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The Impact of Gestational Weight Gain and Diet on Abnormal Glucose Tolerance During Pregnancy in Hispanic Women

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Abstract

Objective—To examine the association of gestational weight gain and dietary factors with abnormal glucose tolerance (AGT).

Methods—We conducted a prospective cohort study among 813 Hispanic prenatal care patients in Massachusetts. Gestational weight gain and oral glucose tolerance test results were abstracted from medical records. Dietary intake was assessed using a semi-quantitative food frequency questionnaire. Target weight gain was based on BMI-specific weekly weight gain rates established by the Institute of Medicine (IOM).

Results—We observed a statistically significant interaction between prepregnancy BMI and weight gain in relation to AGT ($P < 0.01$). Class II/III (BMI ≥ 35 kg/m^2) obese women who had a high rate of weight gain (>0.30 kg/week) or who exceeded target weight were 3–4 times as likely to develop AGT compared to women who gained within IOM ranges (OR = 4.2, 95% CI 1.1–16.0, OR = 3.2 95% CI 1.0–10.5, respectively). Increasing levels of saturated fat and fiber and decreasing levels of energy-dense snack foods and polyunsaturated fat:saturated fat ratio were significantly associated with increased risk of AGT, independent of gestational weight gain.

Conclusions—Weight gain among class II/III obese women and certain dietary components may represent modifiable risk factors for AGT.

Keywords

Abnormal glucose tolerance; Gestational diabetes; Diet; Hispanic; Pregnancy; Weight gain

Introduction

Disturbances in glucose metabolism during pregnancy are common. An estimated 4–12% of pregnancies are complicated by some degree of glucose intolerance, ranging on a continuum from milder abnormal glucose tolerance (AGT) to gestational diabetes mellitus (GDM) [1]. These disorders have been associated with short and long term adverse outcomes for both infant

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and mother, including delivery of large-for-gestational-age infants, [2,3] obesity [4,5] fetal and neonatal death [6] and maternal complications (e.g. caesarean delivery [7] type 2 diabetes) [8]. Emerging research suggests that these adverse maternal and fetal outcomes are also associated with continuous increases in blood glucose levels that are not diagnosed as frank GDM [9–15]. Thus studying risk factors for milder degrees of disturbances in glucose metabolism is important to maternal and offspring health.

Compared to previous decades, American women of childbearing age currently enter pregnancy at a higher weight [16] and are more likely to gain excess weight during pregnancy [17–19]. Recent data from the Pregnancy Risk Assessment Monitoring System in nine states indicate that prepregnancy obesity (BMI > 30.0 kg/m^2) increased between 1993 and 2002, from 13 to 22% [20]. Whereas the association between increasing prepregnancy weight and disturbances in glucose metabolism has been established, [21,22] the relationship between gestational weight gain and risk of these disturbances is less clear.

Although studies on diet during pregnancy are limited, high fat diets have been associated with the development of glucose abnormalities in pregnancy [23] and with the recurrence of GDM in future pregnancies [24]. However, findings have been conflicting with a recent paper finding that consumption of fat and fiber early in pregnancy was not associated with the development of GDM [25]. Therefore, the impact of these macronutrients and food patterns on glucose disturbances during pregnancy remains largely unexplored. In non-pregnant adults, dietary factors such as fat, fiber, glycemic load, fruits and vegetables, and energy-dense snack foods, have been associated with fasting insulin levels and obesity [26,27]. In particular, saturated fatty acids (SFA) have been associated with insulin resistance independent of weight gain [26,28,29]. Fruits and vegetables, which are high in micronutrients and fiber and low in energy density, may contribute to satiety and to the displacement of higher energy-dense foods. In contrast, energy-dense snack foods, which are both high in saturated fat and low in dietary fiber, have been associated with elevated fasting insulin levels and obesity [27]. Given that there is significant evidence from epidemiologic and clinical studies to support the notion that diet influences glucose homeostasis in a non-pregnant population, we would expect this relationship to hold true during pregnancy.

Hispanic women are projected to have the highest birthrates for any minority group in the United States by the year 2009 [30]. In spite of the high prevalence of obesity and excess weight gain during pregnancy among this ethnic group, [16,31,32] little is known about the modifiable risk factors for disturbances in glucose metabolism in this population. Therefore, our goals were to (1) examine the independent association of gestational weight gain with risk of AGT and (2) examine the association between dietary fat, fiber, glycemic load, fruits/vegetables and energy-dense snack foods and AGT among women from the Latina Gestational Diabetes Mellitus Study, a prospective cohort of Hispanic prenatal care patients in Massachusetts.

Methods

Study Design and Population

Details of the study design have been presented elsewhere [33,34]. Briefly, the study was based in the public obstetrics and gynecology clinic and midwifery practice of Baystate Medical Center, a large tertiary care facility in Western Massachusetts. Self-identified Hispanic prenatal care patients were recruited by bilingual interviewers from 2000 to 2003 during the first or second trimester of pregnancy up to 24 weeks gestation (mean, SD = 15.0 ± 5.2 weeks gestation). Interviewers pre-screened eligible patients based on demographic and medical characteristics provided on a daily roster of scheduled patients to generate a list of potential participants. Reasons for exclusion included: ethnicity other than Hispanic; diagnosis of type 2 diabetes, hypertension, heart disease, or chronic renal disease; treatment with medications

thought to adversely influence glucose tolerance (i.e. prednisone or other steroids); multiple gestation pregnancy; less than age 16 or greater than 40 years; ≥24 weeks gestation; or prior participation in the study (to assure inclusion of only one pregnancy per woman.) Less than 1% of women identified as potential participants were excluded based on medical history and 2% were excluded for non-singleton pregnancy. Approximately 2% of potential participants refused to participate.

Overall, 1,231 eligible women were enrolled. For the purposes of this analysis, we excluded women who did not deliver at Baystate Medical Center (n = 123); who miscarried, terminated their pregnancy, or had a preterm birth prior to 28 weeks gestation (n = 48); who were not screened for GDM (n = 54); who were missing data on prepregnancy weight or weight at the time of GDM screening (n = 174), age at time of GDM screen (n = 15) or height (n = 4). After these exclusions, 813 women remained for analysis.

Interviewers informed patients of the study aims and procedures, and each patient read and signed a written informed consent approved by the Institutional Review Boards of the University of Massachusetts-Amherst and Baystate Medical Center.

At the time of recruitment, interviewers collected information on substance use, socio-demographic factors, prepregnancy BMI, and physical activity. Dietary intake was assessed in mid-pregnancy. After delivery, information on incident GDM, AGT, clinical characteristics of the current pregnancy (including weight at each prenatal care visit), and medical and obstetrical history was abstracted from medical records.

Assessment of Abnormal Glucose Tolerance

Prenatal care patients are screened for GDM between the 24th and 28th week of gestation as part of routine hospital protocol. The screening test consists of administering a random 50-g glucose load and a plasma glucose determination one hour later (1-h OGTT). If the plasma glucose value was ≥135 mg/dl, a 3-h glucose tolerance test was performed. A positive screen (≥135 mg/dl) on the 1-h OGTT was used as a measure of AGT.

