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Dietary behaviors and glucose metabolism in young adults at risk for type 2 diabetes

EunSeok Cha, PhD, RN, MPH, MSN, CDE, FAHA[a,b], **Sudeshna Paul, PhD**[b], **Betty J Braxter, PhD, CNM, RN**[c], **Guillermo Umpierrez, MD, CDE, FACP, FACE**[d], and **Melissa Spezia Faulkner, PhD, RN, FAAN**[b,e]

[a]College of Nursing, Chungnam National University, Daejeon, South Korea.

[b.] Nell Hodgson Woodruff School of Nursing, Emory University, Atlanta, USA.

[c.] School of Nursing, University of Pittsburgh, Pittsburgh, USA

[d.] School of Medicine, Emory University, Atlanta, USA

[e.] Byrdine F. Lewis School of Nursing and Health Professions, Georgia State University, Atlanta, USA

Abstract

Purpose: The purpose of the study was to examine the associations between dietary behaviors and glucose metabolism in high-risk young adults to increase the precision of nutrition education to prevent early onset type 2 diabetes (T2D).

Method: Using a descriptive, cross-sectional study design, 106 overweight/obese sedentary young adults ages 18–29 from the metro Atlanta area were recruited to screen diabetes risk. Survey questionnaires, anthropometric assessment, blood pressure, and laboratory data were collected in a clinical research unit. Web–based HOMA2 calculator calculated beta-cell function and insulin sensitivity.

Results: The final sample included 103 subjects. There were similar patterns of diet (caloric intake and dietary quality) between African Americans [AA] and non-AA while AA showed hyperinsulinemia compared to the non-AA. When young adults consumed good quality diet (appropriate carbohydrate intakes; high fiber, low saturated fat but protein rich diet), their insulin resistance was decreased. There was a marginal interaction effect between insulin sensitivity and beta cell function by race. Systolic blood pressure was higher in AA and total cholesterol, triglycerides and LDL-c were higher in non-AA.

Conclusion: Findings are useful to develop age specific nutrition guidelines to prevent early onset T2D in high-risk young adults.

Correspondence: concerning this article should be addressed to EunSeok Cha. Chungnam National University College of Nursing, 266 Munwharo Jungu Daejeon, Korea, 35015, Phone: 82-42-580-8319, Fax: 82-42-580-8329, echa5@cnu.ac.kr or echa5@emory.edu.

Conflict of Interests

None

Keywords

Young adults; Diabetes prevention; Nutrition counseling; Insulin resistance; Insulin sensitivity

Type 2 diabetes (T2D) is a disorder of altered glucose metabolism progressing from normoglycemia to prediabetes and to diabetes.[1] Decreased insulin sensitivity and impaired insulin secretion are key factors mediating the transition.[1, 2] Aging and genetic proclivity are significant contributing factors to altered glucose metabolism.[3–5] Also, obesity related lifestyle such as dietary behaviors, sleep, and physical activity influences insulin sensitivity and insulin secretion. [6–8]

Insulin sensitivity refers to the body's insulin-mediated glucose utilization in peripheral tissues. [9, 10] Under a close interaction between insulin sensitivity and insulin secretory capacity, the glucose concentration is tightly balanced in healthy persons; decreased insulin sensitivity reduces glucose disposal in peripheral tissues resulting in hyperinsulinemia to maintain normoglycemia, which is often called as insulin resistance.[11, 12] When the insulin secretory capacity does not appropriately respond to the body's insulin needs and/or existing insulin does not work appropriately, prediabetes and subsequently T2D occur. [11, 12]

Previous reports show that African Americans adults have lower insulin sensitivity, thereby higher insulin concentration compared to Caucasians. [10, 13, 14] A recent meta-analysis identified racial/ethnic differences in insulin secretion and insulin sensitivity in healthy Caucasians, African Americans and East Asians. [2] In the description of the hyperbolic relationship between insulin sensitivity and insulin response, Caucasians were in the middle between African Americans who were found to have low insulin sensitivity and high insulin secretion, and East Asians who had high insulin sensitivity and low insulin secretion. These variations were present in individuals with normoglycemia and continued in persons with prediabetes and T2D. [2]

