

ETHNICITY AND VARIATION IN BREAST CANCER INCIDENCE

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A breast cancer case-control study in Atlanta and 5 counties of central New Jersey involving interviews with 960 white and 281 black cases younger than 54 years of age enabled assessment of reasons for the varying incidence rates among these 2 ethnic groups. Of interest was why rates of breast cancer are higher among older white women, a trend that is reversed among very young women (<40 years). Calculation of the prevalence of exposure to classic and speculative risk factors and associated relative risks enabled derivation of population attributable risks (PARs) for the various combinations of age and ethnic groups. A higher PAR was derived for older (40–54 years) white (62%) than black (54%) women, which appeared to account for the observed difference in incidence between the 2 ethnic groups. Most of the difference in PARs between older whites and blacks was accounted for by whites having fewer births, later ages at first birth and slightly higher risks associated with reproductive and menstrual factors. Consideration of only well-established breast cancer risk factors showed a PAR among older whites of 57%, an estimate comparable to those previously published. Slightly higher overall PARs were derived when analyses considered several speculative but modifiable risk factors, including years of use of oral contraceptives, body size and alcohol consumption. Many of the analyses among younger women (20–39 years) were limited by available numbers, but it appeared that very little disease occurrence in young black women was associated with the factors studied. *Int. J. Cancer* 73:349–355, 1997.

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It is well recognized that the incidence of breast cancer is higher for white than black women, with population-based data showing approximately a 20% higher rate for white women (Hankey *et al.*, 1993). An anomalous observation is that the reverse is true for women younger than 40 years (Velentgas and Daling, 1994), with black women having rates of breast cancer that are between 10% and 40% higher than those of whites. It has been speculated that socio-demographic and reproductive differences might explain the ethnic disparity in incidence (Krieger, 1990), but studies evaluating the role of specific factors have not been undertaken. A number of case-control studies have examined breast cancer risk factors among black women (Austin *et al.*, 1979; Mayberry and Stoddard-Wright, 1992; Palmer *et al.*, 1995; Schatzkin *et al.*, 1987), but reasons for the generally higher rates in white women or for the ethnic cross-over in incidence by age have not been elucidated.

In a population-based, case-control study that focused on younger women (<55 years of age), 2 of the study sites included relatively large proportions of black women. Because this study concentrated on younger women, there was a unique opportunity for assessing reasons for the differing incidence in breast cancer by age and ethnicity. In the present study, we attempted to pursue the issue by examining age and racial variations in the prevalence of various factors and their associations with risk.

MATERIAL AND METHODS

Although this population-based, case-control study was conducted in 3 different geographic areas (Atlanta, Georgia, 5 counties

of central New Jersey, and Seattle/Puget Sound, Washington), the present analysis includes only the 2 sites (Atlanta and New Jersey) that had sufficient numbers of black study subjects. In New Jersey, the study was confined to women 20–44 years of age, whereas in Atlanta the age range was extended through age 54. All women of these ages newly diagnosed with *in situ* or invasive breast cancer during the period May 1, 1990, through December 31, 1992, were identified through rapid ascertainment systems. Hospital records of eligible patients were abstracted to document details on the clinical and pathologic characteristics of the diagnosed breast cancers.

Controls were ascertained through a series of 13 waves of random digit dialing (Waksberg, 1978). To select a sample of women that approximated the anticipated age distribution of cases, information was sought on female residents who were 20–44 years of age in New Jersey and who were 20–54 years of age in Atlanta. A 90.5% response rate to the telephone screener was obtained from the 10,532 telephone numbers assessed as residential; non-response consisted of a 5.4% refusal to the telephone screener, 0.8% language problems, and 3.3% contact problems.

Structured in-person interviews, which lasted a median of 65 min, collected detailed information regarding demographic factors, reproductive and menstrual history, contraceptive behavior, use of exogenous hormones, medical and screening history, anthropometry and physical activity, adolescent diet, alcohol consumption, smoking, occupations, family history of cancer and certain life style factors and opinions about cancer causation. In addition, subjects were asked to complete a 100-item dietary questionnaire and to consent to a variety of anthropometric measurements.