Assessment of Gestational Weight Gain

The Institute of Medicine (IOM) recommends total gestational weight gain in categories based on prepregnancy BMI. These ranges are: 12.7–18.1 kg for underweight, 11.3–15.9 kg for normal weight, 6.8–11.3 kg for overweight and at least 6.8 kg for obese women (BMI > 29.) The IOM also provides trimester-specific weekly gestational weight gains again upon prepregnancy BMI. Specifically for the second and third-trimesters, the IOM recommends 0.490 kg/week for underweight women, 0.440 kg/week for normal weight women and 0.30 kg/week for overweight women [35]. Since rates of weight gain in the first-trimester are non-linear and because the IOM did not provide specific values, rates used in the prior literature were used (0.27 kg/week for underweight women, 0.183 for normal weight women and 0.083 for overweight women) [36]. Because the IOM only recommends a lower limit of weight gain for obese women, a weight gain of 0.5 kg in the first trimester and a weekly rate of 0.23 kg in the second and third trimesters was used [36].

To accurately capture weight gain up to the time of GDM screening and to compare the observed weight gains to the BMI-specific IOM recommendations, a ‘‘target’’ weight variable was computed for each woman based on the approach used by Siega-Riz et al. [36] and Saldana et al. [36,37]. Target weight was calculated based on the amount of weight each woman was ‘‘expected’’ to gain based on the trimester-specific weekly gestational weight gains above. We then calculated actual weight gain as a percentage of target weight. Given that the literature has not established cut-off points for the percentage of target weight, based on the distribution

of this variable in our cohort, we stratified women into three groups: those who gained less than 3% of target, within ±3% of target, and more than 3% above target weight.

As a secondary measure, rate of weight gain up to the time of GDM screening was calculated as weight at time of GDM screen minus prepregnancy weight divided by gestational age at time of screen. Because previous studies have not established cut point values for rate of weight gain, we stratified women based upon the median value observed in this dataset (less than 0.30 kg/week and greater than 0.30 kg/week).

Dietary Assessment

Dietary information was assessed in mid-pregnancy (mean, SD = 23 ± 8 weeks gestation) among 527 (65%) participants. Women for whom an FFQ was not obtained did not deliver at Baystate Medical Center, experienced a miscarriage, pregnancy termination, or preterm birth, or failed to attend a prenatal care visit or were not located by the interviewer at the clinic or by telephone. Dietary intake was assessed using a semi-quantitative food frequency questionnaire (FFQ) adapted and validated for use with Hispanics in the Northeastern US (where individuals are primarily of Puerto Rican or Dominican origin) [38]. The FFQ was administered in person or over the phone in either Spanish or English to all study participants who could be interviewed during the second trimester by trained bilingual interviewers. Women were asked to report their usual intake since the beginning of their pregnancy.

Completed FFQs were processed by the Dietary Assessment and Epidemiology Research Program at the United States Department of Agriculture, Human Nutrition Research Center for Aging at Tufts University. FFQs were scanned and transferred to electronic files and the Minnesota Nutrient Data System was used to calculate nutrient intake profiles. FFQ forms with more than 12 rows of missing data or forms that resulted in an implausible total energy intake of less than 600 or more than 4,000 kilocalories (kcals) per day [39] were deemed invalid (n = 74), and were excluded from the final dietary sub-sample.

Total SFA, polyunsaturated fatty acids (PUFA) and monounsaturated fatty acids (MUFA) were assessed both as percentages of total energy and as kilocalories from each macronutrient source [40]. Kilocalories from SFA, PUFA and MUFA were calculated by multiplying grams of each by 9 kcal/g. A simple ratio of PUFA:SFA (P:S) was calculated as the quotient of total calories from PUFA and SFA with a higher ratio representing lower amounts of saturated fat and higher amounts of polyunsaturated fat. Total grams of dietary fiber per day were assessed. Fruits (all fruits and fruit juices) and vegetables (including corn, potatoes, root crops and plantains) were converted to daily servings per day and categorized as low (less than 4 servings per day), moderate (4–6 servings per day) and recommended (7 servings or more per day). The average dietary glycemic index value based on a glucose standard was calculated for each participant [41]. Dietary glycemic load was calculated as the product of the dietary glycemic index (reference: glucose) and carbohydrate intake divided by 100.

Energy-dense snack foods considered in this analysis were divided into five food categories: [42] (1) baked goods (cookies, cakes, pies); (2) ice cream (ice cream, ice cream sundaes, sherbet, milkshakes); (3) chips (potato chips and corn chips); (4) candy (chocolate and non-chocolate candy); and (5) soda (only sugar-sweetened). The ice cream category was included because of its high energy content. Energy and nutrients were summed across all foods to obtain total intakes of kilocalories and nutrients for each subject. The percentage of total daily energy from energy-dense snack foods was calculated by adding the kilocalories from each energy-dense snack food and dividing the sum by total daily kilocalories. Kilocalories from energy-dense snack foods were then divided into tertiles for analysis (low: less than 100 kcals per day, moderate: 100–400 kcals per day, and high: greater than 400 kcals per day).

Covariate Assessment

We collected information on risk factors for GDM including maternal age, education (highest level of education completed), annual household income (total household income including salary, tips, welfare and other income), parity, prepregnancy BMI, history of diagnosed GDM, hypertension in the current pregnancy, cigarette smoking during pregnancy, illicit drug use during pregnancy, physical activity (assessed using a modified version of the Kaiser Physical Activity Survey), [43] and family history of type 2 diabetes. For prepregnancy BMI, the National Heart, Lung, and Blood Institute (NHLBI) and World Health Organization (WHO) [44,45] BMI categories were used (<18.5 underweight, 18.5–24.9 normal weight, 25–29.9 overweight, 30–34.9 class I obese and ≥35 class II and III obese) in addition to the IOM BMI categories (<19.8 underweight, 19.8–26 normal weight, 26–29 overweight and >29 obese) [46] since they provide additional levels in the obese category. Place of birth and language preference were used as measures of acculturation.

Statistical Analysis

Data management and analysis were conducted in SAS (version 9.0). We utilized one-way analysis of variance to compare overall means for continuous variables and bivariate analysis using v^2 to test for differences in categorical variables. Logistic regression was used to model the relation between gestational weight gain and AGT. Goodness of fit was assessed using the Hosmer-Lemeshow goodness of fit test. Collinearity was assessed among covariates by computing Pearson's correlation coefficients, tolerance values and variance inflation factors. Confounding was assessed by evaluating the change in the b-coefficients when each covariate was included in the regression model. A change of 10% or greater was used as an indicator of confounding. Because age is considered an important risk factor in the development of glucose disturbances and because prior studies have adjusted for this, [37,47,48] age was retained in the models even though it did not change the estimate by 10%. Based on findings in the published literature, [37] the potential modifying effect of prepregnancy BMI on weight gain was evaluated by testing the interaction between these two variables. Statistical significance was considered present when $P<0.05$.

