we estimate pooled probit, mother FE, and CRE models. In addition to maternal smoking indicator, the pooled probit regression and the CRE model include indicators of mothers' substance use during pregnancy, employment, marital status, age, weight, education, family income, and child's gender, race, ethnicity, and birth order. The mother FE regression selects the covariates that vary by time and across siblings (excludes race and ethnicity indicators). For the regression coefficients of interest, we also present the 95% confidence interval values of the coefficients in squared parentheses. Due to the lack of variation in the cluster specific mean of variable related to smoking a pack or more a day, the variable is dropped from the CRE model. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 6

Estimation of relationship between quantity smoked during pregnancy and the risk of having extreme bodyweight using BMI-for-age distribution for children aged 2-5

	< 5th percentile of WHO distribution			> 85th percentile of WHO distribution			> 95th percentile of WHO distribution		
	Pooled probit	Mother FE	Correlated random effects	Pooled probit	Mother FE	Correlated random effects	Pooled probit	Mother FE	Correlated random effects
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Sample mean		0.093			0.322			0.201	
Quantity smoked									
Smoked (< a pack)	-0.013	-0.056**	-0.056*	0.037**	0.008	0.008	0.024	-0.028	-0.028
	(0.011)	(0.029)	(0.029)	(0.018)	(0.042)	(0.042)	(0.016)	(0.037)	(0.037)
	[-0.035; 0.009]	[-0.112; -0.000]	[-0.112; 0.000]	[0.001; 0.072]	[-0.075; 0.090]	[-0.075; 0.091]	[-0.007; 0.054]	[-0.100; 0.045]	[-0.101; 0.045]
Smoked (≥ a pack)	0.009	-0.041	-0.043	0.051*	-0.004	-0.004	0.023	-0.008	-0.007
	(0.016)	(0.041)	(0.041)	(0.027)	(0.068)	(0.068)	(0.023)	(0.053)	(0.053)
	[-0.022; 0.040]	[-0.120; 0.039]	[-0.123; 0.037]	[-0.002; 0.104]	[-0.137; 0.129]	[-0.137; 0.129]	[-0.022; 0.067]	[-0.112; 0.097]	[-0.111; 0.098]
Cluster-specific means of variables									
Smoked (less than a pack)			-0.012			-0.024			0.032
			(0.041)			(0.072)			(0.055)
Observations		5,328			5328			5328	
Number of mothers		2,878			2,878			2,878	

Notes: The above table presents regression estimates of the relationship between quantity of cigarettes smoked during pregnancy and BFA distribution based bodyweight indicators for preschoolers. The controls included in each specification are similar to the variables incorporated in respective models in Table 5. For the regression coefficients of interest, we also present the 95% confidence interval values of the coefficients in squared parentheses. Due to the lack of variation in the cluster specific mean of variable related to smoking a pack or more a day, the variable is dropped from the CRE model. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 7

Estimation of relationship between maternal smoking during pregnancy and the risk of having extreme bodyweight using weight-for-age distribution for children aged 2-5

	< 5th percentile of WHO distribution			> 85th percentile of WHO distribution			> 95th percentile of WHO distribution		
	Pooled probit	Mother FE	Correlated random effects	Pooled probit	Mother FE	Correlated random effects	Pooled probit	Mother FE	Correlated random effects
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Sample mean		0.061			0.196			0.087	
Smoked	0.010	0.016	0.016	0.012	0.024	0.024	0.002	0.024	0.024
	(0.008)	(0.021)	(0.021)	(0.014)	(0.032)	(0.032)	(0.010)	(0.025)	(0.025)
	[-0.006; 0.025]	[-0.026; 0.058]	[-0.026; 0.058]	[-0.016; 0.040]	[-0.040; 0.087]	[-0.040; 0.087]	[-0.018; 0.022]	[-0.025; 0.074]	[-0.025; 0.074]
Cluster-specific means of variables									
$\overline{\text{Smoked}}$			0.017			-0.016			-0.028
			(0.027)			(0.040)			(0.029)
			[-0.035; 0.070]			[-0.094; 0.063]			[-0.085; 0.029]
Observations		5,784			5,784			5,784	
Number of mothers		3,016			3,016			3,016	

