Published in final edited form as:

Matern Child Health J. 2015 October ; 19(10): 2261–2271. doi:10.1007/s10995-015-1745-7.

Gestational Weight Gain and Health Outcomes 18 Years Later in Urban Black Women

Margaret L. Holland, PhD,

School of Nursing, Yale University, West Haven, CT

Susan W. Groth, PhD, and

School of Nursing, University of Rochester, Rochester, NY

Harriet J. Kitzman, PhD

School of Nursing, University of Rochester, Rochester, NY

Abstract

Objectives—Excessive gestational weight gain (GWG) is associated with higher body mass index (BMI) later in life. Increased BMI is associated with health problems, but there is limited evidence linking GWG directly to later health in black women. We examined the association between GWG and health conditions 18 years after a first birth.

Methods—This study was a secondary data analysis of 467 urban black women, enrolled during pregnancy (1990 – 1991). GWG was the difference between self-reported pre-pregnancy weight and measured weight at delivery. Hypertension, diabetes, obesity, and self-reported health were assessed with self-report and measurements of blood pressure, height, and weight, approximately 18 years after first childbirth.

Results—Higher pre-pregnancy BMI was associated with increased probability of each health condition. Higher GWG was associated with hypertension for women with a pre-pregnancy BMI under 21.3 kg/m² ($P<.05$) and obesity for women with a pre-pregnancy BMI under 25.9 kg/m² ($P<.05$). Diabetes and poor health were not associated with GWG.

Conclusions—GWG may impact a mother's hypertension and obesity status 18 years after childbirth for underweight and normal weight women.

Excessive gestational weight gain (GWG) is a risk factor for greater body mass index (BMI) for the mother in later years (1, 2) and is defined as gaining above the Institute of Medicine (IOM) guidelines (3). The association between excessive GWG and later BMI is strongest in urban black women who were adolescents during their first pregnancy (4–6). Black women are at greater risk of postpartum weight retention than white women (7). In black women parity has been associated with increased BMI 10 years later, as opposed to white and Hispanic women for whom only a first birth was associated with increased BMI (8). Greater BMI is a concern, because chronic medical disorders are associated with overweight and obesity including hypertension, diabetes mellitus, and hyperlipidemia (9, 10). Obesity also

contributes to poor self-reported health status (11) and to an increased risk of mortality (12), primarily due to cardiovascular disease (13). Childbearing has been associated with development of metabolic syndrome (14). For adolescents, childbearing has contributed to persistent atherogenic lipid profiles up to 10 years after birth of a first child (15). However, childbearing was not associated with risk of diabetes up to 20 years later in women with normal glucose tolerance during pregnancy (16).

Less is known about the effect of excessive GWG on long-term health outcomes than the effects of childbearing in general. Excessive GWG has been associated with higher mean blood pressure (BP) 16 years after pregnancy (17). However, in a longitudinal study that adjusted for pre-pregnancy BMI, BP was lower in women up to 20 years following a first birth compared to women with no births (18). In another study, disease 15 years later has been predicted by pre-pregnancy BMI, failure to lose weight following pregnancy, and continued weight gain, but not GWG (19).

Although chronic medical disorders are associated with obesity, it is less clear if they are associated directly with excessive GWG. Combining the known association between GWG and later BMI with the associations between BMI and health outcomes, we hypothesized that greater GWG is associated with greater risks of hypertension, diabetes, obesity and poor self-reported health.

MATERIALS & METHODS

Data on 467 women who participated in the New Mother's Study in Memphis, TN in 1990 – 91 were examined (20). This randomized controlled trial tested the efficacy of a home visiting model, which is now known as the Nurse-Family Partnership, in a low-income urban population. Pregnant women with no previous live births were eligible to enroll if they were no more than 28 weeks pregnant and had 2 of the 3 following risk factors: unmarried, less than high school education, and unemployed. Women were excluded if they had any chronic medical problems that could contribute to fetal growth retardation or preterm birth, including hypertension prior to enrollment, because a study objective was to improve birth outcomes for babies of healthy women. Two arms of the original study have been followed over the past 20 years for periodic interviews. The intervention improved a variety of outcomes for mothers and their children, ranging from mother's spacing of subsequent pregnancies (20) to child academic achievement (21). The study was approved by the University of Rochester institutional review board.

For these analyses, the sample was limited to black women (92%) because differences between races with respect to GWG and health outcomes were expected (22, 23) and the white sample was too small to analyze separately. At randomization, the intervention group included 205 black pregnant women and the control group included 474 black pregnant women. Women with premature deliveries (gestational age < 37 weeks) were excluded from these analyses because GWG would be less than if they delivered at-term. There were a total of 560 full-term births. Of these, 9 mothers (1.6%) died by the time of the 18-year follow-up interview. Data were available for 467 subjects (85%) for these analyses.

Dependent variables

Three health conditions (hypertension, diabetes, and obesity) and self-reported health 18 years after the birth of the first child were used as outcomes. Women were asked if they had “ever been told that you have any of the following” and presented with a list of 23 conditions, including diabetes and high BP. Diabetes was determined by a “yes” on this question for diabetes. Hypertension was defined as “yes” on this question for high BP and/or a measured BP ≥ 140 mmHg for systolic or 90 mmHg for diastolic. Self-reported hypertension (24, 25) and diabetes (26, 27) have been found to be reliable and well correlated with clinical measures. Obesity was defined as BMI ≥ 30 kg/m², calculated from measured weight and height.

