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Racial Disparities in Red Meat and Poultry Intake and Breast Cancer Risk

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Abstract

Purpose—Research on the role of red meat and poultry consumption in breast carcinogenesis is inconclusive, but the evidence in African American (AA) women is lacking. The association between consuming meat and breast cancer risk was examined in the *Women's Circle of Health Study* involving 803 AA cases, 889 AA controls, 755 Caucasian cases, and 701 Caucasian controls.

Methods—Dietary information was collected using a Food Frequency Questionnaire. Odds ratios (OR) and 95% confidence intervals (CI) were obtained from logistic regression models adjusting for potential covariates.

Results—Comparing the fourth vs. the first quartile, among Caucasian women, processed meat (OR=1.48; 95% CI: 1.07–2.04), unprocessed red meat (OR=1.40; 95% CI: 1.01–1.94) and poultry intakes (OR=1.42; 95% CI: 1.01–1.99) increased breast cancer risk. Risk associated with poultry intake was more dominant in premenopausal women (OR=2.33; 95% CI: 1.44–3.77) and for women with ER- tumors (OR=2.55; 95% CI: 1.29–5.03) in the Caucasian group. Associations in AA women were mostly null except for a significant increased risk trend with processed meat consumption for ER+ tumors (OR=1.36; 95% CI: 0.94–1.97, p trend=0.04).

Conclusions—Overall, associations between breast cancer risk and consumption of red meat and poultry were of different magnitude in AA and Caucasian women, with further differences noted by menopausal and hormone receptor status in Caucasian women. This is the first study to examine racial differences in meat and breast cancer risk, and represents some of the first evidence in AA women.

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Keywords

Meat; African American; Breast Cancer; Poultry; Race; Estrogen receptor

Introduction

Breast cancer remains the most commonly diagnosed non-skin cancer in both Caucasian and African American (AA) women in the US (1, 2). Meat consumption in the US has been rising (3), but the impact on breast cancer is inconclusive (4–13), although the evidence is largely based on data from Caucasian women. Nevertheless, due to strong evidence linking red and processed meats to other cancers such as colorectal cancer (14–17), the World Cancer Research Fund and American Institute for Cancer Research recommends limiting red meat intake to less than 500 g/week (18 oz) with very little if any to be processed (14).

Although the overall incidence of breast cancer is higher in Caucasian women, AA women are more likely to be diagnosed at younger ages and present with tumors with more aggressive characteristics, with high grade, lack of expression of estrogen receptor (ER), and basal-like phenotypes (2, 18–20). Despite these disparities, a recent review (21) that summarized the evidence on food, nutrition, and physical activity and breast cancer risk in AA women found no studies on any foods of animal origin and breast cancer risk in AA women. In parallel, there are clear racial differences in red meat and poultry consumption patterns with NHANES data showing higher mean consumption of red meat and poultry in AA compared to Caucasian women (22). The relevance of red meat and poultry consumption to carcinogenesis involves cooking meat at high temperatures and the exposure to heterocyclic amines (HCAs), nitrosamines, saturated fat, and heme iron (14, 23–25).

Most epidemiological evidence supports a positive association between degrees of meat doneness and risk of human cancers (24). A greater proportion of AA also report grilling or barbequing hamburgers and beefsteak than Caucasians in the US based on data adapted from FDA/USDA Consumer Food Safety Surveys (26), which elevates the need to investigate the relationship between meat and breast cancer in a large sample of AA women. We evaluated the role of red meat and poultry intake and breast cancer risk in the *Women's Circle of Health Study*, a case-control study based in New Jersey (NJ) and New York (NY). This is one of the first studies to examine this association in AA women, and the first study to report findings stratified by race.

Methods

Study Population

The *Women's Circle of Health Study* (WCHS) has been described in detail elsewhere (27, 28). In brief, WCHS is a case-control study conducted in NY and NJ involving both Caucasian and AA women. In NY, cases were recruited through major hospitals with large referral patterns for AA women in four boroughs of the metropolitan NYC area (Manhattan, Brooklyn, Bronx, and Queens). Controls were identified through random digit dialing (RDD) of residential telephone and cell phone numbers; recruitment in NYC ended in 2008.

In NJ, data collection was based at The Cancer Institute of New Jersey. Newly diagnosed women with histologically confirmed invasive breast cancer or DCIS (ductal carcinoma in situ) were identified through the NJ State Cancer Registry using rapid case ascertainment in seven NJ counties, including Bergen, Essex, Hudson, Mercer, Middlesex, Passaic, and Union. All AA women meeting the eligibility criteria and a random sample of eligible Caucasian women matched to AA cases by county were identified. Caucasian and AA controls were recruited through RDD supplemented by community recruitment efforts for AA women in the same counties (mainly through churches and health events) with the help of community partners and AA breast cancer advocates (27). Recruitment in NJ concluded in March 2012. The eligibility criteria for cases were: self-identified AA and Caucasian women, 20–75 years of age at diagnosis, no previous history of cancer except non-melanoma skin cancer, recently diagnosed with primary, histologically confirmed breast cancer or DCIS, and English speaking. Controls without a history of any cancer diagnosis other than non-melanoma skin cancer living in the same seven NJ counties as cases were frequency matched to cases by self-reported race and age.

Data Collection

Data collection for the WCHS took place during an in-person interview and included the main study questionnaire (administered by the interviewer) that elicited information on demographics and known and potential risk factors for breast cancer such as physical activity, hormone use, reproductive history, alcohol consumption, smoking, etc. Measures of body composition were collected by bioelectrical impedance analyses using the Tanita scale while height, waist and hip circumferences were measured by the interviewers.

