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Biobehavioral and Psychological Differences Between Overweight Adults With and Without Waist Circumference Risk

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Abstract

Waist circumference (WC) has emerged as an independent predictor of cardiometabolic disease. The purpose of this study was to examine differences between overweight adults with and without WC risk in four domains: demographic, clinical and biological, psychological, and behavioral. The sample ($N = 87$) was primarily sedentary, middle-aged, women, and African-Americans. The majority of participants had WC risk, those with WC risk were older, were women, and had higher body mass index, higher morning salivary cortisol levels, and more depressive symptoms than those without WC risk. Caloric and macronutrient intake did not differ between those with and without WC risk. Our findings could lead to the development of targeted interventions to prevent and/or reduce abdominal obesity, thereby reducing cardiometabolic risk.

Keywords

obesity; depression; lifestyle change; prevention

Abdominal obesity or adipose tissue that is centered at the abdomen, is more closely related to cardiometabolic disease (insulin resistance, diabetes, and cardiovascular disease) than is generalized obesity and may be a better predictor of cardiometabolic disease risk (Grundy et al., 2006; Li, Ford, McGuire, & Mokdad, 2007). Since 1988, the age adjusted prevalence of abdominal obesity [e.g., waist circumference (WC) > 102 cm for men, WC > 88 cm for women; Expert Panel on the Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001; National Institutes of Health [NIH], 1998) has been rising steadily from 29.5% to 42.4% in men and from 47.0% to 61.3% in women; current estimates are that over 50% of U.S. adults have abdominal obesity and are at high risk for obesity-related morbidity and mortality (Li et al., 2007).

Previous research on cardiometabolic disease risk has focused primarily on the influence of bio-psycho-behavioral factors on body mass index (BMI), which is calculated as the ratio of weight in kilograms to height in meters squared (kg/m^2 ; Keys, Fidanza, Karvonen, Kimura, & Taylor, 1972). Little is known, however, about how the influence of bio-psycho-

behavioral risk factors on BMI varies between overweight adults (BMI ≥ 25 kg/m²; NIH, 1998) with and without abdominal obesity. An understanding of how specific risk factors contribute to the development of abdominal obesity and thereby to cardiometabolic disease risk is needed. Therefore, the purpose of this study was to determine if there are significant differences between sedentary overweight adults with and without WC risk in four bio-psycho-behavioral domains: demographic (age, gender, and race), clinical and biological (morning cortisol level, self-reported hypertension, cholesterol levels, and chronic health conditions, and BMI), psychological (depressive symptoms and perceived stress), and behavioral (caloric and macronutrient intake). The conceptual model guiding the study is an adaptation of the risk factor model proposed by Friedman and Brownell (1995), which suggests that risk factors in multiple domains may act independently and/or interdependently to contribute to the development of obesity and health consequences, such as the increased risk of cardiometabolic disease. They conceptualized risk factors as independent, mutually potentiating, and interactive variables, and speculated that risk factors may occur in clusters and vary between subgroups of the obese population. Evidence supporting the role of risk factors clustered into the four domains as they may influence the subgroup, persons with abdominal obesity, is presented below.

DEMOGRAPHIC DOMAIN

Age, gender, and race/ethnicity modify an individual's susceptibility to abdominal obesity. With aging, body weight increases and body composition changes: there is a decrease in the percent of total body weight that is lean and an increase in both subcutaneous and visceral fat (Despres et al., 2000; Gibson, 2005). Women have a higher amount of body fat than men, and following menopause, their body fat distribution shifts to the abdomen (Bhasin, Woodhouse, & Storer, 2003; Douchi et al., 2003; Gibson, 2005). Hispanic and White men have greater total visceral adipose tissue than African-American men (Carroll et al., 2008; Despres et al., 2000), and Hispanic and White women have greater total adipose tissue than African-American women (Despres et al., 2000).

CLINICAL AND BIOLOGICAL DOMAIN

Abdominal obesity is associated with hypertension; dyslipidemia, which is manifested by elevated triglycerides, low high-density lipoproteins (HDL; Carr & Brunzell, 2004), and normal or slightly elevated LDL cholesterol levels (Ginsberg, Zhang, & Hernandez-Ono, 2006); and chronic health conditions (NIH, 1998). The mechanisms linking central adiposity and hypertension are not well understood, however, insulin resistance and the resulting hyperinsulinemia associated with abdominal obesity may interfere with insulin-mediated vascular signaling pathways inhibiting vasorelaxation (Reisin, 2008).

Underlying biologic mechanisms may increase an individual's risk of abdominal obesity. The hypothalamic-pituitary-adrenocortical (HPA) axis plays a role in glucose metabolism and energy regulation (Akil et al., 1999). Cortisol, an objective measure of biologic stress and indicator of HPA activity, is released in healthy adults in a daily rhythm, with a peak at approximately 07:00 a.m. and a trough between 07:00 p.m. and midnight. The diurnal pattern consists of an early morning peak (approximately 30–45 minutes after awakening), a rapid decline over the next few hours, followed by a gradual decline over the rest of the day, with cortisol reaching the lowest levels by bedtime. In women, this diurnal pattern is affected by menstrual cycle, contraceptive use, and menopause status (Duclos, Marquez Pereira, Barat, Gatta, & Roger, 2005). An association between cortisol secretion and abdominal obesity has been shown in men but not in women. In a study of 999 men ages 40–79 years, there was a weak negative correlation between morning cortisol level and WC (Travison, O'Donnell, Araujo, Matsumoto, & McKinlay, 2007). In a study of adults aged

47–59 years, controlling for age and socioeconomic status, an elevated cortisol response to awakening (30 minutes after awakening—wakening value) and a higher average cortisol over the day (30 minutes after waking—minimum evening value) were associated with abdominal obesity, as measured by waist–hip ratio, among middle-aged men but not postmenopausal women (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). High cortisol levels have been linked to cardiometabolic risk factors (for instance, elevated morning cortisol levels are associated with glucose intolerance, hypertension, and dyslipidemia; Reynolds & Walker, 2007), thus may be related to abdominal obesity.

