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# Examining the prevalence of metabolic syndrome among overweight/obese African-American breast cancer survivors vs. matched non-cancer controls

**Patricia Sheean**[1], **Huifang Liang**[2], **Linda Schiffer**[3], **Claudia Arroyo**[4], **Melinda Stolley**[5]

[1]Marcella Niehoff School of Nursing, Loyola University Chicago, Health Sciences Campus, 2160 South First Avenue, Building 120, Room 4527, Maywood, IL 60153, USA

[2]Takeda Development Center Americas, Inc., One Takeda Parkway, Deerfield, IL 60015, USA

[3]Division of Academic Internal Medicine and Geriatrics, University of Illinois at Chicago, 1747 West Roosevelt Road, Room 558, MC 275, Chicago, IL 60608, USA

[4]Division of Academic Internal Medicine and Geriatrics, University of Illinois at Chicago, 1747 West Roosevelt Road, Room 558, MC 275, Chicago, IL 60608, USA

[5]Department of Medicine, Medical College of Wisconsin, Milwaukee, WI, USA

## Abstract

**Purpose—**Metabolic Syndrome (MetS) is more predominant in overweight, obese and minority populations. This study examined the prevalence of MetS in an exclusively African-American (AA) cohort of breast cancer (BC) survivors; an underrepresented group in previous studies demonstrating negative BC outcomes disparities for females with MetS.

**Methods—**Using a case-control design, overweight/obese AA women with treated Stage I–IIIa BC were matched 1:1 on age, race, sex, and body mass index (BMI) category with non-cancer population controls ($n = 444$). Three of the following conditions were used to define MetS: HDL cholesterol <50 mg/dL (1.3 mmol/L), serum triglycerides ≥150 mg/dL (1.7 mmol/L), blood glucose ≥100 mg/dL (or on treatment), waist circumference ≥88 cm, or ≥130 mmHg systolic or ≥85 mmHg diastolic blood pressure (or on treatment). Matched-pairs analyses were conducted.

**Results—**For BC cases, most women had self-reported Stage I ($n = 76$) or Stage II ($n = 91$) disease and were 6.9 (±5.2) years post-diagnosis. MetS was significantly lower in BC survivors vs. their non-cancer population controls (43.2 vs. 51.4 %, respectively; $p < 0.05$). The diagnosis of MetS did not differ by BMI stratification. A lower prevalence of ≥2 risk factors (80.2 vs. 85.6 %, $p < 0.05$) was observed for all cases vs. controls.

**Conclusions—**While MetS occurred less frequently in our BC cases vs. non-cancer controls, our estimates are nearly two times those reported in other BC survivors, suggesting important racial/ethnic differences.

Patricia Sheean, psheean1@luc.edu.

**Conflict of interests** The authors have no conflict of interests to disclose regarding the conduct and report of this work.

**Implications for cancer survivors—**The prognostic implications of MetS among AA BC survivors remain unknown and warrant further investigation.

### Keywords

Breast cancer; Metabolic syndrome; African-American; Case control; Survivorship

## Introduction

In the USA, African Americans (AA) shoulder a disproportionate share of cancer burden. In general, AAs experience the shortest survival and subsequent highest death rate of any racial/ethnic group for most cancers, including breast cancer (BC). [1] Paradoxically, AA women have the second highest incidence of BC (124.3/100,000), yet they exhibit the lowest 5-year survival rate compared to women of all race/ethnicities (81 vs. 91 %, respectively) [2]. The causes of these BC outcome disparities are not yet fully understood and are likely multifactorial. A recent study by Akinyemiju et al. analyzed 456,217 females diagnosed with BC to determine the influence of area-level socioeconomic status, health care access and molecular subtype on individual BC stage, treatment, and mortality across different race/ethnicities [3]. These authors found AA women experienced significantly higher BC mortality compared to white women (hazard ratio 1.39, 95 % confidence interval 1.36–1.43), and although BC molecular subtype was a strong predictor of BC outcomes, area-level and health care access were also significant, independent predictors of BC outcomes disparities. Other investigators have suggested that these disparities may be due to more advanced stage of disease at diagnosis, limited treatment options due to molecular subtypes, lower endocrine therapy use and comorbid conditions, specifically obesity [4–6]. Because obesity is highly prevalent among AA women [7] and independently associated with BC progression and decreased survival [8, 9], it is considered a *modifiable* target to help decrease BC outcome disparities.