The GSEL- Food Frequency Questionnaire (FFQ) developed by the Nutrition Assessment Shared Resource at the Fred Hutchinson Cancer Research Center (FHCRC) queried about both usual frequency and portion size for approximately 125 food items, including red meat and poultry, during the 12 months prior to reference date (date of diagnosis for cases and approximately 97 days prior to date of interview for controls) to ensure comparability in recall period. For each food item, a medium serving size was specified (e.g. 1 cup, 1 tbsp, 1 bar, 2 slices, 4 ounces), and participants were asked if they consumed a small serving (one half or less of the medium serving size), the same quantity as the given medium serving size, or a large serving (1.5 times or more of the medium serving).

The GSEL-FFQ was based on questionnaires used in two large NIH-funded studies, the Selenium and Vitamin E Cancer Prevention Trial (SELECT) and the VITamins and Lifestyle study (VITAL). Validation data for the FFQ used in WCHS also come from the Women’s Health Initiative (WHS), the largest research study in the US with a focus on diet and health also based at the FHCRC. A detailed validation study of the FFQ by Paterson et al (29) demonstrated that the WHI FFQ which was compared to criterion such as the 24-hour dietary recall and 7-day food record had similar or better measurement properties than other popular FFQs such as Block FFQ. Another validation study that specifically assessed the validity and reliability of the WHI FFQ in minority populations observed that when using self-administered FFQs in minority or poorly educated populations, there is a need for participant training (30). To address this concern in the WCHS, although the FFQ was

generally self-administered, it was completed as part of the in-person appointment to allow the interviewer to educate all participants about how to respond to the FFQ.

In addition to querying red meat and poultry consumption, processed red meat items included lunch meats and items such as bacon, sausages, bratwursts, chorizo, salami, and hot dogs. Total red meat was computed as the sum of processed and unprocessed red meat. The poultry variable was created as the summation of two FFQ items: “fried chicken, including nuggets and tenders” and “roasted, stewed, grilled or broiled chicken and turkey”, and hence does not include processed poultry. Total consumption of each food group in grams was calculated as a function of frequency and portion size of intake.

Overall, the participation rate for those who were contacted and eligible was 78.7% and 48.2% in AA cases and controls respectively and 79% and 49% in Caucasian cases and controls respectively. Participation rates for the NJ site were higher (72.5% for AA and 71.6% for Caucasians) than for NY (44.3% in AA and 52% in Caucasian), but the participation rates of cases and controls in each site for each race were similar. A total of 827 AA cases, 905 AA controls, 772 Caucasian cases, and 715 Caucasian controls participated in the study, and over 97% of the participants completed the FFQ resulting in a total of 803 AA cases, 889 AA controls, 755 Caucasian cases, and 701 Caucasian controls available for analyses. Informed consent was obtained from all participants to collect and analyze data, and to publish findings in aggregate. The study was approved by the Institutional Review Boards at the University of Medicine and Dentistry of New Jersey (now Rutgers University), Mount Sinai School of Medicine, and Roswell Park Cancer Institute.

Statistical Analyses

The distribution of selected categorical variables (demographic, socio-economic, and known and potential breast cancer risk factors) for AA and Caucasian cases and controls were summarized using frequencies and proportions. Chi square tests were used to compare the differences in proportions between cases and controls for AA and Caucasian women separately. Red meat and poultry intakes were expressed as a density measure as grams per day for every 1,000 kcal of total energy intake for inclusion in the model that also adjusted for total energy intake as per the multivariate nutrient density method (31). The density measures for each food group were categorized into quartiles based on distribution among all controls with the fourth quartile representing the highest level of consumption. Using race-specific percentiles did not substantially change the case-control distribution for the food groups. Hence, to ease direct comparison of results across both races, we maintained the percentile cut points computed based on distribution from all controls.

Unconditional logistic regression analyses were used to compute odds ratios (OR) and 95% confidence intervals (CI). Tests for linear trend were conducted by including the median intake in each quartile as a continuous variable in regression models. Subgroup analyses included further stratification by menopausal status. Polytomous logistic regression was used to simultaneously model risk of ER positive and ER negative tumors with controls as the reference. Tests of heterogeneity of odds ratios for stratified and polytomous models were computed using the Wald test. We also conducted case-only analyses to model risk of ER negative tumors.

Potential confounders of interest that were included in multivariable models were age, ethnicity (Hispanic or Non-Hispanic), country of origin (“US born”, “Caribbean born”, “Other”), education (“less than 12th grade”, “high school graduate or equivalent”, some college”, “college graduate”, “post-graduate degree”), age at menarche, age at menopause (only for postmenopausal women), menopausal status (if not stratified by this variable), parity (continuous), age at first birth (“0–19”, “20–24”, “25–30”, “≥31”), breastfeeding status (ever/never), history of benign breast disease, family history of breast cancer, hormone replacement therapy (HRT) use, oral contraceptive use (OC), body mass index (BMI), and total energy intake.

In sensitivity analyses, we repeated regression analyses after further adjustment for total fat intake (in a model that excluded total calories) and alcohol. We also further evaluated if associations differed when excluding AA community controls (n=339), non-invasive cases (n=253), HRT users (n=575), and participants with total energy intake of less than 500 calories or more than 4500 calories (n=157). Finally, we repeated analyses also adjusting for fruits and vegetables as potential confounders.

All analyses were conducted using SAS version 9.2 (Cary, North Carolina).

Results

The distribution of demographic and socio-economic characteristics as well as potential risk factors for breast cancer in the entire study population, stratified by race is summarized in Table 1. Overall, Caucasian women tended to have higher education and were less likely to be obese than AA women. As compared to AA and Caucasian controls, both AA and Caucasian cases were more likely to have ever used HRT, have a history of benign breast disease, and family history of breast cancer. Consistent with the literature, a higher percentage of AA cases were diagnosed at stage 2 or higher and with ER negative tumors. Unprocessed red meat intake was higher in Caucasian than in AA women while the opposite was true for processed meat and poultry consumption.

