Maternal Obesity, Gestational Weight Gain, and Offspring Adiposity: The Exploring Perinatal Outcomes among Children Study

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Objective To determine whether adequate vs excessive gestational weight gain (GWG) attenuated the association between maternal obesity and offspring outcomes.

Study design Data from 313 mother-child pairs participating in the Exploring Perinatal Outcomes among Children study were used to test this hypothesis. Maternal prepregnancy body mass index (BMI) and weight measures throughout pregnancy were abstracted from electronic medical records. GWG was categorized according to the 2009 Institute of Medicine criteria as adequate or excessive. Offspring outcomes were obtained at a research visit (average age 10.4 years) and included BMI, waist circumference (WC), subcutaneous adipose tissue (SAT) and visceral adipose tissue, high-density lipoprotein cholesterol, and triglyceride levels.

Results More overweight/obese mothers exceeded the Institute of Medicine GWG recommendations (68%) compared with normal-weight women (50%) ($P < .01$). Maternal prepregnancy BMI was associated with worse childhood outcomes, particularly among offspring of mothers with excessive GWG (increased BMI [20.34 vs 17.80 kg/m^2], WC [69.23 vs 62.83 cm], SAT [149.30 vs 90.47 cm^2], visceral adipose tissue [24.11 vs 17.55 cm^2], and homeostatic model assessment [52.52 vs 36.69], all $P < .001$). The effect of maternal prepregnancy BMI on several childhood outcomes was attenuated for offspring of mothers with adequate vs excessive GWG ($P < .05$ for the interaction between maternal BMI and GWG status on childhood BMI, WC, SAT, and high-density lipoprotein cholesterol).

Conclusion Our findings lend support for pregnancy interventions aiming at controlling GWG to prevent childhood obesity. *(J Pediatr 2014;■:■-■).*

The prevalence of obesity has been increasing dramatically in the US, including among women of reproductive age.[1] Maternal obesity is a major risk factor for gestational diabetes mellitus (GDM) and future type 2 diabetes.[2] Moreover, observational studies suggest an independent association of maternal obesity with excessive fetal growth[3,4] and childhood obesity.[5] Alarmingly, increasing obesity trends are now observed early in life, even among young infants,[6] pointing toward harmful changes in the environment in which contemporary children are born and raised.[7] These and other observations lead to the hypothesis that maternal obesity during pregnancy is associated with lifelong consequences in the offspring[8] and, possibly, over successive generations.[9] It has been suggested that a transgenerational "vicious cycle" results, explaining at least in part, the increases in obesity, GDM, and type 2 diabetes seen over the past several decades.[10] In addition, obese children tend to become obese adults and, once present, obesity and its consequences are expensive and difficult to treat. This makes pregnancy a crucial window of opportunity for obesity prevention in this and the next generation.

The role of gestational weight gain (GWG) on childhood adiposity outcomes is less clear and incompletely studied. Some, but not all,[11,12] epidemiologic studies have found that greater GWG is associated with greater body mass index (BMI) in childhood[13-17] and adolescence[5] and with increased fat mass and poorer metabolic and vascular traits at age 9 years.[18] Some studies have suggested that the association of greater maternal weight gain and offspring obesity persists into adulthood.[12] Maternal prepregnancy BMI and excessive GWG have been linked independently to increased adiposity in the offspring.[11,16,17,19-21] In a group of preschool children, the odds of being categorized as overweight by age 4-5 years was increased by 57% in children exposed to both a maternal prepregnancy BMI greater ≥25 kg/m^2 and excessive weight gain during pregnancy.[16] It remains unclear however, whether the effect of maternal prepregnancy BMI on childhood adiposity outcomes is mitigated by adequate weight gain during pregnancy.

