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Impaired Glucose Regulation is Associated with Poorer Performance on the Stroop Task

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Abstract

Background—Type 2 diabetes is a risk factor for development of cognitive dysfunction. Impairments in glucose regulation have been associated with poorer performance on tests of executive function and information processing speed.

Methods—We administered the Stroop Color Word Task, where higher interference scores are indicative of decreased selective attention, to 98 non-diabetic volunteers (64m; %fat=37±12; age=36±9 y, race=41 NA/30 C/13 H/14 AA) on our inpatient unit. After 3d on a weight maintaining diet, % body fat was measured by DXA and a 75g oral glucose tolerance test (OGTT) was administered. Impaired glucose regulation (*IGR*) was defined as: fasting plasma glucose ≥100 and ≥125 mg/dL and/or 2h plasma glucose between 140 and 199 mg/dL (*IGR*; n = 48; *NGR*; n = 50). Total and incremental area under the curve (AUC) for insulin and glucose were calculated.

Results—Stroop interference scores were not significantly associated with any measure of adiposity or insulin concentrations. Individuals with *IGR* had significantly higher interference scores than those with *normal glucose regulation* (*NGR*; p=0.003). Higher interference scores were significantly correlated with *fasting plasma glucose* concentrations (r=0.26, p = 0.007) and total glucose AUC (r=0.30, p = 0.02) and only trending so for iAUC and 2h *plasma glucose* (r=0.18, p=0.08; r=0.17, p=0.09 respectively). In separate multivariate linear models, *fasting plasma glucose* (p = 0.002) and total glucose AUC (p = 0.0005) remained significant predictors of Stroop interference scores, even after adjustment for age, sex, race, education and %fat.

Conclusions—Individuals with *IGR* had decreased performance on a test of selective attention. *Fasting plasma glucose* was more strongly associated with lower performance scores than 2h *plasma glucose*. Our results indicate that even mild hyperglycemia in the non-diabetic range is associated with attentional processing difficulties in a sample of younger adults. Whether these impairments precede or are induced by impaired glucose regulation is not clear.

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Keywords

executive function; processing speed; interference; obesity; cognitive performance; insulin

1. Introduction

There is mounting evidence of a relationship between obesity and cognitive function. Higher BMI over the life span has been shown to correlate with cognitive impairments in memory, executive function, visuomotor skills [1] [2] and information processing [3–6] (see [7, 8] and [9] for *comprehensive reviews*). Specifically, obese subjects performed more poorly than non-obese on tests measuring decision-making [10, 11] (Iowa Gambling Task), problem solving and working memory (Wisconsin Card Sorting Task), inhibition/attention (Go/No Go task; [12]) and information processing speed [13]. Obese adults [3] [4] and overweight children [5] and adolescents [6] were slower in naming the color of food-related words versus control words in a modified Stroop Task, indicating an enhanced attentional bias to food related stimuli.

Obesity is associated with a range of comorbidities that might account for the association between obesity and cognitive impairments, such as hypertension, high cholesterol levels, cardiovascular disease *and depression*, all of which have been independently linked with cognitive impairments [7,14]. There is a known link between metabolic syndrome (in the absence of diabetes) and cognitive dysfunction [15,16] and in particular, insulin resistance has been linked with poorer performance on a memory task [17] and tests of executive function [18]. Another possible explanation for the link between adiposity and cognitive deficiencies is blood glucose abnormalities. Glucose serves as fuel for the brain and is necessary for cognitive function. Many studies link impaired glucose tolerance [19, 20], hyperglycemia, and diabetes [21] [22] to lower levels of cognitive function on neuropsychological tests [23]. Cross-sectional studies indicate that hyperglycemia may have a negative impact on cognitive functioning in those with type II diabetes (T2DM) [24]. Some, though not all [25] studies have shown that longer illness duration [26] and elevated HbA1c levels [27], are associated with increased severity of cognitive decrements.

In a recent comprehensive review, Lamport and colleagues [23] concluded that worsening glucose tolerance is associated with cognitive impairments; the most prevalent of which are deficits in verbal memory but also include attentional control, executive function [28] *and processing speed deficits* [29]. Prolonged exposure to abnormal glucose levels is associated with cognitive deficits in elderly patients, and individuals with T2DM are at 1.5 to 2.5 fold increased risk for dementia [30]. Furthermore, studies have demonstrated that even mildly increased glucose concentrations in the non-diabetic range are associated with impaired cognition in both younger [31] [32] [33] and older adults [34].

