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The role of anthropometric and nutritional factors on breast cancer risk in African-American women

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Abstract

Objective: While the role of nutrition, physical activity and body size on breast cancer risk has been extensively investigated, most of these studies were conducted in Caucasian populations. However, there are well-known differences in tumour biology and the prevalence of these factors between African-American and Caucasian women. The objective of the present paper was to conduct a review of the role of dietary factors, anthropometry and physical activity on breast cancer risk in African-American women.
Design: Twenty-six research articles that presented risk estimates on these factors in African-American women and five articles involving non-US black women were included in the current review.
Setting: Racial disparities in the impact of anthropometric and nutritional factors on breast cancer risk.
Subjects: African-American and non-US black women.
Results: Based on the few studies that presented findings in African-American women, an inverse association with physical activity was found for pre- and postmenopausal African-American women, while the association for anthropometric and other dietary factors, such as alcohol, was unclear. Studies assessing the effect by molecular subtypes in African-American women were too few and based on sample sizes too small to provide definitive conclusions.
Conclusions: The effect of certain nutrition and lifestyle factors on breast cancer in African-American women is not starkly distinct from those observed in white women. However, there is an enormous need for further research on this minority group to obtain more confirmatory findings.

Keywords
Breast cancer
African American
Nutrition
Obesity
Physical activity

Breast cancer is the most common cancer in women (excluding non-melanoma skin cancer) in the USA, accounting for an estimated 30% of all female cancer cases in 2011[1]. However, there is a clear difference in the disease experience in white and African-American (AA) women. Although the overall incidence of breast cancer is lower in AA than in white women, AA women have worse survival rates from the disease at every stage, are more likely to be diagnosed at younger ages and present with more advanced stage disease[2]. AA women more often present with oestrogen receptor (ER)-negative tumours compared with white women[3]. ER-negative tumours not only have a much poorer response to treatment than ER-positive tumours, but also occur more frequently in premenopausal AA women[3,4].

Known risk factors for breast cancer include a family history of breast cancer, germline mutations in *BRCA1* or *BRCA2* genes, exogenous and endogenous hormone exposure, and increased alcohol intake[5]. The role of lifestyle factors such as diet, physical activity and obesity on breast cancer risk have also been extensively explored but most of these studies have focused on white women[6,7]. Little is known about the epidemiology of modifiable factors in AA women despite disparities in the prevalence of these exposures among races. Being overweight and obese are more common among AA, with 46% and 76% of AA adults being considered obese and overweight, respectively[2]. AA women are also more likely to be physically inactive than white women (52·7% *v.* 35·3%)[2]. National data have shown higher intakes of total fat and cholesterol and lower intakes of dietary fibre and folate in AA women compared with white women[8]. There is also growing evidence that the impact of these lifestyle risk factors could vary by hormone receptor status[3]. As compared with ER-positive breast tumours, tumours that are basal-like subtypes (ER negative) tend to occur in women with higher abdominal adiposity[4]. Hence it is conceivable that the impact of lifestyle factors

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on breast cancer risk may be different in AA than in whites, potentially contributing to the observed racial disparities in disease experience. To our knowledge, the present paper is the first review to summarize the findings on nutrition, obesity and physical activity and breast cancer risk in AA women. We have attempted to put the findings in context by juxtaposing findings in AA and white women in the studies included herein.

Experimental methods

An electronic literature search was conducted using PubMed (US National Library of Medicine, National Institutes of Health) to identify all research studies published up to December 2010 in the English language using a combination of the following keywords: 'race', 'breast cancer', 'Black', 'African American', 'diet', 'nutrition', 'physical activity', 'obesity' and 'body size'. We also carefully searched the tables published in the systematic review on breast cancer[7] conducted in support of the 2007 World Cancer Research Fund (WCRF) Report[6] for studies that included AA. We then complemented this with manual searches of the bibliographies of published articles obtained from the initial search.

The searches resulted in a total of sixty-three abstracts to be considered for inclusion in the present review. Articles were organized under three main topics: 'diet and nutrition', 'physical activity' and 'anthropometry'. Published studies that included AA women, but did not report effect estimates (such as relative risks, odds ratios or hazard ratios) and 95 % confidence intervals for breast cancer risk and any of these three lifestyle variables of interest stratified by race were excluded (n 31). The outcome of interest for the review was breast cancer risk; hence studies that presented race-specific risk estimates for breast cancer mortality were excluded (n 1). Five studies conducted in non-US black populations were included. Thus, a total of thirty-one studies were included in the present review consisting of seven cohorts[9–15], twenty-three case–control studies[16–38] and one case–case study[39].