Completed interviews were obtained from 1,558 of the 1,784 eligible cases (87.3%) and 1,399 of the 1,790 eligible controls (78.2%). Reasons for non-interview included controls refusing to provide telephone screening information (4.9%), refusals to provide interview information (5.9% in cases vs. 13.2% in controls), death (0.4% vs. 0.2%), illness (0.8% vs. 0.2%), a move outside the study area (0.5% vs. 2.2%) and other miscellaneous reasons (0.3% vs. 1.1%). In addition, physician consent for interview was denied for 4.6% of the cases. Among controls, an overall response rate of 70.8% was achieved through multiplication of the telephone screener and interview response rates. It was not possible to calculate response rates by ethnicity among controls (because ethnic information was not asked on the telephone screen). Among cases, whites and blacks had similar response rates (87.9% vs. 87.0%), although older blacks (45+ years) were more reluctant to consent to an interview than other subjects.

For cases to be comparable to the controls who were identified through telephone sampling, the 18 cases who indicated on interview that they did not have a residential telephone were eliminated from analysis. In addition, 18 controls with a history of

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breast cancer were deleted. To reduce effects of detection bias, we eliminated the 261 cases diagnosed with *in situ* carcinomas. After eliminating the 38 cases and 52 controls who reported themselves of an ethnic origin other than white or black, the final analytic sample consisted of 1,241 cases and 1,329 controls.

Because the median interval between diagnosis and interview was 87 days for cases, all information on risk factors was truncated at the date of diagnosis for cases or the date at completion of the telephone screen for controls. Differences in the distribution of risk factors among controls between whites and blacks were investigated by two-way chi-square tests, considering any with p values < 0.05 as statistically significant. Effects on breast cancer risk of ethnicity and effects of other risk factors within each ethnic group were assessed through calculation of odds ratios to approximate relative risks (RRs). Logistic regression analyses were used to obtain maximum likelihood estimates of RRs and their 95% confidence intervals (CI) (Breslow and Day, 1980).

Estimates of population attributable risk (PAR) and associated 95% CI were obtained by an approach based on unconditional logistic regression (Benichou and Gail, 1990; Bruzzi *et al.*, 1985). By combining adjusted RR estimates (derived through logistic regression) and the observed prevalence of the risk factors under study in the cases, this approach yields adjusted PAR estimates. Estimates of PAR usually range between 0 and 1 (or 0% and 100%), but when the factor under study appears protective, a negative PAR is derived. PARs were estimated for each separate risk factor and for combinations of risk factors. By estimating the black-to-white ratio of quantities of 1-PAR for given combinations of risk factors, we could assess the incidence ratio accounted for by these combinations.

RESULTS

A total of 281 of 1,241 cases (22.6%) vs. 296 of 1,329 controls (22.3%) classified themselves as black, resulting in a breast cancer RR of 1.04 (95% CI 0.9–1.3) for being black compared with white (adjusted only for age and study site). The effect of being black varied by age, with the RR being 1.17 (0.8–1.6) for women younger than 40 years of age and 0.95 (0.8–1.2) for those 40 and older.

As an initial step in determining reasons for the differing incidence of breast cancer by ethnicity, among controls we examined the prevalence of various established or suspected breast cancer risk factors for younger (20–39 years) and older (40–54) whites and blacks (Table 1). Among the younger women, there were a number of statistically significant differences between whites and blacks, with the white subjects reporting fewer births, later ages at first birth, longer durations of breastfeeding, smaller body sizes, more frequent consumption of alcoholic beverages and a higher level of education. There were no significant differences between younger whites and blacks in the prevalence of induced abortions, miscarriages, early ages at menarche, extended use of oral contraceptives, family history of breast cancer, breast biopsies or cigarette smoking. Compared with the younger women, older subjects had more births, earlier ages at first birth, fewer abortions, more breast biopsies and more obesity. In general, the same differences in prevalence of risk factors between older whites and blacks were observed as were noted among the younger subjects. One exception was that older white women had longer durations of use of oral contraceptives than older black women.