Women were asked to rate their health status on a 5-point scale (Excellent, Very good, Good, Fair, Poor) using an established item (28). Responses were collapsed into a dichotomous measure of Fair/Poor vs. Excellent/Very Good/Good, following a common (29, 30) and efficient (31) practice. Self-reported health is associated with mortality (29, 30), has been widely used for research (32–34), and is a global measure of health (35).

Independent variables

GWG was calculated from the mother’s self-reported pre-pregnancy weight and measured weight at time of delivery. Self-reported weight is a valid estimate of pre-pregnant weight (36, 37), although it is less accurate for overweight women who tend to underreport their weight. GWG was used as a continuous variable in our primary analyses in order to preserve information and identify any health outcome differences with incremental increases in BMI. We explored categories of GWG as defined by IOM guidelines: (a) underweight; 28–40 pounds; (b) normal weight; 25–35 pounds; (c) overweight; 15–25 pounds and (d) obese; 11–20 pounds (3). Under and over gain were defined as gaining less or more, respectively, than the recommended range. The current 2009 guidelines were used to ensure relevance of the findings to current practice, similarly to recent publications.

Covariates

Pre-pregnancy BMI was included to determine the independent impact of GWG. Maternal age at estimated conception was used to control for weight gain over the lifespan and developmental differences due to the high proportion of adolescent mothers, and provides the best proxy for inception of pregnancy weight gain. Time from enrollment to interview captures the variation in interview timing and is included because this impacts the mother’s age at the interview, as well as the time between pregnancy and interview. Parity (number of live births) was included, because additional pregnancies potentially influence weight. Socioeconomic status (SES) was measured by the percent of months the mother was supported by Aid to Families with Dependent Children from program enrollment until the 18-year interview, using data across all interviews. Smoking history was collapsed into three variables using data across all interviews: no smoking reported; ever reported heavy smoking (≥ 20 cigarettes per day (38)), or ever reported smoking but never reported heavy smoking.

Analysis

The associations between GWG and health outcomes were estimated using multivariable logistic regression. Covariates described above were included in all models. An interaction between GWG and pre-pregnancy BMI was tested, because recommended GWG is dependent on pre-pregnancy BMI (3). Average marginal effects of GWG on the outcomes were calculated across the BMI range (i.e., the predicted increase in probability of having each outcome for each additional pound of GWG, calculated at various levels of pre-pregnancy BMI and average values for all other covariates).

Data on GWG were missing for 7.1% of women in this sample, due to missing pre-pregnancy BMI, GWG, or both. Data were missing for 5% or fewer women on other covariates. Therefore, multiple imputation by chained equations was conducted. Ten imputations were chosen based on the percentage of missing data (39). No outcomes were imputed.

Sensitivity Analyses

Analyses were repeated excluding two mothers who had very high BMI at intake ($>48 \text{ kg/m}^2$), which is 5 standard deviations greater than the mean, and repeated for six mothers with intake BMIs in Obesity Class III ($>40 \text{ kg/m}^2$). Women in this highest obesity category are at risk for multiple health problems and their pre-pregnancy BMI is associated with CVD and all-cause mortality regardless of GWG (40). Analyses were also repeated including the intervention as a covariate, because nurse home visitors encouraged healthy eating, which potentially could affect GWG and other health behaviors. There were no substantial changes to the results from these analyses.

Mediation Analyses

To determine if concurrent and recent BMI were associated with health outcomes, we analyzed models including BMI at years 12 and 18. Because these two BMIs were strongly correlated ($P<.001$), we did not include both in the same model. We tested each of these BMIs as mediators between GWG and later health outcomes, using the Sobel Test to test the significance of the indirect path (GWG to later BMI to health outcomes) (41).

RESULTS

Only 33% of the women gained appropriately per the 2009 IOM guidelines: 39% gained above the current guidelines and 27% gained less (Table 1). Of mothers with normal pre-pregnancy BMI, 35% over gained and 31% under gained. Of underweight mothers, 20% over gained and 37% under gained. Of overweight mothers, 57% over gained and 10% under gained. Of obese mothers, 64% over gained and 20% under gained. The majority (59%) had hypertension at the 18-year interview (self-report of diagnosis only: 9%, measured high BP only: 25%, or both: 25%). At 18 years, 60% were obese. Obesity at 18 years was correlated with hypertension ($P<.001$) and fair/poor health ($P<.006$), but not diabetes ($P<.35$). Poor/fair health was also correlated with hypertension ($P<.001$) and diabetes ($P<.002$).

Pre-pregnancy BMI was consistently associated with later health outcomes. Figure 1 illustrates the increasing prevalence of each health condition with increasing pre-pregnancy BMI. In bivariate analyses, 51% of women normal weight prior to pregnancy had hypertension later, while 89% of obese women had hypertension ($P<.001$). Similar trends were seen with diabetes (5.6% vs. 23%, $P<.001$), obesity (57% vs. 93%, $P<.001$), and poor health (25% vs. 40%, $P<.03$). These associations persisted in multivariate analyses (Table 2).

GWG was directly associated with obesity (OR: 1.04; 95% confidence interval: 1.02, 1.06), but no other health outcomes. However, when an interaction between GWG and pre-pregnancy BMI was included, the average marginal effect of GWG on hypertension and obesity was significant for certain pre-pregnancy BMI ranges (Figure 2). The effect of GWG on hypertension was greater for women with a pre-pregnancy BMI under 21.3 kg/m² and the effect of GWG on obesity was greater for women with a pre-pregnancy BMI less than 25.9 kg/m² ($P<.05$). The effect of GWG on diabetes or poor health did not vary by pre-pregnancy BMI (Figure 2, Panels B and C).