Obesity is associated with a constellation of metabolic factors now referred to as metabolic syndrome (MetS). MetS reflects a group of biochemical abnormalities and clinical conditions that increase the risk for multiple chronic diseases [10] and all-cause mortality [11]. It is prevalent in 23–24 % of the general US population, increases with age and displays variations by race/ethnicity [12, 13]. MetS has been shown to independently influence BC risk [14], and recent data shows that MetS also predicts recurrence and early death in women diagnosed and treated for BC [15]. However, these studies analyze AA BC survivors in aggregate, potentially masking important differences by race/ethnicity. These data are important to highlight since MetS may be an important, yet unrecognized factor in the observed survival disparities among BC survivors. The primary purpose of this study was to examine the prevalence of MetS among an exclusively AA cohort of BC survivors. Because AA populations tend to have greater comorbid conditions (e.g., obesity, HTN), we compared these BC survivors to age, sex, body mass index (BMI), and race-matched non-cancer population controls. This approach allowed us to assess the impact of BC treatment on the occurrence of MetS and to generate prevalence estimates to afford comparisons with other BC investigations.

## Methods

### Breast cancer cases

Participants included overweight/obese AA women recruited as part of a larger randomized controlled trial (R01CA154406). The goal of the larger RCT ($N$ = 244) was to recruit AA breast cancer survivors from communities within Chicago, Illinois for a 6-month cognitive behavioral weight loss intervention. Women were recruited from 2012 to 2015. To be included in these analyses, adult women: (1) self-identified as black or AA; 2) self-reported Stage I–III invasive breast carcinoma; (3) had completed initial treatment at least 6 months *prior to* recruitment (e.g., surgery, chemotherapy, and/or radiation; current adjuvant hormonal therapies acceptable); and (4) were overweight (BMI 25.0–29.9 kg/m$^2$) or obese (BMI ≥30.0 kg/m$^2$). If a potential participant reported plans for moving out the community during the study, were deemed unable to engage in physical activity by their own determination or that of their primary care physician, were diagnosed prior to 18 years of age, were currently pregnant, less than 3 months postpartum or anticipating pregnancy, reported drinking more than two drinks of alcohol/day on a daily basis, were on prescribed weight loss medications or enrolled in a formal weight loss program requiring special foods, had weight loss surgery in the past year or were planning weight loss surgery in the next year, they were excluded from participating in the RCT. The study was approved by the appropriate Institutional Review Boards.

Following an initial phone screen, potentially eligible women were then scheduled for a baseline interview, where the study was described in further detail and informed consent was obtained. At this time, women also completed study-related questionnaires, anthropometric measures (e.g., height and weight), and blood pressure assessment. These interviews and procedures were conducted by trained and study certified personnel to ensure valid and reliable measures. Within1 month of the baseline interview, women returned for phlebotomy and waist circumference measures. For these cases, all clinical data (e.g., blood pressure, lipid panel) were collected at baseline prior to participating in the weight loss intervention.

### Matched non-cancer population controls

A dataset of participants without cancer from the 2007–2012 National Health and Nutrition Examination Surveys (NHANES) was created for comparison using one to one matching on sex, age (within 5 years), race, and BMI [within the same classification category or ±5 kg/m$^2$ for class 3 obesity (BMI ≥40.0)]. AA women who had a BMI, waist circumference, serum triglyceride, serum high-density lipoprotein (HDL) cholesterol, fasting blood glucose, and systolic and diastolic blood pressure in the NHANES 2007–2012, but did not have a history of BC were eligible ($N$ = 473). If one BC case had more than one eligible control, then the record having the same sex, race, minimum age, and BMI difference with the BC case was selected as the control. To guarantee no duplication of non-cancer controls, a non-cancer record was deleted after being selected from the NHANES dataset, and no longer available for further matching. The dataset with non-cancer controls was vertically merged with the BC dataset prior to analyses.