For women with valid dietary data, logistic regression models were used to model the relation between total dietary fat, SFA, PUFA, P:S ratio, fiber, glycemic load, fruits/vegetables, energy-dense snack foods and AGT while adjusting for total energy intake and other possible confounders. Interactions between dietary factors and gestational weight gain in relation to AGT were evaluated by adding each of the interaction terms into the model. An estimate for this parameter was then obtained through maximum likelihood estimation method with 95% Wald confidence limits.

Results

Participants in the final sample (n = 813) did not differ statistically by education, birthplace, language preference, prepregnancy BMI, percentage of target weight, family history of diabetes, history of GDM or illicit drug use during pregnancy as compared to the initial sample (n = 1,231). They were however more likely to be younger ($P = 0.01$) and nulliparous ($P = 0.02$). Among the 813 participants, 90 (11%) met the definition of AGT. The mean age of the participants was 22 ± 4.8 years, with the majority (73%) being less than 25 years of age and 123 (15%) being under age 18 (Table 1). Approximately 55% of women were born in the U.S., 40% of participants were nulliparous, and 34% had a family history of diabetes. In unadjusted analysis, increasing age, education, parity, family history of diabetes, personal history of GDM and smoking were positively associated with AGT risk (Table 1). After adjusting for age, these associations were no longer significant with the exception of smoking and family history of

diabetes. Goodness of fit tests indicated that the fitted models were adequate. Collinearity of variables included in the regression models was not observed.

Gestational Weight Gain

Prepregnancy BMI was positively associated both with AGT in unadjusted and adjusted analysis (Table 2). In age-adjusted analyses, women who were obese prior to pregnancy were 2.3 times as likely to develop AGT (OR = 2.3, 95% CI 1.4–3.9) as compared to normal weight women. A total of 39% of women exceeded their target weight, 36% met their target weight and 25% failed to achieve their target weight (Table 2). Overall the mean rate of weight gain was 0.31 ± 0.21 kg/week. The mean gestational weight gain up to time of GDM screening in the overall sample was 8.3 ± 5.4 and 7.7 ± 5.4 kg for women with AGT. Total and rate of weight gain up to time of GDM screening was greatest for women with a BMI < 18.5 kg/m^2 (10.3 kg, 0.37 kg/week), followed by women with a BMI 18.5–25 kg/m^2 (9.4 kg, 0.35 kg/week), then by women with a BMI 25–29 kg/m^2 (8.4 kg, 0.32 kg/week), then by women with a BMI 30–35 kg/m^2 (6.0 kg, 0.22 kg/week), and finally by women with a BMI ≥ 35 kg/m^2 (4.3 kg, 0.18 kg/week). Neither % of target weight nor rate of weight gain was associated with AGT in age-adjusted (Table 2) or multivariable models (data not shown). However, we observed a statistically significant interaction between prepregnancy BMI and gestational weight gain in multivariable models ($P < 0.01$). Therefore, we examined the association between percent of target weight and rate of weight gain and risk of AGT within strata of prepregnancy NHLBI/WHO BMI categories (Table 3). Due to the sparse number of underweight women, they were excluded for this stratified analysis. A total of 36% of normal weight, 48% of overweight, 46% of class I obese and 32% of class II/III obese women exceeded percent of target weight. In multivariable analyses, class II/III women who exceeded their target weight were 4 times as likely (95% CI 1.1–16.0) to develop AGT compared to those women who were within their target weight but confidence intervals were wide (Table 3). The overall fit of the model was adequate, as assessed by the Hosmer-Lemeshow goodness of fit test ($P = 0.92$). The model also predicted 80% of the cases correctly, a reasonably high percent. For normal weight, overweight, and class I obese women, associations between target weight and AGT were not statistically significant. In multivariable analyses, class II/III obese women who gained more than 0.30 kg/week were 3 times as likely (OR = 3.2, 95% CI 1.0–10.5) to develop AGT compared to those that gained less than 0.30 kg/week (Table 3). Rate of weight gain was not associated with AGT among women in the other BMI strata.

Diet

Participants with complete dietary information (n = 423) did not differ statistically by age, education, birthplace, language preference, prepregnancy BMI, percentage of target weight, family history of diabetes, history of GDM or illicit drug use during pregnancy as compared to those without dietary information. They were however less likely to be parous and less likely to smoke. The distribution of total energy and macronutrient intakes by AGT status is shown in Table 4. Overall, total energy, percent energy from total fat, MUFA and PUFA, total amount of grams of fiber per day, and glycemic load did not differ by AGT status. However, SFA (11.9 ± 2.1 vs. 11.2 ± 2.3) and the P:S ratio (0.8 ± 0.3 vs. 0.7 ± 0.2) were greater among women with normal glucose tolerance compared to women with AGT.

The associations of fats and fiber with the development of AGT are presented in Table 5. The percentage of energy from total dietary fat, PUFA, MUFA, and glycemic load were not associated with AGT. After adjusting for known risk factors for AGT, percent of energy from SFA was associated with an increased risk of developing AGT (OR = 1.3, 95% CI 1.1–1.5.) The P:S ratio was also significantly associated with AGT, with a higher ratio associated with decreased risk of AGT (OR = 0.1, 95% CI 0.02–0.45). Dietary fiber was also inversely associated with risk of AGT (OR = 0.9, 95% CI 0.84–0.99).

With regard to eating patterns, compared to those consuming 4–6 servings per day, women consuming 7 or more servings per day of fruit and vegetable consumption did not have an increased risk of AGT (OR = 1.7, 95% CI 0.7–4.5) (Table 5). For energy from energy-dense snack foods, 16% of normal glucose tolerant women had high consumption of energy-dense snack foods (>400 kcals/day) compared to 24% of women with AGT. Compared to those consuming average amounts of energy-dense snack foods, those with low intakes of energy-dense snack foods had approximately 60% decreased risk of AGT (OR = 0.4, 95% CI 0.1–1.0, $P = 0.05$).

Discussion

In this prospective cohort study, we sought to establish the role of gestational weight gain and diet in the development of AGT. We found that exceeding target weight during pregnancy elevated AGT risk only among women with a BMI greater or equal to 35 kg/m^2. In this BMI group, which corresponds to Class II and Class III obesity, women who exceeded their target weight or who had a high rate of weight gain had a 3–4 fold increased risk of AGT. We did not observe significant associations of total dietary fat, PUFA, MUFA, glycemic load or servings of fruits and vegetables with AGT. However, lower levels of energy-dense snack foods, SFA and P:S ratio and higher intakes of fiber were significantly associated with reductions in AGT risk.