Nevertheless, the majority of studies linking glucose regulation with cognitive deficits have been conducted in the elderly. However, this population often has other co-morbid conditions related to ageing that might affect cognition, confounding the investigation between glucose tolerance and cognitive performance. Thus, additional studies in younger populations are needed to confirm the relationship between glucose concentrations and cognition. *Because processing speed deficits and elevated glucose levels have been documented in obese individuals*, the aim of our study was to investigate the association of impaired glucose regulation with scores on Stroop Task performance, *commonly used in the evaluation of executive functions to measure selective attention and processing speed* [35, 36], in a sample of young to middle-aged non-diabetic adults from 4 inpatient studies. Our

secondary aims were to examine whether: 1) glucose levels following an oral glucose tolerance test (OGTT) and; 2) adiposity were associated with Stroop performance.

2. Methodology

2.1. Recruitment

Ninety-eight non-diabetic volunteers (64m/34f), ages 18–55 years, were recruited from the greater Phoenix area by advertisement to participate in one of four inpatient studies. Prior to participation, all volunteers were fully informed of the nature and purpose of the study and written informed consent was obtained. The experimental protocols were approved by the Institutional Review Board of the National Institute of Diabetes and Digestive and Kidney Diseases.

2.2. Study Design

All subjects were found to be free of medical and psychological disease according to physical examination, medical history, and laboratory testing. Subjects were not taking any medications, were nonsmokers and were excluded if they had any significant health problems, including cancer, hypertension, diabetes mellitus or excess alcohol use (> 3 drinks/d). Those meeting criteria for current substance abuse were also excluded. The first four days of all the studies were identical. Upon admission to the metabolic ward, subjects were fed a standard weight maintaining diet (20%, 30%, and 50% of daily calories provided as protein, fat and carbohydrate, respectively) for 3 d prior to the OGTT. Weight maintenance energy needs (WMEN) on the metabolic ward were calculated for each subject based on weight, gender and BMI [37]. Body composition was measured by dual energy X-ray absorptiometry on the second day of admission (DPX-L, Lunar Corp., Madison, WI) as previously described [38, 39].

2.3. Measures

2.3. a—Participants completed two questionnaires to assess symptoms associated with mood and depression

1. The Inventory for Depressive Symptomatology – Self-Report (IDS SR [40]): a 30-item self-report measure of depressive signs and symptoms. Scores range from 0–90, with scores ≥ 39 indicative of moderate to severe depression.
2. The Physical Anhedonia Scale (PAS; [41]): a 61-item scale to assess sensitivity to reward. A score ≤ 18 indicates anhedonia.

2.3. b The Stroop Test—During the morning of the third day, approximately 1-hour after eating breakfast, participants completed a computerized version of the Stroop Word Color Task, outlined in Denny & Lynch, 2009 [42]. The test was administered using a desktop computer with a 14-inch screen and consisted of three 60-s *conditions* (*color word reading, color naming and color-word naming*). In the first condition the subject read color words (RED, GREEN, BLUE, and YELLOW) written in black letters (*condition 1: color word reading [CW]*), then named the color of a row of four Xs (*condition 2: color naming [CN]*), and finally, named the color of the letters of color words printed in different colors (*condition 3: color – word naming [SW]*). In the color – word naming *condition*, all the stimuli were incongruent (e.g., the word “YELLOW” printed in red letters; see Figure A). The stimulus appeared in the center of the computer screen. The subject gave a verbal response to the stimulus (i.e., read the word or named the color), prior to pressing the space bar to display the next stimulus. A brief, eight-stimulus practice set was presented before the start of each *condition*.

Prior to each *condition*, the subject was given the following instruction by the examiner: “Work quickly but try not to make any mistakes. If you do make an error, try not to correct it. Just go on to the next item.” The computer timed *each condition to last for 60 seconds* and recorded the total number of *trials* completed during *each block*. Subjects typically try to self-correct their errors when they commit one, therefore taking more time to complete the task. As a result, reaction time is also reflected in the total score reported for each *block* (e.g. number of items completed). A member of the study staff was present during the administration to monitor the subjects’ performance.

