Maternal Obesity Is Associated With Younger Age at Obesity Onset in U.S. Adolescent Offspring Followed Into Adulthood

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Abstract

GORDON-LARSEN, PENNY, LINDA S. ADAIR, AND CHIRAYATH M. SUCHINDRAN. Maternal obesity is associated with younger age at obesity onset in U.S. adolescent offspring followed into adulthood. *Obesity.* 2007;15:2790–2796.

Objective: The objective was to test the hypothesis that maternal obesity is associated with younger age of offspring's obesity onset.

Research Methods and Procedures: We used prospective, nationally representative, longitudinal data collected across Waves I (1995; 12 to 20 years), II (1996; 13 to 20 years), and III (2001; 18 to 28 years) of the National Longitudinal Study of Adolescent Health (N = 14,654; 49% female). Interval regression analysis was used to assess the association between maternal obesity and age at offspring's obesity onset (International Obesity Task Force BMI ≥30 equivalent age- and sex-specific cut-off points for adolescents and BMI ≥30 for young adults) using self-reported heights and weights, adjusting for race/ethnicity, sex, parental education, and family income, accounting for complex sampling design.

Results: The net effect of having an obese mother varied by race/ethnicity and was associated with a significantly earlier age at obesity onset ($p = 0.0001$) for whites [$\beta = -8.1$ year, 95% confidence interval (CI), −9.3; −6.9)], blacks ($\beta = -10.8$ years, 95% CI, −12.4; −9.2), Hispanics ($\beta = -7.0$ years, 95% CI, −9.2; −4.8), and Asians ($\beta = -8.6$ years, 95% CI, −13.3; −3.9). Earlier obesity onset (<18 years) was associated with increased severity at young adulthood (mean BMI, 36.0 ± 0.3 kg/m^2) vs. onset after age 18 (mean BMI, 34.4 ± 0.2 kg/m^2; $p = 0.0001$). There were no sex differences in the association of maternal obesity to age at obesity onset.

Conclusions: Having an obese mother was associated with earlier age at obesity onset across all race/ethnic groups, particularly non-Hispanic blacks. Early obesity onset has important health consequences because of its association with more severe adult obesity.

Key words: adolescents, ethnic minorities, family history, longitudinal

Introduction

Offspring of obese parents are at risk for obesity because of shared genes and shared environments. While most studies have investigated the risk of becoming obese given parental obesity (1–3), few studies have examined how parental obesity relates to the age at which offspring become obese.

In the past 3 decades, obesity has tripled among U.S. adolescents (4) and more than doubled among 20 to 39 year olds, (5) with considerable variation by race/ethnicity (6). As longitudinal studies have shown, a substantial amount of weight gain occurs in the transition from adolescence to young adulthood (6,7) with strong tracking from adolescence into adulthood (8–11).

Adolescent obesity is associated with increased mortality and morbidity related to a variety of chronic diseases later in life (12–14). However, less is known about the long-term health effects of age at obesity onset. Research suggests that adults who became obese in childhood and remained obese into adulthood are at higher metabolic risk than those with adult-onset obesity (15). Furthermore, reversal of obesity between childhood and adulthood is associated with decreased metabolic risk in adulthood (15).

In this paper, we use innovative statistical methodology to investigate how maternal obesity influences the age at

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which obesity is first observed in offspring participants in the nationally representative, prospective National Longitudinal Study of Adolescent Health (Add Health).[1] The Add Health data span a lifecycle period of high obesity incidence and include an ethnically diverse sample, which allows comparisons by race/ethnicity over a period of risk. Further, we explore the severity of obesity among those with earlier onset obesity and among those with obese mothers.