PSYCHOLOGICAL DOMAIN

Psychological distress may increase risk of abdominal obesity through biologic and/or behavioral mechanisms (Cowen, 2002). Adults with current depressive symptoms or with a lifetime diagnosis of depression or anxiety are more likely to be obese than adults without either disorder (Strine et al., 2008). Among obese individuals, female gender is the only factor that has been consistently associated with an increased risk of depression, suggesting that obesity and depression may be linked by gender-specific mechanisms (Ma & Xiao, 2010). However, the relationship between depressive symptoms and obesity may be bidirectional. For example, the negative body image, social stigma, and discrimination experienced by many obese individuals may contribute to depression (Puhl & Brownell, 2003). Further, due to the impaired concentration often associated with depression, these individuals may be less able to adhere to dietary and physical activity recommendations (De Wit, Van Straten, Van Herten, Penninx, & Cuijpers, 2009). Results of a recently published cross-sectional study show that in a nationally representative sample of U.S. women, depressive symptoms were positively associated with abdominal obesity as measured by WC, independent of BMI (Ma & Xiao, 2010). Similarly, findings from a longitudinal study of adults showed those with more depressive symptoms at baseline compared to those with fewer depressive symptoms had a faster rate of increase in WC over time (Needham, Epel, Adler, & Kiefe, 2010).

BEHAVIORAL DOMAIN

Overweight individuals report that stress and depressive symptoms can contribute to unhealthy dietary patterns, which may lead to further weight gain and abdominal obesity (Chambers & Swanson, 2006). Negative emotions such as anger and sadness are associated with irregular eating patterns and eating as a distraction, to relax or to feel better (Macht & Simons, 2000). Stress is associated with cravings for high fat, high carbohydrate food, particularly among women (Epel, Lapidus, McEwen, & Brownell, 2001). Depressive symptoms have been associated with increased appetite, excess food intake, preferences for high fat and high carbohydrate foods, and excess alcohol intake, behaviors which contribute to a high BMI in women (Benton, 2002; Siegel, Yancey, & McCarthy, 2000; Strine et al., 2008).

Although dietary patterns may contribute to abdominal obesity, findings from a prospective, longitudinal study of adults aged 50–64 years showed no correlation between total energy intake (kcal) and increase in WC over 5 years (Halkjaer, Tjonneland, Thomsen, Overvad, & Sorensen, 2006), however, macronutrient and macronutrient subgroups were important. For example, there was an inverse relationship between protein intake and 5-year change in WC. Furthermore, a higher intake of protein from animal sources as compared to vegetable sources was associated with a smaller increase in WC. Additionally, among women, there was a positive association between the consumption of foods composed of carbohydrates from refined grains and potatoes and from foods composed of simple or added sugars and an increase in WC over 5 years. In contrast, higher fruit and vegetable intake was associated

with a smaller increase in WC. Findings from a cross-sectional study in a nationally representative sample of 6,988 adults showed that the consumption of energy dense diets, those that are high in fat and simple carbohydrates, was correlated with elevated WC in women and men (Mendoza, Drewnowski, & Christakis, 2007).

ABDOMINAL OBESITY AND CARDIOMETABOLIC DISEASE RISK

Excess abdominal adipose tissue contributes to increased cardiometabolic disease risk through several underlying biologic mechanisms involving glucose metabolism. Visceral adipose tissue is considered to be more metabolically active than subcutaneous adipose tissue, and it may have an enhanced role because it is connected to the liver via the portal circulation, which delivers free fatty acids directly into the liver. Elevated levels of free fatty acids in turn impair insulin signaling and inhibit glucose transport into skeletal muscle, thus contributing to insulin resistance (Boden & Shulman, 2002). Furthermore, visceral adipose conveys the greatest health risk because of the production of hormones that contribute to insulin resistance and cardiometabolic disease, including interleukin-6 (IL-6), tumor necrosis factor- α (TNF- α), and plasminogen activator inhibitor-1 (PAI-1; Greenberg & Obin, 2006). IL-6 inhibits insulin and leptin signaling and decreases adiponectin secretion; TNF- α acts to inhibit glucose uptake and fatty acid oxidation, and it increases the expression of genes involved in de novo synthesis of cholesterol and fatty acids. TNF- α interferes with insulin signaling, which inhibits the uptake of glucose by multiple tissues (Kershaw & Flier, 2004), and PAI-1 inhibits fibrinolysis, contributing to the development of atherosclerosis (Darmon et al., 2006).

Adiponectin, an adipose tissue hormone, regulates lipid and carbohydrate metabolism and protects against cardiovascular disease. Low adiponectin levels are associated with low-HDL levels, high-triglyceride levels, and insulin resistance (Cnop et al., 2003; Havel, 2004). Small adipocytes secrete adiponectin and insulin sensitizing hormone; in contrast, large adipocytes, associated with obesity, undersecrete insulin sensitizing hormone, and oversecrete insulin resistance hormone, thus contributing to insulin resistance (Meier & Gressner, 2004). In addition, cortisol inhibits adiponectin synthesis, further exposing individuals to increased risk of insulin resistance and cardiovascular disease (Fallo et al., 2004).

In summary, the central distribution of adipose tissue contributes to increased cardiometabolic risk over and above that of generalized adiposity. Therefore, identifying risk factors present in the subgroup of the overweight population with abdominal obesity is important. Although researchers have shown that factors from the demographic domain play a role in determining how body fat is distributed, less is known about the role of risk factors from the clinical and biological, psychological, and behavioral domains. The purpose of this study was to examine differences in multiple risk factors across domains among overweight individuals with and without abdominal obesity. The research questions were: Do overweight adults with abdominal obesity differ from those without abdominal obesity by: (a) age, gender, or race (demographic domain); (b) morning cortisol levels, self-reported hypertension, cholesterol levels, chronic health conditions, and BMI (clinical and biological domain); (c) depressive symptoms and perceived stress (psychological domain); and (d) caloric and macronutrient intake (behavioral domain)?

METHODS

Design

A prospective cross-sectional study design was used to determine if there were significant differences in demographic, clinical and biological, psychological, and behavioral domains

between overweight adults with and without abdominal obesity and cardiometabolic disease risk.