## Study variables

The following variables were collected for all cases and controls, unless otherwise specified.

**Demographics—**Data regarding age, sex, race, level of education (high school or less, some college, college graduate, or graduate degree), employment (paid work, not working, out of work or other), health insurance (none, public, private, or other), and self-reported comorbid conditions were collected.

**Anthropometrics—**Height and weight were measured by trained personnel twice using standardized, calibrated equipment, and quality assurance measures were employed. If the two measurements were more than 0.5 cm or 0.2 kg apart for height and weight, respectively, a third measurement was taken. Measurements were recorded to the nearest 0.1 cm or 0.1 kg, respectively. Height and weight were used to calculate BMI ($kg/m^2$) and to classify individuals as overweight/obese.

Currently, there is no universally accepted procedure for the measurement of waist circumference. Although waist circumference measures varied between study groups, both studies utilized the two most common anatomical landmarks—either the natural waist or the umbilicus. For the BC cases, waist circumference was measured by placing the measuring tape (Gulick II Plus) in the horizontal plane (parallel to the floor) around the abdomen at the umbilicus. A second staff member ensured that the measuring tape was parallel to the floor with measurements taken in duplicate to the nearest 0.1 cm and recorded. Additional measurements were taken and recorded until two measurements were within 1.0 cm of each other. For the non-cancer controls, waist circumference was measured by trained personnel just above the uppermost lateral border of the right ilium. The measuring tape (Lufkin steel) was placed in the horizontal plane around the abdomen using a second staff member and a mirror to verify correct placement prior to recording the measurement to the nearest 0.1 cm. [16].

**Blood pressure—**The methodologies to obtain blood pressure in the BC participants were modeled after the NHANES protocol. Utilizing similar procedures, blood pressure was measured with the Omron automated sphygmomanometer for the BC cases and with the mercury or Omron sphygmomanometer for the non-cancer controls [17]. Specifically, arm circumference measures were taken to ensure appropriate blood pressure cuff size. Prior to measurement, participants were seated quietly for at least 5 min with back supported, spine straight, legs uncrossed, and the elbow and forearm resting comfortably on the table with palm turned upward. The brachial artery was located and the blood pressure cuff was placed around the upper arm with the midpoint of the length of the bladder positioned over the brachial artery. Blood pressure was measured in triplicate, recorded, and then averaged.

**Biochemical indices—**Participants were required to fast for a minimum of 8 h prior to phlebotomy. Measures for total cholesterol (TC), [18] triglycerides (TG) [19] and blood glucose [20] in NHANES have been described previously. All BC participants were required to fast for a minimum of 8 h. Blood draws were completed by trained phlebotomists and processed by a chemiluminescent immunoassay (CLIA) certified clinical laboratory.

Specifically, plasma glucose was measured by the modified hexokinase enzymatic assay, TG was ascertained with the glycerokinase assay and cholesterol oxidase assay was used for the determination of HDL.

**Metabolic syndrome—**The National Heart, Lung and Blood Institute criteria was used to evaluate the presence of MetS [21], which was defined as the presence of at least three of the five risk factors listed in Table 1. Medication data for the treatment of hypertension and hyperglycemia were available for all cases and controls. However, because consistent medication data for the treatment of HDL and TG were not available for all participants, the raw values were used in the classification of MetS for all cases and controls.

### Statistical analyses

Means and standard deviations (SDs) were used to describe normally (e.g., age, height, weight, waist circumference, blood pressure), as well as non-normally (HDL, TG, glucose) distributed variables. Non-normally distributed variables were log transformed and then converted back to original scale to facilitate interpretation and presentation of results. Frequencies were used as descriptors for categorical variables. To detect if there were patterns of risk factors by BMI category, the prevalence and 95 % CIs for individual features of the metabolic syndrome were examined by case vs. controls within BMI strata. A paired *t* test or Wilcoxon signed-rank test was used for continuous variables and the McNemar's test, and a Mantel–Haenszel matched-pairs analysis test was used for categorical variables. All analyses were performed using SAS (version 9.4, 2012, SAS Institute, Cary, NC), and a $p$ value <0.05 was used to denote statistical significance.