Tables 2 and 3 show associations between meat consumption and breast cancer risk among Caucasian women, also stratified by menopausal status and ER status. Among Caucasian women overall, consuming processed meat appeared to increase breast cancer risk, when comparing the highest quartile to the lowest (OR=1.48; 95% CI: 1.07–2.04). Similar associations were noted for unprocessed red meat (OR=1.40; 95% CI: 1.01–1.94) and poultry consumption (OR=1.42; 95% CI: 1.01–1.99), although a significant linear trend was only noted for poultry intake (p trend=0.04). Associations with consumption of processed meat were stronger among postmenopausal Caucasian women (OR=1.74; 95% CI: 1.06–2.87) while among premenopausal women, risk was significantly increased with higher unprocessed red meat (OR=1.66; 95% CI: 1.05–2.64) and poultry intakes (OR=2.33; 95% CI: 1.44–3.77). However, significant heterogeneity in odds ratios across menopausal status was only observed for associations with poultry intake (p for heterogeneity=0.03).

When stratifying by ER status among Caucasian women (Table 3), there were no marked differences in risk associated with consumption of red meat although a significant

association was observed for increased risk of ER positive tumors (OR=1.51; 95% CI: 1.02–2.24), with a significant linear trend (p=0.03). The magnitude of associations with processed meat were stronger for ER negative tumors (OR=1.89; 95% CI: 1.00–3.57), while consuming unprocessed red meat in the highest quartile increased risk of ER positive tumors (OR=1.64; 95% CI: 1.10– 2.43), but numbers in these cells were relatively small and tests for heterogeneity of odds ratios across the two ER groups were not significant. Although there were no associations between poultry intake and ER positive breast cancer, higher consumption was significantly associated with risk of ER negative tumors (OR=2.55; 95% CI: 1.29–5.03, p trend=0.01), with a statistically significant p for heterogeneity (p=0.02). In case-only analyses, the increased risk of ER negative tumors associated with poultry intake in the highest quartile remained (OR=2.22; 95% CI: 1.07– 4.61).

Similar analyses for AA women are shown in Tables 4 and 5. In contrast to results observed in Caucasian women, there was no clear evidence of an association between red meat, poultry, and breast cancer risk in AA women overall or when stratified by menopausal status. When evaluating by ER status (Table 5) or in case-only analyses in AA women, there were no significant associations observed except for a borderline increase in risk of ER positive tumors associated with consuming processed meat (OR for quartile 4 vs. 1=1.36; 95% CI: 0.94–1.97) and a significant linear trend (p trend=0.04). None of the tests for heterogeneity of odds ratios were statistically significant.

Overall tests for heterogeneity to assess difference in odds ratios by race resulted in p values of 0.11 for total red meat, 0.39 for processed meat, 0.08 for unprocessed red meat, and 0.27 for poultry (data not shown). Mutual adjustment of processed meat, unprocessed red meat, and poultry did not meaningfully change odds ratios (data not shown). Adjusting for fruit and vegetable intake did not alter any of the associations observed or direction of OR, with the only notable change being an attenuation of OR for processed meat in Caucasian postmenopausal women (OR=1.54; 95% CI: 0.94–2.53), and for total red meat among all Caucasian women (OR=1.29; 95% CI: 0.93–1.81). None of the results from sensitivity analyses altered study conclusions.

Discussion

In this case-control study involving a large sample of AA and Caucasian women, a positive association between intake of processed and unprocessed red meat, poultry and breast cancer risk was limited to Caucasian women. To our knowledge, this is the first investigation of these foods and breast cancer risk in a study of both AA and Caucasian women with evaluation by menopause as well as ER status. Consumption of processed meat in the highest quartile appeared to be more strongly associated with postmenopausal breast cancer while unprocessed red meat and poultry were strongly associated with premenopausal breast cancer in Caucasian women, but odds ratios across menopausal status were significantly different only for poultry intake. Similarly, poultry consumption appeared particularly harmful for ER negative tumors among Caucasian women. Red meat intake in both processed and unprocessed forms increased breast cancer risk in Caucasian women regardless of hormone receptor status, albeit not all risk estimates reached statistical significance. Among AA women, there was no strong evidence relating red meat and poultry

intake to breast cancer risk except for a significant positive linear trend between consuming processed meat and ER positive tumors.

Results from past studies that have investigated the impact of red and processed meats on breast cancer risk are inconsistent and have largely involved only Caucasian women. No significant association between consumption of total meat, red meat and breast cancer risk was observed in the Pooling Project (9) or in the EPIC cohort (10) for red and processed meat. In contrast, increased breast cancer risks associated with consumption of processed meat (6, 11, 12), red meat (11, 12, 32), or total red meat combining both processed and unprocessed meat (5) have been observed, similar to findings in Caucasian women in our study. Among AA women, the null findings are consistent with the only other study on this topic, the Black Women's Health Study (33), which also found no association between any meat type and breast cancer risk in AA women even when stratified by menopausal and hormone receptor status. However, in our study, a significant linear trend was observed for consumption of processed meat and ER positive tumors among AA women, but the odds ratios were not significantly different by hormone receptor status.

Racial differences in the association between diet and cancer risk have been observed for other cancer sites. For example, a high fat/meat/potatoes pattern appeared to increase risk of rectal cancer only in Caucasians and not in AA despite the pattern being observed in both race groups (34). A fruit-vegetable pattern was significantly associated with reduced colon cancer risk only in Caucasians in the North Carolina Colon Cancer Study (35). In our study, the overall test for heterogeneity by race was borderline significant only for fresh red meat intake ($p=0.08$) despite stronger associations in Caucasian women for red meat and poultry consumption. Hence, further evaluation by menopausal and hormone receptor status was conducted.

