Measuring the Implications of Maternal Obesity for Child Development

Heide Jackson University of Wisconsin-Madison

September 21, 2012

Abstract

Childhood obesity is a growing problem in the United States with implications for many aspects of child development; obesity negatively affects a child's health, cognitive abilities, and non-cognitive traits. While an emerging literature has shown how obesity may affect a child's skill acquisition and health, this paper will contribute to this field by showing that maternal obesity has direct consequences for child obesity, health, cognitive capacity, and non-cognitive traits as well as indirect, but nonetheless important, implications for a child's educational attainment. Using data from the National Longitudinal Survey of Youth-Children (1979), preliminary analysis suggests an important effect of maternal obesity on child characteristics. More surprisingly, analysis suggests that childhood obesity does not have a similar influence after controlling for maternal weight status. Results suggest an important intergenerational transfer of obesity which negatively affects child development.

Introduction

This paper explores the cumulative effect of maternal and childhood obesity on a child's likelihood of graduating from high school using data from the NLSY-C (1979), the National Longitudinal Survey of Youth-Children. While previous literature has examined associations between childhood obesity and health, cognitive development, and non-cognitive personality traits, few studies have measured how maternal obesity may affect children [1] . This paper contributes to the literature by looking at the effects of obesity from a life course perspective and examining the influence of both maternal and child obesity on a child's later educational attainment.

Childhood obesity trajectories may begin in-utero. Studies in epigenetics suggest that a child's nutritional intake in the womb influences future nutritional intake [7][3].A mother's diet may be influential in this period as children become familiar with and inclined to eat foods consumed by the mother while she is pregnant and later while she is breast feeding[6]. Even after breastfeeding is concluded, mothers typically serve as the primary providers of food for young children and serve as role models dictating what and how much a child eats [6]. Mothers also determine a child's body image and may even perceive a child who is overweight or obese as being within a normal range.

Children who are obese in childhood are at increased risk for developing: sleep disorders, type 2 diabetes, heart disease, and kidney failure[5]. Children who develop these conditions may have poorer school attendance and poorer cognitive development. Obesity may also negatively affect non-cognitive traits. Obese children may be subjected to discrimination and bullying negatively affecting their social skills and self-esteem [4]. Poor non-cognitive development is associated with lower levels of educational attainment and sub-par labor market performance in adulthood.

This analysis will test two hypotheses: the first is that maternal obesity increases the likelihood of a child becoming obese and obese children will be disadvantaged in their health, cognitive capacity, and behavioral traits. The second hypothesis is that the timing of maternal obesity matters and mothers who are obese around the time they become pregnant are at an increased risk of passing this disadvantage on to their children, independent of their future weight status. My preliminary analysis suggests support for these hypotheses: maternal obesity around the time of pregnancy increases the likelihood of children becoming obese and developing poorer cognitive abilities, non-cognitive traits, and health, but follow-up work will more rigorously test the effect of maternal obesity on children and explore how the timing of maternal obesity may matter.

Data

To study the effect of obesity on educational attainment, I use data

from the National Longitudinal Survey of Youth (NLSY) Studies, the NLSY-1979 and the NLSY-1979(C). Beginning in 1979, researchers with the NLSY 1979 interviewed a nationally representative sample of adolescent youths aged 15-19. The main NLSY 1979 cohort has been interviewed at 2 year intervals from 1979 through the present. Beginning in 1986, information on children born to female members of the cohort was also collected. At subsequent two year intervals, mothers and their children were interviewed. Both NLSY and NLSY-C surveys collect data on respondent health, weight status, cognitive abilities, psychological characteristics, education, and educational aspirations over these three decades. This allows for a unique opportunity to study both maternal and child characteristics and to determine how maternal weight status may influence child weight and health from birth to late adolescence.

Dependent Variables

The dependent variable in this analysis is whether an adolescent graduated from high school. This outcome is chosen because high school graduation is an important predictor of later social standing. The NLSY-C has information on whether the adolescent graduated from high school or received a GED certificate and the date of either certification. For the purposes of this analysis, ged graduates will not be considered high school graduates. GED recipients are excluded because these individuals often resemble high school dropouts in terms of college entry and placement in the labor market [2]. While the timing of high school graduation may also be important for later outcomes, no high school graduates will be excluded based on when they graduated from high school.