Research Methods and Procedures

Survey Design

The study population consisted of >20,000 adolescents enrolled in Wave I (1994 to 1995) of Add Health, a longitudinal, nationally representative, school-based study of U.S. adolescents in grades 7 to 12, supplemented with minority special samples and collected under protocols approved by the Institutional Review Board of the University of North Carolina-Chapel Hill. Wave II (1996) included 14,438 eligible Wave I adolescents who would be still enrolled in high school during 1996, including dropouts, measured between April and August 1996. Thus, older youths who were high school graduates in Wave I were not followed in Wave II. In Wave III, all Wave I respondents were followed (regardless of participation in Wave II). Wave III (2001 to 2002) included 15,197 eligible original Wave I respondents, measured between August 2001 and April 2002. Our final analysis sample included 14,706 respondents (49% female) with relevant data across Waves I, II, and III. In Wave I ages ranged from 12 to 20 years, in Wave II ages ranged from 13 to 20 years, and in Wave III from 18 to 28 years. Exclusions included pregnant females, Native Americans (due to small sample sizes), few participants with outlying ages, and those missing relevant data. The survey design and sampling frame have been described elsewhere (16,17).

Body Mass

Height and weight were self-reported in Waves I, II, and III and measured in Waves II and III during in-home surveys using standardized procedures. We used self-reported data from all 3 waves to maximize the sample size and to have comparably assessed data across the waves. Although there is a tendency for heavier individuals to under-report weight, this phenomenon has little impact on the classification of obesity using BMI. For example, obesity prevalence based on measured vs. self-report at Wave II is 10.3% and 9.2% respectively, and 86.0% of participants were concordantly classified. To deal with the discrepant obesity definitions for adolescents and adults, we used the International Obesity Task Force (IOTF) cut-off points (18) to classify adolescents. The IOTF reference is based on pooled international data (including four national U.S. surveys) and links childhood and adolescent BMI centiles to adult cut-off points of BMI of 25 and 30 kg/m^2. For the young adult population at Wave III (ages 19 to 26), we used the BMI cut-off point of 30 kg/m^2 (19,20).

Study Variables

Race/ethnicity was determined from adolescent and parent questionnaires and categorized as Hispanic, non-Hispanic white, non-Hispanic black, or Asian-American. Age was calculated from date of interview and data of birth. Maternal obesity status, parental education, and family income were reported by parents in the parental home interview.

Statistical Analysis

Our outcome variable was age at onset of obesity. However, we did not fully observe this response variable. Instead, we know only that the adolescent became obese before the Wave I survey, or he or she became obese between the waves of Add Health, or was not obese at the time of the Wave III survey. We also know the exact ages of the individual at the time of each of the survey. For example, for an individual who is 17.5 years of age at Wave I and obese, we know only that this person became obese sometime before age 17.5. Similarly, if a non-obese individual age 15.8 at Wave I is obese at Wave II at age 21.0, we know that this person's age at onset of obesity falls between ages 15.8 and 21.0. For a non-obese individual age 22.4 at Wave III, we know only that this person's age at onset of obesity will be >22.4 years. Such data are called interval censored data (21). For the purpose of our analysis, the upper and the lower bound of age at onset of obesity is tabulated for each individual in the study sample. Examples of such tabulations are shown in Table 1. With these data, our goal was to estimate, for example, a simple linear regression model (additional covariates can be easily added)

$$Y = \hat{a}_0 + \hat{a}_1(\text{mother's obesity status}) + \epsilon \qquad (1)$$

where Y is the age at onset of obesity. For simplicity, we assume that the errors are normally distributed. The regression coefficient β_1 is interpreted in the usual way as the difference in the age at onset of obesity when the mother is obese or not (represented by an indicator variable). The model is estimated using the interval regression procedures in Stata 9.0 (22), including survey procedures to correct for multiple stages of cluster sample design and unequal probability of selection to ensure that our results were nationally representative and that bias in estimates and standard errors was reduced. Descriptive analyses use design-based, adjusted Wald tests to compare subgroup means. In our data,

[1] Nonstandard abbreviations: Add Health, National Longitudinal Study of Adolescent Health; IOTF, International Obesity Task Force.

Table 1. Outcome measure for the multivariate interval regression model

Wave I	Wave II	Wave III	DV1*	DV2*
Non-obese	Non-obese	Obese	15.8	21.0
Obese	Obese	Obese	—	17.5
Non-obese	Non-obese	Non-obese	22.4	—

* DV1 and DV2 represent the grouped dependent variable, age in years at which obesity was first observed given interval, left-, or right-censoring if incidence occurred during study period, if respondent was obese at entry into study, and if respondent remained non-obese throughout study period, respectively.

~8% of participants were already obese when they first entered Add Health, and ~82% were not obese when last observed.