It has been postulated that similar to risk factors such as adiposity, diet in early life may have a different impact on breast cancer risk than diet in later ages; thus indicating potential modification of risks by menopausal status (36). A meta-analysis (37) of 10 studies that investigated the impact of red meat consumption and breast cancer risk focusing on premenopausal women observed a summary relative risk of 1.24 (95% CI: 1.08–1.42). In our study, although risk estimates for processed meat were stronger in postmenopausal Caucasian women, in general, associations for total red meat were similar in pre and postmenopausal Caucasian women, which was also confirmed by tests of heterogeneity. This is consistent with findings from the UK Women's Cohort Study (12) that reported highest risk for highest red meat eaters (processed and unprocessed) in both pre- and postmenopausal groups, with stronger estimates in postmenopausal women, in addition to a Canadian study reporting an elevated breast cancer risk among postmenopausal women with consumption of total meat and processed meat (38).

Further research has been proposed to also investigate the association between consumption of red meat and breast cancer specifically stratified by hormone receptor status (4, 39). Previous studies that evaluated all red meat intake and breast cancer risk stratified by receptor status have observed stronger associations for hormone receptor positive tumors than for hormone receptor negative tumors (36, 40). In our study, although total red meat

was associated with increased risk of ER positive tumors among Caucasian women, processed meat appeared to be more strongly related to increased risk of ER negative tumors. However, tests for heterogeneity of odds ratios suggested no significant difference in the associations by hormone receptor status. Recent results from the Shanghai Breast Cancer Study reported elevated breast cancer risk for all types of meat regardless of hormone receptor status (5). These findings could indicate presence of both estrogenic and non-estrogenic pathways involving meat and carcinogenesis.

There are several potential mechanisms for the role of red meat in the carcinogenesis process. Nitrites that are used to preserve processed meats undergo chemical reactions endogenously to form nitrosamines, also a mutagen and probable carcinogen (14, 41). Cooking red meat at high temperatures (charring, grilling, frying) lead to the production of heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons, which are mutagens and suspected carcinogens (14, 42). HCA content of cooked meat has shown to directly relate to increases in cooking temperature (24). A 4.6-fold increased risk of breast cancer was observed among postmenopausal women who consistently ate well-done red meat in the Iowa Women's Health Study (43). Results from the Nashville Breast Health Study also showed stronger risk for high intake of well-done red meat in both pre and postmenopausal women (44). A past report (45) from the Behavioral Risk Factor Surveillance System observed a much higher percentage of Caucasian respondents reporting consumption of 'pink' hamburgers compared to AA respondents in both NY (24.3% vs. 6.8%) and NJ (26.2% vs. 8.6%), which could indicate increased susceptibility of carcinogens for AA women. However, there is also some evidence that the impact of well-done meat on breast cancer risk could be modified by certain polymorphisms in the NAT1, NAT2, GSTM1, GSTT1, and SULT1A1 genes that encode enzymes involved in HCA activation or detoxification (46). In fact, a case-control study (47) nested in an Iowa cohort of postmenopausal women observed a 3.4-fold greater breast cancer risk among women who consumed well- or very well done meat compared to consuming meat that was rare or medium, and who carried a null GSTM1 or GSTT1 genotype. Interestingly, Caucasians appear to have a much higher frequency of a null GSTM1 or GSTT1 gene than AA (48). Hence, it is possible that there are potential racial differences in gene-environment interactions resulting in differences in the way animal foods and other potential carcinogens are metabolized and their consequential impact on breast tissue.

Other plausible mechanisms include potential estrogenic effects of HCAs shown in animal studies, thus resulting in possible stimulation of ER and PR gene expression (49, 50).

Furthermore, heme iron present in red meat and responsible for its dark red color could have a catalytic effect on endogenous formation of nitrosamines (23), and has been shown to promote estrogen-induced tumors (51). Exposure to meat-derived mutagens has also been observed to be significantly correlated with DNA adducts in breast tissue, potentially leading to unrepaired DNA damage and carcinogenesis (52). A cross-sectional analysis found mean plasma concentrations of sex hormone-binding globulin (SHBG) to be lower with higher intakes of total and fresh red meat (53). Lower SHBG levels correspond with increased bioavailability of estradiol (54, 55). Finally, there is evidence linking exogenous

hormone treatment of beef cattle to increased risk of estrogen-related illnesses even with low levels of exposure in the long term (56).

In our study, there was also a positive association between poultry consumption and breast cancer risk in Caucasian women, with over 2-fold greater risk among premenopausal Caucasian women and among women with ER negative tumors, most commonly seen among premenopausal women (18). Past evidence relating poultry intake and breast cancer risk is inconclusive (5, 8, 9, 14, 44, 52). A recent cross-sectional study reported that intake of chicken, high-fat dairy products, and animal fat may be important determinants of oxidative stress in women with breast cancer (57), and increased breast cancer risk with high poultry consumption was also reported in the Shanghai Breast Cancer Study (5). The null findings in AA women related to poultry consumption are consistent with results from the Black Women's Health Study (33).

Biological mechanisms for the role of white meat in the cancer process involve HCAs (24) and the potential influence of fat in the meat supplemented by meal preparation methods such as frying. Adjusting for fat did not have a major impact on study results. We also repeated analyses adjusting for red meat, but the findings for poultry intake remained unchanged. The largest proportion (∼ 70%) of mean dietary intake of total HCAs in the US appears to be comprised of 2-amino-1-methyl-6-phenylimidazo [4,5-b] pyridine (PhIP), with AA consuming more than Caucasians, which has been attributed to higher intake of chicken and pan-fried meats (26). High PhIP concentrations in well done chicken and beef have also been shown in other studies (24, 25, 58, 59). However, results from the Nashville Breast Health Study on chicken consumption and breast cancer risk were null, and even when poultry consumption was high, there was no correlation between cooking poultry at high temperatures and formation of DNA adducts in breast tissue (44, 52). The null findings in AA women in our study, despite higher poultry intake levels warrants further investigation into potential racial differences in biological pathways involving PhIP metabolism. Nevertheless, findings may have occurred due to chance and need replication in view of potential biases described herein.