Independent Variables

I will include measures of child birth weight and indicators of child health, weight classification, non-cognitive capacity, and cognitive traits at ages 3 to 5, 7 to 9, and 10 to 12 in this analysis. Child health is measured by parental reporting of whether the child has any health limitations which limit daily activities. Non-cognitive and cognitive traits are measured by previously validated scales administered to children during the survey. Family background is also incorporated into the models through adding variables which measure maternal education at the time of the child's birth, the household's income quartile at the time of the child's birth, and the mother's weight classification in the three year's prior to the child's birth, when the child is aged 3 to 5, 7 to 9, and 10 to 12 years of age. For children, weight will be classified based on CDC guidelines for appropriate height for weight. Children will be classified as obese if they fall above the 95th percentile for their age and sex. Maternal weight status will be classified based on bmi with women having a bmi over 30 being classified as obese. Because some weight gain is associated with pregnancy, women known to be in the second or third trimester at each interview wave will be excluded from the bmi measures.

Analytic Approach

I have so far run a series of non-recursive regression models predicting child

characteristics at birth, ages 3 to 5, ages 7 to 9, ages 10 to 12, as well as child educational attainment measured by high school graduation. I run logistic regressions to measure the likelihood of having a health limitation, a clinical behavior problem, and graduating from high school. OLS regressions are used to measure cognitive skills at available ages. For both logistic and ols specifications, standard errors are clustered by mother as the NLSY-C sample includes all children born to women of the NLSY cohort and not controlling for sibship would result in heterogeneity.

The general conceptual model underlying these regressions is shown in Figure 1. Literature suggests that maternal characteristics should have a direct effect on a child's characteristics in early life which affects later cognitive, behavioral, and physical development, and ultimately affects educational attainment. This model is obviously a simplification in that it ignores correlations across these characteristics and lingering direct effects of maternal characteristics on child development, but its purpose is to provide a visual aid for thinking about maternal characteristics while pregnant may have important implications for children that last into adulthood.

Across models, all earlier measures of child health and maternal characteristics will be included so as to measure enduring direct effects of these charactistics on child outcomes Results from these regressions are insightful. Mothers who are obese around the time their child was conceived have an increased likelihood of having a child who becomes obese, experiences a health limitation, has lower cognitive abilities, and a has lower probability of graduating from high school. The exact magnitude of these associations can be seen in Table 1. The coefficients in the regression models are odds ratios for all outcomes except cognition. The cognition coefficients should be interpreted as the standard deviation change in cognition resulting from a one unit change in the relevant covariates. After controlling for maternal bmi, a child's obesity did not have any influence on any of these outcomes. This preliminary finding is interesting and suggests that the intergenerational transmission of obesity is important for child outcomes. However, future models will seek to also control for maternal weight at other ages of the child, to determine how the timing of maternal obesity and variations in maternal weight affecta child's future prospects.

In future analyses, I will more run a formal structural equation model to estimate the total effect of maternal obesity at various ages on a child's outcomes. Additionally I will move to running a fixed effects model to test how changes in maternal weight may affect children and more formally test the proposition that in-utero obesity has an added effect on child outcomes. Then to better speak to the magnitude of maternal obesity's effects, I will create a simulation to explore what would happen to the educational distribution of the NLSY-C 1979 cohort if the prevalence of maternal and child obesity are varied from their current levels. Specifically, I will construct scenarios simulating a cohort of children that has the same characteristics and is subject to the same effects of cognition, health, and non-cognitive traits on educational attainment but has differing prevalence of maternal and child obesity Two extreme scenarios might be of particular interest: in one scenario, the prevalence of obesity will be set

to 0; in the other scenario, the prevalence of obesity for mothers and children will be doubled from the levels observed in the NLSY-C. These extreme scenarios are useful for both better understanding the magnitude of obesity's effects during childhood and on ultimate educational attainment and are useful from a policy perspective. Specifically, they suggest what levels of education might be achieved if obesity were eliminated, and the simulations might suggest what the impact of obesity will be if obesity continues to rise.