## Results

When all matching criteria were applied, data on 222 women with BC (91.4 % of 243) were matched with 222 non-cancer population controls from NHANES ($n = 444$). A general description of the matched participant characteristics is reported in Table 2. BC survivors had achieved significantly higher levels of education ($p < 0.001$), were more likely to be engaged in paid work or seeking employment ($p = 0.02$), and reported more health insurance coverage ($p = <0.001$). WC ($p = 0.001$) and diastolic blood pressure measures ($p < 0.001$) were higher among BC cases, yet they had a lower prevalence of self-reported hypertension ($p = 0.001$) when compared to non-cancer controls. The average BC survivor was diagnosed at 57.2 (±10.2) years of age and was 6.9 (±5.2) years post-diagnosis at the time of enrollment. The majority of the women had self-reported Stage I ($n = 76$) or Stage II ($n = 91$) disease, and 74 and 78 % had received chemotherapy and radiation therapy, respectively.

Table 3 presents the prevalence of one or more risk factors for MetS for BC cases vs. their matched non-cancer population controls overall and stratified by BMI classifications ($n = 222$ pairs). Applying the definition set forth by the NHLBI, the general prevalence of MetS was significantly lower in BC survivors vs. matched controls (43.2 vs. 51.4 %, respectively; $p < 0.05$). However, the frequency of MetS did not differ significantly by BMI category for BC cases vs. non-cancer population controls. Specifically, MetS was diagnosed in 25.0 vs. 37.9 % of those classified as overweight (BMI 25.0–29.9); 38.1 vs. 44.8 % with Class 1 obesity (BMI 30.0–34.9); 64.3 vs. 53.6 % with Class 2 obesity (BMI 35.0–39.9); and in 39.0

vs. 73.2 % with Class 3 obesity (BMI ≥40.0.) However, these differences were not statistically significant.

Table 4 describes the frequency with which the individual components of the MetS occur in the BC cases vs. matched non-cancer population controls overall and then stratified by BMI classifications ($n$ = 222 pairs). BC cases experienced a significantly lower frequency of abdominal obesity (98.2 vs. 99.1 %, respectively; $p < 0.05$), high blood glucose (60.0 vs. 63.1 %, respectively; $p < 0.05$), and high serum triglycerides (9.5 vs. 16.2 %, respectively; $p < 0.05$) when compared to the non-cancer population controls. Significantly, more BC cases with Class I ($n$ = 97 pairs; $p < 0.05$) and Class II (56 pairs: $p < 0.05$) obesity had high blood pressure vs. controls, and all obese participants (Class I–III), regardless of case control status, had a WC ≥88 cm. No consistent patterns were noted for blood glucose or HDL cholesterol levels between BMI categories. However, BC cases with Class 3 obesity (BMI ≥40.0) had a significantly lower frequency of low HDL cholesterol when compared to the non-cancer population controls with Class 3 obesity ($p < 0.05$.)

## Discussion

When we applied the NHLBI definition of MetS [22], which is specific to the US population, the prevalence of MetS was significantly lower in this cohort of AA BC vs. their age, race, sex, and BMI-matched non-cancer controls (43.2 vs. 51.4 %, respectively; $p < 0.05$). Although these case-control findings may at first appear encouraging, they reflect a relatively high prevalence of MetS and potentially confer substantial CVD burden and CVD-related mortality for these minority survivors. Two recent studies report that 15 and 26 % of women with early stage BC met the criteria for MetS [23, 24]. However, in both of these studies only 15 % ($n$ = 132/860) and 3 % ($n$ = 136/4216) of study participants were AA, respectively, and results were not stratified by race/ethnicity. Our prevalence estimates better align with those of the general population, which shows that MetS occurs among 40 % of non-Hispanic black females participating in NHANES (2009–2010) across a broad age spectrum. Thus, our findings highlight important disparities among subpopulations of BC survivors and make a novel contribution to this growing body of literature.