Limitations of this study include lack of information on meat doneness levels that precluded assessment of risks associated with different cooking methods. This study could have been affected by inherent limitations of the case-control study design such as recall and selection bias. For instance, cases could have changed their dietary habits as a result of the cancer diagnosis, and report recent behavior instead of recalling behavior prior to diagnosis. To evaluate the extent of this bias, we evaluated behavioral change after diagnosis (data not shown). A greater proportion of both AA and Caucasian cases reported that they had decreased meat intake and chicken intake since diagnosis than controls. However, this only suggests that we may be underestimating the association with meat and poultry consumption.

To further assess if there were racial differences in recall, we also compared the proportion of AA and Caucasian cases and controls who had increased or decreased meat and chicken intake since the reference date. Although a higher proportion of cases in both races had decreased meat intake and chicken intake since diagnosis as compared to controls, the

differences in proportions were similar in AA and Caucasian women. Self-reported dietary data have also been used to assess racial differences in nutrition by other studies (60, 61). The low response rates, especially among controls in our study could result in potential selection bias, but response rates as low as 50% are not uncommon in population studies (62). Finally, to evaluate selection bias resulting from possible differences by source of controls, we repeated analyses after excluding AA community controls, but results did not change.

The major strength of this study is the large sample of AA women that allowed subgroup analyses to further evaluate associations by menopausal status and hormone receptor status in each race. Extensive collection of all major and potential risk factors as well as in-person interviews to collect detailed dietary data enabled accounting for potential confounding to a large extent.

Conclusions

This study supports an association between processed and unprocessed red meat, poultry meat consumption and increased breast cancer risk in Caucasian women. Although heterogeneity tests across race, menopausal status, and hormone receptor status sub-groups confirmed differences only for poultry intake, magnitude of associations appeared stronger in certain sub-groups, which may be obscured if studies do not stratify but only adjust for these factors. As this is one of the first studies to examine red meat and poultry consumption and breast cancer risk in AA women, further research is needed to make more definitive conclusions in this minority population while taking into account cultural preferences for cooking methods and meat doneness levels.

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Table 1

Distribution of selected characteristics for breast cancer among women participating in WCHS, n=3148

	AA women		Caucasian women	
	Cases (n=803) N (%)	**Controls (n=889) N (%)**	**Cases (n=755) N (%)**	**Controls (n=701) N (%)**
Age at interview (yrs)[1]				
20–34	37 (4.6)	71 (8.0)	23 (3.1)	32 (4.6)
35–44	162 (20.2)	182 (20.5)	140 (18.5)	156 (22.3)
45–54	261 (32.5)	320 (36)	254 (33.6)	257 (36.7)
55–64	262 (32.6)	272 (30.6)	247 (32.7)	251 (35.8)
65–76	81 (10.1)	44 (4.9)	91 (12.1)	5 (0.7)
Education[3]				
<High school	118 (14.7)	112 (12.6)	21 (2.8)	10 (1.4)
High school graduate	241 (30)	227 (25.5)	127 (16.8)	69 (9.8)
Some college	213 (26.5)	259 (29.1)	165 (21.9)	132 (18.8)
College graduate	141 (17.6)	180 (20.2)	230 (30.5)	226 (32.2)
Post-graduate degree	90 (11.2)	111 (12.5)	212 (28.1)	264 (37.7)
Country of origin[1]				
United States	552 (68.7)	711 (80)	639 (84.6)	617 (88)
Caribbean countries	189 (23.5)	129 (14.5)	25 (3.3)	2 (0.3)
Other	62 (7.7)	49 (5.5)	91 (12.1)	82 (11.7)
Ethnicity[1]				
Hispanic	45 (5.6)	26 (2.9)	62 (8.2)	15 (2.1)
Non-Hispanic	758 (94.4)	863 (97.1)	693 (91.8)	686 (97.9)
Marital Status[2]				
Married	287 (35.7)	306 (34.5)	468 (62.1)	477 (68)
Living as married	13 (1.6)	19 (2.1)	22 (2.9)	22 (3.1)
Widowed	74 (9.2)	58 (6.5)	40 (5.3)	19 (2.7)
Separated	62 (7.7)	57 (6.4)	14 (1.9)	16 (2.3)
Divorced	138 (17.2)	136 (15.3)	91 (12.1)	73 (10.4)
Single, never married or never lived as married	229 (28.5)	312 (35.1)	119 (15.8)	94 (13.4)
Age at menarche (yrs)				
<12	228 (28.4)	250 (28.2)	175 (23.4)	157 (22.6)
12–13	365 (45.4)	399 (44.9)	416 (55.6)	368 (53)
>13	210 (26.2)	239 (26.9)	157 (21)	170 (24.5)
Menopausal status				
Premenopausal	408 (50.8)	463 (52.1)	389 (51.5)	385 (54.9)
Postmenopausal	395 (49.2)	426 (47.9)	366 (48.5)	316 (45.1)