Forty-three percent of women gained weight in excess of IOM recommendations, a rate which is comparable to non-Hispanic white populations [17,18]. Our findings that obese Hispanic women of predominately Puerto Rican descent were over 2 times as likely to develop AGT compared to women of normal weight is supported by prior studies conducted among women of other ethnic backgrounds [32,49]. The relationship between gestational weight gain and glucose disturbances has been less clear, however. Of the few studies that have examined this relationship, [32,37,50] only one was conducted among Hispanic women and this population was limited to those of predominantly Mexican descent. To our knowledge, prior studies have not examined gestational weight gain and AGT. Kieffer et al. conducted a cross-sectional study among 552 Hispanic women and found that gestational weight gain up to 28 weeks was not associated with GDM (OR = 1.02, 95% CI 0.985–1.061). The authors failed to observe significant interactions between prepregnancy BMI and weight gain and risk of GDM [32]. The authors did not examine milder forms of glucose disturbance, however. Our findings in a predominantly Puerto Rican population are consistent with these findings.

Our finding of an interaction between prepregnancy BMI and weight gain associated with AGT is consistent with prior research. In a prospective cohort study of 952 pregnant black and white women, [37] Saldana et al. found that gestational weight gain was associated with impaired glucose tolerance (IGT) defined as having one abnormal glucose value on the 3-h OGTT, only among overweight women (BMI ≥ 26–29 kg/m^2) but not among women of other BMI categories. Specifically, women who gained twice the recommended amount of weight had a 2-fold increased risk of IGT compared with women who gained the recommended level. Our findings were similar, but were limited to class II/III obese women.

We found that high percent energy from SFA was associated with an increased risk of developing AGT and that a high P:S ratio was significantly associated with decreased risk of developing AGT. These findings are consistent with some previous studies, although such studies are sparse. Saldana et al. conducted a prospective cohort study among 1,698 pregnant black and white women to study the association between macronutrients measured by FFQ with IGT [23]. The authors found that substituting fat for carbohydrates in statistical models resulted in a significant increased risk (OR = 1.1 95% CI = 1.02–1.12) of both IGT and GDM, although the type of fat considered was not specified [23]. A second cross-sectional study

among 171 pregnant Chinese women found that decreased PUFA intake and low P:S ratio were both associated with IGT and GDM [51]. In a study among 35 Australian pregnant women, women with a recurrence of GDM consumed 41.4% of their energy as fat compared with 33.1% ($P < 0.011$) for women with no recurrence [24]. These studies, however, were not conducted in the US, and did not consider gestational weight gain as a possible confounding factor [24, 51,52]. A more recent study among 1,733 predominantly White pregnant women found that dietary fats, carbohydrate and glycemic load were not associated with GDM or IGT risk, defined as either a failed 1 h-OGTT or one abnormal glucose result on the 3-h OGTT [25]. Our finding that increasing levels of SFA and fiber and decreasing levels of energy-dense snack foods and P:S ratio were significantly associated with increased risk of AGT could be due, in part, to differences in the definition of glucose intolerance and the differences in race/ethnicity.

Beans, the main source of complex carbohydrates in traditional Caribbean Hispanic diets, are high in fiber [53]. We observed that high fiber intake was associated with a decreased risk of developing AGT. Similarly, a recent study by Moses et al. found that women eating lower fiber diets during the second and third trimester gave birth to heavier infants and had higher prevalence of large-for-gestational age infants [54]. These findings are consistent with a low fiber diet inducing disturbances in glucose metabolism and thereby increased glucose transfer to the fetus [54]. An observational study of pregnant women with type 1 diabetes during the second trimester of pregnancy found that insulin requirements were 16–18% lower for women with high fiber intakes compared to women with lower intakes [55]. This observation suggests that higher fiber intakes may prevent glucose disturbances by reducing insulin requirements. Diets that are both high in fiber and low in SFA may be associated with insulin resistance independent of weight gain during pregnancy.

Women in our study who consumed less than 400 kcals of energy-dense snack foods per day had an almost 60% decreased risk of developing AGT compared to women who consumed average quantities. Although the association between energy-dense snack foods and disturbances of glucose metabolism during pregnancy has not been previously explored, studies in adults have demonstrated associations of energy-dense snack foods with elevated fasting insulin levels and obesity [27]. An observational study conducted by Olafsdottir et al. among 406 pregnant women found that eating more sweet foods early in pregnancy increased the risk of excess weight gain (OR = 2.5, 95% CI 1.1–5.8) [56]. Given that energy-dense snack foods are high in SFA and low in PUFA and fiber, our findings that SFA, fiber and a P:S ratio were significantly associated with risk of developing AGT are consistent with these results.

Several limitations of our study are worth noting. First, the relatively small number of observed cases of AGT led to wide confidence intervals, increasing the likelihood of type 2 error, and limiting our ability to detect associations between gestational weight gain and risk of AGT. In addition, we observed small numbers of cases within certain strata of prepregnancy BMI (e.g., 19 cases of AGT among obese patients) limiting our ability to evaluate interaction according to this variable. Second, like most studies of gestational weight gain, prepregnancy weight was self-reported and may have been misreported. A recent validation study of 170 women by Oken et al., however, found an overall correlation coefficient of 0.99 between self-reported pregravid weight and clinically measured weights [57]. In our study, we observed a mean maternal weight gain in early pregnancy (up to time of first visit) of 2.3 kg which is within the range of mean weight gain observed by prior studies which used measured rather than recalled pregravid weight [58–62]. Third, the NHLBI/WHO BMI categories are established for women over the age 18. In our sample 15% were under this age and were less likely to be parous, have a higher education, be born in the US and have a lower BMI. When excluded from the analysis however, the estimates minimally changed; women who exceeded their target weight and who had a BMI ≥ 35 were 3.7 times as likely (95% CI 1.0–14.0) to develop AGT compared to those who stayed within their target weight. Findings were also comparable for rate of weight gain (OR

= 3.5 95% CI 1.04–11.8). Finally, women in the subset with available dietary data were less likely to be parous and less likely to smoke as compared to women in the overall sample. Prior literature has found that people who do not smoke and are nulliparous are more likely to have a better diet quality [63,64].

Our results show that similar to other non-Hispanic populations, obese Hispanic women of predominantly Puerto Rican descent are at increased risk for developing AGT, and that gestational weight gain and diet may be contributing factors. We found that weight gain was associated with AGT among class II/III obese women. This suggests that gestational weight gain may play a role in the development of milder forms of glucose disturbances among obese women. Our observation that class II/III obese women who gain excessive weight during pregnancy are at increased risk for developing AGT, if confirmed would have important implications. Finally, further work is needed to better understand the relationship between diet and glucose disturbances during pregnancy. Once dietary constituents and specific dietary patterns are identified, how best to incorporate culturally appropriate dietary advice into preventive intervention programs should be explored.