Young adults are reported to frequently skip meals and be binge eaters compared to older cohorts. [15] This behavior makes young adults vulnerable to glucose spikes and hyperinsulinemia. Overweight and obese young adults with unhealthy dietary habits, particularly, have greater risk for developing weight gain and early onset T2D compared to individuals with good nutritional intake.[15, 16] Unhealthy food consumptions (e.g., added sugar and processed foods) produce insulin spikes in response to rapid influx of sugar. [17–19] Adipokines (bioactive products secreted by adipose tissues) change the body's metabolism leading to insulin resistance.[20] Increased fat mass increases circulating triglyceride and free fatty acids levels, which reduces insulin sensitivity. [13] Recent research shows that sustained hyperinsulinemia increases the risk for beta cell exhaustion and cardiometabolic diseases that may in part explain health disparities in T2D patients. [4, 13, 21]

The purpose of this study was to examine the associations between dietary behaviors and glucose metabolism in overweight and obese young adults in order to increase the precision of nutrition education to prevent early onset T2D. Specifically, the potential impact of dietary behaviors including calorie intake and dietary quality on: 1) glucose metabolism, insulin sensitivity and insulin secretion (beta cell function); and 2) the development of

prediabetes in overweight and obese young adults was explored. The contributions of metabolic risk factors including hypertension, dyslipidemia, and inflammatory factors (C-reactive protein) to increase diabetes risk were also analyzed as a part of specific aim 2. Differences in race/ethnicity and gender were examined in each model to explore population specific priorities for diabetes prevention program in overweight and obese young adults.

Methods

Study design:

A descriptive cross-sectional study design was used.

Procedure and participants:

Prior to the study, IRB approvals from the appropriate institutions and informed consents from the eligible participants were obtained. Community dwelling young adults aged 18–29 years and students attending colleges in the Atlanta Metro area, USA, were recruited using diverse methods including: recruitment flyers on community bulletin boards, e-mail invitations using a university's student mailing list obtained with permission from the university, and through peers and self-referral for screening of prediabetes and T2D. Inclusion criteria included being overweight/obese (BMI ≥ 25) and having a sedentary lifestyle with a self-reported average leisure time physical activity < 90 minute per week during the past 3 months. Individuals diagnosed with diabetes, cardiovascular diseases, serious illness and unstable conditions requiring physician-supervised dietary and exercise regimens, conditions affecting erythrocyte turnover (e.g., hemolysis, blood loss), or pregnancy were excluded. After a phone screening, eligible young adults were invited to a diabetes screening test in a Clinical Research Unit (CRN) affiliated with the Atlanta Clinical and Translational Science Institute.

Variables and measures:

Self-administered survey questionnaires, anthropometric assessment, blood pressure, and laboratory data were collected.

Socio-demographics: A self-reported questionnaire was used to obtain information regarding race/ethnicity, years of school, age, gender, smoking habits and an average of hours of sleep per night during the past month.

Dietary Behavior: Youth/Adolescent Food Frequency Questionnaire (YFQ) was collected when participants visited the CRN. Based on YFQ responses, [22] nutrient components along with calorie intake, type of calories, and serving sizes were calculated. Then, the dietary quality was quantified as a score of the modified Dietary Quality Index Revised for Young adults (mDQIR-Y), a valid measure of dietary quality. [23–25] The range of a score in the DQIR-Y was 0 (poorest diet quality) to 95 (best diet quality). The detailed information about how to calculate mDQIR-Y score based on the *2010 Dietary Guidelines for Americans* was previously described.[19, 23]

Anthropometric assessment: The research team and nurses in the CRN were trained on anthropometric measures and data collection. Height was measured using a wall-mounted stadiometer and weight was measured using a calibrated scale while participants wore light clothing and stood erect with bare feet. Then, body mass index (BMI) was calculated using weight (Kg) and height (cm) (Kg/m^2). Waist circumference was measured twice at the level of umbilicus with a Gulick II measuring tape while a participant stood upright; the two values should be within 1 inch of each other. Participants were asked to stand with their feet together, place their arms at their side with the palms of their hands facing inward, and breathe out gently during the assessment. For the study, we used an average of two waist circumference readings.