Risks associated with various reproductive and menstrual variables for white vs. black women by age are shown in Table II. Among younger white subjects, there was an inverse relationship of risk with numbers of births. This association, however, was not observed among younger blacks; in fact, nulliparous women were at reduced risk relative to multiparous women. Early menarche was more strongly related to risk among younger white than black subjects. In contrast, factors that were more strongly related to risk

among younger black than white subjects were a late age at first birth and absence of or brief breastfeeding, although interpretation of the latter association was limited by the few number of young, black women who breastfed for extended periods. A history of an induced abortion or a miscarriage did not substantially affect risk in either younger whites or blacks.

Among the older subjects, stronger relationships were observed among whites than blacks with numbers of births, ages at first birth, absence of or brief breastfeeding and ages at menarche. However, in contrast to younger black women, nulliparity was related to an increase in risk among older black women. The only factor among older women that was more strongly related to risk in black than white women was extended use of oral contraceptives, for which usage for 10 or more years was associated with an 80% elevation in risk.

An examination of familial, life style and other factors (Table III) showed that a family history of breast cancer in a first-degree relative was a slightly stronger risk factor in blacks than whites among both younger and older women. In contrast, a history of a breast biopsy was a stronger risk factor in older whites than blacks. Other differences in relative risks between whites and blacks were unremarkable. In general, thinness and heavier consumption of alcoholic beverages tended to be associated with slight elevations in risk in all subgroups examined. Cigarette smoking and years of education were inconsistently related to risk.

Table IV presents the calculation of PARs for identified risk factors for younger and older whites vs. blacks. Among younger white women, PARs of approximately 20% were found for each of the following factors: low numbers of births, later ages at first birth, early ages at menarche, extended use of oral contraceptives and smaller body sizes. Less disease was associated with these factors in young black women. In younger black women, low parity and early ages at menarche were associated with negative PARs, because relative risks for the higher levels of exposure (*i.e.*, few births and early ages at menarche) were less than unity. PARs among younger black women for the other 3 factors (late ages at birth, use of oral contraceptives, body mass) were all closer to 10%, only half the PARs in younger white women. Similar proportions of disease in younger white and black women were associated with a family history of breast cancer (6–8%) and alcohol consumption (7%).

Among older women, whites and blacks had similar PARs for parity (24–26%), family history of breast cancer (8–10%) and alcohol consumption (16–17%). However, more disease in older whites than blacks was associated with late ages at first birth (23% vs. 13%) and early menarche (29% vs. –7%), although the differences were not statistically significant. Only small proportions of disease were associated with previous breast biopsies and body mass, although the PARs were slightly higher for older white than black women. In contrast, the PAR for duration of oral contraceptives use was substantially higher among older black than white women (22% vs. –6%).

We also calculated PARs for combinations of risk factors. Among younger women, the PARs for low number of births combined with later ages at first birth were 40% in whites but non-explanatory in blacks (–13%). However, in older subjects, this combined factor was associated with a sizeable proportion of disease in both whites (30%) and blacks (25%).

Table V shows the PARs for other risk factor combinations. Among younger white women, the addition of age at menarche to the other reproductive variables substantially increased the PAR (52%), but no similar increase was seen among blacks (–22%). Life style factors (years of use of oral contraceptives, body mass, alcoholic drinks) were also associated with higher PARs in whites (36%) than blacks (18%). Because previous analyses have calculated PARs in reference to well-established risk factors (the above 3 reproductive variables, family history of breast cancer, previous breast biopsy) (Bruzzi *et al.*, 1985; Madigan *et al.*, 1995), we

TABLE I – PREVALENCES (%) OF RISK FACTORS AMONG CONTROLS BY AGE AND ETHNICITY, ATLANTA AND NEW JERSEY (1990–1992)