Sample

Eligible participants were overweight adults. The inclusion criteria were: age 18–65 years, BMI $\geq 25 \text{ kg/m}^2$, and able to read and speak English. The exclusion criteria were: engaged in physical activity for ≥ 90 minutes per week; pregnant; breastfeeding; type 1 or type 2 diabetes; engaged in weight loss activities; being treated with steroids, valproic acid, phenothiazines, highly active anti-retroviral therapy, or antidepressants; or history of liposuction, bariatric, or abdominal surgery.

Participants were recruited from community organizations including a university, local businesses, and neighborhood churches, after approval of the protocol and consent by the university Institutional Review Board. Multiple active and passive strategies were used to identify potential participants, such as recruiting adults at community wellness fairs and posting flyers in community settings. Potential participants contacted the principal investigator by telephone, at which time a screening protocol was followed to ensure that participants met the inclusion/exclusion criteria. Potential participants were asked their height and weight to determine if their BMI was $\geq 25 \text{ kg/m}^2$. If eligible, interested participants were met at their home, workplace, or in the research office at the School of Nursing.

The majority of participants were self-referrals (80.2%), and less than a quarter were recruited from health fairs (19.8%). Ninety-one adults consented to participate in the study. Four participants were excluded from data analysis: two participants were unable to complete the study activities, and two were considered extreme outliers (reporting caloric intake > 3 standard deviations below the mean). The final sample ($N=87$) was primarily women ($n = 64$, 73.6%), and African-American ($n = 44$, 50.6%; see Table 1). The majority of women were premenopausal ($n = 46$, 71.9%). The majority of participants had a college degree or higher level of education ($n = 51$, 58.6%), and almost half reported a total combined family income of less than \$60,000 per year ($n = 41$; 47.0%).

Variables and Measures

Demographic domain—Age, gender, race, income, and level of education were obtained by self-report.

Clinical and biological domain—Menopause status, health history including the cardiometabolic risk factors of hypertension, dyslipidemia (NIH, 1998), and chronic health conditions, including asthma, arthritis, angina, heart failure, stroke, and cancer, were obtained by self-report. A chronic health conditions score was calculated as the total number of self-reported health conditions. The number of chronic health conditions was categorized as 0 or ≥ 1 (Ma & Xiao, 2010).

Biologic stress was objectively assessed with a morning salivary cortisol level. A 1 ml sample of saliva was collected using a sampling device called a salivette (Sarstedt, Rommelsdorf, Germany). The salivette consists of a small cotton swab inside a centrifuge tube. Participants were instructed, using a standardized protocol including a demonstration and return demonstration, to collect the saliva sample according to the manufacturers' guidelines. Participants were instructed not to eat, drink, smoke, brush teeth, or chew gum for 30 minutes prior to using the salivette, and they were instructed to collect the sample by lightly chewing on the cotton swab for 3–4 minutes to ensure adequate saturation. The

sample was collected upon awakening before getting out of bed. Participants were provided with a kit including the salivette, a sample collection card, and written instructions.

Saliva cortisol represents the free fraction of cortisol in plasma, and it is highly correlated with plasma cortisol ($r=.91$, $p<.0001$; Salimetrics, 2009). The samples were analyzed using an enzyme immunoassay with an average intra-assay coefficient of variation of 8.7% for low and 5.2% for high concentration, and an average inter-assay coefficient of variation of 2.9% for low and 4.4% for high concentration (Salimetrics).

Anthropometric measures included height, weight, and WC assessed using the National Health and Nutrition Examination Survey anthropometric protocols (National Health and Nutrition Examination Survey (NHANES), 2007). Measured height and weight were used to calculate BMI. WC was measured three times, and the mean was used in the analyses. The intrarater reliability values for height, weight, and WC were .99, .99, and .96, respectively.

Psychological domain—The Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) was used to measure perceived stress. The PSS consists of a 10-item questionnaire that measures the degree to which an individual feels his/her life has been unpredictable or overwhelming in the past month. Possible scores range from 0 to 40, and higher scores on this instrument indicate higher levels of perceived stress. The Cronbach's α ranges from .84 to .86, indicating that the instrument is internally reliable (Cohen et al., 1983). The Cronbach's α in this sample was .89.

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996), a 21-item questionnaire, was used to measure depressive symptoms over the past 2 weeks. Possible scores range from 0 to 63, and higher scores indicate higher levels of depressive symptoms. In a clinical sample, the Cronbach's α was .92, indicating that the instrument is internally reliable (Beck et al.). The Cronbach's α in this sample was .91.

Behavioral domain—Participants weighed their food using the Soehnle 67000 digital food scale (Soehnle Professional GmbH & Co. KG, Murrhardt, Germany), and they recorded all food ingested as well as preparation methods on a standardized food diary for 3 consecutive days including 1 weekend day. The weighed food record is the gold standard in dietary assessment. It is considered the most precise method of estimating the usual food and nutrient intake of individuals as well as the preferred method for correlating intake with biologic measures (Gibson, 2005). Mean caloric intake estimates obtained from 3-day food records are considered to be more accurate than estimates obtained from other forms of dietary assessment, including food frequency questionnaires or 24-hour dietary recalls (De Castro, 1994; Gibson, 2005). The food records were analyzed by a research dietitian for caloric intake (kcal), and macronutrient intake including total and saturated fats (g), total carbohydrates (g), total sugars (g), and protein (g) using the Food Processor SQL Nutrition analysis software (ESHA Research, Salem, OR).

Procedure

Two study visits were completed within a 1-week time period. During the initial visit, the research nurse explained the study procedures and obtained the participant's consent. A standardized protocol was used to teach participants how to weigh and measure their food, how to keep a food record, and how to collect and store the saliva sample. Anthropometric measurements were taken by a trained research nurse using the NHANES procedures (NHANES, 2007). At home, participants completed the study questionnaires and recorded their food intake for 3 consecutive days. A second visit was scheduled at which the research nurse reviewed the questionnaires for completeness, and using a standardized protocol, reviewed the food record to clarify details with the participant. Participants returned the

saliva collection kit, and the research nurse stored the sample in an insulated bag, reviewing the sample collection card with the participant for completeness and accuracy.

Following the second visit, the research nurse hand delivered the saliva sample to the General Clinical Research Center laboratory where it was labeled with the participant's study identification number and the date and time of collection. Samples were centrifuged at 1,000 times gravity for 2 minutes and stored at -20°C . The analysis was performed at the Yerkes National Primate Research Center Biomarkers Core Laboratory using a commercially prepared kit produced by Diagnostic Systems Laboratories (Webster, TX).

