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Risk factors for breast cancer in a black population—The Barbados National Cancer Study

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Abstract

The Barbados National Cancer Study (BNCS) is a nationwide case-control study investigating environmental and genetic factors for breast cancer (BC) in a predominantly African-origin population with similar ancestry as African-Americans. This report evaluates associations of incident BC in the BNCS to various factors, including demographic, anthropometric, reproductive and family history variables, not investigated previously in this population. The BNCS included 241 incident BC cases and 481 age-matched female controls, with mean ages of 57 and 56 years, respectively. In addition to a reported family history of BC in a close relative [odds ratios (OR) = 3.74, 95% CI (1.41, 9.90) in a parent; OR = 3.26 (1.47, 7.21) in a sibling], other factors associated with BC were older age at first full-term pregnancy [OR = 1.04 (1.00, 1.07)] and having a history of benign breast disease [OR = 1.88 (1.19, 2.99)]. Increased parity reduced the risk of BC [OR = 0.34 (0.15, 0.77) among those with ≥3 children]. The reproductive patterns of African-Barbadian (AB) women tended to differ from those of African-American (AA) women (later age of menarche, earlier age at first pregnancy, higher frequency of lactation and infrequent use of exogenous hormones) and could help to explain their considerably lower postmenopausal incidence of BC. The relationship between reported family history and BC, combined with the associations noted for several reproductive and other variables,

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supports the genetic and environmental contributions to BC, which may vary in populations across the African diaspora. Further investigations of other populations may clarify these issues.

Keywords

breast cancer; risk factors; African-origin population

Historically, rates of breast cancer (BC) in Africa have been relatively low, as compared with westernized populations of African origin, such as African Americans (AA), and to European-derived populations.[1] Furthermore, while AA have a lower overall incidence of BC than White-American (WA) women (118 *vs*. 132 per 100,000, respectively), they have a higher frequency of premenopausal disease. The BC mortality of AA is also higher (34 *vs*. 25 per 100,000, respectively),[2] an outcome probably influenced by their earlier age of onset and more advanced stage at diagnosis. However, the reasons for these noted differences remain unclear and strategies to address these disparities require a better understanding of their causes.

Insight into the mechanisms underlying the differential BC patterns of African populations can be gained by studying BC risk factors across the African diaspora. These data, however, have been relatively unavailable. One such population resides in Barbados, West Indies, a highly westernized nation where over 90% of citizens are of African descent. African-Barbadians (AB) and AA share a common ancestry, both having descended from the very select subgroup who survived the long ocean voyage from West Africa during the diaspora.[3] As a British colony and a center of the slave trade in the 17th and 18th centuries, Barbados was an intermediate stop for many ancestors of AA.[3–5] In contrast to other Caribbean islands, Barbados had no indigenous population, remained fairly homogeneous and had limited admixture over time.[6] The combination of social, economic and other environmental differences between AB and AA, yet with a similar heredity, highlights the unique value of the AB population for assessing genetic and non-genetic risk factors for BC. Despite their geographic and socio-cultural differences, AA are more similar to AB than to Africans; thus AB provide a new intermediate, heretofore missing population to allow comparisons across populations of African origin.

The intermediate position of AB regarding BC risk is supported by data from the International Agency for Research on Cancer (IARC)[1] and most recently, from an investigation conducted by Hennis *et al*.[7] According to the IARC, the age-standardized BC incidence rate (per 100,000) in Barbados is approximately midway between the rates noted for West Africa and the US (West Africa 27.8, Barbados 62.5, US 101.1).[1] The corresponding age-standardized (to US population) BC incidence rates in Barbados obtained in a recent report by Hennis *et al*. for 2002–2006 was 78.1 per 100,000,[7] yet were likewise intermediate compared with the IARC and SEER estimates for West African and AA populations.[1,2] Although AB and AA had similar premenopausal rates, AB post-menopausal rates were lower, resulting in an overall lower incidence. Despite this decreased rate, data from that report indicate that the mortality rate for BC in Barbados is 33 per 100,000[7] or more similar to the mortality of AA than of West African women. Although international differences in cancer reporting should be considered, these data suggest that BC may be a more aggressive disease among AB than West Africans and that pertinent risk factors may be prevalent in this population.