	20–39 years of age			40–54 years of age		
	Whites (n = 340)	Blacks (n = 102)	<i>p</i> value	Whites (n = 693)	Blacks (n = 194)	<i>p</i> value
No. of births						
3+	18	27		33	52	
2	32	23		36	22	
1	19	26		16	17	
0	31	24	0.02	15	9	<0.01
Ages at first birth ¹						
<20	14	45		18	42	
20–24	27	28		44	40	
25–29	33	15		25	11	
30+	26	11	<0.01	13	7	<0.01
Years breastfed ²						
1+	29	12		16	14	
<1	38	38		34	25	
None	32	51	<0.01	50	61	0.03
Previous induced abortion ³						
No	68	62		85	83	
Yes	32	38	0.27	15	17	0.35
Previous miscarriage ³						
No	78	75		71	68	
Yes	22	25	0.58	29	32	0.38
Ages at menarche						
14+	21	18		23	21	
13	30	19		30	27	
12	28	34		25	28	
<12	21	29	0.05	22	24	0.76
Years of use of oral contraceptives						
0–<6 months	30	30		28	43	
6 months–<5 years	37	43		36	27	
5–9 years	21	13		22	21	
10+ years	12	14	0.29	13	9	<0.01
Family history of breast cancer ⁴						
No	93	95		93	95	
Yes	7	5	0.50	7	5	0.45
Previous breast biopsy						
No	94	95		86	90	
Yes	6	5	0.79	14	10	0.12
Body mass index ⁵						
>28.8	18	46		23	53	
24.7–28.8	25	27		27	27	
22.0–24.6	25	12		26	13	
<22.0	32	15	<0.01	24	7	<0.01
Alcoholic drinks per week						
None	32	59		37	59	
1–2.9	32	22		32	20	
3–6.9	22	13		17	9	
7+	14	7	<0.01	13	12	<0.01
Cigarette smoking						
No	54	59		51	59	
Yes	46	41	0.43	49	41	0.05
Education						
High school or less	23	27		30	47	
Some college	25	25		25	17	
Technical school	6	14		7	14	
College graduate	31	25		22	14	
Postgraduate	15	8	0.03	16	7	<0.01

¹Restricted to parous women.–²Restricted to women with at least 1 livebirth.–³Restricted to gravid women.–⁴In a first-degree relative (mother, sister or daughter).–⁵Weight in kg divided by height in m².

calculated PARs for these same variables; our results were 55% in younger whites *vs.* –8% in younger blacks. When we added the 3 life style factors to these 5 well-established factors, the PAR increased to 69% in younger whites *vs.* 21% in younger blacks.

Among older subjects, the PAR for reproductive factors was 50% in whites but only 21% in blacks. In contrast, the PAR for life style factors was higher among older blacks (31%) than whites (12%). When only well-established risk factors were considered, the PAR was 57% in whites *vs.* 30% in blacks. However, given the higher PAR among blacks for life style factors, there was less

discrepancy between ethnicities when all factors were considered, the PARs being 62% in older whites *vs.* 54% in blacks.

To assess whether variations in PARs for whites and blacks might reflect differences in stages at diagnosis, we also calculated separate PARs for the 56.4% of cases diagnosed at local stages and the 43.6% diagnosed at regional or distant stages. The few discrepancies between stage-specific PARs appeared to reflect instability of estimates, with the majority of the ethnicity- and age-specific PARs being very similar for the 2 stages at diagnosis (data not shown).