When GWG was included as a categorical variable (i.e. under gain, appropriate gain, over-gain), pre-pregnancy BMI remained a significant predictor of each health outcome and GWG remained a predictor of obesity (Supplemental Table A). The interaction between GWG and pre-pregnancy BMI did not improve model fit in any models with GWG as a categorical variable, and is therefore not included.

BMI at 12 and 18 years post-pregnancy was associated with hypertension: BMI explained 24% (not significant for mediation) and 34% ($P<.03$ for mediation) of the total effect of GWG on hypertension respectively (Table 3). BMI at 12 years was also associated with obesity at 18 years and explained 49% ($P<.004$ for mediation) of the total effect of GWG on obesity.

DISCUSSION

The aim of this study was to combine the association between GWG and later BMI with associations between BMI and health outcomes. Our results support an association between pre-pregnancy BMI and hypertension, diabetes, obesity, and poor self-reported health 18 years after the birth of a first child in urban black mothers. GWG was directly associated with obesity. Body mass index at 18 years mediated the association between GWG and hypertension, and BMI at 12 years mediated the association between GWG and obesity. An interaction between GWG and pre-pregnancy BMI revealed that GWG had different effects on hypertension and obesity for different pre-pregnant BMI ranges.

An association of pre-pregnancy BMI with increased risk of long-term health outcomes is consistent with other studies (17, 19). Pre-pregnancy BMI has been associated with long-term weight retention post-pregnancy (4, 5, 8, 42). Multiple explanations have been proposed to explain this effect. First, women with high BMIs are more likely to gain excessively (8, 17, 43). Second, these women could habitually engage in lifestyle activities detrimental to maintaining a normal body weight that continue across the lifespan (17). Third, a genetic predisposition to gaining excessively could be a factor (44, 45). The genetic

contribution to obesity is complex and little is known about mechanisms that lead to expression of obesity (46). Finally, there may be epigenetic effects stemming from the in utero environment of their mothers that increase their obesity risk (47).

Excessive GWG contributing to obesity risk is also consistent with other studies (1–3, 8, 42) and has been previously reported for adolescents in our sample (5). The interaction between GWG and pre-pregnancy BMI indicates a there is a greater increase in hypertension for those with lower pre-pregnancy BMIs compared to those who with higher BMIs. Fraser et al. reported gaining above recommendations was associated with higher BP 16 years later (17). Conversely, Rooney et al. found a linear effect of increased hypertension with higher BMI 15 years post childbirth (19). Our sample differed from both of these studies in that it only included primiparous poor black women and 60% were obese by year 18, compared to 30% in Rooney et al.'s white, primarily middle-class sample.

More women over gained as the BMI category changed from underweight to obese in our sample. This pattern differs somewhat from three large studies that reported an increase in number who over gained from underweight to overweight categories, but a decrease between the overweight and obese categories (48, 49). Of the women in this sample, fewer over-gained (39%) than reported in recent studies where nearly 50% (43) or more (49) women over-gained. This could be reflective of guideline differences in effect when these women were pregnant. Our sample differed from these studies in that it included only African-American women.

The finding that normal weight women with higher GWG experience a greater negative health impact than women who start out at higher weights has major clinical implications. One potential explanation is that GWG recommendations for normal weight women are higher than for overweight or obese women. If over gain occurs in normal weight women, a higher amount of GWG occurs than if overweight/obese women over gain, which could contribute to long-term weight retention and differences in health effects compared to the heavier women. Relative weight gain could also explain this finding: An equivalent amount of GWG in normal weight compared to obese woman is proportionally more for the normal weight woman. There could be a threshold effect of weight impact on health outcomes, where additional weight gain above some BMI does not substantially increase health risks. This is supported by our finding that 89% of obese women developed hypertension later. It is conceivable that the risk of hypertension from high BMI early in life is so great that GWG does not make a substantial additional impact on this risk. Targeting the GWG of normal weight women to prevent later hypertension requires further evaluation. Our findings differ from Gunderson (2008) where there was a slight decrease in repeated measures of BP for women following pregnancy compared to nulliparous women, although findings were less pronounced for black women (18). Our sample did not include women who were nulliparous for comparison, nor did we have repeated measures of BP.

The effect of GWG on diabetes was non-significant in this sample. Childbearing was not associated with increased risk of diabetes up to 20 years later in women with normal glucose tolerance during pregnancy in another study (16). We did not assess a measure of glucose tolerance during pregnancy. The lack of significance could be due to the low prevalence of

diabetes in these women, which limits the statistical power to detect effects. Although women in this sample had higher prevalence of diabetes than the general population of a similar age and/or race, the numbers are limited. Lifetime risk of diabetes increases with increasing BMI and the younger the age at which someone becomes obese (50). The majority of women in this sample had a mean BMI in the overweight category 6-years post-delivery of their first child when they were in their 20's (4), which would increase their diabetes risk. However, at the time of this study they were relatively young with an average age of 36 years. Diabetes was likely for those with high pre-pregnancy BMI (86% with a BMI between 35–40 kg/m² developed diabetes), suggesting a threshold effect similar to that proposed for hypertension.

The disease burden in this cohort of women is striking. At an average age of 36 years, the prevalence of hypertension (59%) was closer to the U.S. national prevalence for black women ages 45–66 (59.2%) than their own age category (16.8% for ages 20–44) (51); even the more conservative estimate of those reporting diagnosis (34%) is high for their average age. The prevalence of diabetes (9.7%) in this sample is almost twice as high as the national average for black women ages 20–44 (5.2%) (51). Fair or poor health was reported by twice as many women in this sample (24.8%) as the national average for black women ages 25–44 (12.5%), which is closer to the national rate for black women ages 45–64 (26.5%) (51). The prevalence of obesity was similar in this sample (60%) compared to the national average for black women over 20 years old (57.2%) (51).

