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Effect of Glycemic Load on Peptide-YY Levels in a Biracial Sample of Obese and Normal Weight Women

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Abstract

Black women suffer a disproportionately higher rate of obesity than their white counterparts. Reasons for this racial disparity may reflect underlying differences in the appetite suppressing peptide-YY (PYY). The PYY response to food is differentially influenced by macronutrient content but the effect of glycemic load on PYY response is unknown. This study examined whether glycemic load influences fasting and postprandial PYY levels and whether fasting and postprandial PYY levels are lower in obese black women compared to normal weight black women and to white women. Data were collected from 40 women (20 black, 20 white; 10 each normal weight vs. obese) at the University of North Carolina Clinical and Translational Research Center (CTRC). Participants completed in counterbalanced order two 4½-day weight-maintenance, mixed macronutrient high vs. low glycemic load diets followed by a test meal of identical composition. Total PYY levels were assessed before and after each test meal. Results show no differences in fasting PYY levels but significantly less postprandial PYY area under the curve (PYY_{AUC}) in the group of obese black women compared to each other group (race × obesity interaction, $P < 0.04$). PYY_{AUC} was positively related to insulin sensitivity ($P < 0.004$) but was not affected by glycemic load (main and interactive effects, $P > 0.27$). These findings indicate that postprandial PYY secretion is not affected by glycemic load but is blunted in obese black women compared with normal weight black women and with white women; additionally, they begin to address whether blunted PYY secretion contributes uniquely to the pathogenesis of obesity in black women.

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Disclosure

The authors declared no conflict of interest.

INTRODUCTION

Race and obesity

Nearly 65% of US adults are overweight, >30% are obese and the prevalence of obesity is on the rise (1). Non-Hispanic blacks are disproportionately affected, with black women suffering the highest rates of obesity (~50%) overall (2). Compared with white women, black women gain weight at an earlier age, lose less weight with traditional diet and exercise modification, and are especially vulnerable to obesity-related cardiovascular and metabolic complications such as hypertension and diabetes (3–5). Reasons for increased risk of obesity and its comorbid conditions in black women are not fully understood; however, recent work by our group (6) and others (7,8) suggests differences in gastrointestinal peptides involved in short-term appetite regulation may play a role.

PYY and obesity

Peptide-YY (PYY) is a naturally occurring peptide secreted in the distal gastrointestinal tract in response to food intake (9–11). In circulation, PYY is found in two main forms, PYY3–36 and PYY1–36. PYY3–36 is the predominant circulating form and has been shown more clearly to affect food intake and appetite in humans by directly contributing to the achievement and maintenance of satiety during the intermeal period. There is keen interest in identifying factors that regulate PYY levels in the acute postprandial window and in understanding how individual differences in circulating PYY levels contribute to food consumption patterns and weight gain. Recent findings suggest that weight/metabolic status (12–15), dietary (and test meal) macronutrient composition (16–18), sex (19), and race (7,8) may be important factors to consider. For example, exogenous PYY3–36 infusions reduce food intake and ratings of subjective appetite independent of obesity status; yet the endogenous PYY response is diminished in obese compared with normal weight individuals (12,13). In obese subjects, greater PYY3–36 and total PYY (PYY1–36 and PYY3–36) responses have been observed following test meals with relatively higher protein (16,19,20) and fat (16,19) than carbohydrate content in some but not all (17) studies. A diminished nutrient-stimulated total PYY response has also been reported in women relative to men (19) and in blacks compared with whites (7,8). Notably, the findings of diminished nutrient-stimulated total PYY in blacks have been limited to liquid glucose (8) and fat (7) loads, leaving open the question of race differences in endogenous PYY response to mixed nutrient, whole food meals. Also, unknown is whether race differences in PYY vary as a function of obesity.