BMI	Body mass index	IOM	Institute of Medicine
EPOCH	Exploring Perinatal Outcomes among Children	KPCO	Kaiser Permanente of Colorado Health Plan
GDM	Gestational diabetes mellitus	SAT	Subcutaneous adipose tissue
GWG	Gestational weight gain	TG	Triglyceride
HDL-C	High-density lipoprotein cholesterol	VAT	Visceral adipose tissue
HOMA-IR	Homeostatic model assessment	WC	Waist circumference

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The answer to this question is important because it would provide support to the notion that healthier weight-gain patterns during pregnancy may improve the short- and long-term effects on offspring who have been exposed to maternal obesity. To address this question, we used data from the Exploring Perinatal Outcomes among Children (EPOCH) study in Colorado.

Methods

The EPOCH study is an observational historical prospective cohort comprising children born between 1992 and 2002 at a single hospital in Colorado, whose biological mothers were members of the Kaiser Permanente of Colorado Health Plan (KPCO) and who were offspring of singleton pregnancies. All children exposed to maternal GDM were eligible, together with a random sample of children not exposed to GDM. Children were invited to attend an in person-research visit when they were on average age 10.5 years (range, 6-13 years), and approximately 68% agreed to participate.

Included in this analysis were 313 mother-child pairs (141 non-Hispanic white, 145 Hispanic, 27 non-Hispanic African American) who were part of the EPOCH study and had complete data on maternal prepregnancy BMI, GWG, and offspring adiposity outcomes. Children and their mothers completed a research visit between January 2006 and October 2009. Because the EPOCH study was specifically designed to explore the long-term effects of maternal GDM on offspring, the cohort is enriched in offspring of GDM mothers. Because we were exploring specific hypotheses regarding the role of excessive GWG as effect modifier, the small number of offspring of mothers who gained insufficient gestational weight during pregnancy was excluded. The study was approved by the local Institutional Review Board, and all participants provided written informed consent and youth provided written assent.

Maternal Measures

Maternal pregnancy measures (weight, GDM) and offspring birth weight were obtained from the KPCO perinatal database, a linkage of the maternal and perinatal medical record containing prenatal and delivery events for each woman. Maternal prepregnancy weight was measured before the last menstrual cycle preceding pregnancy. Maternal height was collected at the in-person research visit and used to calculate prepregnancy BMI (kg/m^2). BMI was categorized as normal weight (18.5-25 kg/m^2) and overweight/obese ($\geq$25 kg/m^2). Multiple weight measures during pregnancy (on average 4 per participant) were used to model GWG using a longitudinal mixed effects model that included fixed effects for time, time squared, prepregnant BMI, maternal age, gravidity, and a time by prepregnant BMI interaction. The model included subject-specific intercept and slope terms. GWG was estimated using the absolute predicted weight gain for a full-term pregnancy (model predicted weight at term minus model predicted weight at conception). Women were categorized as either exceeding or meeting the 2009 recommended Institute of Medicine (IOM) GWG guidelines (adequate total GWG for normal BMI prepregnancy 11.4-15.9 kg and overweight/obese BMI prepregnancy 5-11.4 kg).[22] Women who gained inadequate weight during pregnancy were excluded from this analysis, according to our a priori hypothesis.

Physician-diagnosed GDM was coded as present if diagnosed through the standard KPCO screening protocol and absent if screening was negative. Since the 1990s, KPCO has routinely screened for GDM in all nondiabetic pregnancies using a 2-step standard protocol and criteria based on the National Diabetes Data Group recommendations.[23]