The current report presents the first nationwide epidemiologic data on BC risk factors in an African-Caribbean population. It is based on results from the Barbados National Cancer Study (BNCS), which was designed to evaluate the contribution of epidemiologic and genetic factors for BC and prostate cancer in the predominantly African-descent population of this island nation.

Material and methods

The BNCS, funded by the National Human Genome Research Institute (NHGRI), with contribution from the Office for Minority Health, includes a Coordinating Center (University Medical Center, Stony Brook, NY), a Clinical Center (Ministry of Health and University of the West Indies, Bridgetown, Barbados, West Indies), a Local Laboratory Center (University of the West Indies), a NHGRI Center (Bethesda, MD) and a Gene Discovery Center (Translational Genomics Research Institute, Phoenix, AZ).

Study population

The BNCS is a population-based case-control study of breast and prostate cancer in Barbados-born residents. Cases for the BC component of the study included all histologically confirmed incident BC that were identified by the country's only Pathology Department at the Queen Elizabeth Hospital between July, 2002 and March, 2006. Controls were women of at least 21 years of age without cancer, who were randomly selected from a national database and frequency matched (2:1) to the cases by 5-year age groups based on the age at the time of diagnosis of the case. Informed consent was obtained from all BNCS participants and the study protocols conformed to the Declaration of Helsinki.

Data collection

The study visit took place at the Winston Scott Polyclinic, Bridgetown, Barbados. Data were collected by certified nursing staff, masked as to case-control status, using standardized forms and procedures. All questions were asked for events prior to the reference date, which was defined as the date of cancer diagnosis for the cases and a similar date for the matched controls. The questionnaire included the following variables.

Personal characteristics consisted of demographic, lifestyle and anthropometric factors such as ancestry, education, marital status, religion, lifetime occupation and related factors (*e.g.*, shift work); smoking history, alcohol use, activity/exercise level and weight history since adolescence. Anthropometry was performed following a strict protocol to assess height (metric measuring tape and right-angled wood block), weight (beam balance scale) and waist and hip circumferences (steel tapes).

Medical history factors included physician-diagnosed diabetes; pulse and blood pressure (BP), hypertension (systolic BP $\geq$140 mmHg or diastolic BP $\geq$90 mmHg or a history of anti-hypertensive treatment); history of heart disease and high cholesterol; use of prescription and over-the-counter medications, use of nutritional supplements; any exposure to dental and chest X-rays; and health care utilization practices, including history of mammography use. Reproductive variables were age at menarche and menopause; menses duration and regularity; age of first pregnancy, number of pregnancies and respective outcomes; history of breast feeding; method of birth control, history of infertility; exogenous hormone use, gynecologic surgeries and mammography history.

Family and personal history of cancer consisted of a battery of questions concerning history of any type of cancer among parents, siblings and children, as well as the participant's own history of cancer and treatment, if applicable.

A blood sample was drawn to evaluate HbA1c and for future analyses of genetic variants; dietary intake was assessed with the administration of a validated food frequency questionnaire.[8,9] Data relating to cancer staging and estrogen receptor status of cases were obtained by chart review at the Queen Elizabeth Hospital.

BNCS participants received a clinical evaluation by the study's physicians, including a complete breast examination. Mammograms were provided for non-pregnant controls.

Statistical analyses

The distribution of demographic factors for cases and controls was compared using chi-square statistics (for categorical variables) and *t*-tests (for continuous variables). Anthropometric measurements [including height, weight, body-mass index (BMI), waist circumference (WC), hip circumference (HC) and waist–hip ratio (WHR)] above the upper quintile were considered high, those below the lowest quintile were classified as low and all others were defined as intermediate. Although standard cutpoints for these variables have been defined, the distribution of body size in Barbados is somewhat different than in other populations.[10,11] As such, quintiles were chosen to better quantify these measurements. To assess associations between BC and potential risk factors, univariate logistic regression models were first used to evaluate variables, including body size, lifestyle and medical/family history. A standard approach of examining bivariate scatterplots for each variable was performed to check for linearity and multivariate logistic regression analyses were mutually adjusted for significant factors ($p < 0.05$) identified by the univariate analyses. On the basis of the logistic regression models, results were presented as odds ratios (OR) and 95% confidence intervals (CI).