Glycemic index reflects the direct effect of a given food at a given portion on blood glucose levels: a higher index indicates a higher blood glucose response. Glycemic load is a method of ranking foods based on their carbohydrate content, glycemic index, and portion size. Consumption of a low glycemic load diet is purported to reduce appetite, and several studies have demonstrated that subjective appetite suppression is sustained longer following consumption of a meal with a low vs. a high glycemic load in obese individuals (21–23). The mechanisms linking low glycemic load with reduced appetite are not clear but may include alterations in appetitive hormones. Prior studies suggest glycemic load-dependent effects on ghrelin (24), and on glucagon-like peptide-1 and cholecystokinin (25,26). The

impact of glycemic load on postprandial PYY response is unknown. Therefore, in this study, we assessed fasting and postprandial PYY after ingestion of a high vs. low glycemic load mixed-macronutrient, high-carbohydrate meal in normal weight and obese black and white women.

Methods and Procedures

Subjects

Individuals were eligible to participate in the study if they were female, age 18 or older, and non-Hispanic black or white race. Race was assessed by self-report using a two-tiered questionnaire in which respondents first indicated their ethnicity (Hispanic, non-Hispanic) and then their race (white, black, or African American, Asian, Native Hawaiian, or other Pacific Islander, American Indian/Alaska Native). Only women who self-identified as non-Hispanic black or non-Hispanic white were included in the study. Participants were recruited from the local community; most (35) were born in the United States (two each black and white were foreign born; one unknown). Women who were pregnant, planning to get pregnant in the next 6 weeks, or lactating; had existing diabetes or reported any other metabolic disorder; were currently using medications that influenced appetite or that had significant weight gain or loss side effects; were underweight BMI <18.5, overweight (BMI = 25–29.9) or morbidly obese (BMI >40); were unable or unwilling to eat animal-derived foods; or were currently exercising vigorously three or more times per week were excluded. Participants were stratified by BMI (normal weight vs. obese), and within each obesity subgroup black and white participants were matched for BMI (±2 kg/m^2) and age (±2 years). The Institutional Review Board of the University of North Carolina at Chapel Hill approved the protocol for this study, and each subject provided written consent before participating in the study.

Outpatient diets and test meals

Participants were evaluated at the University of North Carolina Clinical and Translational Research Center (CTRC) on two separate occasions in counterbalanced order, each following a 4½-day period of consuming a high vs. a low glycemic load, mixed macronutrient (55% carbohydrate, 30% fat, 15% protein) diet. A minimum 1-week washout period separated these two periods. At the conclusion of each outpatient period, participants completed an overnight stay in the CTRC followed by a test breakfast meal with blood samples obtained via indwelling intravenous catheter prior to the meal and thereafter at min 30, 60, 120, and 180. Immediately after each blood draw, participants rated various aspects of subjective appetite (hunger, fullness, urge to eat, and specific food cravings) and meal palatability using 100 mm visual analog scales. Participants were allotted 20 min to complete a test meal and time zero of the postprandial period occurred at the conclusion of that 20-min period.

Meals consumed during each 4½-day lead-in period were designed by the CTRC research dietitian, prepared by the CTRC metabolic kitchen, and consumed by participants on an outpatient basis. Diets of either 2,000, 2,500 or 3,000 kcal were assigned to each subject according to kcal requirements that were based on the participant’s body size. Subjects’

energy requirements were estimated using 35 kcal/kg when BMI <30 and 35 kcal/kg adjusted body weight when BMI >30; estimated energy requirements were then rounded to the nearest 500 kcal for all subjects. Glycemic values were derived using the Food Processor SQL (version 10.2.0.0, ESHA Research, Salem, Oregon) based on previously published values (27). Mean ± s.d. glycemic load was 212.5 ± 31.2 vs. 107.5 ± 25.2 for the high vs. low outpatient diet, respectively. The high and low glycemic load test meals were standardized to 625 kcal (~25% of the total daily intake), 86 g carbohydrate, 21 g fat, and 23 g protein (ProNutra software version 3.2.1.0, Viocare Technologies, Princeton, NJ). Actual mean ± s.d. glycemic load was 59.1 ± 5.9 vs. 31.1 ± 3.6 for the high vs. low test meals, respectively.