Blood pressure: Resting blood pressure was assessed following the American Heart Association guidelines. [26] Participants were seated in a chair with feet on the floor after at least a 5-minute rest and 30-minute abstention from smoking and caffeine. The arm was supported and free of tight, constricting clothing, and the cuff was level with the heart. When the arm circumference exceeded 33 cm, a large cuff was used. [26] If a participant showed an elevated blood (BP) pressure either systolic BP ≥120 mm Hg or diastolic BP ≥80 mm Hg, blood pressure was measured in the other arm. Then, an average of the two readings was calculated.

Laboratory data: Participants were asked to fast at least 8 hours before the CRN visit. Fasting insulin and glucose, A1C, lipids (total cholesterol, triglycerides, HDL-C, and LDL-C), fasting nonesterified fatty acids (NEFA), and C-reactive protein were collected, kept on ice and plasma separated within 1 hour of collection.

Insulin resistance was calculated by homeostasis model of assessment method (HOMA-IR) and adipose tissue insulin resistance [27–29]. The HOMA-IR formula was calculated with the formula: fasting insulin (μU/mL) X fasting glucose (mg/dL) / 405. [29] The adipose tissue insulin resistance was assessed with the equation: fasting nonesterified fatty acids [NEFAs] X fasting insulin. [28]

Insulin sensitivity and beta cell function: The Web–based HOMA 2 calculator was used to calculate beta-cell function and insulin sensitivity (%S) by the Homeostasis Model Assessment (HOMA-B) formula (https://www.dtu.ox.ac.uk/homacalculator/).30–32 HOMA 2 is a mathematical feedback model allowing researchers to estimate beta cell function and insulin resistance without actual insulin assays such as hyperinsulinaemic clamp, the hyperglycaemic clamp, the intravenous glucose tolerance test, and the oral glucose tolerance test. It has been developed in 1976 and diabetes experts acknowledge the usage of the model. [30–32]

Normoglycemia was defined by a fasting glucose < 100mg/dL (5.6mmol/L) and A1C (< 5.7%). Prediabetes was defined by a fasting glucose between100 and 125mg/dL (5.6 and 6.9 mmol/L) or A1C between 5.7% and 6.4%. Type 2 diabetes was defined as a fasting glucose greater than 126 mg/dL (7.0mmol/L) and A1C > 6.5%.

Data Analysis:

Descriptive statistics such as mean, standard deviation, frequencies and percentages were computed in socio-demographic variables, obesity and dietary behaviors, glucose metabolism and metabolic risk factors for the overall sample and sub-groups. Comparisons on the study variables between ethnic groups, normoglycemia and prediabetes groups were performed.

To identify the impact of dietary factors on glucose metabolism (Specific aim 1), linear regression analyses were used; Our main outcomes of interest were insulin sensitivity and beta cell function. For any non-normal outcomes, appropriate transformations were used to satisfy the normal distributional assumption. All dietary factors related to diabetes proved by previous literature (i.e., carbohydrate, protein, fat, saturated fat, unsaturated fat, vitamin D, calcium, added sugar, sodium, fiber) were included as predictors of outcomes of interest in the full model and age and BMI were adjusted. To test for racial differences, we included race and its interactions with dietary factors in the model.

To answer specific aim 2, a logistic regression was fit to identify the predictors of prediabetes. The most parsimonious model was chosen based on stepwise selection and using relative measures such as Akaike information criterion or Bayesian information criterion.[33] A p-value of 0.2 was used as a threshold probability for including or discarding predictors in the stepwise selection method. We used confidence intervals as well as odds ratios to demonstrate strength of relationships between the outcomes and the independent variables because the study included many independent variables (dietary factors, demographics) and a relatively smaller sample size; hypothesis testing was not always meaningful due to insufficient statistical power. Unless noted otherwise, significance level was set at .05 for this study. Statistical analysis was performed using SAS (version 9.2; SAS Institute, Cary, NC) and SPSS 23.0

Results

Socio-demographics.

A total of 106 young adults participated in the diabetes screening; three participants, however, were excluded because one participant had asymptomatic type 2 diabetes and two participants had incomplete data. Therefore, the final data analysis was done with 103 participants only.

The mean age was 24.0± 3.2 years, and mean BMI was 36.6 ± 8.0. The majority of study participants were female (78.6%), African Americans (67.0%), severely obese (BMI >35 kg/m^2=44.7%) and physically inactive (less than 10 METs-h/week =65.0%). About 82% were college/graduates (17.3% were below high school graduate) (See Table 1).