Results

The BNCS included 241 incident cases and 481 female controls with participation rates of 80% and 82% of those eligible, respectively. There were no significant differences in age or parish of residence between participants and eligible non-participants (data not shown). A total of 222 cases and 454 controls (or ~ 93% of BNCS participants) self-reported their ancestry as black or mixed (black and white). Because of the small number of white/other participants in the study, the analyses are based on only those of African-origin. Table I presents the demographic and lifestyle factors for the AB cases and controls. Distributions of age, religion and marital status were similar in the 2 groups. The mean ages of cases and controls were 57 and 56 years, respectively, and just over two-fifths were either currently married or living with their partner. A comparison of lifetime occupation indicated that cases were more likely than controls to be either housewives/homemakers or employed in a professional/managerial occupation. The use of cigarettes or alcohol was relatively infrequent among women in this population and neither factor was a significant predictor for BC; level of physical activity was also unrelated.

Approximately 37% of cases were under 50 years of age at diagnosis. Findings from the chart review indicated that 21%, 18% and 6.5% of cases had stage IIb, III and IV cancer, respectively, and 54% were estrogen receptor (ER) negative.

The distribution of body size and medical/family history factors is presented in Table II. Although height differences between cases and controls did not achieve statistical significance, cases tended to be taller [OR = 1.65 (95% CI 0.97, 2.80); $p = 0.06$]. Cases and controls had no significant differences in weight 5 years prior to the reference date, but weighed less than controls at their study visit. Since this finding is likely to reflect the impact of disease, the weight variable was not included in the subsequent multivariate regression models. With respect to the medical history variables, hypertension tended to be less frequent among cases (51.8% *vs*. 59.5%; $p = 0.06$), whereas cases and controls reported a similar history of diabetes, aspirin use and exposure to X-rays, including mammography.

In the univariate analyses, significant differences in several reproductive factors were noted, with cases having an older age at first full-term pregnancy, fewer children and a more frequent history of benign breast disease than controls. Approximately 15% of cases and 11% of controls

were nulliparous; a similarly high frequency of lactation was found among parous cases and controls. The mean age of menopause was about 50 years and 9% of women from each group reported ever using exogenous hormones after menopause. About one-third of the BNCS participants were pre-menopausal. Separate sub-analyses by menopausal status indicated that increasing parity yielded a significantly protective OR in post-menopausal women [OR = 0.68 (0.51, 0.90)], whereas such a result was not noted in the pre-menopausal group [OR = 1.02 (0.66, 1.55)]. Additionally, a history of benign breast disease was a statistically significant factor in pre-menopausal [OR = 3.89 (1.97, 7.69)] but not post-menopausal women [OR = 1.28 (0.73, 2.26)].

A family history of any cancer, but especially BC, was among the most significant factors in the univariate analysis. More than one-fifth of cases and only 8% of controls reported a family history of BC and the percentage of cases reporting a mother with BC was more than 3 times the percentage reported by controls (6.0% *vs*. 1.8%; $p = 0.005$). Likewise, cases were more likely to report a sister with the disease (8.6% *vs*. 2.6%; $p = 0.001$).

Table III presents the multivariate logistic regression results based on significant factors identified from the univariate analyses. The factors associated with incident BC were older age at first full-term pregnancy [OR = 1.04 (1.00, 1.07) per year], history of benign breast disease [OR = 1.88 (1.19, 2.99)] and a family history of BC [OR = 2.62 (1.58, 4.34)]. Increased parity was negatively associated with BC [OR = 0.40 (0.19, 0.86)]. The ORs decreased from 0.43 (0.20, 0.94) in those having 1–2 children to 0.34 (0.15, 0.77) among those having 3+ children, as compared with nulliparous women. Although not significant in the BNCS univariate analyses, other studies have found that variables such as level of physical activity, age at menarche, use of birth control, history of breast feeding and use of hormone-replacement therapy were significant predictors of BC risk. As such, we conducted additional multivariate analyses adjusting for these variables, as well as those found to be significant in the BNCS, without appreciable impact on the results.

Discussion

The BNCS was designed to evaluate a comprehensive set of risk factors for BC in a predominantly African-origin population. These data represent a ‘‘missing’’ link between results of studies originating in Africa, where incidence of BC has been traditionally low, and those conducted in the US, where incidence and mortality rates among AA women are among the highest in the world. Findings from BNCS indicate that older age at first full-term pregnancy, nulliparity, history of benign breast disease and family history of BC are among the most significant risk factors in this population.