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References

1. Kim C, Berger DK, Chamany S. Recurrence of gestational diabetes mellitus: A systematic review. Diabetes Care 2007;30:1314–1319.10.2337/dc06-2517 [PubMed: 17290037]
2. Gonzalez-Quintero VH, Istwan NB, Rhea DJ, Rodriguez LI, Cotter A, Carter J, et al. The impact of glycemic control on neonatal outcome in singleton pregnancies complicated by gestational diabetes. Diabetes Care 2007;30:467–470.10.2337/dc06-1875 [PubMed: 17327306]
3. Kwik M, Seeho SK, Smith C, McElduff A, Morris JM. Outcomes of pregnancies affected by impaired glucose tolerance. Diabetes Research and Clinical Practice 2007;77:263–268.10.1016/j.diabres.2006.12.004 [PubMed: 17275121]
4. Petitt DJ, Bennett PH, Knowler WC, Baird HR, Aleck KA. Gestational diabetes mellitus and impaired glucose tolerance during pregnancy. Long-term effects on obesity and glucose tolerance in the offspring. Diabetes 1985;34(Suppl 2):119–122. [PubMed: 3996763]
5. Gillman MW, Rifas-Shiman S, Berkey CS, Field AE, Colditz GA. Maternal gestational diabetes, birth weight, and adolescent obesity. Pediatrics 2003;111:e221–e226.10.1542/peds.111.3.e221 [PubMed: 12612275]
6. Schmidt MI, Duncan BB, Reichelt AJ, Branchtein L, Matos MC, Costa e Forti A, et al. Gestational diabetes mellitus diagnosed with a 2-h 75-g oral glucose tolerance test and adverse pregnancy outcomes. Diabetes Care 2001;24:1151–1155.10.2337/diacare.24.7.1151 [PubMed: 11423494]
7. Dodd JM, Crowther CA, Antoniou G, Baghurst P, Robinson JS. Screening for gestational diabetes: The effect of varying blood glucose definitions in the prediction of adverse maternal and infant health outcomes. Australian and New Zealand Journal of Obstetrics and Gynaecology 2007;47:307–312.10.1111/j.1479-828X.2007.00743.x [PubMed: 17627686]
8. Linne Y, Barkeling B, Rossner S. Natural course of gestational diabetes mellitus: Long term follow up of women in the SPAWN study. An International Journal of Obstetrics and Gynaecology 2002;109:1227–1231.
9. Carr DB, Newton KM, Utzschneider KM, Tong J, Gerchman F, Kahn SE, et al. Modestly elevated glucose levels during pregnancy are associated with a higher risk of future diabetes among women without gestational diabetes mellitus. Diabetes Care 2008;31:1037–1039.10.2337/dc07-1957 [PubMed: 18223032]

10. Di Cianni G, Seghieri G, Lencioni C, Cuccuru I, Anichini R, De Bellis A, et al. Normal glucose tolerance and gestational diabetes mellitus: What is in between? Diabetes Care 2007;30:1783–1788.10.2337/dc07-0119 [PubMed: 17416793]
11. Hillier TA, Pedula KL, Schmidt MM, Mullen JA, Charles MA, Pettitt DJ. Childhood obesity and metabolic imprinting: The ongoing effects of maternal hyperglycemia. Diabetes Care 2007;30:2287–2292.10.2337/dc06-2361 [PubMed: 17519427]
12. Hampton T. Study finds newborn outcomes affected by blood glucose levels during pregnancy. Journal of the American Medical Association 2007;298:613–614.10.1001/jama.298.6.613 [PubMed: 17684175]
13. Mello G, Parretti E, Mecacci F, Lucchetti R, Lagazio C, Pratesi M, et al. Risk factors for fetal macrosomia: The importance of a positive oral glucose challenge test. European Journal of Endocrinology 1997;137:27–33.10.1530/eje.0.1370027 [PubMed: 9242198]
14. Lapolla A, Dalfra MG, Bonomo M, Castiglioni MT, Di Cianni G, Masin M, et al. Can plasma glucose and HbA1c predict fetal growth in mothers with different glucose tolerance levels? Diabetes Research and Clinical Practice 2007;77:465–470.10.1016/j.diabres.2007.01.022 [PubMed: 17350135]
15. Group THSCR. Hyperglycemia and adverse pregnancy outcomes. The New England Journal of Medicine 2008;358:1991–10.10.1056/NEJMoa0707943 [PubMed: 18463375]
16. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. Journal of the American Medical Association 2004;291:2847–2850.10.1001/jama.291.23.2847 [PubMed: 15199035]
17. Schieve LA, Cogswell ME, Scanlon KS. Trends in pregnancy weight gain within and outside ranges recommended by the Institute of Medicine in a WIC population. Maternal and Child Health Journal 1998;2:111–116.10.1023/A:1022992823185 [PubMed: 10728266]
18. Helms E, Coulson CC, Galvin SL. Trends in weight gain during pregnancy: A population study across 16 years in North Carolina. American Journal of Obstetrics and Gynecology 2006;194:e32–e34.10.1016/j.ajog.2006.01.025 [PubMed: 16647894]
19. Olson CM, Strawderman MS. Modifiable behavioral factors in a biopsychosocial model predict inadequate and excessive gestational weight gain. Journal of the American Dietetic Association 2003;103:48–54.10.1053/jada.2003.50001 [PubMed: 12525793]
20. Kim SY, Dietz PM, England L, Morrow B, Callaghan WM. Trends in pre-pregnancy obesity in nine states, 1993–2003. Obesity (Silver Spring, Md) 2007;15:986–993.
21. Jensen DM, Sorensen B, Feilberg-Jorgensen N, Westergaard JG, Beck-Nielsen H. Maternal and perinatal outcomes in 143 Danish women with gestational diabetes mellitus and 143 controls with a similar risk profile. Diabetic Medicine 2000;17:281–286.10.1046/j.1464-5491.2000.00268.x [PubMed: 10821294]
22. Virally M, Laloi-Michelin M, Meas T, Ciraru N, Ouled N, Medeau V, et al. Occurrence of gestational diabetes mellitus, maternal and fetal outcomes beyond the 28th week of gestation in women at high risk of gestational diabetes. A prospective study. Diabetes & Metabolism 2007;33:290–295.10.1016/j.diabet.2007.03.003 [PubMed: 17652001]
23. Saldana TM, Siega-Riz AM, Adair LS. Effect of macronutrient intake on the development of glucose intolerance during pregnancy. The American Journal of Clinical Nutrition 2004;79:479–486. [PubMed: 14985225]
24. Moses RG, Shand JL, Tapsell LC. The recurrence of gestational diabetes: Could dietary differences in fat intake be an explanation? Diabetes Care 1997;20:1647–1650.10.2337/diacare.20.11.1647 [PubMed: 9353601]
25. Radesky JS, Oken E, Rifas-Shiman SL, Kleinman KP, Rich-Edwards JW, Gillman MW. Diet during early pregnancy and development of gestational diabetes. Paediatric and Perinatal Epidemiology 2008;22:47–59. [PubMed: 18173784]
26. Marshall JA, Bessesen DH, Hamman RF. High saturated fat and low starch and fibre are associated with hyperinsulinaemia in a non-diabetic population: The San Luis Valley Diabetes Study. Diabetologia 1997;40:430–438.10.1007/s001250050697 [PubMed: 9112020]
27. Mendoza JA, Drewnowski A, Christakis DA. Dietary energy density is associated with obesity and the metabolic syndrome in U.S. adults. Diabetes Care 2007;30:974–979.10.2337/dc06-2188 [PubMed: 17229942]