Bioassays

We used commercially available radioimmunoassays (Linco, St Charles, MO) to measure total PYY (PYY1–36 and PYY3–36) and insulin (assay sensitivities = 5 pg/ml and 1.3 μIU/ml, respectively). Plasma glucose was determined by Ortho Clinical Diagnostics Vitros 950 analyzer (University of North Carolina Hospitals). Due to funding constraints, PYY assays were performed in two phases and in two different laboratories: phase 1, University of North Carolina Endocrine lab (N = 17: 9 black (5 obese), 8 white (3 obese)); phase 2, New York Obesity Research Center Hormone and Metabolite Core (N = 23: 11 black (5 obese), 12 white (7 obese)). Total PYY assay variability was computed separately by each laboratory (intra-assay coefficient of variation ranged from 2.7 to 4.4%; interassay coefficient of variation ranged from 5.1 to 9.3%).

Statistics

Missing data due to sampling and/or assay error (<2%) were estimated using within-diet multiple regression and then imputed prior to analysis. The primary outcome variable was PYY area under the curve (PYY_{AUC}), which was calculated with the trapezoidal method. Race (black, white), obesity (yes, no), and test meal (high, low glycemic load) main and interactive effects on total PYY_{AUC} were assessed by mixed-model ANOVA. Secondary analyses focused on assessing group (race, obesity) and glycemic load main and interactive effects on PYY and subjective appetite levels using repeated measures ANOVA. In both sets of analyses, significant interactive effects were followed by *post hoc* comparison of least squares means for interpretation. Preliminary analyses indicated significant mean total PYY differences by assay phase and significant between-group differences in age; thus, these variables were included as covariates. Participants were encouraged but not forced to eat the entire test meal, resulting in small magnitude individual differences in the macronutrient percentages actually consumed. Thus, to evaluate the influence of individual differences in these factors on total PYY_{AUC}, models were subsequently tested while controlling for actual test meal glycemic load, total kcal, and percentages of protein, carbohydrate, and fat. Insulin sensitivity was determined on the basis of fasting insulin and glucose levels obtained prior to each test meal and was calculated using the quantitative insulin sensitivity check index (28). Pearson correlations, controlling for assay phase and age, were used to examine the relation between total PYY_{AUC} and insulin sensitivity. All analyses were carried out using SAS software (version 9.13, SAS Institute, Cary, NC) and $P < 0.05$ was considered statistically significant.

Results

Baseline measures

Characteristics of the study sample are presented in Table 1. Compared to normal weight subjects, obese subjects were older and they had higher fasting glucose and insulin levels and lower insulin sensitivity (obesity main effect, $P < 0.05$). Blacks and whites were comparable on all measures (race main effects and race × obesity interaction effects, $P > 0.41$). Fasting glucose ($P < 0.0005$) and insulin ($P < 0.02$) levels decreased during study participation. These changes did not differ as a function of diet ($P > 0.81$ and 0.98, respectively) or race ($P > 0.09$ and 0.60, respectively), and were significant among obese but not normal weight participants ($P < 0.0003$; see Table 2). Furthermore, all interactions were nonsignificant (diet × race × obesity interactions, $P > 0.47$; race × obesity interactions, $P > 0.36$), suggesting that fasting insulin and glucose levels did not decrease to a greater extent in any one of the four subgroups. Within subgroup analyses for fasting insulin revealed a greater decrease in normal weight white women on the low vs. high glycemic load diet ($P < 0.05$). In addition, a trend ($P < 0.06$) toward a race × diet effect was observed, with blacks ($n = 20$) tending to show a greater decrease on the high glycemic load diet (−6.9 ± 17.4 vs. −5.7 ± 14.0) and whites ($n = 20$) tending to show a greater decrease on the low glycemic load diet (−3.7 ± 8.2 vs. −4.9 ± 8.3). However, in the absence of a significant diet × race × obesity effect or correction for multiple comparisons these within-subgroup observations should be viewed with considerable caution.