Several theories have been suggested to explain why BC disproportionately affects younger westernized women of African origin, with increased mortality. Most explanations are on the basis of: (*i*) reproductive patterns; (*ii*) genetic influences; (*iii*) tumor biology; and/or (*iv*) other factors such as socioeconomic status and access to health care.

Reproductive patterns

As its well known that young age at menarche,[12] older age at first pregnancy,[13] nulliparity,[13] shorter periods of lactation[12,14] and late menopause have been shown to increase BC risk in AA and other women.[15] These factors would lead to an increase in ovulatory cycles and subsequently higher endogenous estrogen levels over a woman’s lifetime, with an increased vulnerability to environmental carcinogens, and thus increasing BC risk.[16] This explanation for the BC disparities in AA women is supported by some studies in Africa, where BC rates are low, and girls experience menarche at older ages (median 15 years), young ages at first birth (median 19 years), increased parity (mean 5–9 births per woman) and extended periods

of lactation (mean 16 months).[17] However, not all studies have corroborated the findings with regard to each of these individual variables. In the past 15 years, reports including a sizable number of AA participants indicate that the proposed relationship between hormonal exposures and BC may not be so straightforward in women of African origin.[12,18–22]

Although adequate documentation exists on the influence of multiparity and a subsequent long-term protective effect of pregnancy on BC, a transient increase in risk has also been shown to follow pregnancy.[12,23–25] This dual effect, however, has not been consistent in all investigations.[12,26] In the Carolina Breast Cancer Study (CBCS), a population-based case-control study including 1,505 AA and 1,809 WA women, increased parity tended to be associated with a higher BC risk in younger AA women (though not statistically significant) but not among younger WA women.[12] In contrast, multiparity was found to decrease risk in older AA women 50–74 years of age, as expected [adjusted OR = 0.5, 95% CI (0.3, 0.9) for women with ≥5 children]. Results from the Black Women's Health Study (BWHS), which included a prospective cohort of 56,725 participants (349 BC cases), confirmed the CBCS findings among AA women and reported that parity was associated with an increased BC risk in AA women younger than 45 years of age with 4 or more births [incidence rate ratio (IRR) = 2.4 (1.1, 5.1)] and a lower risk among older AA women with 4 or more births [IRR = 0.5 (0.3, 0.9)].[25] The BNCS similarly suggests a protective effect of increased parity. Compared with women who were nulliparous, women with 1–2 children had an OR = 0.43 (0.20, 0.94) and those with 3+ children had an even more protective OR = 0.34 (0.15, 0.77). In further analyses, stratifying by menopausal status and adjusting for other relevant factors this result was statistically significant among post-menopausal AB women; however, no specific pattern was noted in pre-menopausal women. The lack of conclusive findings in the pre-menopausal women may be the result of the low sample size in this group.

Inconsistent data exist on the influence of age at menarche on the BC risk of African-origin women. In these women, the onset of menses at a younger age was reported to be a significant BC risk factor by some studies[12,19,27] but not others.[28–30] Whereas girls in sub-Saharan Africa are known to menstruate at ~15 years,[17] the average age of menarche, based on NHANES III data, was 12.6 years in WA and 12.1 years in AA.[31] Since AA girls tend to begin menstruating approximately (1/2) year earlier than their WA counterparts and a few years earlier than in Africa, this earlier menarche could explain, to some degree, the higher premenopausal rates of BC in AA compared with other groups.[22,32] In our study, the median age of menarche was 13 years in cases and controls, or almost a year older than in AA and was not a significant factor. This later age of menarche may contribute to the intermediate BC risk of AB women, as compared with AA and WA women.