	AA women		Caucasian women	
	Cases (n=803) N (%)	Controls (n=889) N (%)	Cases (n=755) N (%)	Controls (n=701) N (%)
Age at menopause (yrs)[2]				
≤45	36 (9.4)	52 (12.3)	29 (8.1)	27 (8.7)
46–49	60 (15.6)	108 (25.6)	73 (20.4)	71 (22.9)
50–54	247 (64.2)	220 (52.1)	204 (57)	175 (56.4)
>55	42 (10.9)	42 (10)	52 (14.5)	37 (11.9)
Parity (livebirths)				
0	124 (15.4)	148 (16.7)	237 (31.4)	206 (29.4)
1–2	414 (51.6)	438 (49.3)	355 (47)	355 (50.6)
3–4	200 (24.9)	237 (26.7)	146 (19.3)	117 (16.7)
>5	65 (8.1)	66 (7.4)	17 (2.3)	23 (3.3)
Age at first birth (yrs)				
Nulliparous (0 birthcount)	124 (15.5)	148 (16.7)	237 (31.4)	206 (29.4)
≤19	253 (31.6)	294 (33.1)	36 (4.8)	32 (4.6)
20–24	195 (24.3)	220 (24.8)	134 (17.8)	110 (15.7)
25–30	149 (18.6)	120 (13.5)	190 (25.2)	170 (24.3)
>31	81 (10.1)	106 (11.9)	158 (20.9)	183 (26.1)
Breastfeeding[3]				
Never	470 (58.5)	529 (59.5)	430 (57)	355 (50.6)
Ever	333 (41.5)	360 (40.5)	325 (43)	346 (49.4)
Family history of breast cancer[3]				
No	687 (85.6)	786 (88.4)	578 (76.5)	584 (83.3)
Yes	116 (14.4)	103 (11.6)	177 (23.4)	117 (16.7)
Past benign breast disease[1]				
No	547 (68.3)	685 (77.1)	431 (57.6)	466 (66.7)
Yes	254 (31.7)	203 (22.9)	317 (42.4)	232 (33.3)
HRT use				
Never	682 (85.4)	785 (88.5)	559 (74)	540 (77.1)
Ever	117 (14.6)	102 (11.5)	196 (26)	160 (22.9)
Oral contraceptive use[3]				
Never	333 (41.5)	387 (43.6)	261 (34.7)	203 (29)
Ever	470 (58.5)	501 (56.4)	492 (65.3)	498 (71)
BMI				
Underweight/Normal	151 (18.8)	157 (17.7)	342 (45.3)	317 (45.3)
Overweight	235 (29.3)	255 (28.7)	206 (27.3)	191 (27.3)
Obese	416 (51.9)	477 (53.7)	207 (27.4)	192 (27.4)

	AA women		Caucasian women	
	Cases (n=803) N (%)	**Controls (n=889) N (%)**	**Cases (n=755) N (%)**	**Controls (n=701) N (%)**
Stage of presentation				
Non-invasive	119 (22.7)	–	134 (27)	–
Stage 1	158 (30.1)	–	204 (41.1)	–
Stage 2	169 (32.2)	–	124 (25)	–
Stage 3	76 (14.5)	–	33 (6.7)	–
Stage 4	3 (0.6)	–	1 (0.2)	–
Estrogen receptor status				
ER positive	409 (69)	–	413 (82.1)	–
ER negative	184 (31)	–	90 (17.9)	–
Total red meat intake (grams/day/1000 kcal)				
Mean ± SD	25.26 ± 22.22	26.95 ± 22.43	30.09 ± 22.31	28.35 ± 22.22
Median	20.22	21.59	25.13	23.05
Processed meat intake (grams/day/1000 kcal)				
Mean ± SD	11.94 ± 12.55	11.88 ± 11.69	9.72 ± 10.39	9.39 ± 11.45
Median	8.27	8.89	6.43	5.95
Unprocessed red meat intake (grams/day/1000 kcal)				
Mean ± SD	13.32 ± 16.17	15.07 ± 17.33	20.37 ± 18.36	18.99 ± 17.06
Median	8.37	9.29	15.80	14.58
Poultry intake (grams/day/1000 kcal)				
Mean ± SD	27.64 ± 24.35	28.95 ± 24.88	23.38 ± 19.06	21.90 ± 19.14
Median	21.18	22.81	18.82	17.70

[1] chi square p value<0.05 for AA and white women

[2] chi square p value<0.05 in AA women

[3] chi square p value<0.05 in white women

Table 2

Red meat and poultry intake, and breast cancer risk among Caucasian women by menopausal status

	All women				Premenopausal				Postmenopausal			
(grams/day/ 1,000 kcal)	**Ca (N)**	**Co (N)**	**OR**	**95% CI**	**Ca (N)**	**Co (N)**	**OR**	**95% CI**	**Ca (N)**	**Co (N)**	**OR**	**95% CI**
Total red meat												
Q1 (≤10.81)	153	163	Ref		78	89	Ref		75	74	Ref	
Q2 (10.82–22.45)	171	181	1.08	0.78–1.49	96	94	1.56	0.99–2.45	75	87	0.79	0.48–1.30
Q3 (22.46–40.75)	236	169	1.60	1.16–2.20	119	87	2.05	1.31–3.23	117	82	1.41	0.86–2.30
Q4 (>40.75)	195	188	1.24	0.90–1.72	96	115	1.38	0.88–2.19	99	73	1.37	0.83–2.26
P for linear trend			*0.12*				*0.32*				*0.06*	
P for heterogeneity					*0.13*							
Processed meat												
Q1 (≤2.35)	186	211	Ref		97	105	Ref		89	106	Ref	
Q2 (2.36–7.57)	231	184	1.41	1.05–1.89	112	93	1.25	0.82–1.91	119	91	1.69	1.08–2.64
Q3 (7.58–15.19)	167	166	1.13	0.82–1.55	92	108	1.01	0.66–1.54	75	58	1.54	0.93–2.54
Q4 (>15.19)	171	140	1.48	1.07–2.04	88	79	1.39	0.88–2.20	83	61	1.74	1.06–2.87
P for linear trend			*0.07*				*0.27*				*0.08*	
P for heterogeneity					*0.62*							
Unprocessed red meat												
Q1 (≤4.14)	129	148	Ref		64	84	Ref		65	64	Ref	
Q2 (4.15–11.76)	177	142	1.58	1.12–2.24	95	76	1.71	1.05–2.79	82	66	1.51	0.89–2.56
Q3 (11.77–24.70)	207	187	1.40	1.01–1.96	111	104	1.76	1.10–2.80	96	83	1.20	0.71–2.01
Q4 (>24.70)	242	223	1.40	1.01–1.94	119	121	1.66	1.05–2.64	123	102	1.39	0.85–2.28
P for linear trend			*0.29*				*0.16*				*0.42*	
P for heterogeneity					*0.74*							
Poultry												
Q1 (≤10.66)	196	212	Ref		88	127	Ref		108	86	Ref	
Q2 (10.67–19.82)	203	189	1.19	0.88–1.60	112	106	1.62	1.07–2.45	91	83	0.92	0.58–1.47
Q3 (19.83–35.08)	205	176	1.28	0.94–1.73	111	88	2.05	1.33–3.16	94	88	0.84	0.53–1.34

