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Relationship of early pregnancy waist to hip ratio versus body mass index with gestational diabetes and insulin resistance

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Abstract

Objective—To determine the risk of gestational diabetes (GDM) and insulin resistance (IR) in obesity defined by body mass index (BMI), waist-to-hip ratio (WHR) or both combined.

Methods—Secondary analysis of a randomized multicenter trial of antioxidant supplementation versus placebo in nulliparous low-risk women to prevent pregnancy associated hypertension. Women between 9–16 weeks with data for WHR and BMI were analyzed for GDM (n=2300). Those with fasting glucose and insulin between 22–26 weeks (n=717) were analyzed for IR by homeostasis model assessment of insulin resistance (HOMA-IR; normal≤75thpercentile). WHR and BMI were categorized as normal (WHR<0.80; BMI<25kg/m^2); overweight (WHR:0.8–0.84; BMI:25–29.9kg/m^2); and obese (WHR≥0.85; BMI≥30kg/m^2). ROC curves and logistic regression models were used.

Results—Compared with normal, the risks of GDM or IR were higher in obese by BMI or WHR. The subgroup with obesity by WHR but not by BMI had no increased risk of GDM. BMI was a better predictor of IR (AUC-0.71(BMI), 0.65(WHR), p=0.03) but similar to WHR for GDM (AUC-0.68(BMI), 0.63(WHR), p=0.18.

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Conclusion—Increased WHR and BMI in early pregnancy are associated with IR and GDM. BMI is a better predictor of IR compared with WHR. Adding WHR to BMI does not improve its ability to detect GDM or IR.

Trial Registration number—NCT00135707 http://clinicaltrials.gov/

Keywords

Maternal BMI; Waist-to-Hip ratio; gestational diabetes; Insulin resistance

Introduction

Obesity is a major epidemic in the United States, affecting almost one-third of all individuals, and its incidence during pregnancy has doubled over the past 2 decades[1]. Various indices have been developed to characterize obesity and assess its association with adverse health outcomes. The most commonly studied index, body mass index (BMI) [body weight (kilograms)/height^2 (meter^2)] is a measure of total body fat. An elevated BMI has been associated with increased risk of coronary artery disease, hypertension and type II diabetes in the general population[2]. However, BMI values may have different connotations in individuals with diverse ethnic background, short/tall stature, or varied muscle mass, and does not reflect the regional distribution of fat in the body, i.e. subcutaneous versus visceral/central[2]. Such differentiation is important, as subcutaneous fat has fundamentally different metabolic properties than visceral fat.

Visceral fat secretes inflammatory and thrombogenic factors, and inhibits adiponectin production, thus leading to impaired glucose metabolism, diabetes mellitus, hypertension, metabolic syndrome and other cardiovascular diseases[3]. Therefore, measures of central/abdominal obesity such as waist circumference (WC) and waist to hip ratio (WHR) have been compared to BMI for their association with adverse cardiovascular and metabolic consequences. Larger WC is predictive of type 2 diabetes, dyslipidemia, hypertension and coronary artery disease[4]. In non-pregnant females, WHR of >0.85 had a stronger association with type 2 diabetes compared with WC or BMI[5].

During pregnancy, high BMI has been associated with adverse maternal and neonatal outcomes, and is a known risk factor for gestational diabetes[6, 7] and insulin resistance[8]. However, the association between WHR and gestational diabetes and insulin resistance has not been systematically evaluated. The correlation amongst these obesity indices is varied (0.88, 0.34, and 0.44 for BMI-WC, BMI-WHR, WC-WHR, respectively)[9], which suggests that they may provide different information and thus may not be interchangeable. Accordingly, our objective was to determine the risk of GDM and IR in obesity defined by BMI, WHR or both combined and to test the strength of association of WHR versus BMI with subsequent development of GDM or IR.