The outcome variable in the models is the age at which obesity is first observed and the main exposure is maternal obesity. We also tested, and found similar effects when paternal obesity or obesity in either parent was used as the main exposure. Since fewer cases were missing information about mothers, we present results related to maternal obesity only. Similarly, we tested models where the main outcome variable was based on measured weight and height from Waves II and III. Because the results were not substantively different, we present analyses based on the larger sample, which includes self-reported data from Wave I. Control variables included household income, parental education, sex, and race/ethnicity. We tested for interactions of maternal obesity with race/ethnicity and sex, and the non-significant ($p < 0.10$) maternal obesity-sex interaction term was excluded. Geographic region was tested but not included in final models, given that its inclusion did not result in at least a 10% change in the coefficient for age at obesity related to maternal obesity. To illustrate the effects of having an obese mother on age at which obesity is first observed in offspring, we used coefficients from the interval regression models to predict age at obesity onset by race/ethnicity and maternal obesity.

Results

Descriptive Results

At baseline, the mean age of the sample was 15.8 years and 7.1% of males and 9.7% of females were obese (Table 2).

In earlier work, we documented the incidence of obesity between Waves II and III to be 12.7%, which resulted in an approximate doubling of obesity prevalence from Wave II to III, with highest rates in black and Hispanic females (7).

Table 3 shows the percentage of respondents who became obese before, vs. after, the age of 18 years, regardless of the wave of measurement. A substantially higher proportion of young adults with an obese mother became obese before age 18 (Table 3). Further, obesity was more prevalent overall among those with an obese vs. a non-obese mother.

Table 2. Demographic and descriptive statistics* of participants in Add Health (Waves I, II, and II)

	Female (n = 7123)	Male (n = 7531)
Age (yrs) at Wave I [mean (SE)]	15.9 (0.12)	15.7 (0.12)
Age (yrs) at Wave II [mean (SE)]	16.5 (0.12)	16.3 (0.12)
Age (yrs) at Wave III [mean (SE)]	22.2 (0.12)	22.0 (0.12)
Obese† at Wave I [% (SE)]	7.1 (0.48)	9.7 (0.55)
Obese† at Wave II [% (SE)]	7.9 (0.59)	10.3 (0.59)
Obese† at Wave III [% (SE)]	17.8 (0.90)	17.6 (0.81)
Race/ethnicity (%)		
White	57.2	57.0
Black	20.0	21.3
Hispanic	16.7	16.6
Asian	6.1	5.1
Mother obese (%)		
White	19.4	18.8
Black	18.3	16.8
Hispanic	16.1	14.8
Asian	9.4	7.4

Add Health, National Longitudinal Study of Adolescent Health; SE, standard error; IOTF, International Obesity Task Force.
* Weighted for national representation.
† Obesity defined using the IOTF cut-off points at Waves I and II and the adult BMI cut-off point BMI ≥30 kg/m^2 at Wave III.

Table 3. Proportion of participants in Add Health (Waves I, II, and II) with obesity onset prior to the age of 18 years regardless of wave*

	Became obese† before age 18 (n = 1822) (%)	Became obese† after age 18 (n = 2026) (%)
Mother obese		
White	18.9	13.7
Black	26.7	17.3
Hispanic	17.3	17.0
Asian	35.2	8.8
Mother not obese		
White	5.8	7.1
Black	8.7	10.4
Hispanic	8.7	8.2
Asian	3.2	3.0

Add Health, National Longitudinal Study of Adolescent Health; IOTF, International Obesity Task Force.

* Weighted for national representation.

† Obesity defined using the IOTF cut-off points at Waves I and II and the adult BMI cut-off point BMI ≥30 kg/m^2 at Wave III.

Multivariate Analysis Results

Coefficients from the interval regression model are shown in Table 4. In the absence of any interaction with maternal obesity, the regression coefficients represent the difference in the age at onset of obesity between those with obese vs. non-obese mothers. When a race-maternal obesity interaction is included in the model, the net change in the age at onset due to mother's obesity status is the sum of the regression coefficients of the race/ethnicity, maternal obesity and interaction terms. Whites with an obese mother have an estimated age of obesity onset of 8.1 years earlier than whites with a non-obese mother. Compared with whites with a non-obese mother, blacks were estimated to become obese 10.8 years earlier, Hispanics 7.0 years earlier, and Asians 8.6 years earlier.