28. Storlien LH, Baur LA, Kriketos AD, Pan DA, Cooney GJ, Jenkins AB, et al. Dietary fats and insulin action. Diabetologia 1996;39:621–631.10.1007/BF00418533 [PubMed: 8781757]
29. Meyer KA, Kushi LH, Jacobs DR Jr, Folsom AR. Dietary fat and incidence of type 2 diabetes in older Iowa women. Diabetes Care 2001;24:1528–1535.10.2337/diacare.24.9.1528 [PubMed: 11522694]
30. Simpson, C. Reaffirming our commitment to improving health services for Hispanics. Closing the gap: A newsletter of the Office of Minority Health. Vol. 3. U.S. Department of Health and Human Services; 1998.
31. Pawson IG, Martorell R, Mendoza FE. Prevalence of overweight and obesity in US Hispanic populations. The American Journal of Clinical Nutrition 1991;53:1522S–1528S. [PubMed: 2031482]
32. Kieffer EC, Carman WJ, Gillespie BW, Nolan GH, Worley SE, Guzman JR. Obesity and gestational diabetes among African-American women and Latinas in Detroit: Implications for disparities in women's health. Journal of the American Medical Women's Association 2001;56:181–187. 196.
33. Chasan-Taber L, Schmidt MD, Pekow P, Sternfeld B, Manson J, Markenson G. Correlates of physical activity in pregnancy among Latina women. Maternal and Child Health Journal 2007;11:353–363. [PubMed: 17345155]
34. Detjen MG, Nieto FJ, Trentham-Dietz A, Fleming M, Chasan-Taber L. Acculturation and cigarette smoking among pregnant Hispanic women residing in the United States. American Journal of Public Health 2007;97:2040–2047. [PubMed: 17901446]
35. Institute of Medicine, National Academy of Sciences. Nutrition during pregnancy. Washington, DC: National Academy Press; 1990.
36. Siega-Riz AM, Adair LS, Hobel CJ. Institute of Medicine maternal weight gain recommendations and pregnancy outcome in a predominantly Hispanic population. Obstetrics and Gynecology 1994;84:565–573. [PubMed: 8090394]
37. Saldana TM, Siega-Riz AM, Adair LS, Suchindran C. The relationship between pregnancy weight gain and glucose tolerance status among black and white women in central North Carolina. American Journal of Obstetrics and Gynecology 2006;195:1629–1635.10.1016/j.ajog.2006.05.017 [PubMed: 16824460]
38. Tucker KL, Bianchi LA, Maras J, Bermudez OI. Adaptation of a food frequency questionnaire to assess diets of Puerto Rican and non-Hispanic adults. American Journal of Epidemiology 1998;148:507–518. [PubMed: 9737563]
39. Bermudez OI, Velez-Carrasco W, Schaefer EJ, Tucker KL. Dietary and plasma lipid, lipoprotein, and apolipoprotein profiles among elderly Hispanics and non-Hispanics and their association with diabetes. The American Journal of Clinical Nutrition 2002;76:1214–1221. [PubMed: 12450885]
40. Willett, WC. Nutritional epidemiology. Vol. 2. New York, NY: Oxford University Press; 1998.
41. Foster-Powell K, Holt SH, Brand-Miller JC. International table of glycemic index and glycemic load values: 2002. The American Journal of Clinical Nutrition 2002;76:5–56. [PubMed: 12081815]
42. Phillips SM, Bandini LG, Naumova EN, Cyr H, Colclough S, Dietz WH, et al. Energy-dense snack food intake in adolescence: Longitudinal relationship to weight and fatness. Obesity Research 2004;12:461–472.10.1038/oby.2004.52 [PubMed: 15044663]
43. Schmidt MD, Pekow P, Freedson PS, Markenson G, Chasan-Taber L. Physical activity patterns during pregnancy in a diverse population of women. Journal of Women's Health 2006;15:909–918.10.1089/jwh.2006.15.909
44. National Heart, Lung and Blood Institute. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The evidence report. National Institute of Health; Bethesda, MD: 1998. Obesity education initiative, expert panel on the identification, evaluation, and treatment of overweight and obesity in adults. NIH publication No 98-4083
45. World Health Organization. Obesity: Preventing and managing the global epidemic. Report of the WHO consultation of obesity. Geneva: World Health Organization; 1997.
46. Institute of Medicine. Influence of pregnancy weight on maternal and child health: A workshop report. Washington, DC: National Research Council; 2007. p. 1-93.
47. Rudra CB, Sorensen TK, Leisenring WM, Dashow E, Williams MA. Weight characteristics and height in relation to risk of gestational diabetes mellitus. American Journal of Epidemiology 2007;165:302–308.10.1093/aje/kwk007 [PubMed: 17074967]

