The relationship between maternal pre-pregnancy BMI and preschool obesity *

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Abstract

The increasing prevalence of obesity during pregnancy raises concerns over the intergenerational transmission of obesity and its potential to exacerbate the current obesity epidemic. The fetal origins hypothesis posits that the intrauterine environment may have lasting effects on children's outcomes and mother's pre-pregnancy obesity has been associated with pediatric obesity. However, previous research is largely based on comparing individuals across families and hence cannot control for unobservable factors associated with both maternal and child obesity. We use within-family comparisons and instrumental variables to identify the effect of maternal pre-pregnancy obesity on obesity in children. Consistent with extant research, OLS models that rely on across-family comparisons indicate a significant correlation between maternal pre-pregnancy obesity and child obesity. However, maternal fixed effects render those associations insignificant. Instrumenting for mother's BMI with her sisters' BMI confirms the null result indicating that the in utero transmission of obesity is likely not driving the increase in childhood obesity.

1 Introduction and Previous Literature

Childhood obesity has more than doubled in the past 30 years (Ogden et al. 2014). While alarming on its own, recent research has also shown that obesity is persistent and associated with long-term health consequences: obese children are more likely to be obese as adults

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and are at greater risk of type 2 diabetes, heart disease, stroke and certain cancers (Pan et al., 2012; Ogden et al., 2014;). Another study showed that one third of children who were overweight in kindergarten were obese by eighth grade, and almost every child who was obese remained that way as an adult (Cunningham et al., 2014). Recent calculations for the U.S. indicate spending on obesity-related illnesses may be as high as 20 percent of annual health care expenditures (Cawley and Meyerhoefer, 2012).

In addition to the well known health consequences of obesity, research has also linked childhood obesity to poor cognitive outcomes, although the evidence is mixed as to whether the effect is causal (Kaestner and Grossman, 2009; Averett and Stifel, 2010; Zavodney, 2013). The effects of childhood obesity on cognitive outcomes may be particularly acute for lower performing students (Capogrossi et al., 2013). In addition, there is mounting evidence that obese children suffer emotional and behavioral problems (Griffiths et al., 2011). The consensus amongst experts is that it is far easier and less costly to prevent childhood obesity rather than to reverse it (Oken et al., 2003; Whitaker, 2008). Thus, the goal of this paper is to examine potential early determinants of obesity, in particular whether there is a direct, observable link to childhood obesity from maternal obesity prior to pregnancy using a large-scale, national survey.

The hypothesis that maternal obesity would have an effect on childhood obesity has standing in the literature. Scholars from various disciplines have established that pregnancy is a critical time for children's development and that mother's decisions and environmental exposures during pregnancy can have profound effects on birth and later life outcomes. In particular, the fetal origins hypothesis posits that the uterine environment can have far-reaching and lasting impacts on adult health (Almond and Currie, 2011). In this paper, we explore whether this transmission mechanism may explain childhood obesity through

maternal obesity. This paper relates to a large literature that relies on natural experiments to identify the effect of maternal undernutrition during pregnancy on the long-term health of the child (e.g., Almond 2006; Almond and Muzumder 2011; Schulz, 2010; Stein et al., 1995; Prentice, 1983; Ravelli, 1976). This literature finds adverse health consequences for the children of women who were undernourished during pregnancy and these consequences often persist into adulthood. Far less is known about the causal effects of maternal pre-pregnancy obesity on child outcomes.

Maternal obesity has been significantly associated with pregnancy complications. In fact, obesity is fast becoming the most common complication of pregnancy in the U.S. (McDonald, 2010; Lu et al., 2001). Underscoring the magnitude of the problem, pre-pregnancy obesity prevalence continues to increase; in 2009 1 in 5 pregnant women were obese when they became pregnant (Fisher, 2013) and some estimates indicate that nearly 40 percent of pregnant women in the U.S. are obese (Roman et al., 2008).[1] The prenatal period has been identified as a critical period for the development of obesity in children (Oken et al., 2003).