After the second visit the food record was reviewed by the study dietitian. If additional information was needed to analyze the food record, the research nurse contacted the participant, obtained the necessary information, and conveyed this information to the dietitian. The dietitian then analyzed the food record, and these data were saved into an Excel spreadsheet and imported into Statistical Package for the Social Sciences-PC (SPSS, Chicago, IL). Using the Harris-Benedict (Harris & Benedict, 1919) equation, which takes age, gender, height, and activity level (participants were assumed to be inactive) into account, the dietitian calculated the estimated energy expenditure for each participant. The estimated energy expenditure indicates the caloric intake needed to maintain current body weight. To determine the extent of underconsuming and/or underreporting, the dietitian compared the estimated energy intake to the mean reported caloric intake: if estimated energy intake was greater than the mean reported caloric intake, the participant either underreported or underconsumed. The percent of those underreporting or underconsuming is reported. After the second visit, the research nurse scored the BDI-II questionnaire. If the BDI-II score indicated moderate-to-severe depressive symptoms, a standardized protocol was followed to ensure that the participant was referred to mental health services.

Data Analysis

Power analysis and sample size (PASS; Number Cruncher Statistical System (NCSS), Kaysville, UT) 2005 software was used to determine a priori that a sample size of 84 was needed to achieve a power of .80 with an alpha of .05 for an independent t -test detecting a $0.3\text{ }\mu\text{g/dl}$ difference in salivary cortisol levels between those with and without WC risk. The power calculation was based on these assumptions: an equal number of participants with and without WC risk would be enrolled, there would be an equal standard deviation (0.5) between the two groups, and a moderate (0.6) effect size would be observed. Ninety-one participants were recruited to allow for an attrition rate of 7.7% in completing the study activities.

Data were analyzed using SPSS version 15.0 software. Descriptive statistics, Pearson's and point-biserial correlations, chi-squared, t -tests, and Mann-Whitney U -test statistics were used to describe the sample and determine significant differences in order to answer the four research questions. The Mann-Whitney U -test was deemed more appropriate for some variables than the independent t -test to detect differences between those with and without WC risk: the distribution of the data did not meet the normality assumptions of parametric testing (Hintze, 2006).

RESULTS

Demographic Domain

For the total sample, the mean WC was 103.2 ($SD = 14.8$) cm. By gender, the mean WC was 103.4 ($SD = 12.7$) cm and 103.2 ($SD = 14.9$) cm, for men and women, respectively. The majority of participants ($n = 64$, 73.6%) had WC risk, with more women at risk than men

(Table 1). Individuals with WC risk ($M = 42.6$, $SD = 9.9$ years) were significantly older than those without WC risk ($M = 37.8$, $SD = 10.4$ years; $t[85] = -1.994$, $p = .05$).

Clinical and Biological Domain

A small number of participants reported dyslipidemia ($n = 13$, 14.9%), and more than a fifth reported hypertension ($n = 18$, 22.2%). Regarding chronic health conditions, the majority of participants ($n = 75$, 86.2%) reported 0 chronic conditions, and a few ($n = 12$, 13.8%) reported 1–2 chronic conditions. Chi-squared analyses were not appropriate because at least one cell in each table had an expected cell count of less than 5. For the total sample, the mean BMI was 32.1 ($SD = 6.1$) kg/m². By gender, the mean BMI was 30.9 ($SD = 4.5$) kg/m and 32.5 ($SD = 6.5$) kg/m, for men and women, respectively. Participants with WC risk had higher BMIs than those without WC risk (Table 1). BMI was positively correlated with WC (Table 2).

The majority of participants ($n = 64$, 81.0%) had mean morning salivary cortisol levels ($M = 0.68$ µg/dl, $SD = 0.45$ µg/dl) within the normal reference range (0.18–0.95 µg/dl; Salimetrics, 2009). Premenopausal women ($Mdn = 0.63$ µg/dl, range 0.15–2.21 µg/dl) did not differ from postmenopausal women ($Mdn = 0.49$ µg/dl, range 0.18–1.33 µg/dl). Men ($Mdn = 0.51$ µg/dl, range 0.08–2.0 µg/dl) did not differ from women ($Mdn = 0.59$ µg/dl, range 0.15–2.21 µg/dl) on median morning salivary cortisol levels. Because there were no differences based on gender and menopause status, the data were pooled for the analysis. Although cortisol levels were normal (within the reference range), individuals with WC risk had median morning salivary cortisol levels that were higher than those without WC risk (Table 1). A post hoc power analysis using a two-sided Mann–Whitney test assuming a logistic distribution demonstrated that the group sample sizes achieved 53% power to detect a 0.21 µg/dl difference in morning cortisol levels between the group means.

Psychological Domain

In terms of bivariate correlations, perceived stress was positively correlated with WC and depressive symptoms were positively correlated with WC (Table 2). Overall, the median PSS score was 16.0 (range 0–33.0). Individuals with WC risk ($Mdn = 18.0$, range 0–33.0) did not report higher PSS scores than those without WC risk. Overall, the median BDI-II score was 6.0 (range 0–42.0). Although, the majority of participants reported few depressive symptoms, 19 (21.8%) had BDI-II scores indicating mild to severe depressive symptoms (BDI-II scores > 13; Beck et al., 1996). Participants with depressive symptoms ($Mdn = 22.00$, range 11.0–33.0) reported significantly higher levels of stress compared to those without depressive symptoms ($Mdn = 15.00$, range 0–32.0; Mann–Whitney $Z = -3.647$, $p = .01$). In addition, participants with WC risk ($Mdn = 7.00$, range 0–42.0) reported significantly higher BDI-II scores than those without WC risk ($Mdn = 5.00$, range 0–21.0; Mann–Whitney $Z = -2.003$, $p = .05$). A post hoc power analysis using a two-sided Mann–Whitney test assuming a logistic distribution demonstrated that the group sample sizes achieved 77% power to detect a difference of 4.17 in BDI-II scores between the group means.