Implications of Earlier Obesity Onset and Maternal Obesity

Judging by young adult body weight and BMI, the severity of obesity is greatest among those with onset before age 18 (regardless of the wave of measurement) and maternal obesity (Table 5). At Wave III, mean weight was substantially higher among respondents who became obese before age 18 and who had an obese mother (females: 13.3 kg higher; males: 6.7 kg higher) than those with onset after age 18 and without maternal obesity.

Table 4. Predictors of age at which obesity* was first observed over 5 years of follow-up (Wave II to III)† among adolescents and young adults in Add Health

	β coefficient reported as age in years (95% CI)	p
Mother obese	−8.1 (−9.3; −6.9)	0.0001
Black	−2.3 (−3.4; −1.2)	0.0001
Hispanic	−1.0 (−2.6; 0.5)	0.2
Asian	4.6 (0.7; 8.5)	0.02
Black by maternal obesity	−0.4 (−2.4; 1.6)	0.7
Hispanic by maternal obesity	2.1 (−0.4; 4.7)	0.1
Asian by maternal obesity	−5.1 (−10.7; 0.5)	0.07
Parental less than high school education	−3.5 (−5.0; 2.0)	0.0001
Parental high school education/GED	−2.3 (−3.7; −0.8)	0.002
Parental some college education	−1.5 (−2.7; −0.3)	0.02
Parental low income ($0–$26,000/yr)	−2.6 (−3.8; −1.4)	0.0001
Parental middle income ($26,000–$50,000/yr)	−2.3 (−3.4; −1.3)	0.0001
Constant	36.3 (34.3; 38.2)	0.0001

Add Health, National Longitudinal Study of Adolescent Health; CI, confidence interval; GED, general equivalency diploma; IOTF, International Obesity Task Force.

* Obesity defined using the IOTF cut-off points at Waves I and II and the adult BMI cut-off point BMI ≥30 kg/m^2 at Wave III.

† Betas and 95% confidence intervals from a multivariate interval regression model predicting age at obesity onset, which accounts for the sampling strategy by using weights and represents the maternal obesity by race/ethnicity-gender interaction, adjusted for household income, parental education, and sex.

Discussion

In a representative, longitudinal sample of adolescents and young adults in the United States, we found that having an obese mother was associated with younger age of offspring's obesity onset across all race/ethnic groups. This relationship was particularly strong in non-Hispanic black females. Our analysis also suggests that the combination of having an obese mother and an earlier onset of obesity translates into higher BMI and weight at young adulthood.

The impact of our findings for the population is substantial. Our Add Health analysis sample represents ~18.0

Table 5. Body weight and BMI (kg/m^2) at Wave III of Add Health by maternal obesity and offspring category of age at which obesity was first observed regardless of wave*

	Became obese† before age 18 (n = 1822)	Became obese† after age 18 (n = 2026)	Non-obese (n = 10,806)
Females			
Mother obese			
Body weight (kg)	106.3 (2.1)‡	98.0 (1.7)	66.3 (0.7)‡
BMI (kg/m^2)	38.8 (0.7)	35.8 (0.6)	24.4 (0.2)‡
Mother not obese			
Body weight (kg)	97.9 (2.0)	93.0 (1.0)	63.3 (0.2)
BMI (kg/m^2)	37.0 (0.6)	34.7 (0.3)	23.5 (0.1)
Males			
Mother obese			
Body weight (kg)	114.3 (1.7)‡	108.4 (1.3)	80.8 (0.6)‡
BMI (kg/m^2)	36.1 (0.6)‡	34.3 (0.4)	25.0 (0.2)‡
Mother not obese			
Body weight (kg)	106.9 (1.6)	107.6 (1.0)	76.9 (0.4)
BMI (kg/m^2)	33.9 (0.5)	33.4 (0.2)	24.1 (0.1)