These women were all primiparous at study enrollment, although most had subsequent pregnancies. Parity had no effect on BMI 12 and 18 years post-delivery of a first child. This is consistent with studies that indicate a first pregnancy is of greater import than subsequent pregnancies on weight retention (52, 53). Primiparous women gain more weight during pregnancy than multiparous women, which would contribute to risk of postpartum weight retention and subsequent obesity (54). Yet, an association of parity with increased BMI 10 years later in black women (not white or Hispanic women) has been reported (8). An association between parity and weight in black women that is non-linear and most significant at a parity of one has also been reported (55).

The strength of this study is homogeneity and longevity of this sample. The population of urban black women is not as commonly studied as the general population and has disproportionate disease burden (23). In addition, it was not necessary to control for common demographic variables and it is less likely that confounding factors impacted our findings due to the similarities between the women. However, the findings should not be generalized to other populations without further study. In populations with lower disease burden, women may need to be followed to older ages to replicate these findings. The retention rate in this study is high compared to many longitudinal studies.

Nevertheless, there are several limitations. Self-report of hypertension and diabetes has been found to be reliable and correlated to clinical measures. However, there is the risk of under reporting due to lack of diagnosis or the mother's lack of knowledge and recall regarding her own diagnoses. There is also a risk of over reporting if a woman has noted physical symptoms that she surmises are reflective of a particular disease. In addition, hypertension

and diabetes were only reported as present or not, so we were unable to determine GWG impact on disease severity. Although a single measurement of BP is not diagnostic of hypertension, it screens for possible hypertension and 50% of those with elevated BP reported diagnosed hypertension. Pre-pregnancy weight was self-reported, which introduces error to the estimation of GWG with the result that it may be overestimated due to underreporting of actual weight. The GWG data is from the early 1990's and likely reflects a clinical approach to GWG focused on increasing GWG to decrease low birth weight infants. However, this limitation is unavoidable, because long-term effects can only be assessed years after prenatal care. Data were not available on weight loss following pregnancy; those who return to their pre-pregnancy weight may be less likely to develop poor health outcomes than those who retain weight gained during pregnancy. Data on diet and physical activity were not available, but would be useful additions to future studies.

This study provides long-term evidence regarding the health of the mother and suggests that the effects of GWG may be problematic. Other recent literature provides evidence of worse long-term outcomes for children when mothers either under gained or over gained during pregnancy (56). Optimal GWG is a complex issue due to its association with short- and long-term outcomes for both the mother and her child. Pregnancy weight gain may impact maternal health status in low-income minority women for years following a pregnancy, especially for normal weight women in terms of hypertension and obesity. Limiting GWG during pregnancy may have further reaching effects on health beyond weight status.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References

1. Mamun AA, Kinarivala M, O'Callaghan MJ, Williams GM, Najman JM, Callaway LK. Associations of Excess Weight Gain During Pregnancy with Long-Term Maternal Overweight and Obesity: Evidence from 21 Y Postpartum Follow-Up. *Am J Clin Nutr*. 2010; 91(5):1336–41. [PubMed: 20237138]
2. McClure CK, Catov JM, Ness R, Bodnar LM. Associations between Gestational Weight Gain and Bmi, Abdominal Adiposity, and Traditional Measures of Cardiometabolic Risk in Mothers 8 Y Postpartum. *Am J Clin Nutr*. 2013; 98(5):1218–25. [PubMed: 24047920]
3. IOM (Institute of Medicine) and NRC (National Research Council). *Weight Gain During Pregnancy: Reexamining the Guidelines*. Washington, D.C: The National Academies Press; 2009.
4. Groth SW. The Long-Term Impact of Adolescent Gestational Weight Gain. *Res Nurs Health*. 2008; 31(2):108–18. [PubMed: 18181102]
5. Groth SW, Holland ML, Kitzman H, Meng Y. Gestational Weight Gain of Pregnant African-American Adolescents Affects Body Mass Index 18 Years Later. *J Obstet Gynecol Neonatal Nurs*. 2013; 42(5):541–50.
6. Gunderson EP, Striegel-Moore R, Schreiber G, Hudes M, Biro F, Daniels S, et al. Longitudinal Study of Growth and Adiposity in Parous Compared with Nulligravid Adolescents. *Arch Pediatr Adol Med*. 2009; 163(4):349–56.
7. Davis EM, Zyzanski SJ, Olson CM, Stange KC, Horwitz RI. Racial, Ethnic, and Socioeconomic Differences in the Incidence of Obesity Related to Childbirth. *Am J Public Health*. 2009; 99(2): 294–9. [PubMed: 19059856]
8. Abrams B, Heggeseth B, Rehkopf D, Davis E. Parity and Body Mass Index in Us Women: A Prospective 25-Year Study. *Obesity*. 2013; 21(8):1514–8. [PubMed: 23630108]