Notes: The above table presents regression estimates of the relationship between maternal smoking during pregnancy and WFA distribution based bodyweight indicators for preschoolers. For each of the three binary WFA distribution-based indicators, we estimate pooled probit, mother FE, and CRE models. The controls included in each specification are similar to the variables incorporated in respective models in Table 5. For the regression coefficients of interest, we also present the 95% confidence interval values of the coefficients in squared parentheses. Due to the lack of variation in the cluster specific mean of variable related to smoking a pack or more a day, the variable is dropped from the CRE model. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 8

Estimation of relationship between quantity smoked during pregnancy and the risk of having extreme bodyweight using weight-for-age distribution for children aged 2-5

	< 5th percentile of WHO distribution			> 85th percentile of WHO distribution			> 95th percentile of WHO distribution		
	Pooled probit	Mother FE	Correlated random effects	Pooled probit	Mother FE	Correlated random effects	Pooled probit	Mother FE	Correlated random effects
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Sample mean		0.093			0.322			0.201	
Quantity smoked									
Smoked (< a pack)	0.002	0.008	0.009	0.014	0.029	0.028	0.005	0.024	0.024
	(0.009)	(0.022)	(0.022)	(0.016)	(0.033)	(0.033)	(0.011)	(0.027)	(0.027)
	[-0.016; 0.019]	[-0.034; 0.051]	[-0.034; 0.051]	[-0.017; 0.045]	[-0.036; 0.093]	[-0.036; 0.093]	[-0.018; 0.027]	[-0.028; 0.076]	[-0.028; 0.076]
Smoked (≥ a pack)	0.029**	0.049	0.049	0.006	0.001	0.002	-0.005	0.026	0.025
	(0.012)	(0.035)	(0.035)	(0.023)	(0.054)	(0.054)	(0.016)	(0.035)	(0.035)
	[0.005; 0.053]	[-0.019; 0.118]	[-0.019; 0.118]	[-0.039; 0.051]	[-0.105; 0.107]	[-0.104; 0.108]	[-0.036; 0.027]	[-0.043; 0.095]	[-0.043; 0.094]
Cluster-specific means of variables									
Smoked (less than a pack)			0.009			-0.019			0.010
			(0.037)			(0.057)			(0.037)
			[-0.064; 0.083]			[-0.130; 0.092]			[-0.063; 0.082]
Observations		5,784			5,784			5,784	
Number of mothers		3,016			3,016			3,016	

Notes: The above table presents regression estimates of the relationship between quantity of cigarettes smoked during pregnancy and WFA distribution-based bodyweight indicators for preschoolers. The controls included in each specification are similar to the variables incorporated in respective models in Table 5. For the regression coefficients of interest, we also present the 95% confidence interval values of the coefficients in squared parentheses. Due to the lack of variation in the cluster specific mean of variable related to smoking a pack or more a day, the variable is dropped from the CRE model. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 9

Mother fixed effects estimates of maternal smoking during pregnancy on weight by child age

Age-	**1 year old**	**2 years old**	**3 years old**	**4 years old**	**5 years old**
ALL					
Sample mean	21.302	27.466	31.929	36.795	41.831
Smoked	0.449	-2.689**	1.555	0.351	0.150
	(0.851)	(1.160)	(1.312)	(1.921)	(1.551)
Sample size	1,690	1,523	1,750	1,781	1,876
No. of mothers	1,416	1,309	1,472	1,505	1,546
BOYS					
Sample mean	22.155	28.279	32.624	37.441	42.345
Smoked	0.070	-3.137**	3.637*	6.323	1.247
	(1.277)	(1.335)	(2.018)	(4.612)	(2.565)
Sample size	850	754	904	892	964
No. of mothers	775	695	838	814	870
GIRLS					
Sample mean	20.439	26.669	31.187	36.146	41.288
Smoked	1.578	-0.163	4.309	-0.577	1.829
	(2.467)	(2.264)	(2.685)	(2.658)	(3.015)
Sample size	840	713	846	889	912
No. of mothers	770	769	775	809	825

Notes: The above table presents mother FE regression estimates of the relationship between maternal smoking during pregnancy and children's weight at each child age from 1 to 5 years. Controls include contemporaneous information on maternal employment, marital status, education, age, weight, family income, child's height, and birth order. For the combined sample of all children, the regression also controls for child gender. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Table 10

Estimation of the association between maternal smoking during pregnancy and additional pre- and post-natal outcomes