Obesity is believed to be passed from mothers to children through high concentrations of glucose and fatty acids that pass through the placenta. Mothers with high pre-pregnancy BMI and those who gain excessive amounts of weight during pregnancy have more fat and thus deliver greater concentrations of glucose and fatty acids to the developing fetus (Catalano, 2003; Lawlor et al., 2008; Lawlor et al., 2011). The resulting increase in fetal insulin

[1]Medical professionals have long stressed the dangers of obesity and excessive weight gain during pregnancy and highlighted how they might affect the pregnancy and the health of the fetus (Boney 2005; Whitaker 2004). An increasing awareness of these links has led to repeated updating of weight gain recommendations. As recently as 2009 the Institute of Medicine (IOM) issued revised guidelines for healthy pregnancy weight gain. In this paper, we include weight gain as an independent variable, but do not focus on healthy weight gain.

accelerates fetal growth and predisposes the child to weight gain later in life (Lawlor et al., 2008; Lawlor et al., 2011). [2]

Numerous studies have documented a correlation between maternal obesity and childhood obesity (e.g. Salsberry et al., 2007; Whitaker, 2008; Oken et al., 2007; Oken, 2009; Jääskeläinen et al.,2011; Branum et al., 2011; Yu et al., 2013; Ludwig et al., 2013). In particular, studies that focus solely on a cross-section of children generally show that maternal pre-pregnancy obesity and/or excess gestational weight gain (GWG) lead to an increased probability of childhood obesity (e.g. Oken et al., 2003; Oken et al., 2007; Whitaker, 2008).

Across-family comparisons of unrelated children may not reflect only the intrauterine effects of maternal overnutrition but also obesity-promoting or environmental factors that are shared between a mother and her child (Lau et al., 2014; Ludwig et al., 2013). Thus, researchers have compared children of the same mother using mother fixed-effects to control for shared familial influences that may be unobserved, such as genetics and home environment, but do not vary across time. Branum et al., (2011), using data on over 2700 families interviewed in the Collaborative Perinatal Project, find in OLS models that pre-pregnancy weight and GWG are statistically significantly associated with BMI z-scores in four-year old children but this effect disappears in family fixed effects models. Using data on over 146,000 Swedish males, Lawlor et al., (2011) find no association between GWG and BMI at age 18 when comparing siblings. Lawlor et al. (2008) use data on over 4000 families from the U.K. and in OLS models find a significant association between pre-pregnancy BMI and childhood BMI at ages 9 to 11. This effect disappears when they instrument for pre-pregnancy BMI with an obesity genotype as a predictor of pre-pregnancy BMI. In contrast, Ludwig et al.

[2] Animal studies confirm the detrimental effects of maternal obesity on offspring. For example, Samuelsson, et al. (2007) show that diet-induced obesity in pregnant mice results in higher rates of insulin resistance and adiposity in their offspring.

(2013) using data on all school-age children in Arkansas, find evidence in a maternal fixed effects model that high pregnancy weight gain is associated with childhood overweight status. They use this as support for the fetal origins explanation that overnutrition may program the fetus for future weight gain and obesity, though "the magnitude of the effect may be small" (Ludwig, et al. 2013, p. 5).

Given the health costs and potential cognitive consequences associated with childhood obesity in the literature, we seek to add to this literature and identify whether a woman's weight status before pregnancy and her weight gain during pregnancy exert a potentially causal effect on childhood obesity. If there is evidence that obesity is transmitted from mothers to children during pregnancy, policy and practice aimed at reducing maternal weight before pregnancy and controlling for weight gain during gestation may have profound health impacts for not only the women themselves, but also their children. Our study is novel in that we use both maternal fixed-effects (FE) and instrumental variable (IV) estimation in an attempt to ascertain whether the well-established correlation between material pre-pregnancy obesity and childhood obesity is potentially causal. Similar to others (e.g. Oken et al., 2003; Whitaker 2008) we focus on children aged 2 to 4 years to avoid the confounding influence of the school environment and its potential effect on obesity. As far as we know, we are the first to apply the instrumental variable method to data from the United States. Similar to studies to previous studies that have used maternal fixed effects or instrumental variables, we find little evidence that maternal pre-pregnancy BMI exerts a causal impact on obesity among preschool-aged children.

2 Data and outcomes

2.1 Data and Sample Creation

We use the National Longitudinal Survey of Youth 1979 (NLSY79) cohort for our analysis. The NLSY79 sampled 12,686 individuals between the ages of 14 and 21 in 1979 with annual interviews conducted until 1994 and subsequent interviews every other year up to the year 2010 (the most recent year available at the time of this paper). The respondents report data on their labor market experience, births, and marriages every survey round. Children who were born to women in the NLSY79 have been surveyed biannually since 1986. In addition, and crucial to this study, the NLSY collected information on the height and weight of respondents in multiple instances. Height was collected in 1981, 1982, 1985 and 2006, while weight is collected every round. As noted earlier, the most recent data available are from the 2010 survey, when the respondents were ages 45-53. Thus, for nearly all women in the sample, complete fertility histories are observed. In fact, 99.97 percent of births used in this study occur by 2000 and the most recent births we observe in our sample occurred in 2004. These data do not provide a nationally representative sample of children or young adults, although they are appropriately regarded as representative of the population of offspring born to U.S. women who were aged 14–22 in 1979 (Wu and Li, 2005).