MetS is not only implicated in the initiation of BC but also in its progression of BC [25, 26]. Berrino et al. followed 2,092 early stage BC survivors for ~3 years to examine the associations between MetS and BC-related events. Adjusting for age, stage at diagnosis and ER expression, women with MetS were 2.17 (CI 1.31, 3.60) times more likely to experience BC recurrence and 2.45 (CI 1.24, 4.82) more likely to have distant metastases vs. BC survivors without any components of the MetS. Interestingly, the likelihood of these negative occurrences decreased when just one or two components of the MetS were present [15]. Calip et al. also showed that BC survivors with MetS experienced an increased risk of secondary BC events (hazard ratio (HR) = 1.50 (95 % CI 1.08, 2.07) and BC-specific mortality (HR = 1.65, 95 % CI 1.02, 2.69.) [23]. Since AA women present with greater comorbid conditions at the initial time of diagnosis [27], the metabolic alterations of MetS are especially relevant. In the context of these previous investigations, we are inclined to believe that our AA participants with nearly twice the occurrence of MetS would be at significant risk for BC recurrence and reduced overall survivorship. However, our

speculations are greatly tempered by the fact that on average, our participants were enrolled about 7 years after diagnoses. Because women with local or distant recurrence were excluded from participation in the parent weight loss trial, the possibility that women enrolled in this study are inherently 'healthier' than women who did not qualify for the study cannot be dismissed. Further, by design, our participants were required to be overweight or obese to qualify for the weight loss intervention. Including women with a normal BMI (18.5–24.9 $kg/m^2$) would likely lower our estimates.

The exact mechanisms of how MetS influences breast tissue carcinogenesis are not fully understood. Although adipose tissue was once considered an inert substance mainly devoted to energy storage, there are several postulated mechanisms linking MetS to breast tissue carcinogenesis. Essentially, current BC hypotheses model that excess adiposity promotes BC tumor growth by: (1) producing higher concentrations of estrogen and testosterone, [28] (2) contributing to leptin and adiponectin dysregulation, insulin resistance (IR), increased levels of insulin-like growth factor-I (IGF-1) and insulin-like growth factor-binding protein-3 (IGFBP-3) [29–31], and (3) stimulating chronic inflammation [32]. Further, elevated adipose levels are associated with increased aromatase activity, which may work with in conjunction with features of the MetS to decrease plasma levels of sex hormone-binding globulin and free estradiol and testosterone, collectively promoting BC carcinogenesis. These hypotheses have been substantiated by the use of metformin, a biguanide derivative, as it significantly reduces BC risk in persons with diabetes [33]. Metformin may act indirectly to lower circulating insulin levels, or it may act directly on cancer cells by activating AMPK and inhibiting the mammalian target of rapamycin (mTOR) activity [34]. Results of a recent chemoprevention trial by DeCensi et al. showed that metformin (850 mg BID) significantly decreased ki-67 (a marker of BC proliferation) in women with high insulin resistance levels (measured and defined by HOMA scores >2.8) compared to women taking placebo for 4 weeks prior to BC surgery [35]. These results underscore the clinical importance of improving insulin resistance, a hallmark feature of the MetS, for women with BC.

We found that abdominal obesity was prevalent in 100 % of our participants classified as obese, making a significant contribution to the diagnosis of MetS in this cohort. While the use of WC is promoted as a simple, low-cost proxy measure of insulin resistance, it is inherently limited by the fact that it cannot parse out the subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) compartments. VAT is considered the most pathogenic adipose depot since it is associated with inflammation [36] and cardiovascular disease [37]. Previous studies using precise body composition methodologies (i.e., computed tomography, dual energy x-ray absorptiometry) show that AAs possess higher WC measures but possess lower amounts of VAT and higher amounts of abdominal SAT compared to other ethnic populations, even after adjusting for BMI and other covariates [38–40]. Thus, this is an important measurement consideration when examining and interpreting how MetS impacts outcomes in various BC survivors going forward.