Results

Diet and nutrition

Alcohol

Alcohol consumption is an established risk factor exhibiting a dose–response relationship with breast cancer in both pre- and postmenopausal women[6,7]. Alcohol has been proposed to increase risk by interacting with oestrogen levels in the body, diet and other environmental factors[40]. However, the evidence is largely based on studies conducted in white populations.

We found two prospective cohorts[10,11] and three case–control studies[16–18] that reported breast cancer risk estimates for alcohol consumption in AA women (Table 1). Both cohort and case–control studies suggested an increased risk for breast cancer with high levels of alcohol consumption in both races although most confidence intervals crossed unity. However, these studies generally included fewer AA women than whites, which resulted in wider confidence limits for risk estimates and, in general, not significant estimates for AA women.

One study showed a non-significant protective effect of alcohol for breast cancer risk in both white and AA women using average lifetime intake and current alcohol intake of more than 182 g/week as compared with non-drinkers[17]. No study reported meaningful differences in risk, when stratified by race for different types of alcoholic beverages. Regarding consumption, two studies reported reduced rates of high alcohol consumption in AA women[10,16] while two other studies reported similar levels of consumption in both races[17,18]. Decreased alcohol intake in AA (compared with white) women has also been reported in national surveys[40]. Of interest is that even when AA drinkers have reported higher levels of consumption, on average, they had lower levels of urinary ethanol as compared with white drinkers, thus suggesting racial differences in the metabolism of alcohol[41].

One study[18] specifically reported estimates stratified by ER methylation status and suggested a somewhat stronger association for cases with unmethylated ER gene. However, these analyses were based on very small number of AA women and the confidence intervals overlapped. In summary, the impact of alcohol in AA women is currently inconclusive, given the few studies, with relatively small samples and limited range of alcohol consumption.

Vitamins and micronutrients

In general, no firm conclusions have been drawn about associations between any of the vitamins (from foods or supplements) and breast cancer risk[6,7]. We found four studies that examined the role of vitamins in AA women[18–21]. The population-based case–control study evaluated multivitamins and reported no significant associations for any vitamin use including multivitamins, vitamin C, vitamin E, vitamin A and β-carotene for AA or white women[21].

Two studies evaluated blood levels of vitamins and breast cancer risk using a case–control design[19,20]. One of them, a hospital-based study[19], examined the relationship between 1,25-dihydroxyvitamin D ($1,25(OH)_2D$) blood levels and breast cancer risk and reported significant increased risk for those in the lowest quartile of vitamin D, but restricted to white women (OR = 4·5; 95 % CI 2·2, 9·1). There was no association in AA women, but the analysis included only fifty-one women (OR = 0·5; 95 % CI 0·1, 2·7). Of interest was that breast cancer risk associated with lower $1,25(OH)_2D$ levels was elevated in women with ER-positive/progesterone receptor (PR)-positive disease (OR = 5·0; 95 % CI 2·3, 11·0), while there was no significant association in ER-negative/PR-negative

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Table 1 Studies reporting on the association between alcohol consumption and breast cancer risk stratified by race

						W		AA		Adjusted covariates			
Reference	Age (years)	Study design	Sample size	Exposure	Contrast	Risk estimate	95 % CI	Risk estimate	95 % CI	A	B	H	R
Hiatt and Bawol[10]		PC	96 565 (all races)	Level of daily alcohol consumption	≥3 drinks/d *v.* non-drinking women	1·46	1·09, 1·94	1·08	0·62, 1·88	X			
Hiatt *et al.*[11]		PC	68 674 (all races) AA: 76 cases W: 227 cases	Level of daily alcohol consumption	Past drinker *v.* never drank Currently consuming ≥6 drinks/d *v.* non-drinker	2·36 3·55	1·25, 4·81 1·07, 11·79	1·93 2·80	0·70, 5·33 0·35, 22·58	X	X		
Brinton *et al.*[16]	20–54	PCC	AA: 281/296 W: 960/1033	Alcoholic drinks per week	≥7 drinks *v.* none 20–39 years of age 40–54 years of age	 1·1 1·4	 0·6, 1·9 1·0, 2·0	 1·9 1·6	 0·7, 5·5 0·8, 3·0	X			P
Kinney *et al.*[17]	20–74	PCC	AA: 332/328 W: 524/456	Alcohol intake during current age interval Average lifetime consumption	Former drinker *v.* non-drinker ≥182 g/week *v.* non-drinker ≥182 g/week *v.* non-drinker	0·6 1·2 0·9	0·3, 1·2 0·6, 2·3 0·4, 1·9	1·0 0·8 0·7	0·6, 1·7 0·3, 1·8 0·3, 1·6	X	X		P
Zhu *et al.*[18]		PCC	AA: 304/305	Alcohol consumption	Yes *v.* no ER methylation status Methylated Unmethylated Unknown status >0·5 drinks/d *v.* none ER methylation status Methylated Unmethylated Unknown status	N/A		 1·0 1·8 1·6 2·8 3·1 2·0	 0·5, 1.9 0·9, 3.4 0·9, 2·9 0·7, 10·8 0·9, 10·6 0·6, 6·7	X	X	X	P