As we are interested in the explanatory power of mother's weight before pregnancy, we use pre-pregnancy BMI as the key variable of interest. We use the self-reported heights and weights discussed above to create pre-pregnancy BMI and assign respondents to one of four categories using the World Health Organization Cutoffs. Underweight corresponds to a BMI of less than or equal to 18.5; BMI in the recommended range is between 18.5 and 24.9; overweight women are those with a BMI ranging from 25 to 29.9; and obese women have a

BMI greater than or equal to 30. The NLSY also asks women about their gestational weight gain for each pregnancy (GWG), which we control for in all of our models.

In our sample, observations are at the child level. Thus, mothers may appear multiple times, once for each child, and be assigned to different BMI categories for each pregnancy. We start with a sample of all women in the NLSY and their children in the years they are sampled. We calculated each child's age and sex specific BMI percentile using the 2000 CDC reference data (CDC, 2000). Children with a BMI percentile for their age and sex in excess of 85 are considered by health professionals to be at risk of overweight while those with BMI percentiles in excess of 95 are described as overweight. To be consistent with the adult categories of overweight and obese we refer to children with BMI percentiles greater than 85 as overweight and those with BMI percentiles greater than 95 as obese.

The CDC growth charts were devised using survey data from three nationally representative samples of boys and girls aged 2 to 20 during the years 1963 to 1994.[3] This means that when a child is identified as overweight in our sample, it indicates that his BMI is higher than 85% of surveyed children of his age during the reference time period. By definition, then, about 5 percent of children are obese in the reference sample. This metric allows for variation of obesity prevalence over time.

We start with a sample of 8265 NLSY children observed from 1986 to 2010 who are 24 to 59 months old and who have information on height and weight so that we can calculate their BMI percentile score. Because the key explanatory variable of interest is mother's pre-pregnancy BMI we drop the 1348 child years where this information is missing. In addition, because pre-term births have their own set of complications, we further limit our sample to those children who were born preterm (eliminating an additional 845 child-years), and those

[3]These data are compiled from the NHANES I, II, and III surveys.

whose gestation length was in excess of 42 weeks (169 child years) and those born below 500 grams (6 observations) or above 7000 grams (2 observations). We also drop 42 women who reported having diabetes during the year they had a birth. [4]

Because the NLSY is longitudinal, some of the children are observed multiple times in their preschool years. When this is the case we take only their first observation (dropping an additional 1214 observations). We also drop multiple births (34 observations) and those children for whom information on breastfeeding and c-section birth were not reported. those who did not report if they were breastfed (170). This leaves us with a sample of 4435 children. Of those, 1774 have no siblings in the sample. 1758 have one sibling in the sample, 672 have two siblings in the sample, 220 have three siblings in the sample and 35 have four siblings in the sample and 6 have five siblings in the sample. Of the 2661 mothers with more than one child in the sample, 1781 did not change their pre-pregnancy weight category across births. Of those 910 who did change their pre-pregnancy weight category, 213 moved to a lower weight category while the majority moved to a higher weight category.

2.2 Outcomes

Table 1 presents descriptive statistics for the outcome variables by mother's pre-pregnancy BMI category. The unadjusted means reveal that the average BMI percentile scores increase as the mother's BMI category increases indicating a positive correlation between mother's BMI and her child's BMI. We also see that the proportion of children who are overweight or obese increases as the mother's BMI increases.

The NLSY is a particularly rich source of data and we control for many covariates to isolate the effect of mother's pre-pregnancy weight on her preschool-aged child's weight. In

[4]These are similar to the sample inclusion criteria used by Ludwig et al. 2013

particular, we control for mother's age, age at first birth, parity, education, urban residence, marital status and income; these means are shown in Table 2.[5]

We also control for the child's age in months, birth order, birth weight, gender and race. In addition, we include controls for the month of the mother's first prenatal visit, whether she smoked or used alcohol during the pregnancy, whether or not the child was breastfed and the home environment using the Home Observation for Measurement of the Environment (HOME) score. The HOME score is a measure that reflects the cognitive stimulation of the child's environment and the emotional relationship between the mother and child. The HOME score has been linked to childhood obesity by other researchers (Strauss and Knight, 1999).