Materials and Methods

This is a secondary analysis of a randomized multicenter trial of Vitamin C and E supplementation for prevention of pregnancy associated hypertension. The eligibility criteria

for enrollment in the trial were gestational age 9 – 16 weeks with singleton pregnancy in nulliparous women with no history of pre-gestational hypertension, proteinuria, diabetes or other medical problems, substance abuse, fetal abnormalities, uterine bleeding or in-vitro fertilization. A total of 10,154 women were enrolled from 16 clinical centers affiliated with *Eunice Kennedy Shriver* National Institute of Child Health and Human Development Maternal Fetal Medicine Units (MFMU) Network between 2003 and 2008. Of all the patients enrolled in the randomized control trial, 2394 women participated in the "prediction study", and were followed through the pregnancy. A prospective cohort study had been designed to complement the randomized controlled trial and a sub-population of subjects participated in this "prediction study". These women were nulliparous, underwent original randomization between 9–12 weeks and had additional procedures such as blood testing and uterine artery doppler. This was designed to test for various biochemical and biophysical markers for their ability to predict preeclampsia in these women. Of these, 2300 had data for WHR, BMI at enrollment and were analyzed for development of GDM. A subgroup of women who had fasting insulin and glucose levels drawn between 22 – 26 weeks (n=717) were analyzed for development of insulin resistance. Outline of study participants analyzed is shown in figure 1. There was no statistical difference between vitamin treatment groups for development of GDM or IR, so the groups were combined. Full details of the study design and technique of data collection have been described previously[10]. The Institutional Review Boards of each clinical site and the data-coordinating center approved the study.

The waist and hip measurements were standardized and the study personnel were trained to perform the measurements while patient was standing erect, at a horizontal plane, at the level of umbilicus at the end of normal expiration for waist and at the site of maximum extension of the buttocks for hip measurement.

Gestational diabetes was diagnosed at 26 weeks, per the guidelines of each clinical center. Insulin resistance was estimated by using the previously validated surrogate marker of Homeostatic model assessment of insulin resistance (HOMA-IR)[11] as defined below.

$$\mathrm{HOMA-IR}=\text{fasting insulin }(\mu\mathrm{U/mL})\times\text{fasting glucose }(\mathrm{mmol/L})/22.5.$$

The measurements were categorized as normal range (WHR <0.80, BMI <25 kg/m^2); overweight (WHR 0.80 – 0.84, BMI 25 – 29.9 kg/m^2); and obese (WHR ≥0.85, BMI≥30 kg/m^2) based on the WHO criteria[2]. Since HOMA-IR values were not normally distributed, we used percentiles to define elevated HOMA-IR, which was dichotomized into normal (≤75th percentile) versus abnormal (>75 percentile).

The primary outcome of the study was the risk of GDM and insulin resistance in patients classified as normal, overweight and obese by either WHR criteria, BMI criteria or both combined. The secondary outcome was the association of early pregnancy WHR versus BMI with subsequent GDM and insulin resistance.

The Wilcoxon rank sum test was used to analyze continuous variables and chi-square test was used for categorical variables. Receiver operating characteristics curves and logistic regression models adjusting for maternal demographics including maternal age, education,

race, weeks of gestation at enrollment, alcohol and smoking status, were used to evaluate the association of WHR and BMI with GDM and HOMA-IR. Statistical analysis was conducted with SAS software (SAS Institute, Cary, NC). A nominal two-sided P value less than 0.05 was considered to indicate statistical significance and no adjustments were made for multiple comparisons.

Results

Baseline maternal demographic characteristics for the 2300 women are described in Table 1. The demographics of the subset of 717 patients were analyzed and were similar to the rest of the participants in terms of pre-pregnancy weight and BMI at enrollment and smoking status (data not shown).