W, white; AA, African American; PC, prospective cohort; PCC, population-based case–control study; ER, oestrogen receptor; N/A, not applicable.
Key covariates: A, age; B, BMI; H, hormone use; R, reproductive factors (age at menarche, age at menopause, parity); X, adjusted for that covariate; P, partially adjusted for reproductive factors.

disease (OR = 1·1; 95% CI 0·5, 3·0). Current evidence shows that low serum vitamin D levels are not only associated with obesity[42] but are also more prevalent in AA than in non-Hispanic whites[43,44]. Since AA women are more likely to have ER-negative/PR-negative tumours, the effect of vitamin D deficiency on breast cancer risk may be less apparent in this group despite the lower vitamin D levels and higher obesity prevalence.

Similarly, a pilot study investigated the impact of plasma antioxidant micronutrients and breast cancer risk in AA and white women[20]. Although there was a weak inverse association with plasma lycopene levels (a micronutrient rich in tomato-based foods) and a weak positive association with plasma retinol levels for AA women, no significant interactive effect was found for β-carotene, retinol, α-tocopherol and γ-tocopherol. As compared with the highest tertile, breast cancer risk in the lowest tertile of plasma lycopene levels was 0·76 (95% CI 0·07, 7·54) in white women and 2·29 (95% CI 0·10, 58·2) in AA women. The small sample sizes resulting in wide confidence intervals indicate uncertainty in the study findings.

Folate and methionine

Folate intake is known for its consistent interactive effect with alcohol, whereby low folate intake and high alcohol consumption have been shown to increase breast cancer risk[45]. The study of dietary folate has also gained importance due to its potential role as a methyl donor for normal methylation of genes[6]. Abnormal methylation could result in silencing of key cell regulatory genes including ER. A low methyl diet contributing to abnormal gene methylation results from low intakes of methionine (from poultry, fish and dairy products) and folate (from fruits and vegetables). Although the relationship between folate as an independent dietary factor and breast cancer risk has not been confirmed[6], one study examined this association specifically in AA women[18]. An increased risk was suggested for cases with methylated ER and no association for cases with unmethylated ER among women in the lowest quartile of folate (OR = 2·4; 95% CI 0·6, 9·9) and methionine intakes (OR = 1·6; 95% CI 0·4, 6·1), as compared with the highest. However, analyses were based on small numbers and confidence intervals included one.

Fat

The effect of dietary fat on breast cancer risk has been extensively studied but findings have been inconsistent[6,46]. Although higher fat intake could contribute to increased levels of endogenous oestrogens especially after menopause, the evidence for an increased breast cancer risk with increase in dietary fat remains inconclusive[6,46]. We found one study that reported stratified risk estimates by dietary fat intake for AA women[23]. Although AA women had slightly higher median intake of fat than white women, neither total fat nor any of its components such as saturated fat, linoleic acid (polyunsaturated fat) and oleic acid (monounsaturated fat) were significantly associated with breast cancer risk in AA women. However, percentage of energy from total fat (OR = 1·45; 95% CI 1·01, 2·09) and monounsaturated fat (OR = 2·26; 95% CI 1·31, 3·90) appeared to increase risk in white women. The authors attributed this inconsistency in findings between races to residual confounding. Breast cancer risk associated with type of fat used in cooking was similar in all women, except cooking with hydrogenated fat, which significantly increased risk only in white women (OR = 1·41; 95% CI 1·04, 1·92).

Fruits and vegetables

Associations remain inconclusive for both pre- and postmenopausal women when fruits and vegetables have been assessed individually for their influence in breast cancer prevention[6]. Only one study examined this association specifically in AA women[9]. Inverse relationships were observed only for six or more servings weekly of cruciferous vegetables (incidence rate ratio (IRR) = 0·59; 95% CI 0·42, 0·83) and carrots (IRR = 0·71; 95% CI 0·52, 0·97) among premenopausal women, aside from a borderline inverse association for intake of broccoli and citrus fruits. In postmenopausal women, there was a suggestion of a protective association with four or more servings of total fruits and vegetables daily (IRR = 0·76; 95% CI 0·56, 1·04) and for six or more servings of citrus fruits weekly (IRR = 0·74; 95% CI 0·54, 1·01). Furthermore, as compared with less than four servings weekly, total vegetable servings of two or more daily appeared to decrease risk for ER-negative/PR-negative breast cancer (IRR = 0·57; 95% CI 0·38, 0·85).