3 Methods

3.1 OLS

Using the sample of mothers with singleton births over our sample period, we test whether pre-pregnancy obesity and GWG are correlated with obesity among two-, three-, and four-year olds using the following OLS specification:

$$y_{imt} = \alpha + O_{imt}\beta + X_{imt}\phi + Z_m\xi + T_t\theta + \epsilon_{imt} \tag{3.1}$$

where y is the ith child's BMI the first time they appear in the sample between the ages of two and four, for the mth mother in the tth year. O is a vector representing the mother's weight, either as a continuous measure of BMI, or a series of dichotomous variables indicating underweight, overweight or obese status and a control for GWG for each child (pregnancy) i in a given year t. The primary coefficient of interest is the vector β. X_{im} is a vector of

[5]As is common in survey data, a significant number of women do not report their income. Rather than delete them, we measure income categorically including a category for missing. These categories are adjusted for inflation.

variables specific to each child as shown in table 2 (e.g. child's age in months, the HOME score, mother's education, marital status, mother's age at the birth, parity, whether she smoked, used alcohol or prenatal vitamins during the pregnancy and the month of her first prenatal visit, the child's birth weight, whether the child was breastfed, and whether the child was born via c-section). The vector Z includes mother's characteristics that do not vary with each child, which include mother's race and her age at first birth. T_t is vector of year fixed effects.

3.2 Maternal Fixed Effects

As noted earlier, genetics and other time-invariant characteristics of the mother may affect our outcomes of interest. These characteristics might include chronic health conditions, health habits, or environmental exposure. For this reason, we add mother fixed effects to our initial OLS specification in order to account for time-invariant mother characteristics. This specification allows us to compare births across mothers and the effect of pre-pregnancy BMI is now identified off of mothers whose pre-pregnancy BMI status changes over pregnancies. The specification is as listed in 3.1 but with mother fixed effects (γ_m) as follows:

$$y_{imt} = \alpha + O_{imt}\beta + X_{imt}\phi + \gamma_m + T_t\theta + \epsilon_{imt} \tag{3.2}$$

Note that the Z_m vector drops out from this specification because these characteristic do not vary across children. This specification only includes those mothers who had more than one child in the sample. Identification of the parameters on the maternal pre-pregnancy BMI categories comes from discordant siblings (i.e. siblings whose mother changed pre-pregnancy BMI categories).

3.3 Results: OLS and Fixed Effects

Tables 3 and 4 show four OLS specifications starting from the most parsimonious, and then progressively adding relevant controls. Table 3 shows these specification for the dependent variable measured as overweight (BMI percentile¿85) and Table 4 for obese (BMI percentile ¿95). The first column of each table presents results from a model that only includes the mother's pre-pregnancy BMI categories, her GWG and a set of binary indicators for child's age in months. These unadjusted regressions reveal that mothers who begin their pregnancies obese have preschoolers who are 9.5 percentage points more likely to be overweight and 7.3 percentage points more likely to be obese. Both effects are large with the 9.5 percentage point increase in the probability of being overweight translating to a 36.8% increase in the probability of a child falling into the overweight category. ((9.5/25.41)*100=38.7) and the 7.3 percentage point increase in the probability of obesity translating into a 48.8 ((7.3/14.95)*100=48.8 percentage point increase in the probability of preschool obesity.

In column 2, we add in the child's birth weight as a covariate. Previous researchers have found that child birth weight attenuates the effect of mother's pregnancy weight gain on childhood obesity (e.g. Ludwig et al., 2013). Birth weight in pounds is positively and significantly related to both the probability of being overweight and obese as a preschooler. Specifically, an additional pound at birth translates into a 2.1 percentage point (8.6 percent) increase in the probability of overweight and a 1.2 (8.1 percent) increase in the probability of obesity. Consistent with previous literature, adding this variable only slightly attenuates the coefficients on mother's pre-pregnancy BMI.

In column 3 we add a set of year dummy variables (not shown in the table). The coefficients on these year dummies reveal that children born in the later years of our sample are more likely to be overweight or obese which is consistent with the upward trend in

childhood obesity seen in the U.S. Including these year fixed-effects reduces the magnitude of the coefficients on both the pre-pregnancy BMI variables but they remain statistically significant and still exert a sizeable effect.