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	All women				Premenopausal				Postmenopausal			
(grams/day/ 1,000 kcal)	**Ca (N)**	**Co (N)**	**OR**	**95% CI**	**Ca (N)**	**Co (N)**	**OR**	**95% CI**	**Ca (N)**	**Co (N)**	**OR**	**95% CI**
Q4 (>35.08)	151	124	1.42	1.01–1.99	78	65	2.33	1.44–3.77	73	59	0.99	0.59–1.66
P for linear trend			*0.04*				*<0.001*				*0.93*	
P for heterogeneity					*0.03*							

Ca - case; Co- control

Odds ratios are adjusted for age, ethnicity, country of origin, education, age at menarche, menopausal status (when not stratified by this variable), age at menopause (only for postmenopausal women), parity, age at first birth, breastfeeding status, family history of breast cancer, OC use, history of benign breast disease, HRT use, total energy intake, BMI

Table 3

Red meat, poultry intake and breast cancer risk among Caucasian women by hormone receptor status

				ER+ vs. Co		ER− vs. Co		ER− vs ER+	
(grams/day/1,000 kcal)	**ER+ Ca (N)**	**ER− Ca (N)**	**Co (N)**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Total red meat									
Q1 (≤10.81)	74	21	163	Ref		Ref		Ref	
Q2 (10.82–22.45)	92	15	181	1.20	0.81–1.79	0.64	0.31–1.32	0.46	0.21–1.02
Q3 (22.46–40.75)	127	28	169	1.71	1.16–2.53	1.29	0.67–2.46	0.65	0.33–1.31
Q4 (>40.75)	120	26	188	1.51	1.02–2.24	1.31	0.68–2.51	0.86	0.42–1.73
P for linear trend				*0.03*		*0.16*		*0.80*	
P for heterogeneity				*0.41*				–	
Processed meat									
Q1 (≤2.35)	102	23	211	Ref		Ref		Ref	
Q2 (2.36–7.57)	123	25	184	1.35	0.94–1.92	1.23	0.65–2.30	0.92	0.47–1.81
Q3 (7.58–15.19)	91	14	166	1.09	0.75–1.59	0.84	0.41–1.72	0.74	0.34–1.60
Q4 (>15.19)	97	28	140	1.47	1.00–2.15	1.89	1.00–3.57	1.32	0.67–2.61
P for linear trend				*0.12*		*0.06*		*0.35*	
P for heterogeneity				*0.51*				–	
Unprocessed red meat									
Q1 (≤4.14)	65	15	148	Ref		Ref		Ref	
Q2 (4.15–11.76)	88	24	142	1.66	1.08–2.54	1.80	0.88–3.71	0.95	0.43–2.09
Q3 (11.77–24.70)	117	22	187	1.56	1.04–2.35	1.28	0.61–2.65	0.75	0.34–1.64
Q4 (>24.70)	143	29	223	1.64	1.10–2.43	1.67	0.84–3.34	0.98	0.46–2.08
P for linear trend				*0.09*		*0.34*		*0.95*	
P for heterogeneity				*0.86*				–	
Poultry									
Q1 (≤10.66)	107	21	212	Ref		Ref		Ref	
Q2 (10.67–19.82)	112	26	189	1.25	0.88–1.78	1.43	0.76–2.71	1.13	0.57–2.24
Q3 (19.83–35.08)	123	19	176	1.35	0.94–1.93	1.07	0.54–2.15	0.73	0.35–1.55

				ER+ vs. Co		ER− vs. Co		ER− vs ER+	
(grams/day/1,000 kcal)	**ER+ Ca (N)**	**ER− Ca (N)**	**Co (N)**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Q4 (>35.08)	71	24	124	1.09	0.72–1.65	2.55	1.29–5.03	2.22	1.07–4.61
P for linear trend				*0.67*		*0.01*		*0.05*	
P for heterogeneity				*0.02*				–	

Ca - case; Co - control

Odds ratios are adjusted for age, ethnicity, country of origin, education, age at menarche, menopausal status, parity, age at first birth, breastfeeding status, family history of breast cancer, OC use, history of benign breast disease, HRT use, total energy intake, BMI

Table 4

Red meat and poultry intake, and breast cancer risk among AA women by menopausal status