Both the word reading (CW; *Condition 1*) and color naming (CN; *Condition 2*) scores are considered measures of processing speed [43]. “The Stroop Effect” is known as the difference in speed and accuracy (e.g. reaction time) when the color and word are incongruent and is a measure of interference. When the name of a color (e.g., “blue,” “green,” or “red”) is printed in a color not denoted by the name (e.g., the word “red” printed in blue ink instead of red ink), naming the color of the word takes longer and is more prone to errors than when the color of the ink matches the name of the color. We calculated this interference score by taking the number of *trials* achieved (number of items completed) in *Condition 2* (color naming) minus the number of sequences achieved in *Condition 3* (color-word naming) and dividing the difference by the number of *trials* achieved in *Condition 2* (color naming), multiplied by 100 ($[(CN-SW/CN) * 100]$). The result is an interference score that accounts for processing speed and thus provides a truer report of executive function by distinguishing information processing speed from impairments in other domains [44]. Higher calculated interference scores indicate poorer performance and are suggestive of decreased processing speed and impaired executive function [42].

2.4. OGTT

On the day following the Stroop Task (day 4), glucose tolerance was assessed by a 75-g oral glucose tolerance test according to the criteria of the ADA 2003 guidelines [45]. Plasma samples were drawn at 0, 30, 60, 120 and 180 minutes. Only non-diabetic subjects were included in this analysis. Impaired glucose regulation (IGR) was defined as: impaired fasting glucose (IFG): fasting plasma glucose 100 to 125 mg/dL and/or impaired glucose tolerance (IGT): 2h plasma glucose 140 to 199 mg/dL [32]. Total and incremental area under the curve (iAUC) for insulin and glucose were calculated using the trapezoidal method.

2.5. Statistical Analyses

All procedures were carried out using the software of the SAS Institute (SAS version 9.2, Cary, NC). Insulin values were log transformed to approach a normal distribution. Stroop interference scores ranged from -19 to 65 (M 24 ± 13 SD). Because negative interference scores ($n = 6$) are rarely observed, we ran the analyses both with and without the negative scores and the results were similar. Therefore, we included all subjects in the data analysis. Student’s two-tailed T-tests were used to compare differences in Stroop performance between those who had NGR versus IGR.

Spearman’s correlation coefficients were used to describe relationships between Stroop interference score and continuous variables (*fasting plasma glucose*, *2h plasma glucose*, total glucose and insulin AUC, iAUC for glucose and insulin) and Chi-square (χ^2) was used for categorical data. The correlation analyses were controlled for age. Any significant correlations were evaluated further with multivariate linear regression models to determine the relationship between Stroop performance scores and glucose status or continuous variables after adjusting for age, sex, race and %fat. Least squared means with 95%

confidence intervals for categories of glucose status were generated from these multivariate models. Levels of statistical significance were set at $p < 0.05$.

3. RESULTS

3.1. Baseline Characteristics

Participants ranged in age from 19–55 years ($M = 35 \pm 10$). They were largely Native American (42%) and Caucasian (31%) with a smaller percentage of African-Americans (15%) and Hispanics (12%). Fifty-two percent ($n = 51$) had normal glucose regulation (NGR), 15% ($n = 15$) had IFG, 12% ($n = 12$) had IGT and 21% ($n = 20$) had both IFG and IGT (IFG/IGT). Thus, 49% ($n = 47$) of subjects were categorized as having IGR.

Age did not differ between those with NGR (34 ± 10 yrs), IFG only (37 ± 8 yrs), IGT only (33 ± 11 yrs) or those with IFG/IGT (38 ± 9 yrs). Neither mean *fasting plasma glucose*, *2h plasma glucose*, AUC or percent IGR differed by gender or race. Two hour plasma glucose and total AUC glucose, as well as fasting, 2h and AUC insulin, but not %fat or BMI, were all significantly greater in those who did not complete high school compared with those who did (all $p < 0.05$). Baseline subject characteristics are shown in table 1.

3.2 Mood

Scores were in the normal range and similar between NGR and IGR on both measures of depression (11 ± 9 vs. 11 ± 9) and anhedonia (13 ± 8 vs. 15 ± 9), respectively.

3.2. Stroop Performance

Mean interference score was 23 ± 13 , similar to a normal control group (27 ± 7) in a study using the same Stroop methodology [42]. Native Americans had lower interference scores (better performance) compared to Caucasians (18 ± 13 vs. 31 ± 11 , $F = 6.1$, $p = .003$). There were no associations between Stroop performance and gender, age, level of education, *depression or anhedonia scores*.