9. Katzmarzyk PT, Craig CL, Bouchard C. Underweight, Overweight and Obesity: Relationships with Mortality in the 13-Year Follow-up of the Canada Fitness Survey. *J Clin Epidemiol.* 2001; 54(9): 916–20. [PubMed: 11520651]
10. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The Disease Burden Associated with Overweight and Obesity. *JAMA.* 1999; 282(16):1523–9. [PubMed: 10546691]
11. Macmillan R, Duke N, Oakes JM, Liao WJ. Trends in the Association of Obesity and Self-Reported Overall Health in 30 Years of the Integrated Health Interview Series. *Obesity.* 2011; 19(5):1103–5. [PubMed: 21127477]
12. Thomsen M, Nordestgaard BG. Myocardial Infarction and Ischemic Heart Disease in Overweight and Obesity with and without Metabolic Syndrome. *JAMA Internal Medicine.* 2014; 174(1):15–22. [PubMed: 24217719]
13. Thompson D, Edelsberg J, Colditz GA, Bird AP, Oster G. Lifetime Health and Economic Consequences of Obesity. *Arch Intern Med.* 1999; 159(18):2177–83. [PubMed: 10527295]
14. Gunderson EP, Jacobs DR, Chiang V, Lewis CE, Tsai A, Quesenberry CP, et al. Childbearing Is Associated with Higher Incidence of the Metabolic Syndrome among Women of Reproductive Age Controlling for Measurements before Pregnancy: The CARDIA Study. *Am J Obstet Gynecol.* 2009; 201(2):e177–9.
15. Gunderson EP, Schreiber G, Striegel-Moore R, Hudes M, Daniels S, Biro FM, et al. Pregnancy During Adolescence Has Lasting Adverse Effects on Blood Lipids: A 10-Year Longitudinal Study of Black and White Females. *J Clin Lipidol.* 2012; 6(2):139–49. [PubMed: 22385547]
16. Gunderson EP, Lewis CE, Tsai AL, Chiang V, Carnethon M, Quesenberry CP, et al. A 20-Year Prospective Study of Childbearing and Incidence of Diabetes in Young Women, Controlling for Glycemia before Conception - the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Diabetes.* 2007; 56(12):2990–6. [PubMed: 17898128]
17. Fraser A, Tilling K, Macdonald-Wallis C, Hughes R, Sattar N, Nelson SM, et al. Associations of Gestational Weight Gain with Maternal Body Mass Index, Waist Circumference, and Blood Pressure Measured 16 Y after Pregnancy: The Avon Longitudinal Study of Parents and Children (ALSPAC). *Am J Clin Nutr.* 2011; 93(6):1285–92. [PubMed: 21471282]
18. Gunderson EP, Chiang V, Lewis CE, Catov J, Quesenberry CP, Sidney S, et al. Long-Term Blood Pressure Changes Measured from before to after Pregnancy Relative to Nonparous Women. *Obstet Gynecol.* 2008; 112(6):1294–302. [PubMed: 19037039]
19. Rooney BL, Schauburger CW, Mathiason MA. Impact of Perinatal Weight Change on Long-Term Obesity and Obesity-Related Illnesses. *Obstet Gynecol.* 2005; 106(6):1349–56. [PubMed: 16319262]
20. Kitzman H, Olds DL, Henderson CR, Hanks C, Cole R, Tatelbaum R, et al. Of Prenatal and Infancy Home Visitation by Nurses on Pregnancy Outcomes, Childhood Injuries, and Repeated Childbearing Trial - a Randomized Controlled Trial. *JAMA.* 1997; 278(8):644–52. [PubMed: 9272896]
21. Kitzman H, Olds DL, Cole RE, Hanks CA, Anson EA, Arcoleo KJ, et al. Enduring Effects of Prenatal and Infancy Home Visiting by Nurses on Children: Follow-up of a Randomized Trial among Children at Age 12 Years. *Arch Pediat Adol Med.* 2010; 164(5):412–8.
22. Freedman DS, Khan AK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. Racial Differences in Tracking of Childhood Bmi to Adulthood. *Obes Res.* 2005; 13(5):928–35. [PubMed: 15919847]
23. Goran MI. Ethnic-Specific Pathways to Obesity-Related Disease: The Hispanic Vs. African-American Paradox. *Obesity.* 2008; 16(12):2561–5. [PubMed: 19279653]
24. Ahluwalia IB, Tessaro I, Rye S, Parker L. Self-Reported and Clinical Measurement of Three Chronic Disease Risks among Low-Income Women in West Virginia. *J Womens Health.* 2009; 18(11):1857–62.
25. Thawornchaisit P, de Looze F, Reid C, Seubsman S, Sleight A. The Thai Cohort Study Team. Validity of Self-Reported Hypertension: Findings from the Thai Cohort Study Compared to Physician Telephone Interview. *Global Journal of Health Science.* 2014; 6(2):1–11.
26. Jackson JM, DeFor TA, Crain AL, Kerby TJ, Strayer LS, Lewis CE, et al. Validity of Diabetes Self-Reports in the Women's Health Initiative. *Menopause.* 2014; 21(8):861–8. [PubMed: 24496083]