Other dietary findings

The association between phyto-oestrogen intake[22], tea and coffee consumption[14] and breast cancer risk in AA women was examined by two separate studies. No clear associations were found between intake and disease risk in AA women. In general, intake of phyto-oestrogens has been shown to be limited in non-Asian populations[47] while coffee and tea consumption is also lower in AA women than in white women[48].

One study on dietary patterns and breast cancer risk in AA women[15] showed that a prudent diet (with heavy loading on fruits, vegetables, whole grains, fish, poultry and low-fat dairy) appeared to significantly decrease risk in AA women with these characteristics: BMI of less than 25 kg/m^2 (IRR = 0·64; 95% CI 0·43, 0·93), premenopausal (IRR = 0·70; 95% CI 0·52, 0·96) and with ER-negative tumours (IRR = 0·52; 95% CI 0·28, 0·94).

Anthropometry

Adult height

There is probable evidence that adult attained height is a risk factor for breast cancer in premenopausal women

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and convincing evidence that adult height increases risk in postmenopausal women[6,46]. Adult height may be a marker for genetic, environmental, nutritional and hormonal factors that may influence breast cancer risk early in life[6]. The effect of adult height on breast cancer in AA women was assessed in one cohort[12] and three case–control studies[24–26] (Table 2). In one study, adult height was associated with an increased breast cancer risk among premenopausal women, all races combined. But separate analyses by race did not reveal a significant trend for white and AA women, which could be attributed in part to small sample size[26]. In that study, height also appeared to increase risk significantly in premenopausal women for both ER-positive/PR-positive and ER-negative/PR-negative tumours. Associations demonstrating increased risk with increased height were observed in premenopausal AA women in two other studies[12,25]. Despite reduced risk for shorter AA women (less than 61 inches tall) as compared with those of median height in another study, there was limited evidence of an increased risk for AA women taller than 61 inches[24]. Hence, it is difficult to confirm an increased risk of breast cancer with increased adult height in AA women at this time.

Body fatness

The WCRF Report and Continuous Update concluded that body fatness probably decreases breast cancer risk in premenopausal women while the evidence showing increased risk in postmenopausal women was deemed convincing[6,46]. There are multiple mechanisms by which body fatness could affect breast cancer risk, one being that excess fat tissue could raise the availability of circulating oestrogens and increase exposure to endogenous oestrogens, favouring carcinogenesis[6].

A total of seven case–control studies[16,25–30] and one cohort[13] examined the impact of BMI and body weight on breast cancer risk in AA women (Table 3). Four of the studies that also reported risk estimates for white women[16,25,26,30] showed an inverse (albeit not always significant) trend between BMI and breast cancer risk in premenopausal white women consistent with general findings in this group. However, among younger and premenopausal AA women findings were more inconsistent, with three studies[27–29] indicating increased risk with higher BMI and the remaining studies suggesting inverse associations, and only one study[28] showing statistical significance. In one study[25] when analyses were repeated for women younger than 50 years of age (rather than classifying by menopausal status), there was an indication of an inverse association among AA women (OR = 0·5; 95 % CI 0·24, 1·01) with BMI, rendering results comparable to those observed in white women.

In postmenopausal AA women, significant increased risk of more than two times with high BMI was observed only in two studies[27,29]. In contrast, two additional

Table 2 Studies reporting on the association between adult height and breast cancer risk stratified by race

						W		AA		Adjusted covariates			
Reference	Age (years)	Study design	Sample size	Exposure	Contrast	Risk estimate	95 % CI	Risk estimate	95 % CI	A	B	H	R
Palmer *et al.*[24]	25–69	PCC	AA: 681/1155	Height (inches)	≥71 *v.* 64	N/A		1·2	0·5, 2·9	X			P
					<60 *v.* 64			0·3	0·2, 0·7				
				Premenopausal	≥68 *v.* 64–65			0·7	0·4, 1·2				
					<61 *v.* 64–65			0·4	0·2, 0·8				
				Postmenopausal	≥68 *v.* 64–65			1·1	0·6, 2·3				
					<61 *v.* 64–65			0·5	0·3, 1·0				
Hall *et al.*[25]	20–74	PCC	AA: 350/353 W: 523/471	Height (cm)	165·1–188 *v.* 140–160								
					Premenopausal	0·77	0·46, 1·29	2·93	1·44, 5·95	X	X		P
					Continuous	0·99	0·95, 1·02	1·05	1·00, 1·10				
					Postmenopausal	1·63	0·96, 2·76	1·00	0·55, 1·83	X	X		X
					Continuous	1·03	1·00, 1·07	1·00	0·97, 1·04				
Palmer *et al.*[12]	18–69	PC (NCC)	64 530 AA: 910/4535	Adult height (inches)	≥ 70 *v.* ≤61			1·6	1·1, 2·3	X			P
					Premenopausal			2·1	1·2, 3·6	X			P
					Postmenopausal			1·3	0·6, 2·5				
					Only incident cases			3·0	1·3, 6·5	X	X		P
John *et al.*[26]	≥35	PCC	AA: 154/160 W: 143/165	Current height (cm)	Race-specific Q4 *v.* Q1	1·81	0·87, 3·74	1·39	0·65, 2·94	X			P