In column 4 we add the full set of covariates shown in table 2 although for space reasons we only show some of the coefficients. There are several notable findings. First, the addition of these covariates further attenuates the effect of maternal pre-pregnancy obesity on preschool overweight and obesity but the coefficients are still statistically significant. Women who begin their pregnancies obese are 4.6 percentage points more likely to have an obese preschooler than those who being their pregnancies in the recommended BMI range. Second, children of married mothers and those who were breastfed are less likely to be overweight or obese. However, children born via c-section are more likely to be overweight and obese. Hispanic children are more likely to be overweight and obese but we see no effect for black children relative to white children. Birth order is negatively related to obesity but not to overweight. We also find that the HOME environment is not a significant predictor of preschool obesity.

While these OLS results establish a clear correlation between pre-pregnancy BMI and preschool overweight and obesity even after controlling for a rich set of covariates, they do not necessarily establish a causal relationship. To move closer to potentially causal effects, we turn to the results of our maternal fixed effects models which are shown in table 5. We show the FE results in two panels–the top panel has the overweight outcome and the bottom panel the obese outcome. All models include the full set of covariates shown in the table of means and discussed above.

In each panel, the first column presents the OLS results to facilitate comparisons across models. The second column presents the FE model that is identical in specification to the OLS model. This model is identified off of those mother's whose pre-pregnancy BMI category

changed across pregnancies. Women who gain weight versus lose weight across pregnancies might be quite different. Thus, in the third column of this table we present the FE model for only those who gained weight between pregnancies. Finally, because mothers with more than two children might be quite different from mothers with two children (the majority of women in our sample have two children) the last column (5) limits the sample to mothers with only two children who gained weight between their first and second pregnancy. The results from these specifications reveal that regardless of the sample, we find no effect of pre-pregnancy BMI on preschool overweight or obesity. [6]

These maternal FE results indicate no statistically significant effect of pre-pregnancy BMI on our outcomes of interest. The point estimates are smaller than the OLS estimates and often switch signs. As expected, the standard errors are larger. These estimates indicate that once we have controlled for time-invariant, family-specific factors, there is no effect of maternal pre-pregnancy BMI on preschool overweight or obesity.

4 Methods: Instrumental Variables

Our OLS models establish a correlation between maternal obesity and preschool obesity. This association disappears when we use a maternal fixed-effects specification which is consistent with much of the literature as described earlier. While the maternal fixed effects methodology is an improvement over the OLS specifications, this method does not control for time-varying, unobservable factors that may affect both mother and child obesity. These factors include mother's pre- and post-natal behavior that may either reinforce or compensate for child's initial health endowments and concerns about intergenerational transmission

[6]The fixed-effects sample is naturally smaller than the OLS sample given that only mothers with more than one child are in this sample. To be sure that our FE results are not driven by this difference in sample composition, we ran our OLS models on this smaller sample and find qualitatively the same results which we do not show here. This gives us confidence that our FE results are not an artifact of a smaller sample.

of obesity. As a result, we cannot assert definitively that a causal relationship between maternal pre-pregnancy obesity and childhood obesity using either OLS or maternal fixed effects. In order to address the issue of causality, we turn to the method of instrumental variables exploiting the large-scale nature and sampling design of the NLSY79. In particular, among the original respondents to the NLSY are a number of siblings.

Drawing on previous work by Cawley (2004), we instrument the BMI of the mother's in our sample using the BMI of sisters of our NLSY79 mothers, a group we expect to have similar health and obesity status as the mothers in the sample. Unlike Cawley (2004), who used all siblings, our instrument includes only sisters in order to avoid the complications that may arise comparing BMI across genders due to muscle mass and other considerations. Sister obesity status (or aunt obesity, from the perspective of the child) is plausibly exogenous to child obesity; as long as the aunt is not the child's primary caretaker, aunt obesity should influence the child's obesity only through the genetics that are shared between sisters and shared between mothers and children. The model we estimate is given by:

$$O_{imt} = \gamma + B_{imt}\zeta + X_{imt}\phi + Z_m\xi + T_t\theta + \mu_{imt} \tag{4.1}$$

$$y_{imt} = \alpha + \hat{O}_{imt}\beta + X_{imt}\phi + Z_m\xi + T_t\theta + \epsilon_{imt} \tag{4.2}$$

where B_{im} is the average BMI of any sisters interviewed in the year of mother m's birth. y is now the ith child's (child of the mth mother) obesity status in year t. The variable of interest, mother's weight status, O, remains as defined in 3.1. We use the predicted values of O, $\hat{O}$ in the second stage. As before, T_t is a vector of year fixed effects.[7]

Using the average of all sisters interviewed as an instrument is plausibly more exogenous than mother's own BMI, which might be correlated with exercise and eating habits that are

[7]Cawley, 2004 adjusts the instrument for age. Because we use the average of the sisters' BMI we have not made an age adjustment.

also practiced by children. Because we only have one instrument, in this specification we measure mother's pre-pregnancy BMI as a continuous variable as opposed to the categories used in earlier regressions. First stage results of the IV regressions are not shown, but they indicate, as expected, that sister's BMI is a positive and significant predictor of mother's BMI.