The relationship of BC to age at first full-term pregnancy in African-descent women has been debated. In Nigeria, where women tend to give birth earlier, one study reported that age at first full-term pregnancy was not associated with BC,[28] another reported a significant association[29] and a third found an association in premenopausal but not postmenopausal women.[27] Similarly, results of studies in AA have been inconsistent.[12,18,25] The CBCS found that age at first pregnancy was not a risk factor in younger or older AA women,[12] whereas data from the BWHS indicated that older age at first birth (≥30 years) was a significant predictor of BC in younger AA women but not older women in the study.[25] A third study including 490 AA cases and 485 AA controls aged 20–54 also implicated age at first full-term birth as a significant risk factor for BC.[30] In the BNCS, the average age at first birth among cases was ~1 year older than among controls and this represented a statistically significant difference. Additional multivariate-adjusted sub-analyses indicated that later age at first birth confers a significant increase in risk ($p < 0.05$) among AB women 50 years and older [OR = 1.05 (1.01, 1.09)], and although not significant, the finding among younger women (below the age of 50 years) was in the same direction [OR = 1.04 (0.98, 1.10)].

Evidence on the role of lactation on BC risk in AA has been mixed,[12,19] likely due to the overall variability, as well as the frequency and duration of breastfeeding practices in the different studies.[21] What has been substantiated, however, is that WA are more than 2 times as likely to breastfeed as AA.[33] In the CBCS, 20% of younger AA women ever breastfed compared with 41% in the WA women. Whereas only a modest percentage of AA women breastfeed, ~ 96–97% of parous AB women reported ever breast feeding. Perhaps the high rate of lactation is one of the factors that may contribute to the lower incidence of BC in postmenopausal AB than AA and WA women.

Although almost one-half of women in Barbados reported using oral contraceptives, this factor did not influence BC risk. Additionally, about 9% of both cases and controls reported ever using hormones after menopause. Taken together, these results suggest that use of exogenous hormones is not a likely BC risk factor in AB women.

Genetic influences

Some of the strongest known risk factors are a family history of BC[12,19,20,30] and a history of benign breast disease (BBD),[30,34–36] which is also speculated to be a precursor to BC and to involve a familial component.[37] Even while accounting for undoubted reporting bias, the BNCS results strongly suggest genetic influences in BC development in this population. The frequency of BBD history was approximately 2 times higher in cases *vs*. controls and the frequency of BC family history was more than 3 times higher. It has been suggested that perhaps an interaction between these 2 variables exists, thus compounding the risk if both were present. In one study, women with a family history of BC and atypia had an 11-fold increased risk of BC as those without.[38] Such interaction, however, has not been confirmed by others.[34–36] Additional BNCS analyses also did not substantiate an interaction between BBD and family history, although this may be due to sample size issues.

The AB population is more genetically homogeneous than AAs[6] and the lesser admixture in AB than AA is an advantage that facilitates the detection of shared ancestral variants. Genetic analyses in BNCS may assist in identifying such variants. The discovery of such a genetic contribution would assist in clarifying the likely gene–environment interaction influencing breast cancer development and may help to explain why BC disproportionately affects younger, westernized women of African descent.

Tumor biology

Women of African origin may have different tumor biology than other groups. These women tend to present with larger primary tumors at more advanced stages and are more likely to have a higher proportion of lymph node involvement than others.[22,39] Additionally, 40–60% of African women are found to have ER-negative tumors compared with 20–40% in Caucasian populations.[22] Data from the present investigation are consistent with this pattern, as 45% of AB cases had tumors of stage IIB or higher at diagnosis and 54% had ER-negative cancers. These types of tumors are known to be more aggressive and more difficult to treat[40,41]; thus, contributing to the increased mortality among younger African women. It is still unclear, however, why this group is more likely to be ER negative and present with later stage disease at earlier chronological ages than other groups.

Other factors

Although BC cases weighed less than controls at the time of interview, there were no statistically significant differences in weight between the 2 groups 5 years prior to the reference date. In addition, while cases tended to lose weight after their diagnosis, controls tended to gain. These findings suggest that weight loss after diagnosis of BC may be directly related to

the disease and that body weight in adulthood does not appear to significantly influence BC development in this population.

In addition to body weight, it has been suggested that increased BC mortality rates among younger AA women may be due, at least in part, to generally lower socioeconomic factors and a reduced access to health care. In the US, it has been difficult to disentangle the contributions of sociology and biology when evaluating the impact of such factors among AA. The BNCS provided an opportunity to investigate these variables, as AB cover the entire socioeconomic spectrum and the country provides free care to its citizens. Although homemakers/housewives and those in professional occupations tended to be at increased risk in the multivariate analyses, neither education nor occupation (2 indicators of socioeconomic status) was significantly associated with BC in the BNCS. Such associations could not be ruled out, however, particularly with the current sample size that may have had inadequate power to detect modest differences, as suggested by the magnitude of the ORs (ranging from 1.3–1.6) in the multivariable findings. Future investigations with larger sample sizes are required to fully elucidate these possible relationships.