Childhood Measures

All children were invited to an in-person research visit, which included anthropometric measures, questionnaires, a magnetic resonance imaging exam of the abdominal region and a fasting blood sample. Race/ethnicity was self-reported using 2000 US census-based questions and categorized as Hispanic (any race), non-Hispanic white, or non-Hispanic African American. Pubertal development was assessed by child self-report with a diagrammatic representation of Tanner staging adapted from Marshall and Tanner.[24] Youth were categorized as Tanner <2 (prepubertal) and $\geq$2 (pubertal). Total energy intake (kilocalories per day) was assessed using the Block Kid's Food Questionnaire.[25] Self-reported key activities, both sedentary and nonsedentary, performed during the previous 3 days were measured using a 3-day Physical Activity Recall questionnaire.[26] Each 30-minute block of activity was assigned a metabolic equivalent variable to accommodate the energy expenditure. Results were reported as the average number of 30-minute blocks of moderate-to-vigorous activity per day. Current height and weight were measured in light clothing and without shoes. Weight was measured to the nearest 0.1 kg using a portable electronic SECA scale (SECA, Chino, California). Height was measured to the nearest 0.1 cm using a portable SECA stadiometer. Height and weight were measured and recorded twice, and an average was taken. Scales and stadiometers were calibrated every 2 months using standard weights for scales and an aluminum measuring rod for the stadiometer. BMI was calculated as kg/m^2. Waist circumference (WC) was measured to the nearest 1 mm at the midpoint between the lower ribs and the pelvic bone with a metal or fiberglass nonspring-loaded tape measure.

Magnetic resonance imaging of the abdominal region was used to quantify visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) with a 3T HDx imager (General Electric, Waukashau, Wisconsin) by a trained technician. Each participant was placed supine and a series of T1-weighted coronal images were taken to locate the L4/L5 plane. One axial, 10-mm, T1-weighted images at the umbilicus or L4/L5 vertebra was analyzed to determine SAT and VAT content. The analysis technique used was a modification of the technique of Engelson,[27] where adipose tissue regions were differentiated by their signal intensity and location. Images were analyzed by a single reader.

Cholesterol, triglyceride (TG), and high-density lipoprotein cholesterol (HDL-C) were obtained while the patient

was fasting and measured using the Olympus (Center Valley, Pennsylvania) AU400 advanced chemistry analyzer system. Estimated insulin resistance was based on the homeostatic model assessment (HOMA-IR), calculated using fasting glucose and insulin levels collected at the study visit according to the formula: (fasting glucose [mmol/L] × fasting insulin [μU/mL]/22.5).

Statistical Analyses

All analyses were conducted using SAS v9.4 (SAS Institute, Cary, North Carolina). Prepregnant BMI was categorized as normal weight (18.5-25 kg/m^2) and overweight/obese (≥25 kg/m^2). Descriptive analyses compared prepregnant BMI groups using both t tests and χ^2 tests (Table I). Univariate regression was used to examine whether maternal prepregnant BMI was associated with childhood adiposity-related variables (including BMI, WC, SAT and VAT, HDL-C, TGs, and HOMA-IR). An ANOVA model was used to investigate the modifying effect of GWG on the relationship between prepregnancy maternal BMI and childhood adiposity-related variables. All models were controlled for potential confounders, which included current offspring age, sex, race/ethnicity, Tanner stage, birth weight, and maternal GDM status.

Results

Table I shows the characteristics of the study population, according to maternal prepregnant obesity status. Of the 313 eligible mothers, 164 were classified as overweight or obese with a prepregnant BMI >25 kg/m^2. The groups were similar in terms of maternal age, parity status, and gestational age at delivery. Women categorized as overweight or obese were more likely to be diagnosed with GDM compared with normal-weight women, although not significantly (P = .09). Maternal GWG patterns according to maternal prepregnancy overweight/obesity status are also described in Table I. As expected, normal-weight mothers gained significantly more weight in each trimester of pregnancy and overall, compared with overweight/obese mothers. However, 68% of overweight/obese mothers exceeded the 2009 GWG IOM recommendations vs only 50% of normal weight mothers (P < .01).

Children in the 2 groups were similar in terms of age, sex, and pubertal status, but significantly more offspring of overweight/obese mothers were of Hispanic or African American descent (P = .004).

All childhood adiposity measurements were significantly different according to maternal prepregnancy obesity status (Table I). Offspring of overweight/obese mothers had significantly greater BMI, WC, as well as subcutaneous and visceral fat, compared with offspring of normal-weight mothers. TGs and insulin resistance, as estimated by risk HOMA-IR, also were significantly worse for the children exposed to overweight/obesity in utero.