Group differences on study variables.

Overall, dietary quality was fair (mean score 62.49 out of 95) while calorie intake met the 2010 dietary recommendations for Americans (mean ± SD= 1755.5±588 Kcal/day; median: 1671.6 Kcal/day). There were no significant differences in the calorie intake by ethnic

groups. Non-African Americans consumed more caffeine than African Americans, but other nutrients showed no group differences. African Americans consumed lower dietary fiber, magnesium, Vitamin D, calcium and higher added sugar, which showed similar patterns between normoglycemia and prediabetes groups (see Table 2). In this study, calorie intake was positively correlated with dietary quality regardless of racial groups (r=0.53, P<.001 and r=.51, P<.001, respectively).

Fasting glucose was marginally lower (p=.069) in African Americans, while they showed higher fasting insulin and beta cell function (p=.081) compared to non-African Americans. The insulin resistance measures (HOMA-IR, and adipose IR) were similar in African Americans non-African Americans groups. Regarding comparisons between prediabetes and normoglycemic groups, fasting insulin and glucose were higher in prediabetes groups. The degree of beta cell function, however, was almost the same (P=.711). A glucose to insulin ratio was significantly lower in the prediabetes compared to normoglycemic group referring to insulin resistance.

African Americans had significantly higher systolic BP (P=0.032), lower total cholesterol (P=0.004) and triglycerides levels (P<.001) compared to non-African Americans. However, these differences were not found when we compared the values between the normoglycemia and prediabetes groups. Rather, the prediabetes group showed a marginally higher triglyceride than normoglycemia group (P=0.096).

Dietary impact on insulin sensitivity in overweight and obese young adults.

Overweight/obesity measured by body mass index (BMI) was a major contributor of lower insulin sensitivity. The factors increasing in insulin sensitivity were lower calorie intake, including appropriate carbohydrate and protein consumption, which explain 21.6% of the final model. In the model, race/ethnicity was not a statistically significant predictor of insulin sensitivity (See Table 3).

Predictors of beta cell function in overweight/obese young adults

African Americans showed higher beta cell function compared to non-African Americans (Table 4) and is consistent with previous research.[34] Higher beta cell function was also observed in young adults with higher BMI. When insulin sensitivity was higher, the excessive beta cell function was prevented. Interestingly, beta cell function was decreased in persons with greater calorie intake, which may imply a threshold for beta cell function.

In overweight and obese young adults, higher calorie intake was associated with a better-quality diet (r=.54, P<.001). There was a marginal interaction effect between insulin sensitivity and beta cell function by race (P=.064); the rate of an inverse relationship between insulin sensitivity and insulin secretion (herein presented by beta cell function) was higher in the non- African American compared to the African American group. The total variance in beta-cell function explained by the model was 35.9% (see Table 4).

Predictors of prediabetes

Development of prediabetes was significantly associated with lower beta cell function and lower insulin sensitivity although odd ratios were very small (Table 5). Metabolic risk factors such as elevated blood pressure or dyslipidemia, which are often considered co-existing conditions and risk factors for developing type 2 diabetes, did not predict prediabetes in overweight and obese young adults. Also, calorie intake and dietary quality were not directly related to prediabetes in this population.

Discussion

There is awareness of the potential harms of hyperinsulinemia and insulin resistance. [3, 9, 35]; however, a paucity of research exists regarding how hyperinsulinemia contributes to diabetes progression and how to effectively prevent the hyperinsulinemic state in at risk young adults. [8] Findings in this study also reveal the associations of higher fasting insulin and insulin resistance in those with prediabetes, but causation cannot be determined. The study adds scientific evidence for how dietary behavior effects glucose metabolism and how age-specific nutrition education, especially targeting overweight and obese young adults for prevention of early onset T2D, needs to be tailored.

The Treatment Options for Type 2 Diabetes in Adolescents and Youth (TODAY) study showed that maximum preservation of beta cell function before significant loss is essential to delay T2D progression and attain glycemic goals, preventing diabetes complications. [36, 37] Also, recent research found that insulin resistance and the resulting prolonged increased secretion of insulin can lead to early beta cell exhaustion later in life. [2, 10, 21] In our cross-sectional study, dietary behaviors (calorie intake and dietary quality) were influencing factors for progression to prediabetes, although they could not be deemed causative or predictive. Inverse relationships were noted between calorie intake and insulin sensitivity and beta-cell function and insulin sensitivity, respectively.