W, white; AA, African American; PC, prospective cohort; PCC, population-based case–control study; NCC, nested case–control study; Q, quartile; N/A, not applicable.
Key covariates: A, age; B, BMI; H, hormone use; R, reproductive factors (age at menarche, age at menopause, parity); X, adjusted for that covariate; P, partially adjusted for reproductive factors.

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Table 3 Studies reporting on the association between other anthropometric characteristics (current BMI, BMI at age 18 years and WHR) and breast cancer risk stratified by race

Reference	Age (years)	Study design	Sample size	Exposure	Contrast	W		AA		Adjusted covariates			
						Risk estimate	95 % CI	Risk estimate	95 % CI	A	B	H	R
Current/recent BMI													
Schatzkin *et al.*[27]	25–70	PCC	AA: 529/589	BMI (kg/m^2)	≥30 *v.* ≤24	N/A				X	X		P
					Premenopausal			1·2	0·7, 2·1				
					Postmenopausal			2·5	1·5, 4·4				
Mayberry[28]	20–54	PCC	AA: 490/485	BMI (kg/m^2) as an adult	≥32·30 *v.* <24·90	N/A				X	X		P
					20–39 years of age			3·9	1·3, 11·6				
					40–54 years of age			0·8	0·4, 1·4				
Brinton *et al.*[16]	20–54	PCC	AA: 281/296 W: 960/1033	Current BMI (kg/m^2)	<22·0 *v.* >28·8					X			P
					20–39 years of age	1·5	0·9, 2·5	1·3	0·5, 3·1				
					40–54 years of age	1·2	0·9, 1·8	0·8	0·3, 1·9				
Hall *et al.*[25]	20–74	PCC	AA: 350/353 W: 523/471	Current BMI (kg/m^2)	30·13–59·26 *v.* 14·62–24·61								
					Premenopausal	0·46	0·20, 0·80	0·89	0·38, 2·07	X			P
					Postmenopausal	1·08	0·58, 2·00	0·68	0·33, 1·42	X			X
Zhu *et al.*[29]	20–64	PCC	AA: 304/305	BMI (kg/m^2) at reference date	≥30 *v.* <25	N/A							P
					Premenopausal			2·49	0·87, 7·59				
					Postmenopausal			2·32	1·04, 5·19				
Palmer *et al.*[13]	21–69	PC	59 000 AA: 1062 cases	Current BMI (kg/m^2)	≥35 *v.* <25	N/A				X	X		X
					Premenopausal			0·87	0·62, 1·21				
					Postmenopausal			0·99	0·72, 1·36				
John *et al.*[26]	≥35	PCC	AA: 154/160 W: 143/165 (only includes premenopausal women)	Quartile of current weight (kg)	>81·6 *v.* ≤61·2	0·55	0·23, 1·34	1·62	0·26, 1·14	X			P
				Current BMI (kg/m^2)	≥30 *v.* <25	0·60	0·28, 1·30	0·65	0·35, 1·23				
Berstad *et al.*[30]	35–64	PCC	AA: 1622/1661 W: 2953/3021	Recent BMI (kg/m^2)	≥35 *v.* <25					X		X	P
					Premenopausal	0·86	0·57, 1·29	0·81	0·56, 1·19				
					Postmenopausal	0·75	0·53, 1·06	1·26	0·85, 1·85				
BMI at age 18 years													
Mayberry[28]	20–54	PCC	AA: 490/485	BMI (kg/m^2) at age 18	≥30·70 *v.* <23·80	N/A				X	X		P
					20–39 years of age			0·5	0·2, 1·4				
					40–54 years of age			1·5	0·9, 2·7				
Zhu *et al.*[29]	20–64	PCC	AA: 304/305	BMI (kg/m^2) at age 18	≥30 *v.* <25	N/A							
					Premenopausal			1·84	0·27, 12·45				
					Postmenopausal			1·35	0·20, 9·15				
Palmer *et al.*[13]	21–69	PC	59 000 AA: 1062 cases	BMI (kg/m^2) at age 18	≥25 *v.* <20	N/A				X	X		X
					Premenopausal			0·68	0·46, 0·98				
					Postmenopausal			0·53	0·35, 0·81				
Berstad *et al.*[30]	35–64	PCC	AA: 1622/1661 W: 2953/3021	BMI (kg/m^2) at age 18	≥25 *v.* <20					X		X	P
					Premenopausal	0·84	0·61, 1·15	0·67	0·47, 0·96				
					Postmenopausal	0·70	0·49, 1·00	0·80	0·54, 1·19				