	Gestation (weeks)	Vitamin intake	HOME-SF raw score 0-2 years	HOME-SF raw score 3-5 years	HOME-SF standard score 0-5 years
Sample mean	38.586	0.947	139.884	202.878	972.863
Mother smoked	-0.062	-0.014*	-2.011**	-4.946***	-17.240***
	(0.074)	(0.007)	(0.871)	(1.186)	(4.455)
Smoked (< a pack)	-0.073	-0.001	-0.725	-2.046*	-6.905
	(0.082)	(0.008)	(0.955)	(1.241)	(4.742)
Smoked (≥ a pack)	-0.034	-0.039	-5.714***	-12.816***	-46.648***
	(0.117)	(0.010)	(1.431)	(1.991)	(7.641)
Observations	6,940	6,686	4,924	6,607	12,607

Notes: The above table reports regression coefficients obtained from OLS regressions. The robust standard errors are corrected for clustering on mothers. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Figure 1

Mother FE estimates: impact of maternal smoking during pregnancy on weight by child age

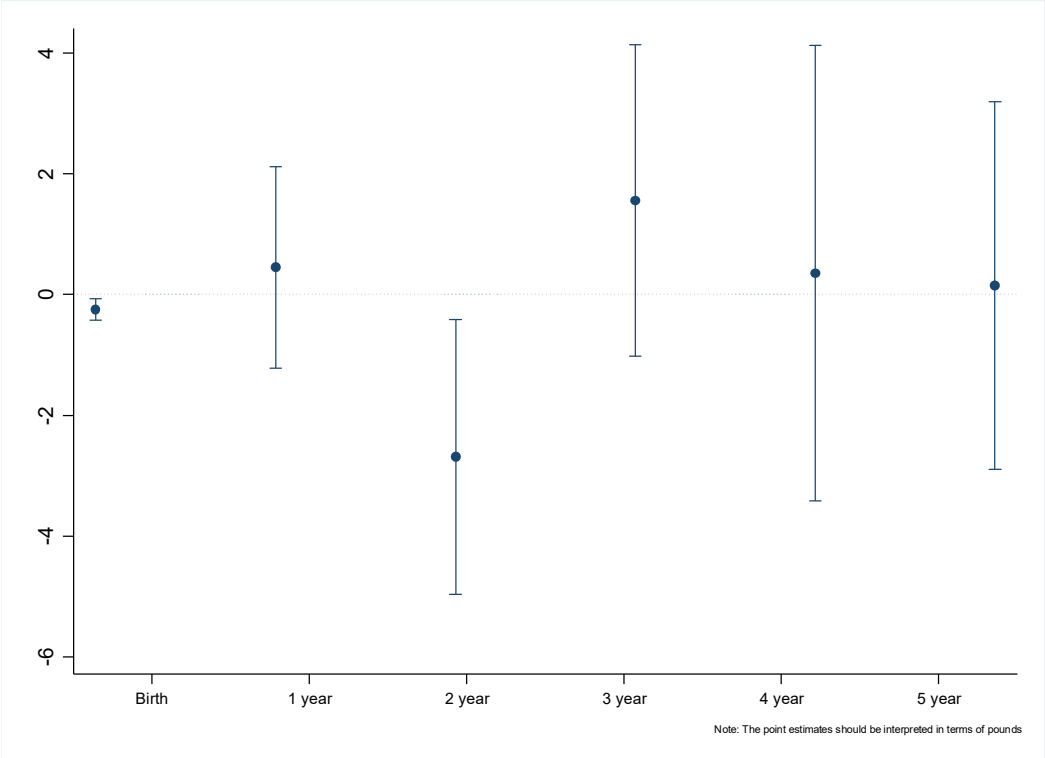
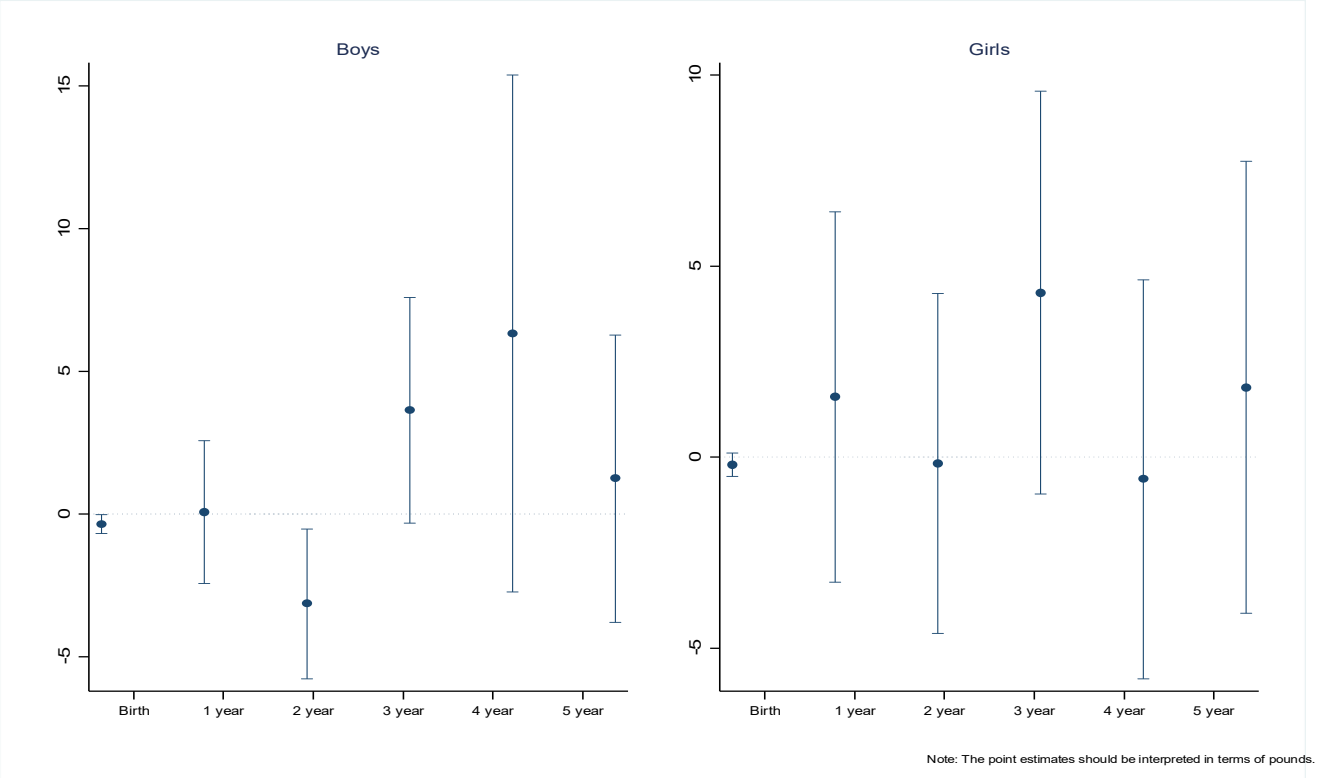