3.5% (80/2300) of the participants developed GDM and 25% (179/717) developed insulin resistance during pregnancy. Mean WHR and BMI of the patients who developed GDM in the study were significantly higher than those who did not develop GDM (0.88 +/− 0.07 versus 0.84 +/− 0.08 for WHR, $p<0.0001$ and 30.85 +/− 8.3 versus 26.06 +/− 6.12 Kg/m2 for BMI, $p<0.0001$, respectively). Mean WHR and BMI were also significantly higher for those who developed IR compared with normal (0.86 ± 0.08 versus 0.83 ± 0.07, $p=0.0001$ for WHR and 30.33 ± 8.15 versus 24.94 ± 5.19, $p<0.0001$ for BMI, respectively.

Women who were overweight or obese by BMI definition (BMI≥25) regardless of their WHR had higher odds of GDM and IR compared to normals (Table 2). Those who were obese by BMI ≥30 had adjusted odd ratios of 4.75 (95% CI 2.65–8.52) and 5.9 (95% CI 3.79–9.19) for GDM and IR, respectively. Those who were obese by WHR regardless of their BMI had increased odds of GDM and IR, (aOR 2.65, 95% CI 1.34–5.25) and (aOR 2.63, 95% CI 1.68–4.13), respectively. However, overweight by WHR was not significantly different from those with normal by WHR for either GDM or IR (Table 2).

Additional regression analyses were done where BMI and WHR were combined, to ascertain their significance when either one or both were abnormal (BMI≥30 and or WHR≥0.85) compared to when both were in non-obese range (BMI<30 and WHR<0.85) in the study population. In this analysis, the aOR in those who were obese by both criteria compared to non-obese by both was 4.28 (95% CI 2.28–8.02) for GDM and 6.03 (95% CI 3.70–9.83) for IR respectively. Rates of GDM and IR were also highest in the group that was obese by both criteria (Figure 2).

Those patients who had obesity by BMI only and had non-obese range WHR also had higher aOR of GDM in comparison with non obese by both criteria, (aOR 3.38, 1.48–7.70). Conversely, those who had obesity by WHR only but with non-obese range BMI were not significantly different than those who were non-obese by both criteria, (aOR 1.79, 0.96–3.31). For IR, obesity by WHR, or BMI, or both showed a significant increase in aOR compared to non-obese by both (Figure 2).

ROC curves analysis showed that BMI and WHR were not significantly different in prediction of GDM (AUC 0.68 and 0.63 for BMI and WHR respectively, $p=0.18$) (Figure 3-

a). However, BMI was a stronger predictor of an abnormal HOMA-IR (AUC 0.71 and 0.65 for BMI and WHR respectively p=0.03) (Figure 3-b).

We also performed an ethnicity-stratified analysis. GDM developed in 7.8% of Hispanics, 5.1% of African Americans and 8.7% of whites with BMI≥30 compared with 2.6%, 0.9% and 1.2% of Hispanics, African Americans, and Whites with normal BMI, respectively. There were significant odds of developing GDM with BMI≥30 in Hispanics (aOR 3.1, 95% CI 1.1–8.6) and whites (aOR 7.7, 95% CI 3.2–18.4) compared with normal BMI. In African Americans, the odds of developing GDM were statistically significant only with WHR≥0.85 (aOR 5.0, 95% CI 1.03–24.3) compared with normal WHR and not BMI ≥30 compared with normal BMI. The trend of developing IR with increasing BMI was statistically significant in all races but with increasing WHR only in Whites (Table 3).

Discussion

In this secondary analysis, we found that BMI ≥30 kg/m^2 and WHR ≥0.85 during early pregnancy are significant risk factors for development of GDM and insulin resistance.

Our findings are consistent with previous studies confirming the association of BMI[6, 11–14] and high WHR[15] in early pregnancy with development of gestational diabetes.

When we compared these indices with each other, BMI was a stronger predictor of IR but similar to WHR for GDM. This finding implies that development of IR during pregnancy may be a function of total body fat and less dependent on the central fat distribution, which is in contrast to the various cardiovascular and metabolic consequences of the latter outside of pregnancy[16, 17].