TABLE II – RELATIVE RISKS OF BREAST CANCER FOR REPRODUCTIVE AND MENSTRUAL RISK FACTORS BY AGE AND ETHNICITY, ATLANTA AND NEW JERSEY (1990–1992)

	20–39 years of age				40–54 years of age			
	Whites		Blacks		Whites		Blacks	
	RR ¹	95% CI	RR ¹	95% CI	RR ¹	95% CI	RR ¹	95% CI
No. of births								
3+	1.0	—	1.0	—	1.0	—	1.0	—
2	1.2	0.8–2.0	1.0	0.4–2.1	1.6	1.1–2.1	1.4	0.8–2.6
1	1.2	0.7–2.2	0.6	0.3–1.3	1.8	1.2–2.6	1.0	0.5–2.0
0	1.4	0.8–2.2	0.6	0.3–1.4	1.7	1.1–2.5	1.8	0.8–4.3
Ages at first birth ²								
<20	1.0	—	1.0	—	1.0	—	1.0	—
20–24	1.0	0.5–2.0	1.6	0.7–3.4	0.9	0.7–1.3	1.0	0.6–1.7
25–29	1.3	0.7–2.4	2.1	0.8–5.6	1.4	0.9–2.0	1.0	0.4–2.2
30+	1.0	0.5–2.0	1.2	0.3–4.5	1.8	1.2–2.8	1.1	0.4–2.8
Years breastfed ³								
1+	1.0	—	1.0	—	1.0	—	1.0	—
<1	1.4	0.8–2.2	4.6	0.9–24.4	1.2	0.8–1.7	0.7	0.3–1.6
None	1.2	0.7–2.0	5.3	0.9–27.8	1.2	0.8–1.7	0.9	0.4–1.7
Previous induced abortion ⁴								
No	1.0	—	1.0	—	1.0	—	1.0	—
Yes	0.8	0.5–1.3	0.9	0.5–1.9	1.1	0.8–1.5	1.0	0.5–1.7
Previous miscarriage ⁴								
No	1.0	—	1.0	—	1.0	—	1.0	—
Yes	0.9	0.6–1.4	0.9	0.5–2.0	0.8	0.6–1.0	1.0	0.6–1.6
Ages at menarche								
14+	1.0	—	1.0	—	1.0	—	1.0	—
13	1.1	0.7–1.7	0.8	0.3–2.2	1.3	0.9–1.8	1.0	0.5–1.8
12	1.4	0.9–2.3	0.9	0.3–2.1	1.7	1.2–2.3	1.0	0.6–1.9
<12	1.5	0.9–2.5	1.1	0.4–2.7	1.6	1.2–2.2	0.8	0.4–1.6
Years of use of oral contraceptives								
0–<6 months	1.0	—	1.0	—	1.0	—	1.0	—
6 months–<5 years	1.2	0.8–1.9	0.7	0.4–1.6	1.0	0.8–1.4	1.1	0.6–2.2
5–9 years	1.3	0.8–2.1	2.3	0.9–5.7	0.7	0.5–1.0	1.4	0.7–2.8
10+ years	1.8	1.1–3.1	1.2	0.5–3.2	1.0	0.7–1.6	1.8	0.8–4.0

¹RRs adjusted for age (as a continuous variable), for study site and where appropriate for a combined variable of numbers of births and ages at first birth.—²Restricted to parous women.—³Restricted to women with at least 1 livebirth.—⁴Restricted to gravid women.

DISCUSSION

It has long been recognized that white women have higher incidence rates of breast cancer in general than black women. An anomalous observation, however, is the reverse trend among younger women (<40 years). In the present study, we attempted to understand reasons for this ethnic variation in incidence of breast cancer by age. Among older women (40–54 years), it appeared that most of the difference between whites and blacks could be attributed to varying prevalences and effects of well-recognized reproductive and menstrual factors. However, among younger women (20–39), we were less successful in understanding reasons for ethnic differences.

Reasons for the ethnic variation in breast cancer incidence have not been well pursued, especially with respect to age patterns. Relatively few studies have addressed how risk factors might operate in black women. Two studies (Austin *et al.*, 1979; Palmer *et al.*, 1995) involving 127 and 524 black study subjects, respectively, found epidemiologic risk profiles similar to those previously identified in studies focused on white women. Neither study included a white comparison series. In one investigation—the Cancer and Steroid Hormone Study (CASH), in which both black and white cases were included (involving 490 non-Hispanic black and 3,934 non-Hispanic white cases younger than 55 years of age) (Mayberry and Stoddard-Wright, 1992)—some differences were noted with respect to the effects of family history of breast cancer, duration of breastfeeding and age at menarche. However, other analytic approaches, including segregation analyses, from this same study failed to support the notion of ethnic differences in familial effects (Amos *et al.*, 1991). None of these studies of black women, however, have considered intervening effects of age, which as we saw in our study can influence both the prevalence of

exposures and the magnitude of risks, thereby affecting proportions of disease attributable to different risk factors.