27. Schneider ALC, Pankow JS, Heiss G, Selvin E. Validity and Reliability of Self-Reported Diabetes in the Atherosclerosis Risk in Communities Study. *Am J Epidemiol.* 2012; 176(8):738–43. [PubMed: 23013620]
28. Ware JE, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey - Construction of Scales and Preliminary Tests of Reliability and Validity. *Med Care.* 1996; 34(3):220–33. [PubMed: 8628042]
29. Dalen JD, Huijts T, Krokstad S, Eikemo TA. Are There Educational Differences in the Association between Self-Rated Health and Mortality in Norway? The HUNT Study. *Scand J Public Health.* 2012; 40(7):641–7.
30. McGee DL, Liao YL, Cao GC, Cooper RS. Self-Reported Health Status and Mortality in a Multiethnic US Cohort. *Am J Epidemiol.* 1999; 149(1):41–6. [PubMed: 9883792]
31. Manor O, Matthews S, Power C. Dichotomous or Categorical Response? Analysing Self-Rated Health and Lifetime Social Class. *Int J Epidemiol.* 2000; 29(1):149–57. [PubMed: 10750617]
32. Ahnquist J, Wamala SP, Lindstrom M. Social Determinants of Health - a Question of Social or Economic Capital? Interaction Effects of Socioeconomic Factors on Health Outcomes. *Soc Sci Med.* 2012; 74(6):930–9. [PubMed: 22305807]
33. Doubeni CA, Schootman M, Major JM, Stone RAT, Laiyemo AO, Park Y, et al. Health Status, Neighborhood Socioeconomic Context, and Premature Mortality in the United States: The National Institutes of Health-AARP Diet and Health Study. *Am J Public Health.* 2012; 102(4):680–8. [PubMed: 21852636]
34. Hasson D, Schwarz UV, Lindfors P. Self-Rated Health and Allostatic Load in Women Working in Two Occupational Sectors. *J Health Psychol.* 2009; 14(4):568–77. [PubMed: 19383657]
35. Mavaddat N, Kinmonth AL, Sanderson S, Surtees P, Bingham S, Khaw KT. What Determines Self-Rated Health (SRH)? A Cross-Sectional Study of Sf-36 Health Domains in the Epic-Norfolk Cohort. *J Epidemiol Commun H.* 2011; 65(9):800–6.
36. Perry GS, Byers TE, Mokdad AH, Serdula MK, Williamson DF. The Validity of Self-Reports of Past Body Weights by U.S. Adults. *Epidemiology.* 1995; 6(1):61–6. [PubMed: 7888448]
37. Stevens-Simon C, Roghmann KJ, Mcanarney ER. Relationship of Self-Reported Prepregnant Weight and Weight-Gain During Pregnancy to Maternal Body Habitus and Age. *J Am Diet Assoc.* 1992; 92(1):85–7. [PubMed: 1728630]
38. Pierce JP, Messer K, White MM, Cowling DW, Thomas DP. Prevalence of Heavy Smoking in California and the United States, 1965–2007. *JAMA.* 2011; 305(11):1096–112.
39. White IR, Royston P, Wood AM. Multiple Imputation Using Chained Equations: Issues and Guidance for Practice. *Statistics in Medicine.* 2010; 30(4):377–99. [PubMed: 21225900]
40. Mongraw-Chaffin ML, Anderson CM, Clark JM, Bennett WL. Prepregnancy Body Mass Index and Cardiovascular Disease Mortality: The Child Health and Development Studies. *Obesity.* 2014; 22(4):1149–56. [PubMed: 24124023]
41. Ender, PE. Program to Compute Sobel-Goodman Mediation Tests. UCLA Academic Technology Services; 2012. Sgmediation. ats.ucla.edu/stat/ado/analysis
42. Gunderson EP. Childbearing and Obesity in Women: Weight before, During, and after Pregnancy. *Obstet Gyn Clin N Am.* 2009; 36(2):317–32.
43. Deputy NP, Sharma AJ, Kim SY, Hinkle SN. Prevalence and Characteristics Associated with Gestational Weight Gain Adequacy. *Obstetrics & Gynecology.* 2015; doi: 10.1097/AOG.0000000000000739
44. Groth SW, Morrison-Beedy D. Gnb3 and Fto Polymorphisms and Pregnancy Weight Gain in Black Women. *Biological Research For Nursing.* 2014; doi: 10.1177/1099800414561118
45. Stuebe AM, Lyon H, Herring AH, Ghosh J, Wise A, North KE, et al. Obesity and Diabetes Genetic Variants Associated with Gestational Weight Gain. *Am J Obstet Gynecol.* 2010; 203(3)
46. Albuquerque D, Stice E, Rodríguez-López R, Manco L, Nóbrega C. Current Review of Genetics of Human Obesity: From Molecular Mechanisms to an Evolutionary Perspective. *Mol Genet Genomics.* 2015; doi: 10.1007/s00438-015-1015-9
47. Campión J, Milagro FI, Martínez JA. Individuality and Epigenetics in Obesity. *Obesity Reviews.* 2009; 10(4):383–92. [PubMed: 19413700]

48. Johnson J, Clifton RG, Roberts JM, Myatt L, Hauth JC, Spong CY, et al. Pregnancy Outcomes with Weight Gain above or Below the 2009 Institute of Medicine Guidelines. *Obstet Gynecol.* 2013; 121(5):969–75. [PubMed: 23635732]
49. Simas TAM, Liao X, Garrison A, Sullivan GMT, Howard AE, Hardy JR. Impact of Updated Institute of Medicine Guidelines on Prepregnancy Body Mass Index Categorization, Gestational Weight Gain Recommendations, and Needed Counseling. *J Womens Health.* 2011; 20(6):837–44.
50. Narayan KMV, Boyle JP, Thompson TJ, Gregg EW, Williamson DF. Effect of Bmi on Lifetime Risk for Diabetes in the Us. *Diabetes Care.* 2007; 30(6):1562–6. [PubMed: 17372155]
51. Centers for Disease Control and Prevention. [Accessed December 26, 2012] Health Data Interactive. www.cdc.gov/NCHS/hdi.htm
52. Gunderson EP, Abrams B. Epidemiology of Gestational Weight Gain and Body Weight Changes after Pregnancy. *Epidemiol Rev.* 2000; 22(2):261–74. [PubMed: 11218377]
53. Gunderson EP, Abrams B, Selvin S. The Relative Importance of Gestational Gain and Maternal Characteristics Associated with the Risk of Becoming Overweight after Pregnancy. *Int J Obesity.* 2000; 24(12):1660–8.
54. Nohr EA, Vaeth M, Baker JL, Sorensen TIA, Olsen J, Rasmussen KM. Pregnancy Outcomes Related to Gestational Weight Gain in Women Defined by Their Body Mass Index, Parity, Height, and Smoking Status. *Am J Clin Nutr.* 2009; 90(5):1288–94. [PubMed: 19759164]
55. Lee SK, Sobal J, Frongillo EA, Olson CM, Wolfe WS. Parity and Body Weight in the United States: Differences by Race and Size of Place of Residence. *Obes Res.* 2005; 13(7):1263–9. [PubMed: 16076997]
56. Gage SH, Lawlor DA, Tilling K, Fraser A. Associations of Maternal Weight Gain in Pregnancy with Offspring Cognition in Childhood and Adolescence: Findings from the Avon Longitudinal Study of Parents and Children. *Am J Epidemiol.* 2013; 177(5):402–10. [PubMed: 23388581]