Figure 2

Mother FE estimates: impact of maternal smoking during pregnancy on weight by child age and gender



Appendix

Table A.1

Maternal smoking and risk of extreme bodyweight based on objective weight and height measurements

	Indicators based on BFA distribution (aged 2-5)					
Panel A:	<5th percentile		>85th percentile		>95th percentile	
	Pooled Probit	Mother FE	Pooled Probit	Mother FE	Pooled Probit	Mother FE
Sample mean:	0.092		0.282		0.153	
Smoked	0.002	-0.006	0.057**	0.003	0.036*	0.071
	(0.016)	(0.068)	(0.025)	(0.093)	(0.019)	(0.074)
	[-0.029; 0.033]	[-0.140; 0.127]	[0.009; 0.106]	[-0.180; 0.186]	[-0.001; 0.074]	[-0.075; 0.216]
Observations	2,090					
Number of mothers	1,487					

	Indicators based on WFA distribution (aged 2-5)					
Panel B:	<5th percentile		>85th percentile		>95th percentile	
	Pooled Probit	Mother FE	Pooled Probit	Mother FE	Pooled Probit	Mother FE
Sample mean:	0.062		0.180		0.072	
Smoked	0.014	0.043	-0.013	0.098	0.018	0.047
	(0.112)	(0.047)	(0.021)	(0.064)	(0.015)	(0.047)
	[-0.010; 0.038]	[-0.049; 0.134]	[-0.055; 0.029]	[-0.027; 0.224]	[-0.046; 0.015]	[-0.046; 0.139]
Observations	2,258					
Number of mothers	1,573					

Notes: The above table replicates pooled probit and mother FE regressions estimated in Table 5 (Panel A) and Table 7 (Panel B) using indicators constructed from objective measurements of children 's height and weight. The objective measurements are identified from NLS-CYA information based on whether the reported height and weight estimates are based on tape- and scale-based measurements. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Table A.2