Among older women, we derived a higher PAR for whites than blacks (62% vs. 54%). These overall PAR estimates could account for an excess incidence of breast cancer of as much as 21% among older white women $[(1 - .54)/(1 - .62) = 1.21]$. Most of the ethnic difference among older women rests in the larger PAR for the main reproductive factors (numbers of births, ages at first birth, ages at menarche) among whites (50%) than blacks (21%). The difference in incidence might have even been higher had blacks not had a higher PAR for life style factors, which resulted from stronger associations with duration of oral contraceptive use. Although our point estimates need to be cautiously interpreted due to their relatively low precision, especially among black women, they point to a potential explanation for the excess breast cancer incidence observed among older white women. Notably, the higher prevalence of white women with limited numbers of births and late ages at first birth, coupled with slightly higher RR estimates in white women for late ages at birth and early ages at menarche, seems to be chiefly responsible for the ethnic difference in PARs.

To our knowledge, only one other investigation has attempted to assess reasons for varying incidence rates in white vs. black women (Rockhill *et al.*, 1996). Similar to our results in older women, this investigation also found a higher PAR for white vs. black women. However, their estimated PARs of 20% in white women and 6% in black women for benign breast biopsy, history of breast cancer in a first-degree relative, menarche before age 12 and nulliparity or first birth at age 30 or later are considerably lower than our estimates. Although the populations studied were not entirely similar (given their focus on somewhat older women), it may be that some of the difference in PARs between the investigations reflected their use of

TABLE III – RELATIVE RISKS OF BREAST CANCER FOR FAMILIAL, LIFE STYLE AND OTHER RISK FACTORS BY AGE AND ETHNICITY, ATLANTA AND NEW JERSEY (1990–1992)

	20–39 years of age				40–54 years of age			
	Whites		Blacks		Whites		Blacks	
	RR ¹	95% CI	RR ¹	95% CI	RR ¹	95% CI	RR ¹	95% CI
Family history of breast cancer								
No	1.0	—	1.0	—	1.0	—	1.0	—
Yes	1.9	1.1–3.4	2.6	0.9–8.1	2.6	1.8–3.7	3.0	1.3–6.9
Previous breast biopsy								
No	1.0	—	1.0	—	1.0	—	1.0	—
Yes	1.1	0.5–2.1	1.1	0.3–4.0	1.6	1.2–2.3	1.1	0.5–2.6
Body mass index ²								
>28.8	1.0	—	1.0	—	1.0	—	1.0	—
24.7–28.8	1.4	0.9–2.4	0.8	0.4–1.8	1.0	0.7–1.5	0.9	0.5–1.7
22.0–24.6	1.0	0.6–1.7	2.3	0.9–5.6	1.2	0.8–1.7	1.4	0.6–3.0
<22.0	1.5	0.9–2.5	1.3	0.5–3.1	1.2	0.9–1.8	0.8	0.3–1.9
Alcoholic drinks per week								
None	1.0	—	1.0	—	1.0	—	1.0	—
1–2.9	1.1	0.8–1.7	1.1	0.5–2.2	1.3	0.9–1.7	1.6	0.9–2.6
3–6.9	1.1	0.7–1.7	0.9	0.4–2.4	1.2	0.9–1.7	1.3	0.6–2.8
7+	1.1	0.6–1.9	1.9	0.7–5.5	1.4	1.0–2.0	1.6	0.8–3.0
Cigarette smoking								
No	1.0	—	1.0	—	1.0	—	1.0	—
Yes	1.1	0.8–1.5	0.9	0.5–1.6	1.1	0.8–1.4	0.9	0.6–1.6
Education								
High school or less	1.0	—	1.0	—	1.0	—	1.0	—
Some college	0.7	0.4–1.1	1.2	0.5–2.6	1.3	0.9–1.7	1.3	0.7–2.2
Technical school	0.6	0.3–1.4	0.6	0.2–1.6	0.9	0.5–1.4	0.7	0.3–1.4
College graduate	0.6	0.4–1.0	0.8	0.3–1.8	1.0	0.8–1.4	0.8	0.4–1.6
Postgraduate	0.8	0.5–1.4	1.6	0.5–5.2	1.2	0.8–1.6	1.6	0.7–3.4