5 Results: IV

In table 6, we present the results of our IV regressions. We also show OLS and FE models in this table to facilitate comparisons. Columns 1 and 2 are the OLS model on the full sample with the full set of covariates. Columns 3 and 4 are the FE model on the full sample and columns 5 and 6 are the IV models. Because the IV model is estimated on a smaller sample size (only on those women who have a sister in the NLSY) we limit our OLS and FE results to that sample to compare. We also do not include gestational weight gain these models as we only have on instrument and GWG is likely endogenous as well. In these models, we see no evidence of an impact of mother's pre-pregnancy BMI on the probability that her preschooler is overweight or obese.

As expected, the IV standard errors are larger than the OLS standard errors though comparable to those of the FE when using the same sample. The IV specifications do not point to a causal relationship between mother's BMI and childhood BMI.

We have focused our attention on overweight and obese since these are the conditions associated with health concerns. In appendix 1, we also examine the child's BMI percentile as an outcome. We show OLS, FE and IV models. The same pattern holds here in that we find a strong positive effect of mother's pre-pregnancy BMI on her child's preschool BMI percentile. This effect disappears in FE and IV models.

6 Discussion and Conclusions

Evidence from numerous studies establishes a strong relationship between maternal obesity and childhood obesity. Here, we examine a snapshot of obesity before the mother becomes pregnant and attempt to determine whether these correlations stand up to stricter identification strategies including one depending on within-family variation and another on an instrumental variable. We extend the literature by focusing on a national sample of births. We consider childhood obesity before the child enters school, thus eliminating estimation problems that may arise from heterogeneous content and quality of schooling.

We find, as in other studies, that there is a positive and significant relationship between mother's obesity status before she becomes pregnant and her child's obesity during the preschool years. These correlations, however, disappear in maternal fixed-effects models and in instrumental variables specifications.

These results suggest that other time-invariant, mother-specific characteristics, such as exercise habits and healthy eating, may be just as or more important than GWG or pre-pregnancy BMI for determining healthy child outcomes. However, we cannot control for unobservable, time-variant, mother-specific characteristics in the FE specifications. If mothers who changed weight categories from one pregnancy to the next were, for instance, aware of the dangers associated with weight gain and engaged in compensatory behavior to counteract the potential adverse effects of their pre-pregnancy weight status, we may not see an effect on the probability of overweight or obesity. Our instrumental variables results reinforce the FE results; they also indicate no effect of maternal BMI on child obesity. Our genetic instrument, however, is most likely to be informative about the genetic channels of inter-generational transmission of obesity.

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7 Tables

Table 1: Sample outcome means and proportions on children's obesity status by mother's pre-pregnancy BMI.

Sample Means (proportions) of outcome variables by Mother's Pre-pregnancy BMI					
		Mom's Pre-Pregnancy BMI			
	All Children	BMI<18.5	18.5< BMI<24.9	25< BMI<29.9	BMI>30
BMI percentile>95	.1495	.1152	.1385	.1714	.2096
(obese)	(.3566)	(.3197)	(.3455)	(.3771)	(.4075)
BMI percentile>85	.2541	.2242	.2367	.2913	.3253
(overweight)	(.4354)	(.4177)	(.4251)	(.4546)	(.4691)
BMI percentile	50.897	45.5234	49.6133	54.686	56.5969
	(35.4814)	(35.6036)	(35.2521)	(35.1987)	(36.2998)
Observations	4435	330	2873	817	415