Table 3 *Continued*

Reference	Age (years)	Study design	Sample size	Exposure	Contrast	W		AA		Adjusted covariates			
						Risk estimate	95 % CI	Risk estimate	95 % CI	A	B	H	R
WHR													
Hall *et al.*[23]	20–74	PCC	AA: 350/353 W: 523/471	WHR	0·86–1·34 *v.* 0·60–0·77								
					Premenopausal	2·44	1·17, 5·09	2·50	1·10, 5·67	X	X		P
					Continuous	1·04	1·01, 1·08	1·05	1·00, 1·10				
					Postmenopausal	1·64	0·88, 3·07	1·62	0·70, 3·79	X	X		X
					Continuous	1·03	1·00, 1·06	1·03	0·99, 1·07				
Palmer *et al.*[13]	21–69	PC	59 000 AA: 1062 cases	WC (inches)	≥37 *v.* <28	N/A				X	X		X
					Premenopausal			1·04	0·73, 1·48				
					Postmenopausal			1·05	0·73, 1·51				
				WHR	≥0·87 *v.* <0·71					X	X		X
					Premenopausal			1·19	0·87, 1·64				
					Postmenopausal			0·99	0·72, 1·37				
John *et al.*[26]	≥35	PCC	AA: 154/160 W: 143/165 (only premenopausal)	Quartile of WHR	>0·85 *v.* ≤0·77	1·35	0·47, 3·86	0·82	0·39, 1·74	X			P
				Quartile of WC (cm)	>98 *v.* ≤78·7	0·90	0·37, 2·17	1·09	0·47, 2·52	X			P

WHR, waist-to-hip ratio; W, white; AA, African American; PCC, population-based case–control study; PC, prospective cohort; WC, waist circumference; N/A, not applicable.
Key covariates: A, age; B, BMI; H, hormone use; R, reproductive factors (age at menarche, age at menopause, parity); X, adjusted for that covariate; P, partially adjusted for reproductive factors.

case–control studies[25,30] provided little support for an association with BMI for postmenopausal breast cancer in AA or white women. An additional cohort study in AA women[13] also failed to find an association. Possible explanations for the contradictory results of these studies with the overall body of literature in postmenopausal (mostly white) women[6] were postulated as possible effect modification by hormone replacement therapy[25] and the relatively younger ages sampled even in the postmenopausal category[30]. After stratifying by ER status, high BMI was associated with increased risk of ER-positive/PR-positive tumours only among AA women, whereas it seemed to decrease risk for ER-negative/PR-negative tumours particularly among white women[30]. Similar findings were reported in a prospective study among AA women[13], which suggested that high BMI ($\geq 30\,kg/m^2$) increased risk for ER-positive/PR-positive tumours while decreasing risk for ER-negative/PR-negative tumours. However, these analyses were based on very small number of cases and confidence intervals included the null value.

Four studies also assessed the effect of BMI at young adulthood on breast cancer risk. Of these, two studies[28,29] reported no association in both pre- and postmenopausal women while two studies reported inverse associations in these women[13,30].

Overall, the evidence is inconclusive at the present time. More studies are needed with sufficient power to stratify by menopausal status and ER subtypes, which are important modifiers of the association between BMI and breast cancer risk.

Measures of central obesity

Abdominal fatness has been generally measured through waist and hip circumferences and commonly through waist-to-hip ratio (WHR), and is a probable risk factor for breast cancer in postmenopausal women[6]. As compared with WHR, a single measure of waist circumference has been recommended to be a better measure of subcutaneous fat and intra-abdominal fat[6]. Two case–control studies[25,26] and one prospective cohort[13] reported WHR and waist circumferences to examine the impact of central obesity on breast cancer risk in AA women. In general, there appeared to be no racial differences in the way central adiposity affected breast cancer risk. For example, positive associations with greater central adiposity were observed in both AA and white premenopausal women[25] while no significant associations were reported for the same in either of the races in the remaining studies[13,26], with the exception of an inverse association with ER-positive/PR-positive tumours in general[26].