Table II shows the association between maternal prepregnancy BMI and various offspring adiposity-related outcomes. The analysis is stratified by whether the mothers

Table I. Characteristics of study population (mean ± SD) according to maternal prepregnancy overweight/obesity status

	Normal weight (n = 149)	Overweight or obese (n = 164)	*P* value
Maternal characteristics			
Maternal age at delivery, y	30.38 ± 5.73	30.66 ± 5.84	.67
Nulliparous, n (% yes)	42 (28.19)	50 (30.49)	.66
Prepregnant BMI, kg/m^2	21.70 ± 1.99	30.76 ± 5.29	<.0001
GDM, n (%)	24 (16.11)	39 (23.78)	.09
First-trimester weight gain, kg	3.98 ± 1.07	3.18 ± 1.51	<.0001
Second-trimester weight gain, kg	4.88 ± 1.08	4.08 ± 1.53	<.0001
Third-trimester weight gain, kg	5.68 ± 1.07	4.89 ± 1.51	<.0001
Total GWG, kg	16.52 ± 3.55	13.58 ± 5.41	<.0001
Exceeded IOM recommended GWG, n (%)	74 (49.66)	111 (67.68)	.0012
Child characteristics			
Gestational age at delivery, wk	38.86 ± 1.79	38.74 ± 2.43	.62
Birth weight, g	3211.30 ± 551.00	3250.10 ± 653.30	.57
Current age, y	10.48 ± 1.41	10.39 ± 1.50	.59
Male sex, n (%)	81 (54.36)	91 (55.49)	.84
Race/ethnicity, n (%)			.004
Non-Hispanic white	80 (53.69)	61 (37.20)	
Hispanic	62 (41.61)	83 (50.61)	
Non-Hispanic African American	7 (4.70)	20 (12.20)	
Tanner stage ≥2, n (%)	77 (46.95)	87 (53.05)	.25
BMI, kg/m^2	17.80 ± 3.31	20.34 ± 5.14	<.0001
Waist, cm	62.83 ± 9.02	69.23 ± 13.50	<.0001
SAT, cm^2	90.47 ± 78.05	149.30 ± 119.50	<.0001
VAT, cm^2	17.55 ± 9.98	24.11 ± 15.80	<.0001
TG, mg/dL	82.72 ± 37.91	93.51 ± 43.80	.02
HDL-C, mg/dL	52.72 ± 9.44	50.46 ± 11.14	.06
HOMA-IR	36.69 ± 26.92	52.52 ± 39.95	<.0001
Physical activity, blocks/day	4.52 ± 2.93	3.88 ± 2.88	.05
Total daily calories, kcal/d, mean (95% CI)	1795.60 ± 549.60	1839.40 ± 531.30	.47
Percent calories from fat, %	35.99 ± 5.11	36.20 ± 4.80	.70

Table II. The association between maternal prepregnancy BMI and offspring outcomes, stratified by GWG status*

	Adequate GWG, β (95% CI)	Excessive GWG, β (95% CI)	Interaction, *P* value
BMI, kg/m^2	0.13 (0.02-0.253)	0.34 (0.25-0.44)	.004
Waist, cm	0.38 (0.10-0.65)	0.83 (0.58-1.08)	.01
SAT, cm^2	3.49 (0.89-6.08)	7.26 (4.90-9.62)	.03
VAT, cm^2	0.37 (0.004-0.74)	0.72 (0.39, 1.06)	.16
TG, mg/dL	0.16 (−0.98, 1.31)	1.12 (0.08, 2.16)	.21
HDL-C, mg/dL	0.16 (−0.12, 0.44)	−0.34 (−0.60, −0.07)	.01
HOMA-IR	0.55 (−0.33, 1.43)	1.66 (0.86, 2.45)	.06

β = the unit increase in offspring parameter for every 1 kg/m^2 increase in maternal prepregnancy BMI.
P values for each univariate model reported.
*Adjusted for age, sex, race/ethnicity, Tanner stage, birth weight, and maternal GDM.

were meeting or exceeding the 2009 IOM recommendations for GWG. Increasing maternal prepregnancy BMI was associated with significantly worse childhood outcomes in both GWG groups. However, the effect of maternal prepregnancy BMI on several childhood outcomes was attenuated for offspring of mothers with adequate vs excessive GWG. This trend was observed for all explored variables and was statistically significant for childhood BMI, WC, SAT, and HDL-C. Additional adjustment for current diet and physical activity did not materially change the results.