PYY effects

All main and interactive effects of glycemic load on fasting total PYY and total PYY_{AUC} were not significant, indicating that glycemic load neither affected PYY directly (diet, $P > 0.27$) nor differentially as a function of obesity (diet × obesity, $P > 0.82$) or race (diet × race, $P > 0.29$) (see Table 3). However, fasting total PYY and total PYY_{AUC} differed across subgroups (race × obesity interactions, Ps < 0.03). Obese black women exhibited lower fasting total PYY levels compared to normal weight black women (age- and assay phase–adjusted mean ± s.e.: 98.4 ± 5.0 pg/ml vs. 114.3 ± 5.1 pg/ml, respectively; $P < 0.04$) but neither group differed significantly from obese white (109.6 ± 4.9) or normal weight white (102.7 ± 4.9) women. Total PYY_{AUC} was significantly lower in black compared to white women (race effect, $P < 0.03$) and in obese compared to normal weight women (obesity effect, $P < 0.04$), with obese black women having significantly lower total PYY_{AUC} compared to all others (race × obesity interaction, $P < 0.003$); see Figure 1). The race × obesity interaction for total PYY_{AUC} remained significant after controlling separately for individual differences in test meal kcal ($P < 0.03$), fat percent ($P < 0.002$) and carbohydrate percent ($P < 0.005$). As shown in Figure 2, PYY levels differed between obese black women and other women at all postprandial time points (overall main group effect, $P < 0.002$). Total PYY_{AUC} was associated with insulin sensitivity ($r = 0.33$, $P < 0.004$; Figure 3), but there were no clear differences in this association by race/obesity subgroup.

Subjective appetite effects

Participants rated visual appeal, taste, and palatability higher for the high vs. the low glycemic load meal (test meal main effect, Ps < 0.02), but otherwise there were no consistent

main or interactive effects of race, obesity, or glycemic load on ratings of subjective appetite (data not shown).

Discussion

PYY provides a signal for the achievement and maintenance of satiety following acute feeding episodes. Deficits in PYY have been demonstrated in obese compared to normal weight individuals (12) as well as in blacks compared to whites (7,8). Based on these observations, diminished PYY secretion has been implicated as a potential factor in race differences in obesity risk. This is the first study, to our knowledge, to explicitly test for PYY deficits in obese black women relative to age- and BMI-matched obese white women and to normal weight black and white women. Results indicate significantly lower postprandial PYY secretion in obese black women compared to all others. Thus, our results confirm previous findings of race- and obesity-related deficits in PYY secretion and extend these findings in an important way. Specifically, the current study suggests that race differences in PYY secretion are largely due to the subset of blacks who are obese.

In our sample, PYY_{AUC} was significantly related to insulin sensitivity—a finding that is consistent with some (14,29) but not all (8,17,30) prior reports. This may be due to differences in sample characteristics, specifically age of the participants (adolescents in the negative studies, adults in the present study). Increasing age is related to progressive decrements in glucose tolerance (31) and decrements in β-cell function (32). The mean ± s.d. fasting glucose/fasting insulin ratios were 6.5 ± 2.5 vs. 8.0 ± 5.3 in black vs. white children, respectively (8) but in the present study were 10.4 ± 8.2 vs. 12.1 ± 10.8 in black vs. white adults, respectively. Thus, it is plausible that the wider range of insulin sensitivity exhibited by subjects in the present study facilitated detection of an insulin sensitivity-PYY association. Low circulating PYY levels have been linked to insulin resistance in healthy humans with increased familial risk of type 2 diabetes (14), and PYY knockout mice hypersecrete insulin in response to glucose challenge (33). These findings suggest that PYY deficits may contribute to hyperinsulinemia and insulin resistance. Furthermore, postprandial PYY secretion is diminished in healthy humans with vs. without increased familial risk of type 2 diabetes (29). Women in this study were relatively young and exhibited normal fasting insulin and glucose levels, but their familial history of diabetes was not assessed. Further, prospective studies that incorporate more rigorous assessments of insulin/glucose regulation and family history of type 2 diabetes are needed to understand whether PYY deficits contribute to increased rates of insulin resistance and diabetes observed in black compared to white women (34,35).