The higher beta cell function observed by study participants with the most elevated BMI and who were African Americans indicates a compensatory response for heightened insulin resistance that may eventually lead to beta cell exhaustion, the development of prediabetes and eventually T2D. The positive relationship between calorie intake and diet quality supports the importance of educating at risk young adults to avoid only decreasing calorie intake to promote improved metabolic health, but rather to ensure adequate caloric intake that includes a balanced diet from good nutritional sources. Future research questions need to address how to: 1) improve young adults' health literacy to have adequate calorie intake (i.e., serving size calculation) [38]; 2) encourage them to have a balanced and optimal quality diet despite widespread dietary myths (e.g., low-carb, high-fat diet benefits preventing weight gain and diabetes); and 3) prevent their binge eating habits. [15, 19, 39] Age specific social marketing strategies should be considered to enhance behavioral change.

Our findings are particularly important for overweight and obese young women within our study age range who are in a need of preconceptual lifestyle modification counseling but limited research is available. [40, 41] During the pregnancy, all women experience alternations in glucose and lipid metabolisms as a part of normal pregnancy. [42–44] Recent research shows

that overweight and obese pregnant women may experience different trajectory in these alternations [45]; pregestational obesity, dyslipidemia or metabolic syndrome increases the risk for pregnancy complications and adverse outcomes such as pregnancy induced hypertension, preeclampsia, gestational diabetes, later life cardiovascular diseases, preterm birth, and large for gestational age neonates. [44–46] Questions remain whether :1) mild metabolic abnormal conditions (e.g., prehypertension, hyperinsulinemia, or prediabetes) but not a disease diagnosed yet (e.g., hypertension, diabetes) in presentational phase still increases the risk for developing adverse pregnancy outcomes; 2) maternal hyperinsulinemia in preconceptual phase increases the risk for gestational diabetes[47] ; and 3) preconceptual nutrition modification (i.e., a good quality diet with adequate calorie) can prevent adverse pregnancy outcomes.[48] A future research should be followed in these areas.

The findings of the current study revisited a need of differentiation on nutrition counseling between early stage (prevention) and late stage (diabetes management) which many lay persons are confused. As our findings show, total consumption of carbohydrate (% of total calories) does not directly affect serum glucose level in young adults with normoglycemia or prediabetes since glucose regulation feedback loop is working well. Rather, the strict reduction of carbohydrate intake often significantly increases saturated fat and decreases dietary fiber consumption, which increases the risk for insulin resistance and impairment in insulin sensitivity.[49] For individuals with T2D, meticulous monitoring of carbohydrate, a direct factor to increase serum glucose level, necessitates the reduction of glucose variation and the achievement of optimal glycemic control. [50] A modified nutrition education accompanied by disease progression is essential in diabetes care.

We acknowledge the study limitation of using a cross-sectional study design and the use of an indirect web-based calculator to estimate beta cell function and insulin sensitivity, despite the validity of this measure. [30–32] We also acknowledge limited accuracy in evaluating dietary impact based on self-reported of dietary and physical activity behaviors. Finally, the values of HOMA-IR were almost identical in African Americans and non-African Americans groups in spite of African Americans having higher fasting insulin and lower fasting glucose. These results raise questions on the clinical application of HOMA-IR assessing insulin resistance. Although it has been shown that hyperinsulinemia is independent of insulin resistance, HOMA-IR may be a useful tool when measured longitudinally rather than cross-sectionally. A future prospective, longitudinal population-based study with an objective dietary measure using advanced technology (e.g., mobile apps for recording real-time dietary intake and analyses) is warranted.

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Table 1.