Standard errors for continuous variables in parentheses

Table 2: Sample means and proportions for control variables

Means of Control Variables					
		Mom's Pre-Pregnancy BMI			
	All	<18.5 Underweight	18.5<BMI<24.9 Recommended	25<BMI<29.9 Overweight	>30 Obese
GWG	31.9186	34.0939	32.5889	31.6805	26.0169
	(13.8158)	(14.422)	(12.782)	(14.8585)	(16.3877)
Age of child, months	39.4724	40.5697	39.6453	39.1322	38.0723
	(9.3746)	(9.4725)	(9.4432)	(9.2579)	(8.8898)
Hispanic	.1921	.1576	.1883	.246	.1398
Black	.2573	.2061	.2381	.2889	.3687
Child is male	.5094	.4879	.5089	.5275	.494
Age, first birth	22.7017	21.4879	22.6878	22.9645	23.2458
	(4.8097)	(4.0725)	(4.7926)	(4.8535)	(5.2154)
Age, birth of child	26.1964	24.2273	25.9565	26.9829	27.8747
	(4.7268)	(4.4674)	(4.6463)	(4.67)	(4.8118)
Birth order	2.0045	1.8121	1.9527	2.1432	2.2434
	(1.0962)	(1.0837)	(1.0535)	(1.1622)	(1.1962)
HOME score	44.7227	44.8455	46.9074	39.7442	39.3012
	(30.3902)	(30.878)	(30.3789)	(29.6492)	(29.7897)
Education years	12.7278	12.3848	12.7767	12.7638	12.5907
	(2.3527)	(2.1804)	(2.3594)	(2.4969)	(2.114)
Married	.7055	.6091	.7156	.7283	.6675
Sep./Div./Wid.	.143	.2121	.1403	.1248	.1422
Income missing	.138	.1424	.1406	.1285	.1349
Low Income	.2949	.3879	.2851	.2852	.3084
Middle Income	.2924	.2545	.2865	.3121	.3253
Urban residence	.7475	.7182	.7466	.7576	.7566
Month 1st prenatal	2.552	2.5393	2.5517	2.5117	2.644
	(1.6676)	(1.6234)	(1.644)	(1.6766)	(1.8411)
Prenatal vitamins?	.9452	.9455	.9499	.9351	.9325
Alcohol Use During Pregnancy					
Unknown	.0005	0	.0003	.0012	0
<1/month	.2401	.2636	.2482	.2215	.2024
Monthly	.0399	.0515	.04	.0404	.0289
Weekly	.0408	.0182	.0466	.0269	.0458
Cigarette Use During Pregnancy					
Unknown	.0032	.0061	.0035	.0024	0
No smoking	.9968	.9939	.9965	.9976	1
Breastfed	.5274	.4727	.5506	.4982	.4675
C-section	.2228	.1455	.1998	.2864	.3181
Observations	4435	330	2873	817	415

Standard deviations in parentheses

Table 3: OLS results with full controls on overweight status (BMI percentile>85).

	(1)	(2)	(3)	(4)
Pre-Preg. BMI<18.5	-0.014	-0.008	-0.004	0.002
	(0.023)	(0.023)	(0.023)	(0.023)
Pre-Preg. 25<BMI<29.9	0.055***	0.050***	0.044**	0.034*
	(0.018)	(0.019)	(0.019)	(0.018)
Pre-Preg. BMI>30	0.095***	0.088***	0.075***	0.062**
	(0.024)	(0.024)	(0.025)	(0.025)
GWG	0.001**	0.001*	0.001*	0.001
	(0.000)	(0.001)	(0.001)	(0.001)
Birth weight, lbs		0.021***	0.019***	0.022***
		(0.006)	(0.006)	(0.006)
Hispanic				0.048**
				(0.020)
Black				0.001
				(0.020)
Child is male				0.020
				(0.013)
Age, first birth				-0.004
				(0.003)
Age, birth of child				0.009***
				(0.004)
Birth order				-0.014
				(0.011)
HOME score pctile				-0.000
				(0.000)
Yrs of education				-0.006
				(0.004)
Married				-0.049**
				(0.024)
Breastfed				-0.025*
				(0.015)
C-section				0.038**
				(0.017)
Constant	0.231***	0.083	0.055	0.074
	(0.044)	(0.062)	(0.062)	(0.102)
Year FE	No	No	Yes	Yes
Age in Months Fe	Yes	Yes	Yes	Yes
Table 2 controls	No	No	No	Yes
Observations	4,435	4,435	4,435	4,435
R-squared	0.017	0.020	0.030	0.044

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 4: OLS results with full controls on obese status (BMI percentile>95).