Low glycemic load diets are thought to support weight maintenance and/or loss through several mechanisms including lowering postprandial insulin and glucose responses (21), increasing postprandial time to subsequent food intake (21), improving body composition (23), and reducing hunger (23). However, the clinical utility of low glycemic load diets remains controversial in light of long-term studies indicating no advantage in terms of initial weight loss (36) or weight-loss maintenance (37) as well as recent contradictory findings of greater satiety, desire to eat, and prospective food consumption following high vs. low glycemic load challenges (38,39). In this study, manipulating glycemic load had no

appreciable effect on total PYY, nor did it appear to influence total PYY to any greater or lesser extent as a function of obesity or race, suggesting the potential appetite and weight regulation benefits of a low glycemic load diet are not mediated via alterations in total PYY.

Fat digestion is critical for stimulation of PYY (40) and postprandial PYY secretion in humans is sensitive to macronutrient content (16,17,19,20). Whereas high-protein intake is generally more satiating than high fat or high carbohydrate intake (41–43), the PYY response to macronutrient manipulation is less straightforward. In a sample of 18 obese adults (14 women), total PYY_{AUC} was greater following a high fat/low carbohydrate meal vs. a high carbohydrate/low fat meal (19). In eight obese women, PYY_{AUC} was not assessed; instead, PYY3–36 level was consistently greater after a high fat vs. a high carbohydrate liquid meal (16). In this same study, the high-carbohydrate meal evoked an immediate and sustained postprandial increase in PYY3–36, whereas the high-protein meal evoked a delayed increase in PYY3–36. In a third study of obese and lean men (20), $PYY3–36_{AUC}$ was greatest overall after a high-protein meal as well as significantly greater after a high fat compared to a high-carbohydrate meal in normal weight males but not in obese males. Lastly, in a recent study of adolescent girls, PYY3–36 responses differed (were lower) between obese and lean girls after a high fat meal but not after a high protein meal or high-carbohydrate meal (17). Thus, the macronutrient stimulus-PYY response is complex and apparently influenced by multiple factors including meal composition (i.e., whole food vs. liquid), obesity, sex, and age. To the extent that these complex relations replicate in future studies, they may justify further investigations of glycemic load effects on PYY that involve males, younger participants, and liquid meal challenges. Liquid meals, in particular, may be useful in circumventing potential confounding due to subjective differentiation of taste and palatability of low vs. high glycemic load meals. Like other appetitive hormones (44), PYY may demonstrate cephalic phase activity (45); thus, individual differences in perceived taste and palatability may influence observed group differences in PYY response.

Strengths of this study are the close matching of black and white participants within obesity subgroups on BMI and age. Limitations of the study include the small sample size; the potential for individual variability in actual consumption across test meals; reliance on BMI as our sole measure of obesity risk; lack of consideration of psychological factors that may affect appetite hormone responses; our focus on total PYY given that PYY3–36, in particular, has been linked to subjective satiety; lack of control for menstrual cycle; and lack of assessment of meal-stimulated PYY response before each 4½ day outpatient diet period. Whole food challenges such as the one used in this study have the benefit of ecological validity but are vulnerable to individual variability in actual caloric and macronutrient consumption. In this study, these factors did not likely account for diminished PYY_{AUC} observed in obese black women. Compared to obese white women, for example, obese black women consumed relatively the same average number of calories (627.8 vs. 625.8) and % fat (29.2 vs. 28.2). It should be acknowledged, however, that our approach differed from others' who adjusted test meal caloric content on the basis of body size or metabolic requirements (7,17,19). The impact that such an approach might have on our findings warrants further investigation. Recent findings suggest that postprandial release of the appetite-stimulating hormone, cholecystokinin, is greater following a high vs. a low glycemic load meal and moderated by the psychological factors of cognitive dietary restraint