Stroop interference scores were not correlated with any measures of adiposity (weight, BMI, waist, %fat). There were no differences between those with IGR and NGR on color word (*condition 1*) or color naming (*condition 2*) trials, but compared to those with NGR, the IGR group performed worse on the more difficult, color-word naming trial (*condition 3*; $F = 6.4$, $P = 0.02$; see figure 2a). Those with IGR also had significantly higher interference scores, indicative of poorer performance, compared to those with NGR ($p = 0.002$; see figure 2b). In a multivariate linear model controlling for age, sex, race, education and %fat, the marginal means of the four separate groups demonstrated that individuals with only IFG (25 [19, 31; 95%CI]), only IGT (28 [21, 35]) and those with IFG/IGT (28 [22, 35]) had similar interference scores, but that all were greater than individuals with NGR (18 [15, 22]) ($F = 3.9$, $p = 0.01$).

Higher interference scores were positively correlated with *fasting plasma glucose* concentrations ($r = 0.27$, $p = 0.007$) and total glucose AUC ($r = 0.24$, $p = 0.02$; see Figures 3a and 3b). The correlations between interference scores and *2h plasma glucose* ($r = 0.18$, $p = 0.08$) and iAUC did not meet our criteria for significance ($r = 0.17$, $p = 0.09$). When total glucose AUC, iAUC, and *2h plasma glucose* were added separately to the model with *fasting plasma glucose*, remained significant. Interference scores were not associated with any measure of insulin (data not shown).

In separate linear models, *fasting plasma glucose* ($F = 5.9$, $p = 0.002$) and total AUC for glucose ($F = 3.7$, $p = 0.01$) remained significant predictors of Stroop interference scores, even after adjustment for age, sex, race, education and % fat.

4.0. DISCUSSION

Poorer performance of incongruent color-naming sequences on the Stroop Task was associated with IGR and of the glucose variables, most strongly with fasting plasma glucose. This indicates that even elevated glucose levels in the non-diabetic range are associated with decreased selective attention. However this effect seems specific to glucose as we did not observe a relationship between insulin levels and Stroop performance.

Our findings are consistent with previous studies showing relationships between even modest levels of glucose intolerance [19, 20] hyperglycemia, and diabetes [21] [22] with lower levels of cognitive function on neuropsychological tests [23], [29]. Other studies, however have failed to find a relationship between fasting glucose levels in the non-diabetic range and cognitive function in either middle aged [46] or older adults [47] [48]. For those studies that have demonstrated an association between glucose and cognitive function, many of these have been conducted in the elderly, who have other co-morbid conditions and other ageing related diseases which could confound the investigation between glucose tolerance and cognition.

Our findings are unique in that we observed a relationship between mild hyperglycemia and decreased selective attention in a younger cohort, averaging 35 years in age, *consistent with previous studies showing the effects of poor glucose regulation on tasks associated with frontal cortex functions* [18, 28]. Interestingly, in our sample, age was not associated with poorer Stroop performance and the negative association between IGR and performance was strong even after statistical adjustment for age. In addition, a wide range of ethnic groups and educational levels were well-represented. *Also, while there were differences between ethnic groups on Stroop performance, there were no differences in glucose metabolism between these groups. Our findings of the relationship between glucose regulation and Stroop performance were significant after adjustment for ethnic group and age, indicating that our findings are not limited to specific demographic characteristics.*

4.1. Stroop and Adiposity

Despite this relationship between poorer performance on the Stroop Task and increasing glucose concentrations, we did not observe a relationship between measures of adiposity with Stroop scores and the effects of glucose on Stroop Interference scores were independent of adiposity in our model. Other studies have observed delayed processing of food related stimuli, *but not to negative or neutral stimuli, during a modified Stroop task* in obese adults [3] adolescents [6] and children [5]. This suggests an information processing bias in obesity that reflects a specific hypersensitivity for food cues [5], *although this was not assessed in our study.* However, while the food Stroop might be related to adiposity, the attentional deficits observed in the current study appear more strongly associated with fasting glucose concentrations than with adiposity.