Socio-demographics by race/ethnicity or prediabetes conditions (N=103)

	Non-AA (n=34)	AA (n=69)	*P*-value	Normo-glycemia (n=71)	Prediabetes (n=32)	*P*-value
Social demographics						
Male	13(59.1%)	9(40.9%)	.005**	16 (72.7%)	6 (27.3%)	.797
Female	21(25.9%)	60(74.1)		55 (67.9%)	26(32.1%)	
Age (years)	24.5±3.3	23.8±3.2	.274	23.7±3.1	24.6±3.5	.229
Self-reported sleep hours	6.9±1.2	6.3±1.3	.050	6.5±1.3	6.6±1.4	.649
Median of physical activity (MET-h/wk)	5.2	6.1	.123	6.8	2.2	.013*
Obesity/visceral obesity						
BMI	33.5±6.3	38.2±8.4	.002**	35.7±7.7	38.9±8.5	.064
Waist Circumference (inch)	40.4±5.8	42.7±7.1	.102	41.5±6.5	43.1±7.3	.274

* *P*<.05

** *P*<.01

AA: African Americans

Table 2.

Comparisons on dietary behaviors and clinical variables by race/ethnicity (African Americans [AA] vs. non-African Americans) and prediabetes (N=103)

	Non-AA (n=34)	AA (n=69)	*P*-value	NG (n=71)	pDM (n=32)	*P*-value
Dietary behaviors						
Calories (Kcal)	1773.0±591.8	1746.9±590.7	.833	1750.8±567.5	1766.0±641.3	.904
Dietary Quality	62.8±11.0	62.0±11.5	.739	62.8±11.8	61.1±10.3	.477
% of carb of total calorie intake	53.5±7.3	53.9±5.4	.748	53.6±6.0	54.2±6.1	.619
% of protein of total calorie intake	16.3±3.5	15.5±3.1	.229	16.1±3.3	15.1±209	.161
% of total fat of total calorie intake	30.9±4.6	31.2±5.2	.759	30. 9±5.2	31.5±4.7	.632
% of saturated fat of total calorie intake	10.6±2.1	10.3±2.3	.500	10.3±2.2	10.5±2.3	.573
Dietary fiber (gm)	18.3±8.9	16.1±7.0	.179	17.3±8.1	15.8±6.9	.388
Added sugar (gm)	55.4±31.6	59.8±26.4	.459	55.3±25.8	65.1±32.2	.104
Total sugar (gm)	100.3±49.1	107.2±41.2	.453	102.4±43.7	110.6±44.4	.385
Sodium (mg)	2186.0±847.4	2058.0±774.9	.447	2054.5±776.2	2202.0±847.0	.388
Magnesium (mg)	263.9±102.1	242.36±92.8	.287	257.5±100.7	231.6±83.4	.206
Vitamin D (IU)	194.6±157.2	195.7±179.8	.975	204.0±180.1	176.1±153.3	.449
Calcium (mg)	777.8±327.0	742.0±324.1	.601	774.3±350.4	708.3±257.7	.341
Cholesterol (mg)	216.8±111.4	231.7±104.4	.508	229.4±102.1	220.9±117.0	.590
Alcohol (mg)	2.08±2.4	2.0±2.4	.879	2.12±2.5	1.8±2.0	.712
Caffeine (mg)	65.2±49.1	30.5±30.7	<.001***	42.9±41.6	39.8±40.1	.728
Glucose metabolism						
Fasting insulin (pmol/L)	119.2±52.0	118.3±9.5	.398	116.4±60.2	149.8±64.5	.012*
Fasting glucose (mg/dL)	88.3±10.4	84.3±10.4	.069	83.1±8.6	91.2±12.0	<.001***
A1C (%)	5.3±0.4	5.5±0.4	.164	5.2±0.3	5.9±0.3	<.001***
Beta cell function (%)	166.8±59.2	194.4±81.4	.081	183.4±71.5	189.4±85.1	.711
Insulin sensitivity (%)	62.3±45.5	57.0±33.0	.505	65.1±42.0	44.61±18.2	.001**
Ratio of glucose to insulin	6.8±4.1	5.9±3.1	.230	6.6±3.8	5.2±2.1	.015*
Insulin resistance						
- HOMA-IR	3.7±1.7	3.8±2.1	.672	3.4±1.8	4.7±1.9	.001**
- Adipose IR	8.1±4.6	10.2±10.6	.269	9.2±10.4	10.4±5.3	.542
Metabolic risk factors						
Systolic blood pressure (mmHg)	114.7±14.5	121.8±16.1	.032**	119.9±15.5	118.3±16.9	.650
Diastolic blood pressure (mmHg)	71.7±8.0	73.5±11.2	.403	72.9±8.5	72.9±13.5	.988
Total cholesterol (mg/dL)	172.3±34.4	151.6±32.7	.004**	155.7±36.0	164.5±30.6	.231
Triglyceride (mg/dL)	126.8±73.6	73.3±34.4	<.001***	84.1±43.5	104.2±77.4	.096
HDL-C (mg/dL)	42.2±8.8	42.8±9.1	.744	42.7±9.0	42.5±9.0	.926
LDL-C (mg/dL)	113.9±30.8	97.4±29.8	.010*	101.1±32.6	106.7±27.3	.404
Nonesterified fatty acids (NEFA)	.499±.167	.560±.418	.412	.554±.407	.510±.201	.564
C-reactive protein	4.7±5.1	6.5±7.6	.201	5.5±6.7	6.7±7.4	.422