Physical activity

In the 2007 WCRF Report and 2010 WCRF Continuous Update, the evidence for physical activity reducing breast cancer risk was found to be probable for postmenopausal women and limited for premenopausal women[6,7,46].

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Physical activity even at moderate levels results in increased energy expenditure, favouring maintenance of a healthy weight. Furthermore, regular moderate physical activity has been shown to decrease levels of endogenous sex hormones and insulin levels, and create a supportive environment for apoptosis, which could have a potential protective effect on breast cancer development[6,46].

The association between physical activity and breast cancer risk in AA women was investigated in four case–control studies[31–34] (Table 4). Physical activity was reported in h/week in two studies while the remaining studies reported annual MET-h/week, exercise levels and weekly minutes of activity. With the exception of lifetime recreational activity (measured in one study)[32], all other physical activity measures trended towards being protective against breast cancer in AA women. This association was consistent for premenopausal and postmenopausal breast cancers. In fact, studies that included AA and white women tended to suggest a stronger inverse association for AA women as compared with white women for the same level of physical activity[32–34]. Physical activity of 3 h/week or more was significantly inversely related to breast cancer for all AA women in two studies[33,34]. Although one study showed that the protective effect of physical activity could start with just 3 h/week or more[33], no dose–response associations were found in any of the studies. In summary, a protective effect of physical activity against breast cancer risk was consistently shown in AA women.

Studies on non-US black women

Breast cancer epidemiology and tumour biology in women in Africa have been found to be mostly similar to AA women[49]. Extending the review to include studies among women of African descent outside the USA could provide a comprehensive view of factors influencing disease risk in the AA population. We found five studies[35–39] related to our review that were conducted in non-US black women; all five studies focused on anthropometric factors.

In the Barbados National Cancer Study[37], increased height appeared to increase risk especially in women older than 50 years of age. Greater waist circumference and WHR seemed to interact with age by increasing risk in older women and decreasing risk in women younger than 50 years of age.

Anthropometry in Nigerian women was measured through WHR in two studies[35,38] and BMI in the third study[36]. While positive associations were observed between WHR and breast cancer risk in postmenopausal women, the findings among premenopausal women were inconsistent. Adult height emerged as a significant risk factor for breast cancer in both pre- and postmenopausal urbanized Nigerian women; however no significant association between BMI and breast cancer risk was found[36].

A study that assessed the relationship between BMI and triple-negative tumours mostly involved women born in the Caribbean[39]. Although both obese and non-obese black women had higher number of triple-negative breast cancer (ER negative/PR negative/Her2 negative) than non-black obese and non-obese women, BMI did not appear to influence triple-negative status[39].

Discussion

The breast cancer literature has consistently shown that AA women are more likely to be diagnosed at younger ages, with a higher occurrence of ER-negative/PR-negative tumours that are associated with poorer prognosis[2,3,50]. Nutritional and lifestyle differences among the different racial groups in the USA continue to exist, potentially contributing to disparities in breast cancer aetiology and survival. National data indicate that even after controlling for socio-economic factors, AA women were found to be more physically inactive, have a higher BMI and have a poorer diet quality than white women[8]. Our current understanding of the role of modifiable risk factors on breast cancer risk comes from research studies mostly involving white women. Hence, reviewing studies that focus on AA women and summarizing what we know about the impact of lifestyle factors in these women will further understanding of breast cancer prevention in this minority group.

Physical activity appears to have a beneficial effect for AA women even at relatively low levels of 3 h/week or more. The potential protective effect observed in premenopausal women is especially relevant among AA women since strenuous to moderate levels of physical activity have also been suggestive of lowering risk of ER-negative tumours[3,34].

Few racial differences in nutrition and dietary factors were observed from the available evidence although the number of studies presenting data on AA women was not sufficient to draw definitive conclusions. For example, although heavy consumption of alcohol should be avoided for all women, the risk may not manifest in a similar manner for AA women. On the other hand, AA women could be encouraged to follow a healthy/prudent dietary pattern due to its potential beneficial effect on ER-negative tumours, which has also been supported elsewhere[3].