4.2. Potential Mechanisms

Whether or not the development of cognitive impairment precedes or follows impaired glucose regulation is unclear. One recent study demonstrated improved memory performance following weight loss after bariatric surgery [49]. Although glucose concentrations were not reported, it is likely that they decreased significantly following bariatric surgery [50]. Several studies have found that reducing glucose levels through

treatment with medication resulted in improvements in cognitive performance [51] [52], including Stroop-word naming [53]. Whether or not higher glucose levels are related to the attentional biases specifically towards food-cues as observed in previous studies [3] [4, 5] [54] is unclear. It is also not known whether longitudinal changes in cognitive performance scores are related to changes in glucose concentrations or weight or an interaction between the two.

Cortisol dysregulation is associated with T2DM and has been shown to impact cognitive function. Both exogenous and endogenous cortisol elevations can reduce declarative memory performance [55], possibly from glucocorticoid-mediated deficits in neurogenesis and synaptic plasticity [56] or overall stress-induced allostatic burden to the brain [57]. Whether or not cortisol levels mediate the relationship between glucose levels and Stroop performance has not been studied.

The mechanism by which elevated glucose concentrations might affect cognitive performance is not clearly understood. One possibility is that glucose transfer across the blood brain barrier (BBB) and between intra and extracellular fluid in the brain is impaired by hyperglycemia [23], thus impacting cognitive performance due to lack of glucose availability. Cognitive dysfunction could result from a reduction in availability of acetylcholine caused by the decreased transport of glucose across the BBB that occurs with even mild degrees of hyperglycemia [23]. *In support of this, whole brain glucose metabolism appears to be activated maximally during fasting in NGR but not IGR individuals [58]. This may explain why fasting plasma glucose is more strongly associated with interference scores than post-prandial hyperglycemic measures. Fasting plasma glucose also represents a more chronic level of hyperglycemia, and may explain why decreased Stroop performance is associated with higher hbA1C (a measure of chronic hyperglycemia) in those with diabetes.* However, the greater variability of 2h plasma glucose compared with fasting plasma glucose might have weakened the association.

4.3 Study Limitations

Limitations of this study include a decreased ability to detect associations with adiposity due to a restricted BMI range. Less than one-fifth of our sample was classified as normal weight and between group differences may have been more difficult to detect with uneven sample sizes. We also acknowledge that the associations between glucose concentrations and interference score were modest but feel it could still be important when considering the influence of such a relationship over time.

Our volunteers were tested 1 hour after breakfast and it is unclear how the effects of their metabolic state may have contributed to the similarities and/or discrepancies between this and other studies. Methodologies are inconsistent in the literature, so while some studies investigating the Stroop task in similar populations were conducted within 2–4 hours following a meal (e.g. [3] [54]) or an overnight fast [28], others do not mention meal timing or metabolic state (e.g. [4] [6]). Future studies should control for, and report on meal timing and metabolic status to further evaluate their impact on cognitive performance.

The version of the Stroop Task used in the current study is based on Golden's 3 card version [59] and is similar to the one used in many previously published studies ([54], [6], [42]). It is possible that the formatting difference between the version used here and in other publications might have influenced thereby limiting our ability to compare our results to other studies (see Salo 2001 [60] for a discussion). And, as previously mentioned, we did not calculate reaction time per se, whereas other studies using the Stroop utilize a measure reaction time in milliseconds based on how long it takes participants to tap the correct color on a keyboard compared to the verbal response elicited from our volunteers. While these

two factors might explain some of the discrepancies between our results and those reported in other studies, it is unclear exactly how our results might differ.

Our study sample consisted of individuals who were able to spend between 10–30 days on our inpatient unit and therefore might not be typical of the general population. However, they were recruited from the general Phoenix area and are ethnically diverse, unlike many other studies. Lastly, there was a subgroup of six participants who obtained negative interference scores, indicating better performance on the harder task (*condition 3*) compared to a simpler task (*condition 2*). While negative interference scores are rarely observed in studies using the Stroop, our results did not change when these individuals were excluded and there was nothing to justify removing them from the analysis. While it is possible that these individuals may not have performed at full effort or may have been distracted during the testing period, a staff member was present during the test administration to ensure the appropriate procedures were followed.

4.4 Summary

In conclusion, we observed a relationship between impaired glucose regulation and decreased performance on the Stroop/Word Color Task in a group of young to middle aged adults. These findings indicate that individuals with higher glucose levels, even within the non-diabetic range, are less able to inhibit automatic responses, indicative of attentional processing difficulties. This association was primarily driven by fasting rather than 2 hour glucose concentrations. Whether or not higher glucose levels are related to the attentional biases specifically towards food-cues as observed in previous studies should be a focus of future investigations. Future studies should also examine whether or not reducing even modestly elevated glucose levels through medication or dietary intervention might improve cognitive response and whether cognitive remediation might decrease risk for future cognitive decline.