and disinhibition, with cholecystokinin being blunted in participants reporting both high cognitive restraint and high disinhibition (38). Future studies designed to replicate or extend the present study might benefit by including measures of psychological attributes related to eating. PYY3–36 infusion reduced appetite and food consumption 2 h later at a buffet meal by ~30% in obese subjects (12,13), whereas PYY1–36 increased the postprandial insulin response but had no appreciable effect on energy intake (15). Notably, however, the effect of PYY1–36 on subjective appetite was dependent on dose and body composition: low dose PYY1–36 reduced hunger and perceived ability to eat in lean subjects but had the opposite effect in obese subjects; high dose PYY1–36 increased hunger and decreased satiety before food intake but had the opposite effect after food intake. Although intriguing, these findings are difficult to interpret because obese vs. lean subjects were defined on the basis of BMI (rather than body composition) and because there were disproportionately more obese subjects who received the high dose treatment. Further studies are needed to understand how endogenous levels of PYY1–36 vs. PYY3–36 relate to subjective appetite and subsequent energy intake and how these relationships differ as a function of obesity and race. In light of findings suggesting that the ovarian hormone milieu influences the food-inhibition effect of PYY (46), PYY3–36 receptor binding (47), energy consumption patterns (48), and food transit (49), future studies should control for menstrual cycle effects, preferably by limiting testing to the follicular phase. Finally, although we did not observe group differences in fasting PYY, it is still possible that our findings were influenced by the 4½-day outpatient diets. Future studies should consider evaluating meal-stimulated PYY response both before and after dietary manipulation to delineate the precise nature and extent of PYY deficiency in obese black women.

So far, race differences in PYY have been demonstrated only in studies of black and white adult women and children. Moreover, there is some indication that obesity-related deficits in PYY are limited to adults (8,12). Larger studies, which also include men, other race/ethnic minorities, and children, are needed to determine whether diminished PYY activity plays a unique role in the pathogenesis of obesity among black women. Studies that include adult participants and their offspring may be helpful in addressing potential genetic and gene-environment contributions to race differences in PYY. Finally, because PYY is sensitive to variations in macronutrient content and to acute exercise (50), studies that closely measure daily food intake and physical activity in individuals in their natural environments may be instrumental in understanding how PYY is regulated and ultimately relates to individual risk for obesity.

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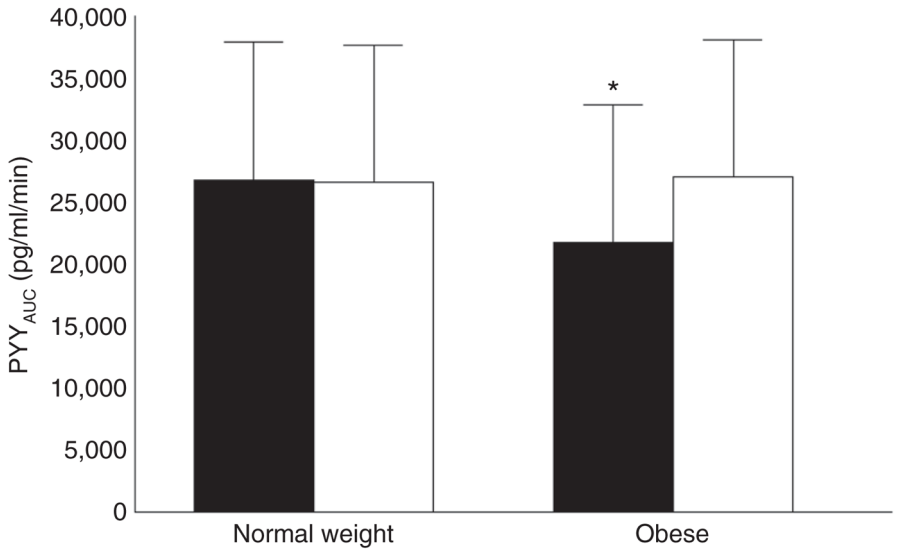

Figure 1.