Behavioral Domain

Those with WC risk did not report a higher intake of kilocalories and macronutrients than those without WC risk (Table 3). The macronutrient distribution for overweight adults with WC risk compared to those without WC risk was 49.5% versus 48.0% carbohydrates, 35.0% versus 34.5% fat, and 15.5% versus 17.5% protein. The recommended macronutrient composition is 45–65% of calories to be derived from carbohydrates, 20–35% from fat, and 10–35% from protein sources (U. S. Department of Health and Human Services & U.S. Department of Agriculture, 2005).

DISCUSSION

The findings document demographic, clinical and biological, and psychological differences between those with and without WC risk. Almost three-quarters of all participants had a WC above the gender specific cutpoints, indicating that these individuals were at high to very high relative risk for obesity-associated cardiometabolic diseases, and more women were at risk than men. Those with WC risk were older than those without WC risk.

The results show a strong positive association between BMI and WC, which is consistent with data from a Canadian study showing strong positive correlations between BMI and WC in both men and women (Pouliot et al., 1994). Findings from the World Health Organization (WHO) survey MONItoring trends and determinants in CARdiovascular disease (MONICA) using pooled data from 19 male and 18 female populations showed that BMI explained 77% and 75% of WC variance in men and women, respectively (Molarius, Seidell, Sans, Tuomilehto, & Kuulasmaa, 1999).

Hypertension, reported by 25% of the participants with WC risk, indicates many had substantial risk for cardiovascular disease progression. Although the mechanisms linking hypertension and abdominal obesity are not well understood, the adipose tissue hormones TNF- α , IL-6, PAI-1, and C-reactive protein play a role in initiating and sustaining the low-grade inflammation (endothelial dysfunction and oxidative stress) associated with the progression of hypertension and arteriosclerosis (Reisin, 2008).

The PSS scores indicated that the participants reported a level of stress comparable to that of other working adults (Brunner, 1997). The majority of participants were well educated and reported incomes of \$60,000 or above, suggesting that they had access to resources, such as health information, that could mitigate the negative effects of stress due to weight issues.

The BDI-II scores suggest that the group as a whole was not clinically depressed (Beck et al., 1996). However, 21.8% ($n = 19$) were considered to have mild-to-severe depressive symptoms which is higher than the 4–5% reported in the general population, and is similar to the prevalence reported among adults with chronic conditions (Egede, 2007). Participants who had more depressive symptoms also reported greater perceived stress. Those with WC risk had more depressive symptoms than those without WC risk.

Although cortisol levels were within the normal reference range, individuals with WC risk had significantly higher morning cortisol levels compared to those without WC risk. This subtle shift within the normal reference range is a concern because previous research suggests that higher morning cortisol levels are associated with cardiovascular risk factors and disease (Reynolds & Walker, 2007). For example, Bhattacharyya, Malloy, and Steptoe (2008) found morning cortisol levels were higher among individuals with coronary artery disease (CAD) than those without CAD.

Individuals with abdominal obesity may be more susceptible to HPA axis dysregulation due, in part, to the high prevalence of glucocorticoid receptors and the high-lipoprotein lipase (LPL) activity in abdominal adipose tissue (Bjorntorp, 1991). Cortisol and insulin stimulate LPL activity; within the adipose tissue capillary walls, LPL breaks down the triglyceride contained in its carrier lipoprotein. The adipocytes take up the free fatty acids, reassembling them into triglyceride for storage (Duclos et al., 2005; Qi & Rodrigues, 2007). In addition, cortisol stimulates the sympathetic nervous system to release neuropeptide Y which acts in the periphery to facilitate adipose tissue growth through angiogenesis, as well as the proliferation and differentiation of new adipocytes (Kuo et al., 2007). Cortisol, in processes mediated by LPL and neuropeptide Y, contributes to abdominal adiposity and increased cardiometabolic disease risk. Because higher cortisol levels may be linked to cardiovascular

disease, understanding the factors contributing to elevated cortisol levels in those with abdominal obesity will better enable healthcare providers to target interventions aimed at decreasing cardiometabolic disease risk.

Caloric intake and macronutrients did not differ significantly between those with and without WC risk, and the reported macronutrient composition fell within the USDA recommended guidelines. Underreporting is a challenge in dietary assessment, and despite rigorous methods to minimize underreporting caloric intake, over half of the participants (58.6%) either under consumed or underreported their caloric intake, potentially masking important relationships. This form of dietary assessment requires motivation, and it is possible that participants altered their dietary pattern to simplify the weighing and recording process resulting in under consuming on the study days (Gibson, 2005). Findings from a large nationally representative study showed that the mean energy intake for US adults is 2,146 kcal per day with 51.9% of calories derived from carbohydrates, 32.7% from fat, and 14.7% from protein (Wright, Wang, Kennedy-Stephenson, & Ervin, 2003). Based on this contextual information, the mean values for this sample (2,029.8 kcal and 49.0% from carbohydrates) suggest that participants under consumed and/or underreported their carbohydrate intake, which would affect the overall distribution of macronutrients.

The findings demonstrate associations of both depressive symptoms and morning cortisol levels with WC risk. Taking into account the nonparametric adjustment, the observed power was 77% to detect differences in depressive symptoms between those with and without WC risk, and indicates with reasonable confidence that a type II error has not been committed. Although there was moderate power (53%) to detect differences between morning cortisol level and WC risk, the power was lower than anticipated due to several factors: the actual difference between the group means (effect size) was smaller than anticipated and the standard deviation of the cortisol values was higher than expected. By collecting saliva samples over multiple days in future research, variability would be minimized, thus improving the observed power. Larger sample sizes and more rigorous cortisol sampling protocols also are needed to examine the relationship between morning cortisol values and WC risk.

A limitation of the study is the use of convenience sampling to enroll participants, with the result that the final sample was biased toward well-educated adults with relatively high incomes. In addition, the study activities required highly motivated participants, and people who chose to enroll may differ from non-participants in motivation. The use of the cross-sectional design limits the ability to address direction or causality of the relationship found between abdominal obesity and both depressive symptoms and morning cortisol levels. Due to problems with inadequate specimen collection, salivary cortisol data were missing on 10.3% of the sample, which also contributed to low power. Depressive symptoms were assessed using a self-report questionnaire, and participants may have over or under reported their depressive symptoms. There may be other important variables, such as previous episodes of depression and comorbid anxiety, which were not measured yet confounded relationships among study variables (Strine et al., 2008; Ma & Xiao, 2010). Finally, measured WC was used to assess abdominal obesity, and these anthropometric data were not triangulated with other methods of assessing body composition.