Strengths and limitations

The major strengths of this population-based case-control study include its nationwide ascertainment, standardized and comprehensive protocols and high rates of participation. Limitations include the possible biases inherent to long-term recall of relevant factors and the modest sample size, resulting in reduced power, especially among pre-menopausal women.

Conclusions

Rates of BC vary among AA, AB and West African women, despite the fact that both AA and AB originated from the same geographic regions of West Africa. The variability is likely due to different exposures to certain risk factors, of which the major ones include genetic, reproductive and other environmental variables. The reproductive patterns of AB women are more similar to those of West African women, yet AB and AA share a more common westernized culture compared with women from West Africa; as such, Barbados represents an intermediate group among the three. The unique features of this population have helped to identify factors which may be influencing BC in this and other populations of African origin. The results of the present investigation confirm that later age at first birth, nulliparity, a history of benign breast disease and a family history of BC are significant risk factors for the disease in this population. Other reproductive variables such as age at menarche (which is later in AB than AA and WA girls), lactation (which is a common practice in AB) and use of exogenous hormones (which is infrequent in post-menopausal AB women) were not significant predictors and may be likely factors influencing the lower incidence of BC in postmenopausal AB than AA women. A strong association between family history and BC suggests that genetics plays a significant role, as well. Overall, it is generally accepted that BC is a complex, multifactorial disease and is likely the result of interacting genetic and environmental factors. Further investigations are required to disentangle the true contribution of each possible risk factor and to identify common variants that may be particularly important in the development of BC in this and other populations of African origin.

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TABLE I

DEMOGRAPHIC AND LIFESTYLE FACTORS FOR BNCS AFRICAN-BARBADIAN (BLACK + MIXED) BREAST CANCER CASES AND CONTROLS

Demographic factors	**Cases (*n* = 222)**	**Controls (*n* = 454)**	***p*-value**
Age (yrs) mean ± sd (median)	56.8 ± 14.3 (54.5)	55.8 ± 14.1 (53.0)	0.41
Religion (%)			0.15
Anglican	41.3	34.4	
Pentecostal	17.8	23.0	
Other	40.8	42.6	
Marital Status (%)			0.46
Single and never married	30.2	35.7	
Married or living together	42.3	41.0	
Separated or divorced	14.9	11.9	
Widowed	12.6	11.4	
Education mean ± sd (median)	12.1 ± 3.8 (11.0)	11.7 ± 3.3 (11.0)	0.13
Occupation (%)			0.01
Housewife/homemaker	11.3	7.1	
Prof/admin/managerial	19.4	13.2	
Other	69.4	79.7	
Regularly work at night (%)	21.2	24.0	0.41
Ever smoke cigarettes (%)	5.9	4.0	0.27
Ever drink alcohol (%)	21.6	25.1	0.22
Physical activity level (%)			0.70
Inactive	2.3	1.3	
Not very active	13.7	15.9	
Somewhat active	30.6	29.0	
Very active	53.4	53.8	