This study has numerous limitations. First, since only women who were overweight/obese were recruited for the weight loss trial, data on women with a normal BMI (18.5–24.9 $kg/m^2$) are not included. Therefore, these findings cannot be extrapolated to women of all BMI categories. Considering that current population estimates classify 77 % of AA women

as overweight/obese [7], we do not believe this severely restricts generalizability to a broader group of AA BC survivors. Second, while participants were recruited from neighborhoods of varying socioeconomic status, many of our participants were well-educated and had health insurance. The lower frequency of MetS among the BC cases likely reflects better access to health care, since only 3 % ($n$ = 7) of the BC cases vs. 14 % ($n$ = 32) of the non-cancer controls reported lacking health insurance. The possibility that women with BC were being treated for reduced HDL cholesterol or elevated TGs may explain differences in the prevalence estimates between groups; however, as seen in Table 4, these particular risk factors were much less prevalent in both cases and controls. Third, different methods were used to obtain WC in the cases and the controls. The implications of these differing measurement techniques are simply unknown. Fourth, this study did not include any information on dietary intake, and recent work shows a protective relationship between healthy eating and MetS [41]. Finally, the prognostic implications of MetS in this cohort of AA BC survivors cannot yet be determined.

## Conclusions

MetS reflects a constellation of physiologic abnormalities associated with increases in body weight, and as such, confers substantial risk for atherosclerotic CVD. This study demonstrated that the prevalence of MetS was lower (43 vs. 51 %) among a cohort of overweight/obese AA BC survivors compared to their sex, age, race, and BMI-matched non-cancer population controls. Although we can only speculate on the reasons behind these differences (e.g., access to medical care), it is critical to recognize that these prevalence estimates are highly problematic and considerably higher than those reported in other BC studies. The presence of MetS among BC survivors is now emerging as an important predictor of BC outcomes disparities, including recurrence and mortality. Going forward, clinicians should be aware of these potentially different risk profiles, promoting medication and lifestyle adherence. Future investigators should examine MetS outcomes by race/ethnicity and considered differences in adipose tissue depots not depicted by WC measures, as we pursue the prognostic implications of MetS among AA BC survivors.

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**Table 1**

Clinical features used to evaluate the presence of the metabolic syndrome

| | |
|---|---|
| Reduced HDL cholesterol | <50 mg/dL (1.3 mmol/L) |
| Elevated triglycerides | ≥150 mg/dL (1.7 mmol/L) |
| Elevated blood glucose | ≥100 mg/dL or use of medication for hyperglycemia |
| Elevated waist circumference | ≥88 cm |
| Elevated blood pressure | ≥130 mmHg systolic blood pressure or ≥85 mmHg diastolic blood pressure or use of medication for hypertension |

**Table 2**

Clinical characteristics of African-American breast cancer survivors and their sex, age, race and body mass index matched non-breast cancer controls from NHANES[ab] 2007–2010 (*n* = 222 pairs)