* $P<.05$;

** $P<.01$;

*** $P<.001$

NG refers to the normoglycemic group while pDM refers to the prediabetes group.

Table 3:

Predictors of (log) insulin sensitivity in overweight and obese young adults without diabetes (N = 103)

Parameters	B	SE	Beta	P-value	95% CI	
					Lower bound	Upper bound
†Non-African Americans (vs. AA)	−1.32	.79	−1.22	.099	−2.885	.252
Calorie intake *	−.001	.001	−1.36	.034	−.002	−.000
Age	.01	.02	.08	.394	−.017	.042
Dietary Quality	−.01	.008	−.23	.191	−.026	.005
Carbohydrate intake	.006	.003	1.00	.054	.000	.012
Dietary fiber	.01	.01	.17	.351	−.012	.034
Protein intake *	.01	.006	.62	.036	.001	.024
Body mass index (BMI) ***	−.03	.007	−.42	<.001	−.039	−.013
Race/ethnicity (Non AA) X dietary quality	.01	.009	.70	.194	−.006	.030
Race/ethnicity X BMI	.01	.02	.42	.369	−.016	.042

† AA refers to African Americans.

‡ R^2 Squared = .293 (Adjusted R^2 = .216).

* P<.05 ;

** P<.01;

*** P<.001.

Table 4.

Predictors of beta cell function in overweight and obese young adults without diabetes (N = 103)

Parameters	***B***	***SE***	***Beta***	***P-value***	***95% CI***	
					Lower bound	***Upper bound***
Non-African Americans *	−49.20	24.59	−.31	.048	−98.02,	−0.38
Insulin sensitivity ***	−1.20	.25	−.59	<.001	−1.68,	−0.71
BMI	1.53	.85	.16	.073	−0.15,	3.21
Age **	−5.21	1.96	−.22	.009	−9.09,	−1.32
Calorie intake *	−.03	.01	−.21	.030	−.05,	−.003
Dietary quality	.77	.66	.12	.247	−.54,	2.08
Race/ethnicity X Insulin Sensitivity	.63	.33	.32	.064	−.04,	1.29

R^2 = .403 (Adjusted R^2 = .359).

Reference group for the race ethnicity is African Americans.

* *P*<.05;

** *P*<.01;

*** *P*<.001.

Table 5:

Predictors of prediabetes in overweight and obese young adults. (N = 103)

Parameters	*B*	*S.E.*	*Odds ratio (OR)*	*95% CI for OR*	
				Lower	*Upper*
Ethnicity (African Americans)	.71	.69	2.04	.53	7.86
Beta cell *	−.01	.005	.99	.98	1.00
Insulin sensitivity **	−.06	.02	.94	.90	.98
Calorie intake	.00	.001	1.00	.99	1.00
Dietary quality	.00	.03	1.00	.95	1.05
Total cholesterol	.06	.04	1.06	.99	1.14
Triglyceride	.002	.008	1.00	.99	1.02
HDL	−.06	.05	.95	.85	1.05
LDL	−.06	.04	.95	.88	1.01
SBP	−.02	.02	.98	.93	1.02
DBP	−.005	.03	1.00	.94	1.06
C-reactive protein	−.01	.04	.99	.91	1.07
Free fatty acid (NEFA)	.70	1.28	2.02	.16	24.83
Age	.28	.82	1.32	.26	6.64
Constant	4.96	3.83	142.24		

* $P<.05$;

** $P<.01$.