The **Figure** illustrates the relationship between maternal prepregnancy BMI and childhood adiposity-related outcomes and the effect of adequate vs excessive GWG on this relationship. In each panel, the association of prepregnancy BMI and child outcome (BMI, WC, SAT, and HDL-C) is attenuated for offspring of mothers who gained adequate vs those who gain excessive gestational weight.

Discussion

In this cohort of more than 300 mother-child pairs, women who were overweight or obese before their pregnancy were more likely to exceed the IOM recommendations compared with women who began their pregnancy at a normal BMI. Furthermore, greater prepregnancy BMI was associated with worse adiposity and metabolic risk markers in their offspring at an average age of 10 years, including greater BMI and WC, SAT and VAT fat deposition, and abnormal lipid markers. Several of these relationships were significantly attenuated, however, for offspring of women that gain the recommended amount of weight, compared with those who gained excessive weight during pregnancy.

This analysis extends previous observations of an association between maternal prepregnancy BMI and offspring adiposity outcomes later in life.[11,20,21,28,29] A clear relationship between maternal weight status before pregnancy has been linked to offspring obesity as early as 2-4 years of age in a retrospective cohort of low-income Women, Infants, and Children participants,[11] in a national representative cross-sectional sample of 6-8-year-olds[20] and extended to early adolescence period in a prospective sample of more than 200 white mother-child pairs.[21] Various adiposity indicators in children have been explored, including BMI percentile[11,20] and fat mass via dual X-ray absorptiometry measurements.[21] Offspring of mothers who had a BMI ≥30 kg/m^2 prepregnancy had a greater odds of being categorized as obese (≥95th percentile) at age 2 years (OR 2.2 [95% CI 1.8-2.6]), 3 years (OR 2.6 [95% CI 2.2-3.1]), and 4 years (OR 2.6 [95% CI 2.2-3.1]).[11] Similarly, for every one-unit increase in maternal pregnancy BMI, fat mass measured by dual X-ray absorptiometry increased by 0.26 (95% CI 0.04-0.48) in boys and 0.42 (95% CI 0.29-0.56) in girls at age 9 years.[21]

Several mechanisms that are not mutually exclusive may explain these associations. These include shared genes, shared familial socioeconomic and lifestyle factors, as well as specific intrauterine effects. Work, particularly from the Pima Indian population, suggests that the effect of maternal pregnancy diabetes on offspring obesity risk is not fully explained by genetic factors. In a small nuclear family study (52 families, 182 siblings) conducted in the Pima Indian population, obesity was greater among nondiabetic offspring born after the mother had been diagnosed with type 2 diabetes (ie, overnutrition resulting from exposure to increased intrauterine glucose levels) than in their siblings born before their mothers' diagnosis (ie, exposed to lower intrauterine glucose levels).[30] In another study the prevalence of obesity among 2- to 18–year-old siblings born after maternal biliopancreatic surgery was 52% lower than among age-matched siblings born when their mother was obese.[31] Because siblings discordant for intrauterine exposures carry a similar risk of inheriting the same susceptibility genes and share a similar postnatal environment, such studies provide strong evidence that part of the excess risk of childhood obesity associated with overnutrition in utero reflects specific intrauterine effects.