Figure 1.

Prevalence of health conditions (hypertension, diabetes, poor health, and obesity) 18 years after the birth of a first child, by pre-pregnancy body mass index (BMI) and gestational weight gain.

HTN: hypertension, DM: diabetes, PH: poor health, OBS: obesity. For hypertension, “No” indicates that the participant did not report a diagnosis of hypertension and her measured blood pressure was not elevated. For diabetes, “No” indicates that she did not report a diagnosis of diabetes. For poor health, “No” indicates that she reported excellent, very good, or good health. For obesity, “No” indicates a BMI less than 30 kg/m².

Figure 2.

Average marginal effect of interaction between gestational weight gain and pre-pregnancy BMI on health conditions 18 years after the birth of a first child.

Table 1

Sample description

Variable	%	Mean (SD)	Min	Max
Gestational Weight Gain				
Weight gain (pounds)		31.5 (15.4)	-14	96
Underweight (<18.5 kg/m ²)		31.6 (9.0)	14	46
Normal (18.5 to 24.9)		31.6 (14.2)	-2	90
Overweight (25 – 29.9)		32.5 (18.2)	-7	96
Obese (≥ 30)		27.5 (21.5)	-14	91
Over gain ^b	39			
Recommended gain ^b	33			
Under gain ^b	27			
Health Outcomes at 18 years				
Hypertension	59			
Diabetes	9.7			
Poor health	25			
Obese	60			
Covariates				
Smoking: ever heavy	13.5			
Smoking ever, but not heavy	26.2			
Pre-pregnancy BMI category				
Underweight (<18.5 kg/m ²)	11.5			
Normal (18.5 to 24.9)	61.5			
Overweight (25 – 29.9)	17.3			
Obese (≥ 30)	9.7			
Pre-pregnancy body mass index (BMI; kg/m ²)		23.3 (5.0)	15.2	51.9
Age at conception of first child (years) ^e		17.4 (2.9)	12	33
Time from conception to 18 year interview (years)		18.6 (0.7)	18	21

Variable	Time assessed	%	Mean (SD)	Min	Max
Parity at 18 years	all interviews after 12 months ^f		3.3 (1.7)	1	11
% of months on AFDC at 18 years (SES)	all interviews		33 (28)	0	94

^aInterview at the time of study enrollment, or shortly after enrollment. Enrollment occurred during or before the 28th week of pregnancy.

^bWeight gain categories based on the 2009 IOM guidelines.

^cInterview scheduled near the study child's 18th birthday.

^dInterviews were conducted during pregnancy at enrollment and at 28 and 36 weeks; and through the child's life at 6, 12, and 24 months, 3.5, 6, 9, 12, and 18 years. Variables marked "all interviews" combined information gathered at each of these times.

^e70% of the mothers were adolescents (less than 19 years old) at the estimated date of conception of their first child.

^fInterview scheduled near the study child's 1st birthday

Table 2

Associations between gestational weight gain (continuous) and health conditions 18 years after the birth of a first child.^a

	Hypertension		Diabetes		Poor Health		Obesity	
	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 1 OR (95% CI)	Model 2 OR (95% CI)
Gestational Weight Gain (pounds)	1.01 (1.00, 1.02)	1.07 (1.00, 1.15)	1.01 (0.99, 1.03)	1.00 (0.92, 1.07)	1.01 (1.00, 1.02)	0.99 (0.93, 1.06)	1.04 (1.02, 1.06)*	1.18 (1.04, 1.33)**
Pre-pregnancy body mass index (BMI)	1.13 (1.07, 1.18)***	1.22 (1.09, 1.36)***	1.12 (1.06, 1.18)***	1.10 (1.01, 1.21)*	1.06 (1.01, 1.10)**	1.04 (0.96, 1.12)	1.44 (1.32, 1.58)***	1.69 (1.39, 2.04)***
GWG × Prepreg BMI		1.00 (1.00, 1.00)		1.00 (1.00, 1.00)		1.00 (1.00, 1.00)		0.99 (1.00, 1.00)
Age at conception of first child (years)	1.08 (1.00, 1.16)*	1.07 (1.00, 1.15)*	1.08 (0.97, 1.20)*	1.08 (0.97, 1.20)*	1.00 (0.92, 1.08)*	1.00 (0.92, 1.08)*	0.87 (0.79, 0.96)*	0.87 (0.79, 0.96)*
Conception to 18 year interview (years)	0.97 (0.73, 1.28)	0.96 (0.72, 1.27)	1.00 (0.63, 1.59)	1.01 (0.63, 1.61)	0.92 (0.67, 1.26)	0.92 (0.67, 1.27)	0.90 (0.63, 1.27)	0.89 (0.62, 1.26)
Parity at 18 years	1.00 (0.88, 1.13)	0.99 (2.37, 1.12)*	1.12 (0.92, 1.35)	1.12 (0.92, 1.36)	1.10 (0.96, 1.25)	1.10 (0.96, 1.26)	1.11 (0.94, 1.30)	1.11 (0.94, 1.30)
% of months on AFDC at 18 years (SES)	0.97 (0.45, 2.10)	0.99 (0.45, 2.14)	2.76 (0.80, 9.48)	2.71 (0.78, 9.36)	2.24 (0.97, 5.16)	2.21 (0.96, 5.10)	0.69 (0.26, 1.84)	0.67 (0.25, 1.79)
Smoking: ever heavy	0.81 (0.44, 1.50)*	0.83 (0.45, 1.52)	0.43 (0.14, 1.35)	0.42 (0.13, 1.34)	1.39 (0.74, 2.62)	1.38 (0.73, 2.61)	0.27 (0.12, 0.58)*	0.26 (0.12, 0.58)*
Smoking ever, but not heavy	0.85 (0.54, 1.34)	0.84 (0.53, 1.32)	1.01 (0.49, 2.08)	1.02 (0.50, 2.09)	1.09 (0.66, 1.80)	1.09 (0.66, 1.81)	0.23 (0.13, 0.41)	0.22 (0.12, 0.40)*