48. Dudhbhai M, Lim L, Bombard A, Juliard K, Meenakshi B, Trachelenberg Y, et al. Characteristics of patients with abnormal glucose challenge test and normal oral glucose tolerance test results: Comparison with normal and gestational diabetic patients. American Journal of Obstetrics and Gynecology 2006;194:e42–e45.10.1016/j.ajog.2005.11.031 [PubMed: 16647897]
49. Solomon CG, Willett WC, Carey VJ, Rich-Edwards J, Hunter DJ, Colditz GA, et al. A prospective study of pregravid determinants of gestational diabetes mellitus. Journal of the American Medical Association 1997;278:1078–1083.10.1001/jama.278.13.1078 [PubMed: 9315766]
50. Brennand EA, Dannenbaum D, Willows ND. Pregnancy outcomes of First Nations women in relation to pregravid weight and pregnancy weight gain. Journal of Obstetrics and Gynaecology Canada 2005;27:936–944. [PubMed: 16411008]
51. Wang Y, Storlien LH, Jenkins AB, Tapsell LC, Jin Y, Pan JF, et al. Dietary variables and glucose tolerance in pregnancy. Diabetes Care 2000;23:460–464.10.2337/diacare. 23.4.460 [PubMed: 10857935]
52. Feskens EJ, Virtanen SM, Rasanen L, Tuomilehto J, Stengard J, Pekkanen J. Dietary factors determining diabetes and impaired glucose tolerance. A 20-year follow-up of the Finnish and Dutch cohorts of the Seven Countries Study. Diabetes Care 1995;18:1104–1112.10.2337/diacare.18.8.1104 [PubMed: 7587845]
53. Bermudez OI, Falcon LM, Tucker KL. Intake and food sources of macronutrients among older Hispanic adults: Association with ethnicity, acculturation, and length of residence in the United States. Journal of the American Dietetic Association 2000;100:665–673.10.1016/S0002-8223(00) 00195-4 [PubMed: 10863569]
54. Moses RG, Luebcke M, Davis WS, Coleman KJ, Tapsell LC, Petocz P, et al. Effect of a low-glycemic-index diet during pregnancy on obstetric outcomes. The American Journal of Clinical Nutrition 2006;84:807–812. [PubMed: 17023707]
55. Kalkwarf HJ, Bell RC, Khoury JC, Gouge AL, Miodovnik M. Dietary fiber intakes and insulin requirements in pregnant women with type 1 diabetes. Journal of the American Dietetic Association 2001;101:305–310.10.1016/S0002-8223(01)00080-3 [PubMed: 11269608]
56. Olafsdottir AS, Skuladottir GV, Thorsdottir I, Hauksson A, Steingrimsdottir L. Maternal diet in early and late pregnancy in relation to weight gain. International Journal of Obesity (London) 2006;30:492–499.10.1038/sj.ijo.0803184
57. Oken E, Taveras EM, Kleinman KP, Rich-Edwards JW, Gillman MW. Gestational weight gain and child adiposity at age 3 years. American Journal of Obstetrics and Gynecology 2007;196:322.e1–8. [PubMed: 17403405]
58. Clapp JF 3rd, Seaward BL, Sleamaker RH, Hiser J. Maternal physiologic adaptations to early human pregnancy. American Journal of Obstetrics and Gynecology 1988;159:1456–1460. [PubMed: 3207124]
59. Forsum E, Sadurskis A, Wager J. Resting metabolic rate and body composition of healthy Swedish women during pregnancy. The American Journal of Clinical Nutrition 1988;47:942–947. [PubMed: 3376909]
60. Forsum E, Sadurskis A, Wager J. Estimation of body fat in healthy Swedish women during pregnancy and lactation. The American Journal of Clinical Nutrition 1989;50:465–473. [PubMed: 2773826]
61. Van Loan MD, Kopp LE, King JC, Wong WW, Mayclin PL. Fluid changes during pregnancy: Use of bioimpedance spectroscopy. Journal of Applied Physiology 1995;78:1037–1042. [PubMed: 7775296]
62. Clapp JF 3rd, Little KD. Effect of recreational exercise on pregnancy weight gain and subcutaneous fat deposition. Medicine and Science in Sports and Exercise 1995;27:170–177.10.1249/00005768-199502000-00004 [PubMed: 7723638]
63. Bodnar LM, Siega-Riz AM. A Diet Quality Index for Pregnancy detects variation in diet and differences by sociodemographic factors. Public Health Nutrition 2002;5:801–809.10.1079/PHN2002348 [PubMed: 12570888]
64. Margetts BM, Thompson RL, Speller V, McVey D. Factors which influence 'healthy' eating patterns: Results from the 1993 Health Education Authority health and lifestyle survey in England. Public Health Nutrition 1998;1:193–198. [PubMed: 10933418]

Table 1

Odds ratios for abnormal glucose tolerance according to participant characteristics

	Total population n = 813[a]		AGT cases n = 90[a]		Unadjusted odds ratios		Age-adjusted odds ratios	
	No.	%	No.	%	OR	95% CI	OR	95% CI
Age groups (years)								
15–19	289	35.6	16	17.8	1.0	Referent	n/a	n/a
20–24	304	37.4	32	35.6	2.0	1.1–3.7		
25–29	144	17.7	24	26.7	3.4	1.8–6.7		
30–39	76	9.4	18	20.0	5.3	2.6–11.0		
					P_{trend}	<.0001		
Education								
Less than high school	419	55.9	38	47.5	1.0	Referent	1.0	Referent
High school, trade/technical school	240	32.0	24	30.0	1.1	0.7–1.9	0.9	0.5–1.6
College graduate	91	12.1	18	22.5	2.5	1.3–4.6	1.5	0.8–2.9
					P_{trend}	<.01	P_{trend}	0.2
Birthplace								
U.S. born	411	55.0	42	52.5	1.0	Referent	1.0	Referent
Puerto-Rico or foreign-born	337	45.0	38	47.5	1.1	0.7–1.8	0.9	0.5–1.4
Language preference								
English only	540	67.3	55	61.8	1.0	Referent	1.0	Referent
English and Spanish	113	14.1	17	19.1	1.6	0.9–2.8	1.7	1.0–3.2
Spanish only	149	18.6	17	19.1	1.1	0.6–2.0	0.9	0.5–1.6
					P_{trend}	0.4	P_{trend}	0.9
Parity								
0	330	40.6	27	30.0	1.0	Referent	1.0	Referent
1	249	30.6	25	27.8	1.3	0.7–2.2	0.8	0.4–1.5
>2	234	28.8	38	42.2	2.2	1.3–3.7	0.9	0.5–1.7
					P_{trend}	<.01	P_{trend}	0.8
Family history of diabetes								
No	261	33.7	19	22.4	1.0	Referent	1.0	Referent
Yes	513	66.3	66	77.7	1.9	1.1–3.2	2.0	1.2–3.5

Matern Child Health J. Author manuscript; available in PMC 2009 July 1.

	Total population n = 813[a]		AGT cases n = 90[a]		Unadjusted odds ratios		Age-adjusted odds ratios	
	No.	%	No.	%	OR	95% CI	OR	95% CI
History of gestational diabetes								
No	781	96.2	82	91.1	1.0	Referent	1.0	Referent
Yes	31	3.9	8	8.9	3.0	1.3–6.8	2.2	0.9–5.5
Smoked during pregnancy								
No	602	79.3	76	91.6	1.0	Referent	1.0	Referent
Yes	157	20.7	7	8.4	0.3	0.1–0.7	0.4	0.2–0.8
Illicit drug use during pregnancy								
No	724	94.5	80	95.2	1.0	Referent	1.0	Referent
Yes	42	5.5	4	4.8	0.8	0.3–2.4	1.1	0.4–3.2

[a]Sample size varies due to missing data

Table 2

Characteristics of gestational weight gain & odds ratios of abnormal glucose tolerance according gestational weight gain