| Characteristics[c] | Mean ± SD or *n* (% of sample) | | *P* value |
|---|---|---|---|
| | Breast cancer *n* = 222 | Non-breast cancer *n* = 222 | |
| Age (years) | 57.2 ± 10.2 | 57.3 ± 10.2 | 0.076 |
| Race—African American | 222 (100 %) | 222 (100 %) | 1.000 |
| Weight (kg) | 94.4 ± 17.5 | 93.9 ± 17.5 | 0.509 |
| Body mass index (kg/m$^2$) | 35.5 ±5.9 | 35.4 ±5.9 | 0.200 |
| 25<30 | 28 (12.6 %) | 28 (12.6 %) | 1.000 |
| 30<35 | 97 (43.7 %) | 97 (43.7 %) | |
| 35<40 | 56 (25.2 %) | 56 (25.2 %) | |
| ≥40 | 41 (18.5 %) | 41 (18.5 %) | |
| Waist (cm) | 111.9 ± 14.1 | 109.9 ± 12.5 | 0.001 |
| SBP (mm Hg) | 127 ±18 | 130 ±21 | 0.250 |
| DBP (mm Hg) | 79 ± 10 | 71 ± 15 | <0.001 |
| TG (mg/dL) | 98.6 ± 47.7 | 107.7 ±66.3 | 0.073 |
| HDL (mg/dL) | 61.8 ± 15.1 | 59.3 ± 14.5 | 0.070 |
| Glucose (mg/dL) | 108.9 ±35.9 | 119.0 ±50.0 | 0.073 |
| Education level | | | <.001 |
| High school or less | 53 (23.9 %) | 109 (49.1 %) | |
| Some college | 86 (38.7 %) | 75 (33.8 %) | |
| College graduate or graduate degree | 83 (37.4 %) | 38 (17.1 %) | |
| Employment | | | 0.019 |
| Paid work | 108 (48.6 %) | 98 (44.1 %) | |
| Not working | 89 (40.1 %) | 113 (50.9 %) | |
| Out of work | 22 (9.9 %) | 11 (5.0 %) | |
| Other | 3 (1.4 %) | 0 (0.0 %) | |
| Health insurance, *n* (%) | | | <.001 |
| None | 7 (3.2 %) | 32 (14.4 %) | |
| Public | 98 (44.5 %) | 104 (46.8 %) | |

| Characteristics[c] | Mean ± SD or *n* (% of sample) | | *P* value |
|---|---|---|---|
| | Breast cancer *n* = 222 | Non-breast cancer *n* = 222 | |
| Private | 115 (52.3 %) | 78 (35.1 %) | |
| Other | 0 (0.0 %) | 8 (3.6 %) | |
| Self-reported comorbidities | | | |
| Hypertension | 129 (58.1 %) | 154 (69.4 %) | 0.001 |
| Diabetes | 52 (23.4 %) | 57 (25.7 %) | 0.631 |

[a] *NHANES* National Health and Nutrition Examination Survey

[b] Breast cancer survivors and non-breast cancer counterparts were matched on sex, age (within 5 years), race (African-American), and body mass index (within the same BMI categories and ±5 kg/m$^2$)

[c] *SBP* systolic blood pressure; *DPB* diastolic blood pressure; *TG* triglyceride; high-density lipoprotein cholesterol

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**Table 3**

Prevalence of one or more risk factors for the metabolic syndrome [percentage (95 % confidence interval)]

| | ≥1 risk factors | | ≥2 risk factors | | ≥3 risk factors | | ≥4 risk factors | |
|---|---|---|---|---|---|---|---|---|
| | **Cases** | **Controls** | **Cases** | **Controls** | **Cases** | **Controls** | **Cases** | **Controls** |
| Total *N* = 222 pairs | 99.6 (97.5, 100) | 99.1 (96.8, 99.9) | 80.2[b] (74.3, 85.2) | 85.6[b] (80.3, 89.9) | 43.2[b] (36.6, 50.0) | 51.4[b] (44.6, 58.1) | 12.6 (8.6, 17.7) | 14.0 (9.7, 19.2) |
| BM1 category[a] | | | | | | | | |
| Overweight *n = 29* | 96.4 (81.7, 99.9) | 93.1 (77.2, 99.2) | 57.1 (37.2, 75.5) | 75.9 (56.5, 89.7) | 25.0 (10.7, 44.9) | 37.9 (20.7, 57.7) | 10.7 (2.3, 28.2) | 0 (0, 0) |
| Class 1 obesity *n* = 96 | 100.0 (100.0, 100.0) | 100.0 (100.0, 100.0) | 83.5 (74.6, 90.3) | 83.3 (74.4, 90.2) | 38.1 (28.5, 48.6) | 44.8 (34.6, 55.3) | 13.4(7.3,21.8) | 14.6 (8.2, 23.3) |
| Class 2 obesity *n* = 56 | 100.0 (100.0, 100.0) | 100.0 (100.0, 100.0) | 85.7 (73.8, 93.6) | 89.3 (78.1,96.0) | 64.3 (50.4, 76.6) | 53.6 (39.7, 67.0) | 17.9 (8.9, 30.4) | 13.6 (−0.7, 28.0) |
| Class 3 obesity *n* = 41 | 100.0 (100.0, 100.0) | 100.0 (100.0, 100.0) | 80.5 (65.1,91.2) | 92.7 (80.1,98.5) | 39.0 (24.2, 55.5) | 73.2 (57.1, 85.8) | 4.9 (0.6, 16.5) | 26.8 (14.2, 42.9) |