It is known that WHR has a weak correlation with BMI (r=0.4)[18, 19]. However we found that WHR was still comparable to BMI for its association with diabetes. Therefore, it could be hypothesized that combining the two as a collective risk assessment tool may improve their ability to predict GDM and IR. We found that those who were obese by both criteria (BMI≥30/WHR≥0.85) represented the highest risk of GDM and IR compared to non obese by both. However, overall, using both obesity measures together gave similar adjusted odds ratios to using BMI as the only measure of obesity. Thus, BMI should remain as the standard clinical guide to establish the risk of GDM and IR during pregnancy.

It is important to note that the ability of various obesity indicators to predict diabetes varies by ethnicity, likely due to differences in percentage of total body fat and body fat distribution[20 – 23]. In our ethnicity stratified analysis, we noted that in Hispanics and whites, BMI was associated with increased odds of developing GDM, while in African Americans, WHR was associated with GDM. This may suggest a higher role of central adiposity in development of GDM in African Americans that the total body fat alone. BMI ≥30 was associated with increased odds of developing insulin resistance in all races. In whites only, WHR≥0.85 was also associated with insulin resistance. The lack of significance in some categories may be due to small numbers and the wide confidence intervals suggest that validation in other cohorts would be desirable.

Our findings imply that being a strong predictor of GDM and IR, elevated BMI should continue to be a significant risk factor requiring early screening for effective management of pregnancy. In African Americans however, WHR can also be used as an independent risk factor for development of GDM.

Strengths of this study are that it includes data collected by trained staff prospectively on a large number of women. However, our study was limited by the lack of a more precise gestational age range when BMI and WHR were calculated. It ranged from 9 – 16 weeks, and there may be concerns that as the pregnancy progresses, these indices may be influenced by gestational weight gain in lean tissues, thus limiting their use in pregnancy. An alternative would be to use pre-pregnancy BMI or WHR as an indicator of obesity in pregnancy, but that calculation may be frequently self-reported and inaccurate. In addition, due to the waist measurement taken at the level of the umbilicus, the measurements may be confounded in those in whom the umbilicus descended below the waist along with the panniculus.

In conclusion, BMI and WHR are significant risk factors for development of gestational diabetes and insulin resistance. This association varies among different ethnicities. Combining these 2 indicators does not significantly improve the detection of GDM or IR in the general population compared to using only BMI as a measure of obesity status. Thus, WHR should not replace BMI as a predictor of GDM and IR during pregnancy

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Abbreviations

aOR	Adjusted Odds Ratio
AUC	Area Under the Curve
BMI	Body Mass Index
CI	Confidence Interval
GDM	Gestational Diabetes Mellitus
HOMA-IR	Homeostasis Model Assessment of Insulin Resistance
IR	Insulin Resistance
ROC	Receiver Operating Characteristics