Implications

Nurses and other healthcare providers, in a variety of settings and from a variety of specialties, should be aware that depressive symptoms are associated with abdominal obesity (Ma & Xiao, 2010; Needham et al., 2010). The temporal sequence of this association has not been established (Needham et al.), but early identification of and intervention for those at risk for depressive symptoms may improve prevention and treatment of

cardiometabolic disease (Strickland et al., 2002), although this needs to be tested in clinical trials. Mild-to-moderate depressive illnesses are common, yet are often undetected and untreated (Egede, 2007). Nurses could use assessment of abdominal obesity to identify individuals at risk of depressive symptoms and refer these individuals to qualified mental health professionals. The Patient Health Questionnaire 2, a two-item questionnaire, is a short tool that can be administered during brief office encounters to screen adults for depression (Whooley & Simon, 2000). If an individual answers yes to one or both questions, further assessment and follow up is recommended.

Routinely incorporating anthropometric measures such as WC into health assessments requires careful measures of height and weight to calculate an accurate BMI. By providing patients with accurate information about their BMI and WC, they are better informed about their cardiometabolic disease risk. This may be of particular importance among individuals with normal BMI, as the risk of mortality in individuals with normal BMIs and elevated WC is 20% higher than that of individuals with normal BMIs and normal WC (Koster et al., 2008). Furthermore, communicating personalized risk information to patients has been associated with risk-reducing behaviors (Roach & Marrero, 2005), and lifestyle modification is important among adults with WC risk.

In summary, these findings showed subtle differences in overweight adults with abdominal obesity compared to those without abdominal obesity in age and gender (demographic domain), morning cortisol levels and BMI (clinical and biological domain), and depressive symptoms (psychological domain), but not in the behavioral domain. Further research is needed to better understand how the biological and psychological risk factors interact and contribute to abdominal obesity. This may lead to the development and testing of targeted interventions aimed at preventing and reducing abdominal obesity, thereby reducing cardiometabolic disease risk.

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REFERENCES

- Akil, H.; Campeau, S.; Cullinan, WE.; Lechan, RM.; Toni, R.; Watson, SJ.; Moore, RY.
Neuroendocrine systems I: Overview—Thyroid and adrenal axes. In: Zigmond, MJ.; Bloom, FE.;

- Landiz, SC.; Roberts, JL.; Squire, LR., editors. Fundamental neuroscience. San Diego, CA: Academic Press; 1999. p. 1127-1150.
- Bhattacharyya MR, Molloy GJ, Steptoe A. Depression is associated with flatter cortisol rhythms in patients with coronary artery disease. *Journal of Psychosomatic Research*. 2008; 65:107–113. [PubMed: 18655854]
- Beck, AT.; Steer, RA.; Brown, GK. BDI-II, 2nd edn.. San Antonio, TX: The Psychological Association; 1996. Manual for the Beck Depression Inventory.
- Benton D. Carbohydrate ingestion, blood glucose and mood. *Neuroscience and Biobehavioral Reviews*. 2002; 26:293–308. [PubMed: 12034132]
- Bhasin S, Woodhouse L, Storer TW. Androgen effects on body composition. *Growth Hormone and Igf Research*. 2003; 13(Suppl A):S63–S71. [PubMed: 12914730]
- Bjorntorp P. Adipose tissue distribution and function. *International Journal of Obesity*. 1991; 15(Suppl 2):67–81. [PubMed: 1794941]
- Boden G, Shulman GI. Free fatty acids in obesity and type 2 diabetes: Defining their role in the development of insulin resistance and beta-cell dysfunction. *European Journal of Clinical Investigation*. 2002; 32(Suppl 3):14–23. [PubMed: 12028371]
- Brunner, RL. The Perceived-Stress Scale. In: Jeor, SSt, editor. Obesity assessment: Tools, methods, interpretations: A reference case: The RENO Diet-Heart study. New York, NY: Chapman & Hall; 1997. p. 471-478.
- Carr MC, Brunzell JD. Abdominal obesity and dyslipidemia in the metabolic syndrome: Importance of type 2 diabetes and familial combined hyperlipidemia in coronary artery disease risk. *Journal of Clinical Endocrinology and Metabolism*. 2004; 89:2601–2607. [PubMed: 15181030]
- Carroll JF, Chiapa AL, Rodriquez M, Phelps DR, Cardarelli KM, Vishwanatha JK, Cardarelli R. Visceral fat, waist circumference, and BMI: Impact of race/ethnicity. *Obesity (Silver Spring, MD)*. 2008; 16:600–607.
- Chambers JA, Swanson V. A health assessment tool for multiple risk factors for obesity: Results from a pilot study with UK adults. *Patient Education and Counseling*. 2006; 62:79–88. [PubMed: 16431069]
- Cnop M, Havel PJ, Utzschneider KM, Carr DB, Sinha MK, Boyko EJ, Kahn SE. Relationship of adiponectin to body fat distribution, insulin sensitivity and plasma lipoproteins: Evidence for independent roles of age and sex. *Diabetologia*. 2003; 46:459–469. [PubMed: 12687327]
- Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *Journal of Health and Social Behavior*. 1983; 24:385–396. [PubMed: 6668417]
- Cowen PJ. Cortisol, serotonin and depression: All stressed out? *British Journal of Psychiatry*. 2002; 180:99–100. [PubMed: 11823315]
- Darmon P, Dadoun F, Boullu-Ciocca S, Grino M, Alessi MC, Dutour A. Insulin resistance induced by hydrocortisone is increased in patients with abdominal obesity. *American Journal of Physiology-Endocrinology and Metabolism*. 2006; 291:E995–E1002. [PubMed: 16772320]
- De Castro JM. Methodology, correlational analysis, and interpretation of diet diary records of the food and fluid intake of free-living humans. *Appetite*. 1994; 23:179–192. [PubMed: 7864611]
- Despres JP, Couillard C, Gagnon J, Bergeron J, Leon AS, Rao DC, Bouchard C. Race, visceral adipose tissue, plasma lipids, and lipoprotein lipase activity in men and women: The Health, Risk Factors, Exercise Training, and Genetics (HERITAGE) family study. *Arteriosclerosis, Thrombosis and Vascular Biology*. 2000; 20:1932–1938.
- De Wit LM, Van Straten A, Van Herten M, Penninx BW, Cuijpers P. Depression and body mass index, a U-shaped association. *BMC Public Health*. 2009; 9:14. [PubMed: 19144098]
- Douchi T, Kosha S, Uto H, Oki T, Nakae M, Yoshimitsu N, Nagata Y. Precedence of bone loss over changes in body composition and body fat distribution within a few years after menopause. *Maturitas*. 2003; 46:133–138. [PubMed: 14559384]
- Duclos M, Marquez Pereira P, Barat P, Gatta B, Roger P. Increased cortisol bioavailability, abdominal obesity, and the metabolic syndrome in obese women. *Obesity Research*. 2005; 13:1157–1166. [PubMed: 16076984]