	Total population n = 813		AGT cases n = 90		Unadjusted odds ratios		Age-adjusted odds ratios	
	No.	%	No.	%	OR	95% CI	OR	95% CI
Prepregnancy BMI IOM categories (kg/m^2)								
<19.8	99	12.2	6	6.7	—		—	
19.8–26	375	46.1	27	30.0	1.0	Referent	1.0	Referent
26–29	112	13.8	16	17.8	2.1	1.1–4.1	1.8	0.9–3.6
>29	227	27.9	41	45.6	2.8	1.7–4.8	2.3	1.4–3.9
					P_{trend}	<.0001	P_{trend}	<.01
Prepregnancy BMI NHLBI/WHO categories (kg/m^2)								
<18.5	51	6.3	3	3.3	—			
18.5–24.9	365	44.9	23	25.6	1.0	Referent	1.0	Referent
25–29.9	201	24.7	27	30.0	2.3	1.3–4.1	1.9	1.1–3.5
30–34.9	112	13.8	18	20.0	2.8	1.5–5.5	2.4	1.2–4.7
35	84	10.3	19	21.1	4.3	2.2–8.4	3.4	1.7–6.8
					P_{trend}	<.0001	P_{trend}	<.001
Target weight								
Did not achieve	200	24.6	21	23.3	1.1	0.6–2.2	1.2	0.6–2.2
Met	295	36.3	30	33.3	1.0	Referent	1.0	Referent
Exceeded	318	39.1	39	43.3	1.1	0.7–1.9	1.1	0.7–1.9
					P_{trend}	0.5	P_{trend}	0.6
Rate of weight gain								
≤0.30 kg/week	406	49.9	49	54.0	1.0	Referent	1.0	Referent
>0.30 kg/week	407	50.0	41	46.0	0.8	0.5–1.3	0.8	0.5–1.3
Total wt. gain up to GDM screen kg. (mean, SD)	8.3	5.4	7.7	5.4				

Table 3

Odds ratios for abnormal glucose tolerance relative to gestational weight gain, stratified by prepregnancy BMI

BI/WHO categories (kg/m^2)[a]		Total N		Cases (n = 90)		Age-adjusted		Multivariate adjusted[b]		Rate of weight gain	Total N		Cases (n = 90)		Age-adjusted		Multivariate adjusted[b]	
	Target weight	No.	%	No.	%	OR	95% CI	OR	95% CI		No.	%	No.	%	OR	95% CI	OR	95% CI
eight (18.5–24.9	Did not achieve	100	27.4	9	40.0	1.8	0.6–5.5	1.6	0.5–5.3									
	Met	134	36.7	7	30.0	1.0	Referent	1.0	Referent	≤0.30 kg/week	157	36.0	12	*52.0*	1.0	Referent	1.0	Referent
	Exceeded	131	35.9	7	30.0	1.2	0.4–3.7	1.2	0.4–3.8	>0.30 kg/week	208	43.0	11	*48.0*	0.9	0.3–2.1	0.9	0.3–2.1
t (25–29.9 kg/m^2)	Did not achieve	37	18.4	3	12.0	0.3	0.07–1.7	0.4	0.07–1.8									
	Met	68	33.8	12	44.0	1.0	Referent	1.0	Referent	≤0.30 kg/week	99	49.3	14	*52.0*	1.0	Referent	1.0	Referent
	Exceeded	96	47.8	12	44.0	0.7	0.3–1.7	0.6	0.2–1.5	>0.30 kg/week	102	50.7	13	*48.0*	0.8	0.3–1.9	0.7	0.3–1.6
ss I (30–34.9 kg/	Did not achieve	20	17.9	4	22.0	1.5	0.3–7.5	1.9	0.4–9.6									
	Met	41	36.6	5	28.0	1.0	Referent	1.0	Referent	≤0.30 kg/week	72	64.3	11	*61.0*	1.0	Referent	1.0	Referent
	Exceeded	51	45.5	9	50.0	1.6	0.4–6.0	1.9	0.5–7.4	>0.30 kg/week	40	35.7	7	*39.0*	1.4	0.4–4.3	1.5	0.5–4.9
III Obese (≥35 kg/	Did not achieve	21	25.0	2	11.0	0.6	0.10–3.6	0.5	0.1–3.2									
	Met	36	42.8	6	32.0	1.0	Referent	1.0	Referent	≤0.30 kg/week	61	72.6	10	*53.0*	1.0	Referent	1.0	Referent
	Exceeded	27	32.1	11	58.0	3.8	1.0–13.4	4.2	1.1–16.0	>0.30 kg/week	23	27.4	9	*47.0*	3.0	1.0–9.4	3.2	1.0–10.5

category had insufficient cases for analysis

ted for age, smoking, history of GDM and family history of diabetes

Table 4

Diet characteristics by glucose status of 423 women with dietary data

	NGT n = 382 (mean ± SD)	AGT n = 41 (mean ± SD)	*P*-value*
Total energy (kcal)	2,444.8 ± 800.6	2,467.9 ± 693.2	0.86
Total fat, %	33.1 ± 4.7	33.1 ± 4.0	0.93
Polyunsaturated fat, %	8.9 ± 2.1	8.3 ± 1.7	0.06
Monousaturated fat, %	10.5 ± 1.8	10.5 ± 1.6	0.94
Saturated fat, %	11.2 ± 2.3	11.9 ± 2.1	0.04
P:S ratio	0.8 ± 0.3	0.7 ± 0.2	0.01
Protein, %	14.4 ± 2.2	15.0 ± 2.3	0.12
Carbohydrate, %	53.6 ± 6.2	53.0 ± 5.6	0.58
Fiber, g	17.8 ± 8.5	16.9 ± 5.8	0.57
Glycemic load	155.6 ± 54.3	151.6 ± 42.3	0.65
Energy-dense snack foods (kcal)	242.9 ± 220.2	269 ± 196.1	0.52

* *P* values from two tailed *t*-test

Table 5

Odds ratios for abnormal glucose tolerance according to dietary factors

Variable	Age adjusted		Age and total calorie adjusted		Fully adjusted model[a]	
	OR	95% CI	OR	95% CI	OR	95% CI
Polyunsaturated fat, %	0.9	0.7–1.0	0.9	0.7–1.0	0.8	0.7–1.0
Monounsaturated fat, %	1.1	0.9–1.3	1.1	0.9–1.3	1.2	0.9–1.5
Saturated fat, %	1.2	1.02–1.4	1.2	1.02–1.4	1.3	1.1–1.5
P:S ratio	0.2	0.05–0.77	0.2	0.04–0.8	0.1	0.02–0.45
Fiber, g	0.98	0.94–1.03	0.95	0.9–1.0	0.9	0.84–0.99
Glycemic load	1.0	0.99–1.01	1.0	.98–1.01	0.99	0.97–1.01
Fruits and vegetables						
Less than 4 servings per day	1.3	0.6–3.0	1.4	0.6–3.2	1.5	0.6–3.7
4–6 Servings a day	1.0	Referent	1.0	Referent	1.0	Referent
7 Servings or more per day	1.5	0.6–3.5	1.4	0.6–3.5	1.7	0.7–4.5
Energy-dense snack (EDS) foods						
Low EDS (<100 calories/day)	0.5	0.2–1.2	0.5	0.2–1.2	0.4	0.1–1.0
Average EDS (100–400 calories/day)	1.0	Referent	1.0	Referent	1.0	Referent
High EDS (>400 calories/day)	1.9	0.8–4.5	2.1	0.85–5.0	2.3	0.9–6.3

[a]Models are adjusted for total calories, pre-pregnancy BMI, age, history of GDM, family history of diabetes, parity, gestational weight gain up to time of GDM screen, smoking and physical activity