Severe obesity and WHR have been shown to explain 27 % of observed racial differences in stage at diagnosis of the disease[51]. An interesting finding from the present review was that although AA women tended to be of larger stature and have higher BMI and central adiposity, anthropometric differences between the two races did not emerge as a critical factor in the racial disparities in breast cancer risk. Most of the studies in AA women reported statistically non-significant associations. This may be in part due to limited heterogeneity in BMI categories and menopausal status in this minority group or residual confounding. For example, breast-feeding patterns could influence prevalence of basal-like breast cancer in younger

Table 4 Studies reporting on the association between physical activity and breast cancer risk stratified by race

						W		AA		Adjusted covariates			
Reference	Age (years)	Study design	Sample size	Exposure	Contrast	Risk estimate	95 % CI	Risk estimate	95 % CI	A	B	H	R
Adams-Campbell *et al.*[31]	21–69	PCC	AA: 704/1408	Strenuous exercise in high school (h/week)	≥7 *v.* <1	N/A				X	X		P
					Premenopausal			1·0	0·6, 1·6				
					Postmenopausal			1·1	0·6, 2·1				
				Strenuous exercise at age 21 (for diagnosis at age ≥30)	Premenopausal			0·5	0·3, 0·8				
					Postmenopausal			0·6	0·3, 1·2				
				Strenuous exercise at age 30 (for diagnosis at age ≥40)	Premenopausal			0·5	0·2, 1·1				
					Postmenopausal			0·5	0·2, 1·2				
				Strenuous exercise at age 40 (for diagnosis at age ≥50)	Premenopausal			Not provided					
					Postmenopausal			0·3	0·1, 0·9				
John *et al.*[32]	35–79	PCC	AA: 409/461	Lifetime physical activity (since menarche) (h/week)									
			W: 449/499	Total activity									
				Premenopausal	≥20·8 *v.* <9·1	0·76	0·36, 1·61	0·68	0·35, 1·34	X	X		X
				Postmenopausal	≥21·7 *v.* <9·6	0·91	0·60, 1·41	0·71	0·47, 1·07	X			X
				Total moderate activity									
				Premenopausal	≥16·7 *v.* <6·8	0·44	0·20, 0·99	0·77	0·38, 1·56	X	X		X
				Postmenopausal	≥17·8 *v.* <7·6	1·02	0·66, 1·59	0·60	0·40, 0·92	X			X
				Recreational activity									
				Premenopausal	≥4·0 *v.* <1·5	0·79	0·39, 1·60	1·37	0·73, 2·55	X	X		X
				Postmenopausal	≥2·7 *v.* <0·7	1·13	0·76, 1·70	1·04	0·69, 1·57	X			X
				Total daily living activity									
				Premenopausal	≥9·5 *v.* <4·0	0·71	0·30, 1·67	0·81	0·38, 1·73	X	X		X
				Postmenopausal	≥10·5 *v.* <4·3	1·10	0·71, 1·71	0·91	0·58, 1·44	X			X
				Mostly moderate or strenuous jobs									
				Premenopausal	≥10·0 *v.* 0	0·87	0·40, 1·91	0·67	0·35, 1·28	X	X		
				Postmenopausal	≥9·1 *v.* 0	0·62	0·39, 1·00	0·77	0·51, 1·16	X			
Bernstein *et al.*[33]	35–64	PCC	AA: 1605/1646	Lifetime exercise activity	Annual h/week					X	X		
			W: 2933/3033		≥3·0 *v.* inactive	0·83	0·70, 0·98	0·75	0·61, 0·93				
					Annual MET-h/week ≥15·2 *v.* inactive	0·81	0·69, 0·96	0·77	0·62, 0·95	X			
Ratnasinghe *et al.*[34]		PCC	AA: 88/406	Physical activity						X	X		
			W: 1463/2406	time/week	>3 *v.* <1	0·46	0·39, 0·56	0·21	0·11, 0·39				
				min/time	>30 *v.* <10	0·51	0·42, 0·62	0·27	0·14, 0·52				
				min/week	>150 *v.* <30	0·55	0·46, 0·67	0·25	0·14, 0·52				

W, white; AA, African American; PCC, population-based case–control study; MET, metabolic equivalent task; N/A, not applicable.
Key covariates: A, age; B, BMI; H, hormone use; R, reproductive factors (age at menarche, age at menopause, parity); X, adjusted for that covariate; P, partially adjusted for reproductive factors.

AA women[4] or certain black populations may have unusually higher prevalence of *BRCA1* mutations[52].

In summary, the current evidence suggests that the impact of dietary factors, body size and physical activity on breast cancer risk in AA women may not be starkly different from that in white women. However, one of the main findings from the present review is the disproportionately fewer studies that have evaluated these factors among AA women. Given the racial disparities in tumour biology and lifestyle factors, a better understanding of the role of diet, obesity and physical activity in AA women is crucial to achieve more effective breast cancer prevention strategies.

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