Total PYY area under the curve by race and BMI status. Age- and assay phase–adjusted mean total PYY_{AUC} (s.e.), collapsing across glycemic load, in black (solid bar) vs. white (open bar) obese and normal weight women (black ($n = 20$) < white ($n = 20$), $P < 0.02$; obese ($n = 20$) < normal weight ($n = 20$), $P < 0.05$; *obese black < each other group, Ps < 0.003).

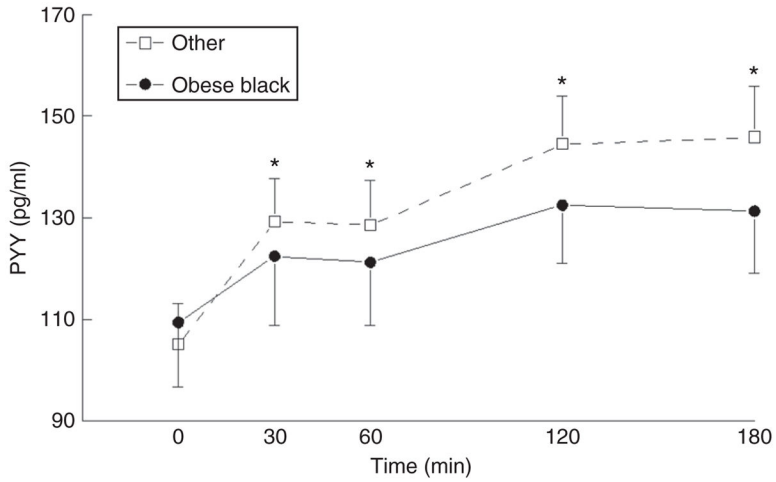

Figure 2.

Total PYY levels in black obese vs. other women. Change in postprandial total PYY levels (age- and assay phase–adjusted mean values ± s.e.) in obese black women ($n = 10$; closed circles) vs. others ($n = 30$; obese white, normal weight white and black combined). Data are collapsed across glycemic load conditions. Overall group effect, $P < 0.002$; *within time point group difference, $P < 0.001$.

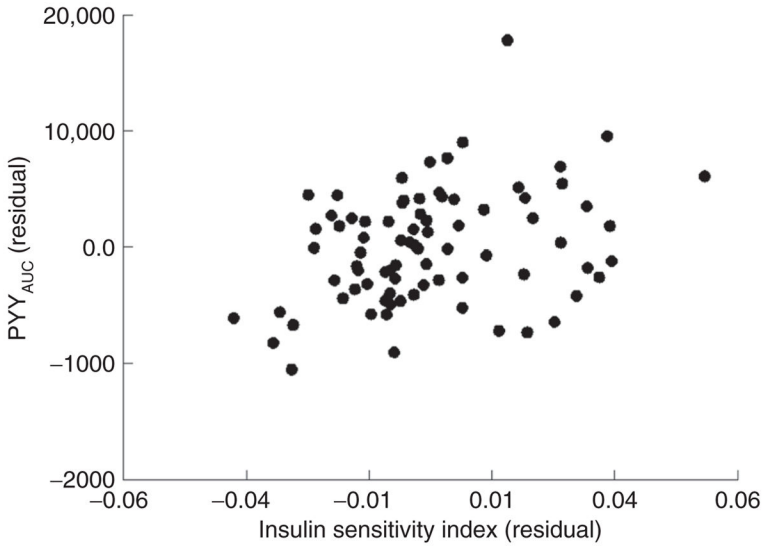

Figure 3.