	All women				Premenopausal				Postmenopausal			
(grams/day/ 1,000 kcal)	**Ca (N)**	**Co (N)**	**OR**	**95% CI**	**Ca (N)**	**Co (N)**	**OR**	**95% CI**	**Ca (N)**	**Co (N)**	**OR**	**95% CI**
Total red meat												
Q1 (≤10.81)	228	235	Ref		119	126	Ref		109	109	Ref	
Q2 (10.82–22.45)	209	216	1.17	0.89–1.55	104	106	1.36	0.90–2.04	105	110	1.03	0.69–1.55
Q3 (22.46–40.75)	212	229	1.10	0.82–1.46	103	116	1.22	0.80–1.84	109	112	1.01	0.67–1.53
Q4 (>40.75)	154	209	0.96	0.70–1.30	82	115	1.15	0.74–1.78	72	95	0.79	0.50–1.25
P for linear trend			*0.58*				*0.76*				*0.29*	
P for heterogeneity					*0.67*							
Processed meat												
Q1 (≤2.35)	185	187	Ref		89	91	Ref		96	96	Ref	
Q2 (2.36–7.57)	185	213	1.03	0.76–1.39	104	116	1.04	0.67–1.60	81	97	1.02	0.65–1.59
Q3 (7.58–15.19)	196	232	1.12	0.82–1.52	101	124	1.12	0.72–1.75	95	107	1.14	0.73–1.78
Q4 (>15.19)	237	257	1.21	0.89–1.64	114	132	1.26	0.80–1.99	123	126	1.12	0.72–1.73
P for linear trend			*0.18*				*0.26*				*0.59*	
P for heterogeneity					*0.97*							
Unprocessed red meat												
Q1 (≤4.14)	253	250	Ref		135	131	Ref		118	119	Ref	
Q2 (4.15–11.76)	237	254	0.95	0.73–1.24	109	125	0.96	0.66–1.42	128	129	1.05	0.72–1.53
Q3 (11.77–24.70)	186	210	0.98	0.74–1.30	93	111	1.04	0.69–1.56	93	99	0.99	0.65–1.50
Q4 (>24.70)	127	175	0.84	0.61–1.14	71	96	0.97	0.63–1.50	56	79	0.75	0.47–1.19
P for linear trend			*0.28*				*0.95*				*0.19*	
P for heterogeneity					*0.77*							
Poultry												
Q1 (≤10.66)	176	185	Ref		92	92	Ref		84	93	Ref	
Q2 (10.67–19.82)	196	209	1.09	0.81–1.46	99	99	1.13	0.73–1.74	97	110	1.09	0.71–1.69
Q3 (19.83–35.08)	212	221	1.12	0.83–1.51	115	130	1.07	0.71–1.61	97	92	1.22	0.78–1.90

	All women				Premenopausal				Postmenopausal			
(grams/day/ 1,000 kcal)	**Ca (N)**	**Co (N)**	**OR**	**95% CI**	**Ca (N)**	**Co (N)**	**OR**	**95% CI**	**Ca (N)**	**Co (N)**	**OR**	**95% CI**
Q4 (>35.08)	219	274	0.92	0.69–1.23	102	142	0.83	0.55–1.25	117	131	0.99	0.65–1.51
P for linear trend			*0.40*				*0.21*				*0.81*	
P for heterogeneity					*0.87*							

Ca - case; Co - control

Odds ratios are adjusted for age, ethnicity, country of origin, education, age at menarche, menopausal status (when not stratified by this variable), age at menopause (only for postmenopausal women), parity, age at first birth, breastfeeding status, family history of breast cancer, OC use, history of benign breast disease, HRT use, total energy intake, BMI

Table 5

Red meat, poultry intake and breast cancer risk among AA women by hormone receptor status

				ER+ vs. Co		ER− vs. Co		ER− vs ER+	
(grams/day/1,000 kcal)	**ER+ Ca (N)**	**ER− Ca (N)**	**Co (N)**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Total red meat									
Q1 (≤10.81)	105	50	235	Ref		Ref		Ref	
Q2 (10.82–22.45)	102	46	216	1.26	0.89–1.78	1.13	0.71–1.81	0.96	0.57–1.62
Q3 (22.46–40.75)	108	59	229	1.24	0.87–1.77	1.30	0.82–2.06	1.13	0.68–1.87
Q4 (>40.75)	94	29	209	1.29	0.89–1.86	0.73	0.42–1.24	0.64	0.36–1.16
P for linear trend				*0.26*		*0.26*		*0.18*	
P for heterogeneity				*0.13*				–	
Processed meat									
Q1 (≤2.35)	91	39	187	Ref		Ref		Ref	
Q2 (2.36–7.57)	84	41	213	0.94	0.64–1.37	1.03	0.62–1.73	1.11	0.63–1.97
Q3 (7.58–15.19)	101	47	232	1.17	0.80–1.70	1.16	0.70–1.95	0.96	0.54–1.70
Q4 (>15.19)	133	57	257	1.36	0.94–1.97	1.23	0.74–2.05	0.92	0.53–1.59
P for linear trend				*0.04*		*0.36*		*0.59*	
P for heterogeneity				*0.90*				–	
Unprocessed red meat									
Q1 (≤4.14)	115	60	250	Ref		Ref		Ref	
Q2 (4.15–11.76)	127	52	254	1.09	0.78–1.50	0.83	0.54–1.28	0.85	0.52–1.37
Q3 (11.77–24.70)	91	45	210	1.05	0.74–1.49	0.96	0.61–1.52	0.98	0.59–1.64
Q4 (>24.70)	76	27	175	1.08	0.74–1.57	0.69	0.41–1.17	0.71	0.40–1.29
P for linear trend				*0.77*		*0.24*		*0.35*	
P for heterogeneity				*0.40*				–	
Poultry									
Q1 (≤10.66)	79	45	185	Ref		Ref		Ref	
Q2 (10.67–19.82)	108	43	209	1.30	0.90–1.88	0.97	0.60–1.58	0.71	0.41–1.22
Q3 (19.83–35.08)	106	51	221	1.24	0.86–1.78	1.13	0.71–1.80	1.00	0.59–1.70

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				ER+ vs. Co		ER− vs. Co		ER− vs ER+	
(grams/day/1,000 kcal)	**ER+ Ca (N)**	**ER− Ca (N)**	**Co (N)**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Q4 (>35.08)	116	45	274	1.06	0.74–1.51	0.79	0.49–1.28	0.77	0.45–1.32
P for linear trend				*0.75*		*0.31*		*0.60*	
P for heterogeneity				*0.61*				–	

Ca - case; Co - control

Odds ratios are adjusted for age, ethnicity, country of origin, education, age at menarche, menopausal status, parity, age at first birth, breastfeeding status, family history of breast cancer, OC use, history of benign breast disease, HRT use, total energy