- Egede LE. Major depression in individuals with chronic medical disorders: Prevalence, correlates and association with health resource utilization, lost productivity and functional disability. *General Hospital Psychiatry*. 2007; 29:409–416. [PubMed: 17888807]
- Epel E, Lapidus R, McEwen B, Brownell K. Stress may add bite to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology*. 2001; 26:37–49. [PubMed: 11070333]
- Expert Panel on the Detection Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001; 285:2486–2497. [PubMed: 11368702]
- Fallo F, Scarda A, Sonino N, Paoletta A, Boscaro M, Pagano C, Vettor R. Effect of glucocorticoids on adiponectin: A study in healthy subjects and in Cushing's syndrome. *European Journal of Endocrinology*. 2004; 150:339–344. [PubMed: 15012619]
- Friedman MA, Brownell KD. Psychological correlates of obesity: Moving to the next research generation. *Psychological Bulletin*. 1995; 117:3–20. [PubMed: 7870862]
- Gibson, RS. 2nd edn.. New York, NY: Oxford University Press; 2005. Principles of nutritional assessment.
- Ginsberg HN, Zhang YL, Hernandez-Ono A. Metabolic syndrome: Focus on dyslipidemia. *Obesity* (Silver Spring, MD). 2006; 14(Suppl 1):41S–49S.
- Greenberg AS, Obin MS. Obesity and the role of adipose tissue in inflammation and metabolism. *The American Journal of Clinical Nutrition*. 2006; 83:461S–465S. [PubMed: 16470013]
- Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Costa F. Diagnosis and management of the metabolic syndrome: An American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Current Opinion in Cardiology*. 2006; 21:1–6. [PubMed: 16355022]
- Halkjaer J, Tjonneland A, Thomsen BL, Overvad K, Sorensen TI. Intake of macronutrients as predictors of 5-y changes in waist circumference. *The American Journal of Clinical Nutrition*. 2006; 84:789–797. [PubMed: 17023705]
- Harris, JA.; Benedict, FG. A biometric study of basal metabolism in man. Washington, DC: Carnegie Institute of Washington; 1919.
- Havel PJ. Update on adipocyte hormones: Regulation of energy balance and carbohydrate/lipid metabolism. *Diabetes*. 2004; 53(Suppl 1):S143–S151. [PubMed: 14749280]
- Hintze, JL. Kaysville, UT: Statistical and Power Analysis Software; 2006. Power analysis and sample size system (PASS) user's guide.
- Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. *Journal of Clinical Endocrinology and Metabolism*. 2004; 89:2548–2556. [PubMed: 15181022]
- Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL. Indices of relative weight and obesity. *Journal of Chronic Diseases*. 1972; 25:329–343. [PubMed: 4650929]
- Koster A, Leitzmann MF, Schatzkin A, Mouw T, Adams KF, Van Eijk JT, Harris TB. Waistcircumference andmortality. *AmericanJournal of Epidemiology*. 2008; 167:1465–1475. [PubMed: 18417494]
- Kuo LE, Kitlinska JB, Tilan JU, Li L, Baker SB, Johnson MD, Zukowska Z. Neuropeptide Y acts directly in the periphery on fat tissue and mediates stress-induced obesity and metabolic syndrome. *Nature Medicine*. 2007; 13:803–811.
- Li C, Ford ES, McGuire LC, Mokdad AH. Increasing trends in waist circumference and abdominal obesity among US adults. *Obesity* (Silver Spring, MD). 2007; 15:216–224.
- Ma J, Xiao L. Obesity and depression in US women: Results from the 2005–2006 National Health and Nutritional Examination Survey. *Obesity* (Silver Spring, MD). 2010; 18:347–353.
- Macht M, Simons G. Emotions and eating in everyday life. *Appetite*. 2000; 35:65–71. [PubMed: 10896762]
- Meier U, Gressner AM. Endocrine regulation of energy metabolism: Review of pathobiochemical and clinical chemical aspects of leptin, ghrelin, adiponectin, and resistin. *Clinical Chemistry*. 2004; 50:1511–1525. [PubMed: 15265818]