Relation between total PYY_{AUC} and insulin sensitivity. Scatter plot of residuals depicting significant relation between total PYY_{AUC} and insulin sensitivity ($r = 0.33$, $P < 0.004$).

Table 1

Characteristics of the study sample

	White normal	White obese	Black normal	Black obese
N	10	10	10	10
Age (years) **	29.4 (7.9)	34.1 (10.5)	26.5 (6.1)	35.6 (7.6)
BMI (kg/m^2) ***	22.9 (1.4)	34.2 (2.8)	22.8 (1.4)	35.1 (2.8)
Fasting glucose (mg/dl) *	84.9 (7.4)	90.4 (16.1)	83.7 (8.8)	96.5 (10.8)
Fasting insulin (μU/ml) **	7.1 (4.1)	18.9 (17.4)	8.5 (5.3)	25.2 (27.2)
Insulin sensitivity ***	0.16 (0.02)	0.14 (0.01)	0.16 (0.02)	0.14 (0.02)

Data are expressed as mean (s.d.).

Obese (n = 20) vs. normal weight (n = 20):

* $P < 0.05$,

** $P < 0.01$,

*** $P < 0.001$.

Black vs. white: all Ps > 0.44. Obesity × Race interactions: Ps > 0.40.

Table 2

Effect of glycemic load on fasting glucose and insulin

	White normal	White obese	Black normal	Black obese
N	10	10	10	10
Fasting glucose (mg/dl) *				
High GL	84.1 (8.7)	85.6 (9.4)	80.8 (5.4)	86.9 (7.8)
Low GL	82.8 (9.0)	87.2 (14.9)	79.8 (6.8)	88.2 (7.2)
Δ Fasting glucose (mg/dl)				
High GL	−0.8 (4.0)	−4.8 (8.6)	−2.9 (7.8)	−9.6 (6.9)
Low GL	−2.1 (4.0)	−3.2 (5.8)	−3.9 (7.9)	−8.3 (7.0)
Fasting insulin (μU/ml) **				
High GL	8.2 (4.6)	13.3 (8.9)	9.6 (3.1)	14.0 (7.8)
Low GL	5.6 (2.9)	13.0 (7.9)	9.6 (5.3)	18.6 (12.6)
Δ Fasting insulin (μU/ml)				
High GL	−0.8 (2.9)	−6.6 (10.8)	−0.6 (6.4)	−13.2 (22.5)
Low GL	−2.7 (2.5)	−7.2 (11.3)	−0.9 (5.9)	−10.5 (18.2)

Δ = Change relative to level at study entry (Table 1). Data are expressed as mean (s.d.).

GL, glycemic load.

Obese ($n = 20$) vs. normal weight ($n = 20$):

* $P < 0.02$,

** $P < 0.0003$.

Blacks ($n = 20$) vs. whites ($n = 20$): Ps > 0.09. Obesity × Race interactions: Ps > 0.36. Diet × obesity × race interactions: Ps > 0.47.

Table 3

Effect of glycemic load on fasting PYY and PYY_{AUC}

	White normal	White obese	Black normal	Black obese
N	10	10	10	10
Fasting PYY (pg/ml)				
High GL	95.0 (6.9)	106.8 (6.9)	116.5 (7.0)	95.9 (7.0)
Low GL	110.4 (6.9)	112.4 (6.9)	112.1 (7.0)	100.9 (7.0)
PYY_{AUC}(pg/ml)				
High GL	26,358 (11,101)	27,105 (11,104)	26,565 (11,107)	21,667 (11,104)
Low GL	26,812 (11,101)	26,910 (11,104)	27,007 (11,107)	21,843 (11,104)

Data are expressed as least squares mean (s.e.), adjusted for assay phase and age.

AUC, Area under the curve, collapsed across assay phase; GL, glycemic load.

All main and interactive effects are nonsignificant (*P*s > 0.27).