Add Health, National Longitudinal Study of Adolescent Health; IOTF, International Obesity Task Force. Values are mean (standard error).
* Weighted for national representation.
† Obesity defined using the IOTF cut-off points at Waves I and II and the adult BMI cut-off point BMI ≥30 kg/m^2 at Wave III.
‡ Comparison of offspring category of age at which obesity was first observed (before age 18, after age 18, or non-obese) by maternal obesity vs. maternal non-obesity ($p \leq 0.01$).

million 13- to 20-year-old students at public and private schools in the U.S. and indicates that >1.6 million adolescents became obese before the age of 18 years. A substantial proportion (20%) of offspring of an obese mother became obese before the age of 18 years, while only 6.5% of those with a non-obese mother had early obesity onset. The transition from adolescence to young adulthood is a period of high incidence and maintenance of obesity, with maternal obesity playing a major role in observed age of obesity onset.

Differences in the long-term consequences of early- and later-onset obesity are not fully understood. While some studies show increased risk of adult (vs. child) BMI on adult risk of chronic disease (23–25), there is evidence that the cumulative effects of long-term obesity may be more deleterious than later-onset obesity (13,15). In a southwestern Ohio cohort followed over the full life cycle from birth into later adulthood, Wisemandle et al. (26) found that starting at age 4 and through adulthood, individuals with onset of overweight (BMI ≥25) before the age of 25 years had higher weight and BMI than those with later-onset or no overweight. Early obesity in black adolescent females has been shown to be associated with higher fasting blood glucose levels and increased risk of impaired fasting glucose in young adulthood (27). Further, preadolescent central adiposity that continues into young adulthood was associated with young adult metabolic risk factors, such as elevated triglycerides, glucose, and blood pressure (28). In addition, weight gain after 2 years of age has been shown to be related to impaired glucose tolerance later in life (29).

Offspring of obese parents are at high risk for obesity themselves (30–32) due to the effects of both shared genes and shared environments. There is familial aggregation of physical activity and diet patterns, attributable to genetic and non-genetic factors (33–41). Furthermore, those with Class III or extreme obesity (BMI ≥40 kg/m^2) have been shown to have significantly heavier parents than those with Class I or II obesity (BMI <40 kg/m^2) (42).

Although our study is one of the first to look at the combined effect of race/ethnicity and maternal obesity in observed age at onset of obesity in a nationally representative, prospective cohort, our analytical strategy is not without caveats. Our analysis assessed the age at which obesity was first observed once participants entered the Add Health study but was limited to the age distribution of the Add Health sample. While left- and right-censoring allows prediction of the age distribution not surveyed in Add Health, we are limited in not having data across the full spectrum of

ages. The interval regression approach, like survival analysis approaches, holds the assumption that all respondents will become obese at some point up to infinity. Ultimately, we rely on the distribution of available data (within the studied age range) to predict probability of being obese outside of the studied age range. In addition, interval regression assumes normal error distribution. We think that this is a tenable assumption because the normal error in our case may be with low dispersion. We acknowledge that, if a large proportion of the full population were never to become obese, our parameter estimates might overestimate the average time to obesity among those who become obese. Further, while the absolute numbers might differ with other distributional assumptions, it is highly likely that we would still find the race/ethnicity differences and the relative effects of maternal obesity. The interval regression model is specifically designed to address interval censoring and, thus, is a well-suited methodological approach to take advantage of the strengths of the Add Health data.

The race/ethnic variation in this large, nationally representative, prospective cohort observed during an understudied age range with data from adolescent respondents and their parents, provides an outstanding opportunity for assessing these relationships. Another limitation is the use of self-report data on height and weight. However, we conducted separate comparative analyses using measured- vs. self-report data and found that our results changed very little with the use of measured height and weight. Similarly, we used only data on maternal obesity but conducted separate comparative analyses using paternal obesity, and both maternal and paternal obesity and again found that our results changed very little depending on the measure used. To maximize sample size and to use consistent measures, we opted to use self-report weight and height data and data on maternal obesity alone.

The public health implications of the trend toward earlier onset obesity are substantial. The trend foreshadows higher rates of diabetes and nutrition-related chronic degenerative diseases, emerging at younger ages (43,44). Given that risk seems to be higher in those with long-term vs. adult-onset obesity and that reversal of obesity seems to protect against metabolic risk, early identification and intervention to reduce obesity are likely to have major health implications.

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