TABLE II

POTENTIAL RISK FACTORS RELATED TO BODY SIZE, MEDICAL AND FAMILY HISTORY AMONG AFRICAN-BARBADIAN (BLACK + MIXED) WOMEN

Factors	Cases (*n* = 222) mean ± sd (median)	Controls (*n* = 454) mean ± sd (median)	OR (95% CI)
Body size[1]			
Height (cm)	161.8 ± 6.1 (162.0)	160.8 ± 6.5 (161.0)	1.65 (0.97, 2.80)
Weight 5 yrs prior (kg)	72.7 ± 16.5 (70.5)	73.2.0 ± 15.8 (70.5)	0.76 (0.38, 1.52)
Current Weight (kg)	72.0 ± 15.9 (69.5)	75.3 ± 16.8 (73.2)	0.49 (0.29, 0.82)*
Body mass index (kg/m^2)	27.4 ± 5.8 (26.7)	29.1 ± 6.2 (28.6)	0.48 (0.29, 0.80)*
Waist circumference (cm)	90.2 ± 12.6 (90.0)	91.4 ± 12.9 (91.0)	0.72 (0.43, 1.21)
Hip circumference (cm)	105.7 ± 11.3 (104.0)	107.8 ± 12.5 (106.0)	0.62 (0.37, 1.05)
Waist–hip ratio	0.85 ± 0.08 (0.86)	0.85 ± 0.07 (0.85)	1.15 (0.69, 1.92)
Medical History			
Hypertension (%)	51.8	59.5	0.73 (0.53, 1.01)
Antihypertensive treatment	41.6	44.3	0.90 (0.65, 1.24)
Diabetes history (%)	17.6	15.9	1.13 (0.74, 1.73)
HbA1c	5.8 ± 1.4 (5.5)	6.0 ± 1.3 (5.7)	0.92 (0.81, 1.05)
Regular use of aspirin (%)	10.8	13.4	0.78 (0.47, 1.29)
Chest X-rays (%)	25.7	20.7	1.32 (0.91, 1.93)
Dental X-rays (%)	51.0	43.1	1.37 (0.99, 1.91)
Number of mammograms	2.8 ± 3.4 (2.0)	2.8 ± 4.2 (1.0)	1.00 (0.94, 1.07)
Reproductive history			
Age at menarche (yrs)	13.2 ± 1.8 (13.0)	13.1 ± 1.8 (13.0)	1.04 (0.95, 1.14)
History of fertility problems (%)	9.8	7.1	1.42 (0.80, 2.52)
Age at first full-term pregnancy (yrs)[2]	22.3 ± 5.7 (21.0)	21.3 ± 5.2 (20.0)	1.04 (1.00, 1.07)*
Number of children			0.79 (0.63, 0.99)*
0	15.3	11.0	
1–2	43.2	40.5	
3+	41.4	48.5	
Ever breast feed (%)[2]	97.3	96.3	1.41 (0.50, 3.94)
History of benign breast disease (%)	21.9	12.4	1.98 (1.30, 3.04)*
Ever used oral contraception (%)	47.8	46.8	1.03 (0.86, 1.22)
Age at menopause (yrs)[3]	51.0 ± 4.0 (50.5)	50.2 ± 4.0 (51.0)	1.05 (0.98, 1.13)
Ever used post-menopausal hormones (%)	9.1	9.2	0.99 (0.57, 1.74)
Family history of Cancer			
Family history of any cancer (%)	58.6	42.1	1.95 (1.40, 2.69)*
Family history of breast cancer:			
Any family history (%)	20.7	7.9	3.04 (1.90, 4.86)*
Parent (%)	6.0	1.8	3.39 (1.38, 8.32)*
Sibling (%)	8.6	2.6	3.45 (1.64, 7.24)*

* $p < 0.05$.

[1] OR (95% CI) represents high (values above the upper quintile) versus low (values below the lower quintile) for each of the body size variables.

[2] Among parous women.

[3] Among menopausal women (74 cases, 155 controls).

TABLE III

MULTIVARIATE LOGISTIC REGRESSION RESULTS FOR ASSOCIATIONS OF BREAST CANCER AMONG AFRICAN-BARBADIAN (BLACK + MIXED) WOMEN

Factors	OR (95%CI)
Age (per yr)	1.00 (0.99, 1.02)
Occupation	
Professional occupation	1.36 (0.83, 2.24)
Housewife/homemaker	1.58 (0.86, 2.89)
Other occupation	1.00
Age at first full-term pregnancy	1.04 (1.00, 1.07)*
Parity[1]	0.40 (0.19, 0.86)*
3 + children	0.34 (0.15, 0.77)*
1–2 children	0.43 (0.20, 0.94)*
Nulliparity	1.00
History of benign breast disease	1.88 (1.19, 2.99)*
Family history of breast cancer	
Any	2.62 (1.58, 4.34)*
In parent	3.74 (1.41, 9.90)*
In sibling	3.26 (1.47, 7.21)*

* $p < 0.05$ based on logistic regression models. Number of children and family history of BC in parents and siblings were entered in separate models.

Parity was defined as nulliparous *vs*. parous.