From a public health prevention perspective, distinguishing between specific intrauterine mechanisms and shared familial genetic/behavioral effects is essential for the development of randomized trials aimed at testing effective pregnancy interventions to reverse the obesity epidemic. In the absence of definite evidence provided by a randomized clinical trial, this question can be tested by exploring whether GWG is a potential effect modifier of the relationship between maternal BMI and child outcomes. We found that adequate GWG significantly reduces the association between maternal prepregnancy BMI and offspring outcomes. For most childhood adiposity-related outcomes, the association with maternal prepregnancy BMI was still significant even if mothers gained the recommended amount of weight during pregnancy, likely reflecting the other causal pathways described previously (shared familial genetic and nongenetic effects); however, all these associations were substantially reduced (by 50%-60%) if women gained the recommended amount of weight during pregnancy. Of note, the inverse association between maternal BMI and offspring HDL-C levels

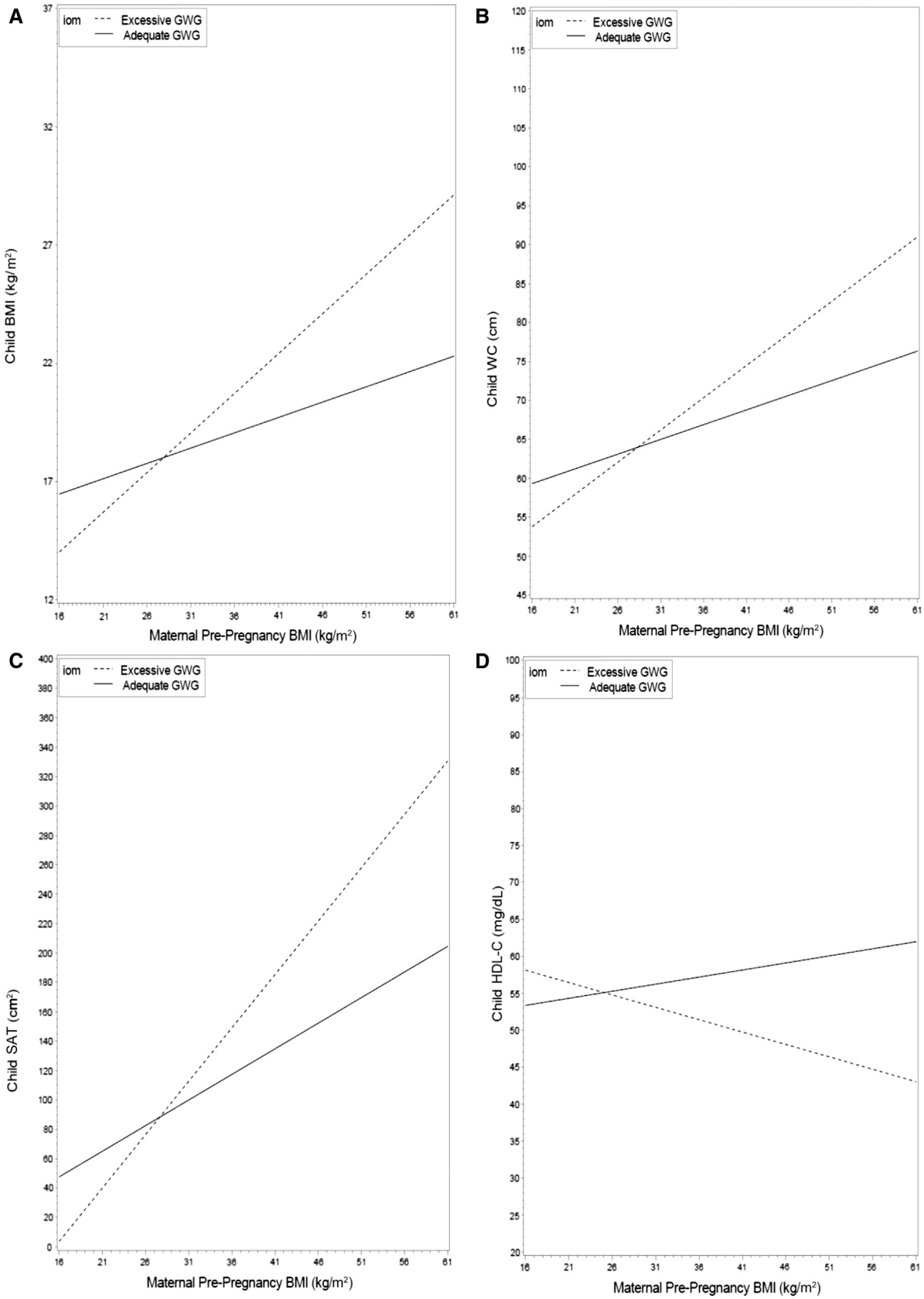

Figure. **A-D,** GWG modifies the association between maternal prepregnancy BMI and childhood adiposity-related measurements.