	(1)	(2)	(3)	(4)
Pre-Preg. BMI<18.5	-0.023	-0.020	-0.016	-0.009
	(0.018)	(0.018)	(0.018)	(0.018)
Pre-Preg. 25<BMI<29.9	0.032**	0.029*	0.023	0.014
	(0.015)	(0.015)	(0.015)	(0.015)
Pre-Preg. BMI>30	0.073***	0.069***	0.058***	0.046**
	(0.022)	(0.022)	(0.022)	(0.022)
GWG	0.001**	0.001	0.001	0.001
	(0.000)	(0.000)	(0.000)	(0.000)
Birth weight, lbs		0.012**	0.010*	0.013**
		(0.005)	(0.005)	(0.005)
Hispanic				0.053***
				(0.016)
Black				0.011
				(0.017)
Child is male				0.016
				(0.011)
Age, first birth				-0.005*
				(0.003)
Age, birth of child				0.010***
				(0.003)
Birth order				-0.019**
				(0.009)
HOME score pctile				-0.000
				(0.000)
Mom's yrs of education				-0.005
				(0.003)
Mother is married				-0.051**
				(0.020)
Breastfed				-0.023*
				(0.012)
C-section				0.026*
				(0.014)
Constant	0.137***	0.055	0.025	0.026
	(0.036)	(0.052)	(0.052)	(0.084)
Year FE	No	No	Yes	Yes
Age in Months Fe	Yes	Yes	Yes	Yes
Table 2 controls	No	No	No	Yes
Observations	4,435	4,435	4,435	4,435
R-squared	0.013	0.015	0.027	0.044

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 5: Fixed Effects results on child overweight and obese status using mother's pre-pregnancy BMI

	OLS	Maternal FE			
		Overweight			
	(1)	(2)	(3)	(4)	(5)
Pre-Preg. BMI<18.5	-0.000	0.043	0.044	0.030	0.067
	(0.023)	(0.061)	(0.070)	(0.083)	(0.099)
Pre-Preg. 25<BMI<29.9	0.032*	-0.077*	-0.085*	-0.072	-0.099
	(0.018)	(0.042)	(0.050)	(0.057)	(0.065)
Pre-Preg. BMI>30	0.052**	-0.042	-0.051	0.036	-0.039
	(0.024)	(0.067)	(0.075)	(0.101)	(0.103)
GWG	0.001	-0.001	-0.001	-0.001	-0.002
	(0.000)	(0.001)	(0.001)	(0.001)	(0.002)
Table 2 controls	Yes	No	Yes	Yes	Yes
Observations	4,435	4,435	4,224	1,758	1,674
R-squared	0.051	0.066	0.070	0.097	0.110
Number of CASEID		2,910	2,830	879	837
		Obese			
	(1)	(2)	(3)	(4)	(5)
Pre-Preg. BMI<18.5	-0.010	0.031	0.058	0.089	0.143**
	(0.018)	(0.046)	(0.046)	(0.068)	(0.070)
Pre-Preg. 25<BMI<29.9	0.012	-0.057	-0.077*	-0.006	-0.037
	(0.015)	(0.036)	(0.040)	(0.053)	(0.058)
Pre-Preg. BMI>30	0.039*	0.011	-0.012	0.049	-0.004
	(0.022)	(0.061)	(0.070)	(0.096)	(0.100)
GWG	0.000	-0.002**	-0.002**	-0.002	-0.002*
	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)
Table 2 controls	Yes	Yes	Yes	Yes	Yes
Observations	4,435	4,435	4,224	1,758	1,674
R-squared	0.050	0.075	0.083	0.112	0.124
Number of CASEID		2,910	2,830	879	837

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 6: IV, OLS, and FE results using mother's BMI as a continuous variable. The mother's sisters' average BMI serves as the instrument for mother's BMI in the IV regressions.

	IV		OLS		FE	
	overweight	obese	overweight	obese	overweight	obese
Mom's Pre-Preg. BMI	-0.013	-0.008	0.003	0.002	-0.007	-0.004
	(0.010)	(0.008)	(0.003)	(0.002)	(0.010)	(0.009)
Table 2 controls	Yes	Yes	Yes	Yes	Yes	Yes
Observations	1,456	1,456	1,456	1,456	1,456	1,456
R-squared	0.052	0.071	0.078	0.086	0.142	0.156
Number of CASEID					952	952

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 7: Appendix: IV, OLS, and FE results using mother's BMI as a continuous variable on a continuous measure of child's obesity: BMI percentile. The mother's sisters' average BMI serves as the instrument for mother's BMI in the IV regressions.

	IV	OLS	FE
	y=Child's BMI percentile		
Pre-Preg. BMI	-1.275	0.558***	-0.109
	(0.887)	(0.116)	(0.446)
Table 2 controls	Yes	Yes	Yes
Observations	1,456	4,435	4,435
R-squared	0.036	0.053	0.061
Number of CASEID			2,910

Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1