Mother fixed effects estimates: impact of maternal smoking on BMI by child age

Age-	Birth	1 year old	2 years old	3 years old	4 years old	5 years old
ALL						
Sample mean:	12.956	19.035	17.981	19.858	16.359	16.793
Smoked	-0.583**	1.138	-3.666	5.006	1.102	1.469
	(0.261)	(1.218)	(4.767)	(8.254)	(0.715)	(1.653)
Sample size	6,477	1,690	1,523	1,750	1,780	1,876
No. of mothers	3,832	1,416	1,309	1,472	1,504	1,546
BOYS						
Sample mean:	12.996	19.496	18.220	22.618	16.482	16.160
Smoked	-0.756	2.013	-0.588	-1.572	2.777*	0.352
	(0.519)	(1.572)	(1.082)	(2.357)	(1.637)	(1.195)
Sample size	3,313	850	754	904	891	964
No. of mothers	2,507	775	695	838	813	870
GIRLS						
Sample mean:	12.916	18.571	17.745	16.908	16.236	17.462
Smoked	-0.432	-8.254**	1.371	3.197	-0.047	0.934
	(0.365)	(3.244)	(1.918)	(2.108)	(0.832)	(0.985)
Sample size	3,164	840	769	846	889	912
No. of mothers	2,464	770	713	775	809	825

Notes: The above table presents mother FE regression estimates of the relationship between maternal smoking during pregnancy and children's BMI at each child age from 1 to 5 years. Controls include contemporaneous information on maternal employment, marital status, education, age, weight, family income and child's height, and birth order. For the combined sample of all children, the regression also controls for child gender. ***, **, * denote the coefficients are significantly different from zero at the 10%, 5%, and 1% level respectively.

Figure A.1

Mother FE estimates: impact of maternal smoking during pregnancy on BMI by child age

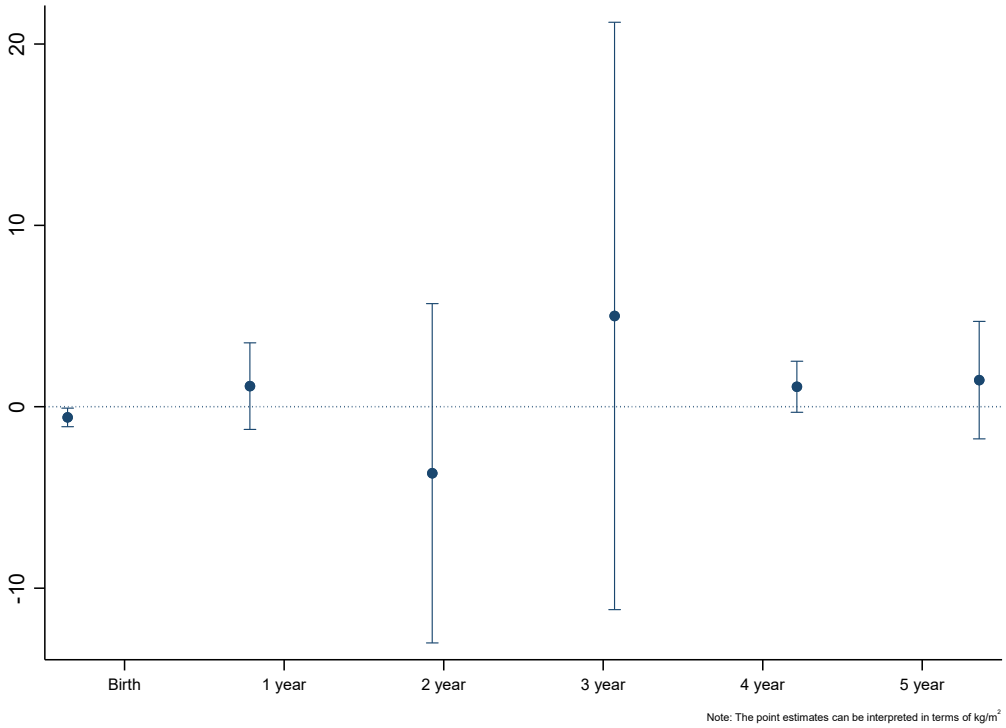


Figure A.2

Mother FE estimates: impact of maternal smoking during pregnancy on BMI by child age and gender