WC	Waist Circumference
WHR	Waist-to-Hip ratio

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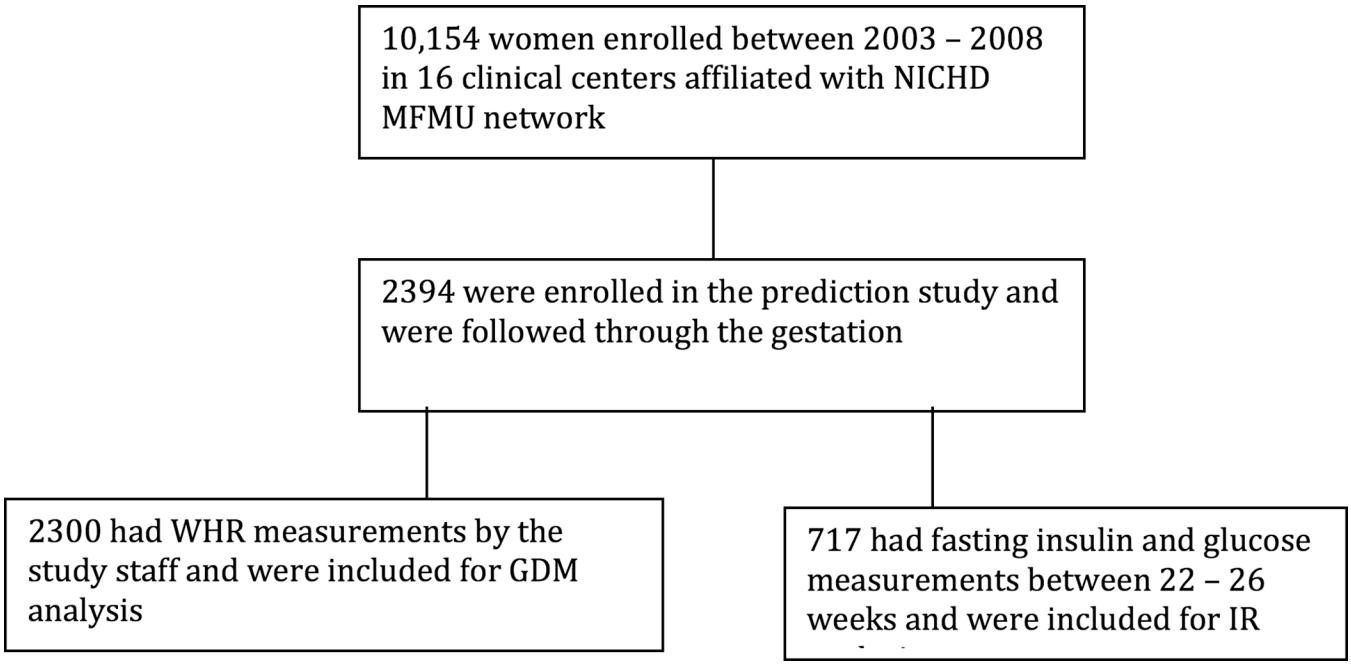

Figure 1.
Study participants analyzed for Gestational Diabetes Mellitus and Insulin Resistance

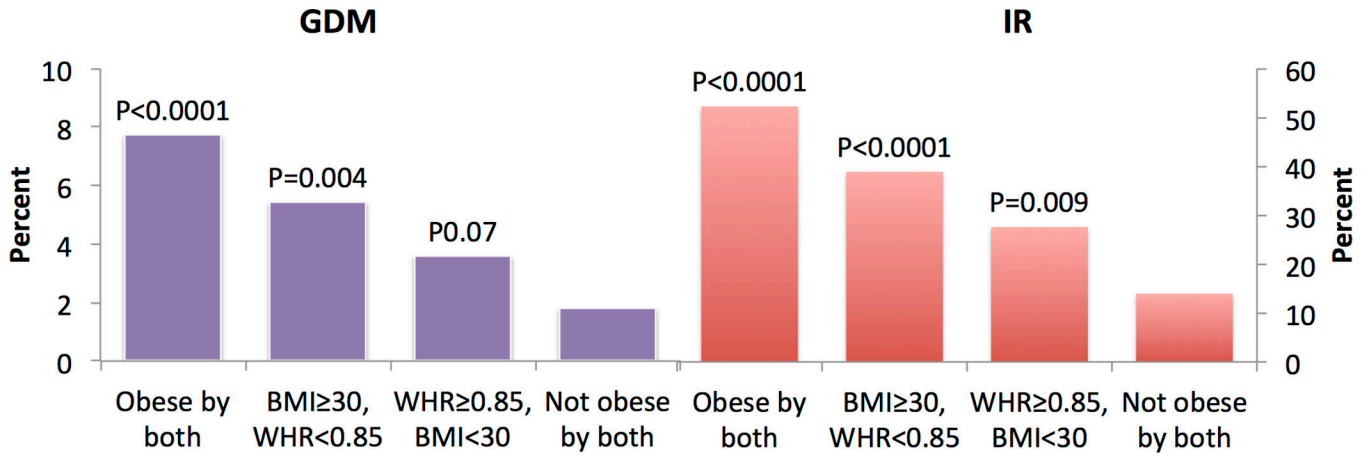

Figure 2.