- 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. [PubMed: 17229942]
- Molarius A, Seidell JC, Sans S, Tuomilehto J, Kuulasmaa K. Waist and hip circumferences, and waist–hip ratio in 19 populations of the WHO MONICA Project. *International Journal of Obesity and Related Metabolic Disorders*. 1999; 23:116–125. [PubMed: 10078844]
- National Health and Nutrition Examination Survey. Anthropometry procedures manual. 2007. Retrieved September 13, 2010, from www.cdc.gov/nchs/data/nhanes/nhanes_07_08/manual_an.pdf
- National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The evidence report (National Institutes of Health (NIH) publication; No. 98–4083). Bethesda, MD: National Institutes of Health, National Heart Lung and Blood Institute; 1998.
- Needham BL, Epel ES, Adler NE, Kiefe C. Trajectories of change in obesity and symptoms of depression: The CARDIA Study. *American Journal of Public Health*. 2010; 100:1040–1046. [PubMed: 20395582]
- Pouliot MC, Despres JP, Lemieux S, Moorjani S, Bouchard C, Tremblay A, Lupien PJ. Waist circumference and abdominal sagittal diameter: Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *The American Journal of Cardiology*. 1994; 73:460–468. [PubMed: 8141087]
- Puhl RM, Brownell KD. Psychosocial origins of obesity stigma: Toward changing a powerful and pervasive bias. *Obesity Reviews*. 2003; 4:213–227. [PubMed: 14649372]
- Qi D, Rodrigues B. Glucocorticoids produce whole body insulin resistance with changes in cardiac metabolism. *American Journal of Physiology, Endocrinology and Metabolism*. 2007; 292:E654–E667. [PubMed: 17077342]
- Reisin, E. Obesity-related hypertension. In: Izzo, JL.; Sica, DA.; Black, HR., editors. *Hypertension primer: The essentials of high blood pressure: Basic science, population science, and clinical management*. 4th edn. Philadelphia, PA: Lippincott Williams & Wilkins; 2008. p. 149–150.
- Reynolds RM, Walker BR. Can cortisol predict the future in obesity? *Clinical Endocrinology*. 2007; 67:1–2. [PubMed: 17466008]
- Roach P, Marrero D. A critical dialogue: Communicating with type 2 diabetes patients about cardiovascular risk. *Vascular Health and Risk Management*. 2005; 1:301–307. [PubMed: 17315602]
- Salimetrics. High sensitivity salivary cortisol enzyme immunoassay kit. 2009. Retrieved September 13, 2010 from <http://www.salimetrics.com/assets/documents/products-and-services/salivary-assays/Cort-D%20insert,%20booklet,%20for%20web,%2024%20p,%20no%20tp,%20rev%2010-09.pdf>
- Siegel JM, Yancey AK, McCarthy WJ. Overweight and depressive symptoms among African-American women. *Preventive Medicine*. 2000; 31:232–240. [PubMed: 10964637]
- Steptoe A, Kunz-Ebrecht SR, Brydon L, Wardle J. Central adiposity and cortisol responses to waking in middle-aged men and women. *International Journal of Obesity and Related Metabolic Disorders*. 2004; 28:1168–1173. [PubMed: 15211363]
- Strickland PL, Deakin JF, Percival C, Dixon J, Gater RA, Goldberg DP. Bio-social origins of depression in the community. Interactions between social adversity, cortisol and serotonin neurotransmission. *British Journal of Psychiatry*. 2002; 180:168–173. [PubMed: 11823330]
- Strine TW, Mokdad AH, Dube SR, Balluz LS, Gonzalez O, Berry JT, Kroenke K. The association of depression and anxiety with obesity and unhealthy behaviors among communitydwelling US adults. *General Hospital Psychiatry*. 2008; 30:127–137. [PubMed: 18291294]
- Travison TG, O'Donnell AB, Araujo AB, Matsumoto AM, McKinlay JB. Cortisol levels and measures of body composition in middleaged and older men. *Clinical Endocrinology*. 2007; 67:71–77. [PubMed: 17466009]
- U. S. Department of Health and Human Services & U.S. Department of Agriculture. *Dietary guidelines for Americans*, 2005. 6th edn.. Washington, DC: U.S. Government Printing Office; 2005.

- Whooley MA, Simon GE. Managing depression in medical outpatients. *New England Journal of Medicine*. 2000; 343:1942–1950. [PubMed: 11136266]
- Wright, JD.; Wang, CY.; Kennedy-Stephenson, J.; Ervin, RB. Dietary intake of ten key nutrients for public health, United States: 1999–2000. (Advance Data from Vital and Health Statistics, no. 334). Hyattsville, MD: National Center for Health Statistics; 2003.

Table 1

Median Values for Demographic, Clinical and Biological Differences Between Those With and Without Waist Circumference Risk (N = 87)

Variable	No WC Risk, <i>n</i> = 23	WC Risk, <i>n</i> = 64	Chi-Squared Statistic	Mann-Whitney Z
	Median (Range) or Count (%)	Median (Range) or Count (%)		
Gender			14.551 **	
Male	13 (56.5)	10 (43.5)		
Female	10 (15.6)	54 (84.4)		
Race			0.032	
AA	12 (27.3)	32 (72.7)		
White	11 (25.6)	32 (74.4)		
BMI (kg/m ²)	26.7 (24.6–33.4)	33.1 (25.1–52.0)		–4.914 **
Salivary cortisol (mg/dl)	0.41 (0.08–1.73)	0.59 (0.15–2.21)		–2.286 *
Waist circumference (cm)	89.63 (78.27–100.90)	105.37 (89.0–140.0)		–5.862 **

Note: WC, waist circumference; AA, African-American; BMI, body mass index.

* *p* .05.

** *p* .01.

Table 2

Correlation Matrix (Pearson's or Biserial)

Variable	Gender	Age	BMI	PSS	BDI-II	Cortisol ^a	WC
Race	.14	-.16	.05	.01	.07	-.12	-.07
Gender	1	.17	.11	.03	.14	.10	-.01
Age		1	-.01	-.31**	-.09	.24**	.09
BMI			1	.23*	.31**	-.04	.86*
PSS				1	.60**	-.17	.23**
BDI-II					1	-.08	.25**
Cortisol ^a						1	.00
WC							1

Note: BMI, body mass index; PSS, perceived stress scale; BDI-II, Beck Depression Inventory; Cortisol, morning salivary cortisol value; WC, waist circumference.

^a *n* = 79.
* *p* .05.
** *p* .01.

Table 3

Median Macronutrient Values for Those With and Without Waist Circumference Risk (N = 87)

Variable	No WC Risk, n = 23	WC Risk, n = 64	Mann-Whitney Z
	Median (Range)	Median (Range)	
Energy (kcal)	1,855.5 (1,828.7)	1,966.9 (2,665.3)	-0.808
Total fat (g)	72.1 (118.8)	74.1 (149.1)	-0.731
Saturated fat (g)	20.2 (35.7)	23.4 (48.3)	-0.505
Total carbohydrates (g)	226.8 (350.8)	238.1 (504.0)	-1.001
Total sugars (g)	77.8 (197.9)	87.7 (256.4)	-1.410
Protein (g)	76.8 (153.6)	75.3 (118.0)	-0.703

Note: WC, waist circumference; kcal, kilocalories; g, grams. There were no significant group differences.