	(1) LBW	(2) Obesity 3 to 5	(3) Obesity 7 to 9	(4) Health Limits 7 to 9	(5) Behavior Probs 7 to 9	(6) Cognition 7 to 9	(7) Obesity 12 to 14	(8) Health Limits 12 to 14	(9) Behavior Probs 12 to14	(10) Cognition 12 to 14	(11) HS or Ged
HH Lowest Income	1.409*	0.857	1.135	1.047	1.512*	-0.229***	1.440	1.154	0.949	0.821***	1.030
	(2.48)	(-0.98)	(0.74)	(0.29)	(2.29)	(-3.96)	(1.48)	(0.85)	(-0.26)	(-4.03)	(0.11)
Mother Obese Near Birth	0.774	1.828***	3.759***	1.599***	1.270	-0.246***	5.031***	1.365*	1.231	0.850***	0.688
	(-1.87)	(4.91)	(9.54)	(3.81)	(1.49)	(-5.32)	(7.84)	(2.11)	(1.19)	(-4.04)	(-1.40)
Mom No HS or GED	1.543***	1.467**	1.008	1.014	2.564***	-0.547***	1.383	0.803	1.276	0.771***	0.489**
	(3.51)	(2.96)	(0.05)	(0.10)	(6.03)	(-10.92)	(1.49)	(-1.38)	(1.39)	(-6.02)	(-2.96)
LBW		0.941	1.066	1.556*	1.214	-0.221**	0.719	1.141	0.808	0.912	2.214
		(-0.27)	(0.24)	(2.38)	(0.81)	(-3.05)	(-0.76)	(0.55)	(-0.71)	(-1.34)	(1.86)
Obesity 3to 5			5.194***	1.465*	1.331	-0.160*	1.346	1.010	0.836	0.957	1.708
			(10.48)	(2.19)	(1.40)	(-2.54)	(1.05)	(0.04)	(-0.63)	(-0.67)	(1.12)
Obesity 7 to 9							11.08***	0.730	0.978	0.896	1.931
							(9.92)	(-1.40)	(-0.09)	(-1.79)	(1.32)
Health Limits 7 to 9							1.456	10.30***	2.451***	1.005	0.831
							(1.21)	(15.36)	(4.28)	(0.09)	(-0.55)
Behavior Probs 7 to 9							1.307	1.110	6.086***	0.934	1.115
							(0.89)	(0.47)	(9.31)	(-1.09)	(0.35)
Cognition 7 to 9							0.904	0.956	0.732***	1.708***	1.141
							(-0.92)	(-0.66)	(-3.54)	(25.88)	(0.96)
Obesity 12 to 14											0.947
											(-0.16)
Health Limits 12 to 14											1.001
											(0.00)
Behavior Probs 12 to 14											0.505**
											(-2.60)
Cognition 12 to 14											2.582***
											(6.08)
Constant						0.350***					
N	4659	3460	2780	3160	2971	(12.11)	1380	2108	2040	1991	1124

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

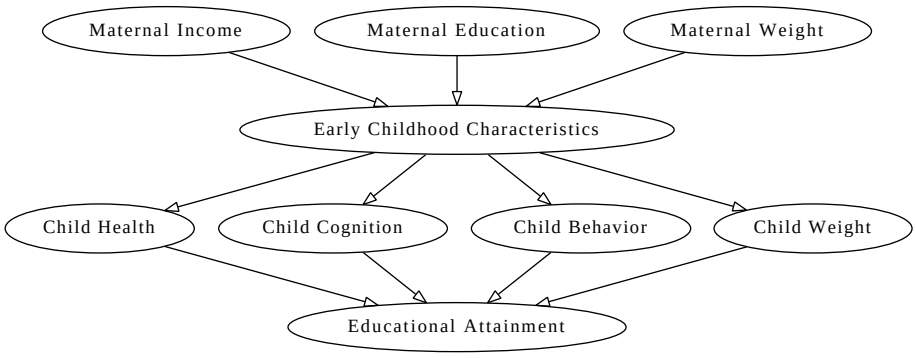

Figure 1: Pathways Linking Obesity to Education

Bibliography

[1] Edward C. Norton Lisa M. Powell Euna Han. Direct and Indirect Effects of Teenage Body Weight on Adult Wages. *Euna Han, Edward C. Norton, Lisa M. Powell*, 2009.

[2] James J. Heckman and Yona Rubinstein. The Importance of Noncognitive Skills: Lessons from the GED Testing Program. *The American Economic Review*, 91(2):145–149, 2001.

[3] B.T. Heijmans, E.W. Tobi, A.D. Stein, H. Putter, G.J. Blauw, E.S. Susser, P.E. Slagboom, and LH Lumey. Persistent epigenetic differences associated with prenatal exposure to famine in humans. *Proceedings of the National Academy of Sciences*, 105(44):17046, 2008.

[4] C. Katharina Spiess John Cawley. Obesity and Skill Attainment in Early Childhood. *NBER Working Paper No. 13997*, 2008.

[5] Dejian Lai PhD Jennifer Turner BA Tim Poffenbarger Jonathan M. Sorof and Ronald J. Portman. Overweight, Ethnicity, and the Prevalence of Hypertension in School-Aged Children. *Pediatrics*, 113(1):475–482, 2004.

[6] J.S. Savage, J.O. Fisher, and L.L. Birch. Parental influence on eating behavior: conception to adolescence. *The Journal of law, medicine & ethics*, 35(1):22–34, 2007.

[7] R.A. Waterland and K.B. Michels. Epigenetic epidemiology of the developmental origins hypothesis. *Annu. Rev. Nutr.*, 27:363–388, 2007.