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Highlights

- Examined glucose regulation with Stroop Task scores in young non-diabetic adults
- Stroop interference scores were not significantly associated with adiposity or insulin
- Poorer performance was most strongly correlated with fasting plasma glucose concentrations
- Those with impaired glucose regulation did more poorly than those with normal glucose regulation.
- Fasting and AUC glucose during an OGTT predicted Stroop performance

Figure 1A. Stroop Word Color Task

Based on the methodology by Denny and Lynch, 2009

Condition 1 (CW: color word reading): read color words (Red, Green, Blue, etc) written in black letters

Condition 2 (CN: color naming): name the color of a row of four XXXX

Condition 3 (SW: color – word naming): name the color of letters of color words printed in different colors (the word “**Yellow**” printed in red ink)

Figure 2a: **Figure 2b:** **Figure 2.**

Figure 2a. Differences in Processing Speed

Mean number of correct responses on each trial of Stroop task adjusted for age, sex, race, BMI, education

Error bars represent 95% CI

CW = *Condition 1*: color word reading

CN = *Condition 2*: color naming

SW = *Condition 3*: color – word naming (Stroop Word)

There were no differences between IGR and NGR on color word (*Condition 1*) or color naming (*Condition 2*) but the NGR group performed better on the Stroop-word (*Condition 3*)

* $F = 6.4$, $P = 0.02$

Figure 2b. IGR (black bar) group had higher interference scores compared to those with NGR (gray bar)

$M \pm 95\%$ CI after adjustment for age, sex, race, BMI, education

19 [CI 15–22] vs. 27 [23–32]

* $F = 10.6$, $P = 0.002$

Figure 3a

Figure 3b

Figure 3.

Figure 3a. Fasting Plasma Glucose (mg/d; x axis) is positively associated with Stroop interferences scores (y axis).

Interference score calculated as: $([Condition\ 2 - Condition]/Condition) * 100$ ([CN-SW/CN] * 100).

Unadjusted Spearman Correlation: $R=0.27$, $p = 0.007$

Figure 3b: AUC Glucose (mg/d; x-axis) is positively associated with Stroop interferences scores (y-axis).

Interference score calculated as: $([Condition\ 2 - Condition\ 3]/Condition\ 3) * 100$ ([CN-SW/CN] * 100).

Unadjusted Spearman Correlation: $R=0.24$, $p = 0.02$

Table 1

Demographic and Metabolic Characteristics

	NGR	IGR	p [*]
Education (years)	12.9 ± 1.4	13.0 ± 1.4	0.43
Age (y)	34 ± 10	37 ± 10	0.29
<i>Ethnicity</i>			0.93 [‡]
<i>Native American</i>	<i>N = 20 (39%)</i>	<i>20 (42%)</i>	
<i>Caucasian</i>	<i>16 (31%)</i>	<i>15 (32%)</i>	
<i>African-American</i>	<i>9 (18%)</i>	<i>6 (13%)</i>	
<i>Hispanic</i>	<i>6 (12%)</i>	<i>6 (13%)</i>	
BMI (kg/m ²)	30 ± 7	36 ± 7	0.0005
Body Fat (%)	34 ± 13	40 ± 10	0.02
Waist circumference (cm)	40 ± 7	45 ± 5	0.003
Fasting Glucose (mg/dL)	92 ± 5	102 ± 6	< 0.0001
2-h Glucose (mg/dL)	108 ± 20	148 ± 26	< 0.0001
AUC Glucose (mg/dL)	23737 ± 3965	24062 ± 3579	0.74
iAUC Glucose (mg/dL)	6001 ± 6832	6262 ± 2746	0.50
Fasting Insulin (uU/ml)	12 ± 11	18 ± 10	0.05
2-h Insulin (uU/ml)	96 ± 92	126 ± 87	0.18
AUC Insulin (uU/ml)	15931 ± 11889	19619 ± 11177	0.20
iAUC Insulin (uU/ml)	14006 ± 10124	15555 ± 9139	0.60

Values are presented as mean ± SD except for ethnicity

* T-test

‡ Chi Square