observed with excessive weight gain during pregnancy became nonsignificant among the group who met the GWG recommendations.

Our study had numerous strengths, including directly measured pregnancy exposures, state-of the art measures of childhood adiposity, and the ability to readily explore associations between pregnancy exposures and childhood adiposity outcomes later in life. Limitations include the observational (rather than experimental) nature of the study and, likely, the relatively limited size of the cohort, which may have resulted in some nonsignificant interactions. We were underpowered to additionally explore whether insufficient GWG modifies the association between maternal BMI and offspring outcomes. However, the majority of research in this area has found no or little association between inadequate GWG and childhood risk of obesity.[32] Finally, our cohort has oversampled women with GDM and thus our findings may not be completely generalizable to a lower-risk population.

In conclusion, our findings suggest that the effect of maternal prepregnancy BMI on several childhood adiposity-related outcomes is attenuated for offspring of mothers with adequate vs excessive GWG. Therefore, pregnant women should be encouraged to follow the IOM recommendations of weight gain for their given prepregnancy BMI. Finally, our study lends support for pregnancy interventions aiming at controlling GWG to prevent childhood obesity. Carefully designed randomized clinical trials are needed to determine whether improved weight gain patterns can be achieved throughout pregnancy that would prevent the short and long-term consequences on the offspring, and curb the obesity epidemic. ■