Percentage with GDM and IR by obesity category when BMI and WHR were combined. P-values presented pertain to adjusted odds ratios for GDM and IR from multivariable logistic regression analysis. The obesity category is compared with the reference category of non-obese (WHR<0.85 and BMI <30).

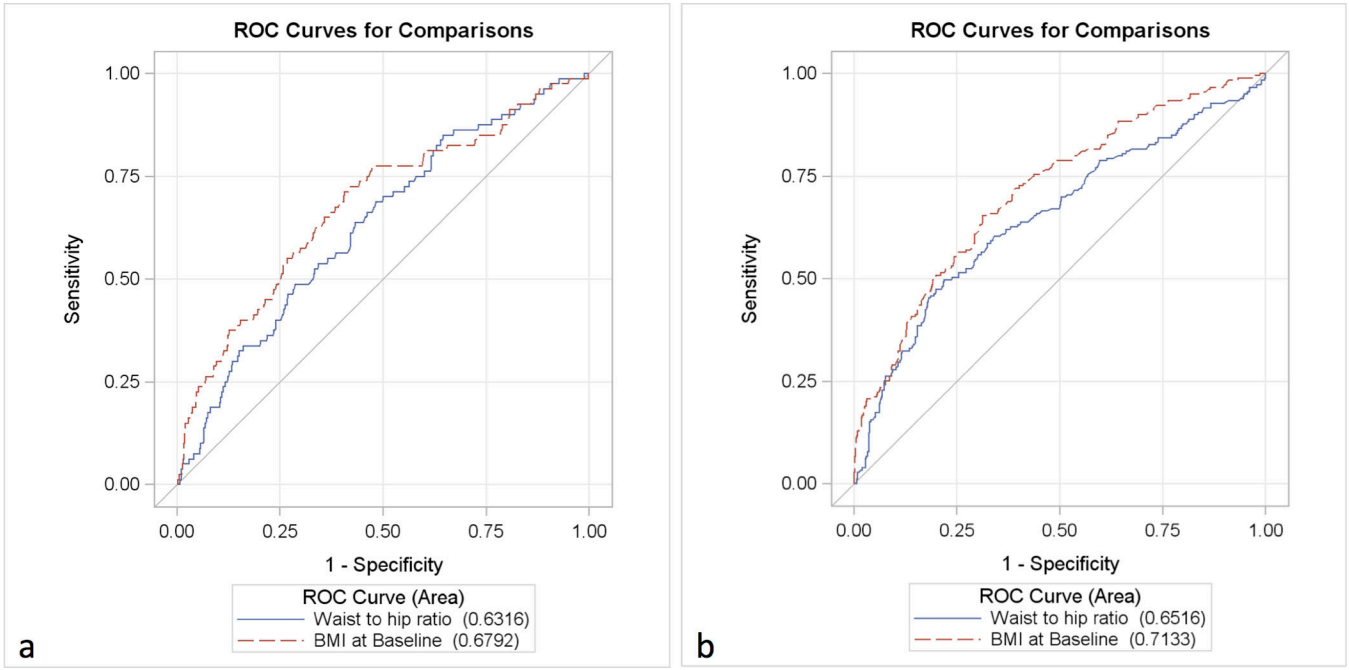

Figure 3.

Receiver operating characteristics curves showing the association of WHR versus BMI with GDM (a) and HOMA-IR (b).