* $P < .05$,** $P < .01$,*** $P < .001$ ^aFor each health outcome, Model 1 includes the covariates and Model 2 additionally includes an interaction between GWG and pre-pregnancy body mass index (BMI). All models used logistic regression with multiple imputation by chained equations with 10 imputations and a sample size of 467.

Table 3

Associations between gestational weight gain (continuous) and health conditions 18 years after the birth of a first child with BMI at 12 and 18 years as mediators.^a

	<u>Hypertension</u>		<u>Diabetes</u>		<u>Poor Health</u>		<u>Obesity</u>
	Model 3 OR (95% CI)	Model 4 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)	Model 3 OR (95% CI)	Model 4 OR (95% CI)	Model 3 OR (95% CI)
Gestational Weight Gain (pounds)	1.06 (0.98, 1.15)	1.05 (0.97, 1.13)	0.98 (0.90, 1.06)	0.99 (0.92, 1.07)	1.00 (0.94, 1.07)	0.99 (0.93, 1.05)	1.18 (1.05, 1.33)
BMI at 12 years	1.04 (1.00, 1.08)*		1.04 (0.99, 1.10)		1.00 (0.97, 1.04)		1.45 (1.32, 1.59)*
BMI at 18 years		1.09 (1.05, 1.13)*		1.00 (0.95, 1.05)		1.03 (1.00, 1.07)	
Pre-pregnancy body mass index (BMI)	1.15 (1.02, 1.30)*	1.08 (0.97, 1.21)	1.03 (0.91, 1.17)	1.09 (0.97, 1.22)	1.04 (0.95, 1.15)	1.00 (0.92, 1.09)	1.26 (1.03, 1.54)*
GWG × Prepreg BMI	1.00 (0.99, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)	0.99 (0.99, 1.00)*
Age at conception of first child (years)	1.04 (1.01, 1.18)*	1.04 (1.02, 1.19)*	1.06 (1.00, 1.25)	1.06 (0.97, 1.21)	1.04 (0.93, 1.09)	1.04 (0.93, 1.09)	1.06 (0.82, 1.04)
Conception to 18 year interview (years)	1.17 (0.71, 1.31)	1.16 (0.75, 1.34)	1.30 (0.54, 1.50)	1.27 (0.65, 1.66)	1.19 (0.68, 1.34)	1.18 (0.67, 1.27)	1.26 (0.56, 1.41)
Parity at 18 years	0.99 (0.86, 1.13)	0.97 (0.85, 1.10)	1.11 (0.90, 1.37)	1.11 (0.91, 1.36)	1.10 (0.95, 1.27)	1.09 (0.95, 1.25)	0.94 (0.76, 1.17)
% of months on AFDC at 18 years (SES)	0.84 (0.37, 1.91)	0.94 (0.42, 2.08)	3.26 (0.88, 12.05)	2.58 (0.74, 9.00)	2.05 (0.86, 4.89)	2.20 (0.95, 5.10)	0.39 (0.10, 1.43)
Smoking: ever heavy	1.08 (0.56, 2.09)	1.16 (0.62, 2.20)	0.51 (0.16, 1.66)	0.42 (0.13, 1.36)	1.35 (0.69, 2.64)	1.58 (0.82, 3.05)	0.74 (0.28, 1.97)
Smoking ever, but not heavy	0.92 (0.56, 1.51)	1.03 (0.64, 1.67)	1.08 (0.50, 2.36)	0.97 (0.46, 2.02)	1.10 (0.65, 1.87)	1.19 (0.71, 1.98)	0.30 (0.14, 0.64)*
Sobel test for mediation	.06	.03*	.22	.81	.82	.12	.004**
Proportion of total effect that is mediated	24%	34%	-56%	2%	6.8%	-72%	49%

* $P < .05$,

** $P < .01$,

*** $P < .001$

^aFor each health outcome, Model 3 includes the BMI at 12 years and Model 4 includes BMI at 18 years. There is no Model 4 for obesity, because this outcome is based on BMI at 18 years. All models used logistic regression with multiple imputation by chained equations with 10 imputations and a sample size of 467.