[a]Body mass index (BMI; kg/m$^2$) classifications are as follows: overweight = BMI 25.0–29.9, Class 1 obesity = BMI 30.0–34.9, Class 2 obesity = BMI 35.0–39.9, Class 3 obesity = BMI ≥40.0

[b]$p < 0.05$

**Table 4**

Prevalence of individual risk factors for the metabolic syndrome [percentage (95 % confidence interval)]

| | Abdominal obesity | | High blood pressure | | High blood glucose | | High triglycerides | | Low HDL cholesterol[a] | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Case | Controls | Case | Controls | Case | Controls | Case | Controls | Case | Controls |
| Total *N* = 222 pairs | 98.2[c] (95.5, 99.5) | 99.1[c] (96.8, 99.9) | 73.3 (67.0, 79.0) | 48.7 (41.9, 55.4) | 60.0[c] (51.4, 68.2) | 63.1[c] (56.4, 69.4) | 9.5[c] (6.0, 14.2) | 16.2[c] (11.6, 21.7) | 21.2 (16.0, 27.1) | 25.7 (20.1, 32.0) |
| BMI category[b] | | | | | | | | | | |
| Overweight *n* = 28 pairs | 85.7 (67.3, 96.0) | 93.1 (77.2, 99.2) | 64.3 (44.1, 81.4) | 51.7 (32.5, 70.6) | 63.6 (30.8, 89.1) | 41.4 (23.5, 61.1) | 7.4 (0.9, 24.3) | 13.8 (3.9, 31.7) | 10.7 (2.3, 28.2) | 6.9 (0.9, 22.8) |
| Class 1 obesity *n* = 97 pairs | 100.0 (100.0, 100.0) | 100.0 (100.0, 100.0) | 76.3” (66.6, 84.3) | 44.8” (34.6, 55.3) | 53.5 (39.9, 66.7) | 59.4 (48.9, 69.3) | 9.3 (4.3, 16.9) | 17.7 (10.7, 26.8) | 21.7 (13.9, 31.2) | 21.9 (14.1, 31.5) |
| Class 2 obesity *n* = 56 pairs | 100.0 (100.0, 100.0) | 100.0 (100.0, 100.0) | 85.5” (73.3, 93.5) | 51.8” (38.0, 65.3) | 73.0 (55.9, 86.2) | 66.1 (52.2, 78.2) | 12.5 (5.2, 24.1) | 12.5 (5.2, 24.1) | 26.8 (15.8, 40.3) | 25.0 (14.4, 38.4) |
| Class 3 obesity *n* = 41 pairs | 100.0 (100.0, 100.0) | 100.0 (100.0, 100.0) | 56.1 (39.8, 71.5) | 51.2 (35.1, 67.1) | 55.9 (37.9, 72.8) | 82.9 (67.9, 92.9) | 7.3 (1.5, 19.9) | 19.5 (8.8, 34.9) | 19.5” (8.8, 34.9) | 48.8 “ (32.9, 64.9) |

[a] High-density lipoprotein cholesterol

[b] Body mass index (BMI; kg/m$^2$) classifications are as follows: overweight = BMI 25.0–29.9, Class 1 obesity = BMI 30.0–34.9, Class 2 obesity = BMI 35.0–39.9, Class 3 obesity = BMI ≥40.0

[c] $p < 0.05$