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Table 1

Demographic and baseline characteristics

	Waist hip ratio ≥ 0.80	**Waist hip ratio < 0.80**	**P-value**	**BMI ≥ 25**	**BMI < 25**	**P-value**
	N=1656	**N=644**		**N=1104**	**N=1196**	
Maternal age (years)	23.3 ± 4.7	23.4 ± 4.9	0.99	23.2 ± 4.7	23.5 ± 4.8	0.08
Gestational age at enrollment (weeks)	11.4 ± 1.1	11.4 ± 1.1	0.21	**11.3 ± 1.1**	**11.5 ± 1.0**	**0.003**
Race/Ethnicity						
Non-Hispanic white	**754 (45.5)**	**354 (55.0)**	**<0.001**	**286 (25.9)**	**644 (53.9)**	**<0.001**
African American	**340 (20.5)**	**215 (33.3)**		**337 (30.5)**	**218 (18.2)**	
Hispanic	**529 (31.9)**	**63 (9.8)**		**464 (42.0)**	**306 (25.6)**	
Other	**33 (2.0)**	**12 (1.9)**		**17 (1.5)**	**28 (2.3)**	
Smoking	287(17.3)	92 (14.3)	0.08	**204 (18.5)**	**175 (14.6)**	**0.01**
Education >=12 years	**1218 (73.6)**	**549 (85.3)**	**<0.001**	834 (75.5)	933 (78.0)	0.16

Data are presented as mean ± standard deviation or n (%).

Table 2

Incidence and adjusted odds ratio for development of gestational diabetes mellitus and insulin resistance in normal, overweight and obese women defined by WHR versus BMI

	Gestational diabetes mellitus				Insulin resistance			
	WHR		BMI		WHR		BMI	
	N/Total (%)	aOR (95% CI)	N/Total (%)	aOR (95% CI)	N/Total (%)	aOR (95% CI)	N/Total (%)	aOR (95% CI)
Normal	11/644 (1.7)	1.0	19/1196 (1.6)	1.0	36/229 (15.7)	1.0	51/382 (13.4)	1.0
Over-weight	17/591 (2.9)	1.64 (0.76,3.54)	26/601 (4.3)	**2.72 (1.49, 4.98)**	38/205 (18.5)	1.14 (0.69, 1.91)	50/172 (29.1)	**2.57 (1.63, 4.05)**
Obese	52/1065 (4.9)	**2.65 (1.34, 5.25)**	35/503 (7.0)	**4.75 (2.65, 8.52)**	105/283 (37.1)	**2.63 (1.68, 4.12)**	78/163 (47.9)	**5.9 (3.79, 9.19)**

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Table 3

Obesity and development of GDM and Insulin Resistance by Ethnicity. Adjusted odds ratios (aOR) and 95% CI are presented for patients categorized as obese by WHR or BMI compared with normal WHR (<0.80) and BMI (< 25), respectively.

	Gestational diabetes mellitus				Insulin resistance			
	WHR≥0.85		BMI≥30		WHR≥0.85		BMI≥30	
	N/Total (%)	aOR (95% CI)	N/Total (%)	aOR (95% CI)	N/Total (%)	aOR (95% CI)	N/Total (%)	aOR (95% CI)
Hispanic	23/423 (5.44)	3.22 (0.42,24.71)	8/103 (7.77)	**3.09 (1.11,8.62)**	30/67 (44.78)	2.22 (0.68,7.30)	10/19 (52.63)	**3.52 (1.10, 11.31)**
African American	12/212 (5.66)	**5.0 (1.03, 24.32)**	10/198 (5.05)	4.77 (0.95,23.87)	23/51 (45.10)	1.90 (0.72,5.01)	26/45 (57.78)	**6.18 (2.33, 16.45)**
Whites	17/412 (4.13)	1.82 (0.76,4.33)	17/195 (8.72)	**7.71 (3.23, 18.37)**	49/160 (30.63)	**2.7 (1.50, 4.87)**	40/97 (41.24)	**6.04 (3.36, 10.86)**