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References

1. Yeh J, Shelton JA. Increasing prepregnancy body mass index: analysis of trends and contributing variables. Am J Obstet Gynecol 2005;193:1994-8.
2. Chu SY, Callaghan WM, Kim SY, Schmid CH, Lau J, England LJ, et al. Maternal obesity and risk of gestational diabetes mellitus. Diabetes Care 2007;30:2070-6.
3. Okun N, Verma A, Mitchell BF, Flowerdew G. Relative importance of maternal constitutional factors and glucose intolerance of pregnancy in the development of newborn macrosomia. J Matern Fetal Med 1997;6:285-90.
4. Baeten JM, Bukusi EA, Lambe M. Pregnancy complications and outcomes among overweight and obese nulliparous women. Am J Public Health 2001;91:436-40.
5. Oken E, Rifas-Shiman SL, Field AE, Frazier AL, Gillman MW. Maternal gestational weight gain and offspring weight in adolescence. Obstet Gynecol 2008;112:999-1006.
6. Kim J, Peterson KE, Scanlon KS, Fitzmaurice GM, Must A, Oken E, et al. Trends in overweight from 1980 through 2001 among preschool-aged children enrolled in a health maintenance organization. Obesity (Silver Spring) 2006;14:1107-12.
7. Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, Koplan JP. The continuing epidemic of obesity in the United States. JAMA 2000;284:1650-1.
8. Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. Lancet 2002;360:473-82.
9. Catalano PM. Obesity and pregnancy—the propagation of a viscous cycle? J Clin Endocrinol Metab 2003;88:3505-6.
10. Pettitt DJ, Knowler WC. Diabetes and obesity in the Pima Indians: a cross-generational vicious cycle. J Obesity Weight Regul 1988;7:61-75.
11. Whitaker RC. Predicting preschooler obesity at birth: the role of maternal obesity in early pregnancy. Pediatrics 2004;114:e29-36.
12. Koupil I, Toivanen P. Social and early-life determinants of overweight and obesity in 18-year-old Swedish men. Int J Obes (Lond) 2008;32:73-81.
13. Wrotniak BH, Shults J, Butts S, Stettler N. Gestational weight gain and risk of overweight in the offspring at age 7 y in a multicenter, multiethnic cohort study. Am J Clin Nutr 2008;87:1818-24.
14. Moreira P, Padez C, Mourao-Carvalhal I, Rosado V. Maternal weight gain during pregnancy and overweight in Portuguese children. Int J Obes (Lond) 2007;31:608-14.
15. Oken E, Taveras EM, Kleinman KP, Rich-Edwards JW, Gillman MW. Gestational weight gain and child adiposity at age 3 years. Am J Obstet Gynecol 2007;196:322.e1-e8.
16. Robinson CA, Cohen AK, Rehkopf DH, Deardorff J, Ritchie L, Jayaweera RT, et al. Pregnancy and post-delivery maternal weight changes and overweight in preschool children. Prev Med 2014;60:77-82.
17. Ensenauer R, Chmitorz A, Riedel C, Fenske N, Hauner H, Nennstiel-Ratzel U, et al. Effects of suboptimal or excessive gestational weight gain on childhood overweight and abdominal adiposity: results from a retrospective cohort study. Int J Obes (Lond) 2013;37:505-12.
18. Fraser A, Tilling K, Macdonald-Wallis C, Sattar N, Brion MJ, Benfield L, et al. Association of maternal weight gain in pregnancy with offspring obesity and metabolic and vascular traits in childhood. Circulation 2010;121:2557-64.
19. Hinkle SN, Sharma AJ, Swan DW, Schieve LA, Ramakrishnan U, Stein AD. Excess gestational weight gain is associated with child adiposity among mothers with normal and overweight prepregnancy weight status. J Nutr 2012;142:1851-8.
20. Salsberry PJ, Reagan PB. Dynamics of early childhood overweight. Pediatrics 2005;116:1329-38.
21. Gale CR, Javaid MK, Robinson SM, Law CM, Godfrey KM, Cooper C. Maternal size in pregnancy and body composition in children. J Clin Endocrinol Metab 2007;92:3904-11.
22. Weight gain during pregnancy: Reexamining the guidelines. Washington (DC): National Academy of Sciences; 2009.
23. National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and the other categories of glucose intolernance. Diabetes 1979;28:1039-57.
24. Marshall WA, Tanner JM. Growth and physiological development during adolescence. Annu Rev Med 1968;19:283-300.
25. Block G, Murphy M, Roullet J, Wakimoto P, Crawford PB, Block T. Pilot validation of a FFQ for children 8-10 years. In: Proceedings of the Fourth International Conference on Dietary Assessment Methods, September 17-20, 2000; Tuscon, Arizona.
26. Pate RR, Ross R, Dowda M, Trost SG, Sirard J. Validation of a 3-day physical activity recall instrument in female youth. Pediatr Exerc Sci 2003;15:257-65.
27. Hu HH, Nayak KS, Goran MI. Assessment of abdominal adipose tissue and organ fat content by magnetic resonance imaging. Obes Rev 2011;12:e504-15.
28. Li C, Kaur H, Choi WS, Huang TT, Lee RE, Ahluwalia JS. Additive interactions of maternal prepregnancy BMI and breast-feeding on childhood overweight. Obes Res 2005;13:362-71.
29. Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, et al. Early life risk factors for obesity in childhood: cohort study. BMJ 2005;330:1357.

30. Dabelea D, Pettitt DJ. Intrauterine diabetic environment confers risks for type 2 diabetes mellitus and obesity in the offspring, in addition to genetic susceptibility. J Pediatr Endocrinol Metab 2001;14:1085-91.
31. Kral JG, Biron S, Simard S, Hould FS, Lebel S, Marceau S, et al. Large maternal weight loss from obesity surgery prevents transmission of obesity to children who were followed for 2 to 18 years. Pediatrics 2006;118:e1644-9.
32. Nehring I, Lehmann S, von Kries R. Gestational weight gain in accordance to the IOM/NRC criteria and the risk for childhood overweight: a meta-analysis. Pediatr Obes 2013;8:218-24.