¹RRs adjusted for age, for study site and for a combined variable of numbers of births and ages at first birth.

TABLE IV – POPULATION ATTRIBUTABLE RISKS (PARs) (%) FOR SELECTED BREAST CANCER RISK FACTORS BY AGE AND ETHNICITY, ATLANTA AND NEW JERSEY (1990–1992)¹

	20–39 years of age				40–54 years of age			
	Whites		Blacks		Whites		Blacks	
	PAR	(95% CI)	PAR	(95% CI)	PAR	(95% CI)	PAR	(95% CI)
Limited no. of births	19	(–10, 48)	–30	(–95, 35)	26	(14, 39)	24	(6, 43)
Late ages at first birth	20	(–21, 61)	8	(–31, 47)	23	(3, 44)	13	(–12, 38)
Early ages at menarche	20	(–7, 46)	–6	(–75, 63)	29	(13, 44)	–7	(–51, 38)
Extended use of oral contraceptives	20	(–1, 42)	8	(–38, 54)	–6	(–24, 13)	22	(1, 44)
Family history of breast cancer	6	(1, 11)	8	(0, 16)	10	(6, 13)	8	(2, 13)
Previous breast biopsy	0	(–4, 4)	–1	(–10, 9)	6	(2, 11)	1	(–7, 8)
Low body mass index	21	(–9, 51)	12	(–19, 42)	5	(–15, 24)	–2	(–23, 19)
High alcoholic consumption	7	(–17, 31)	7	(–17, 30)	16	(3, 29)	17	(0, 33)

¹Calculations based on categories, reference levels and results as shown in Tables I to III. PARs adjusted for age, for study site and where appropriate for a combined variable of numbers of births and ages at first birth.

wider reference groups, which would yield smaller PARs (Wacholder *et al.*, 1994). In our study, the reference categories used were narrowly defined for most variables (*e.g.*, age at menarche of 14+ years and age at first birth of <20 years) because our primary aim was to determine how much potential difference in PARs and associated incidence rates could be accounted for by established and speculative risk factors. Among older subjects in our study, there were only small differences in the RRs for most factors for black *vs.* white women; therefore, most of the differences in PARs by ethnicity were accounted for by higher “exposure” probabilities in whites, as has been seen by Mayberry and Stoddard-Wright (1992). In addition to white women having fewer births and later ages at first birth compared to blacks, they also reported longer use of this oral contraceptives, heavier consumption of alcoholic beverages, and more thinness, a risk factor for early onset breast cancers (Swanson *et al.*, 1996).

Our overall PAR estimate for older white women of 62% is somewhat higher than in other studies (Bruzzi *et al.*, 1985; Madigan *et al.*, 1995). In the study by Bruzzi *et al.* (1985), the

factors of age at first birth, age at menarche, breast biopsies and family history of breast cancer accounted for approximately 55% of disease occurrence. Although some of the discrepancy might be due to our focus on relatively young women (40–54 years), it is of interest that restriction of our analysis to these same risk factors derived a very similar figure (57%) to past studies, supporting the notion that our higher estimates were due to the consideration of a variety of more speculative risk factors (including years of use of oral contraceptives, body mass and alcohol consumption). Such an increase in attributable risk after consideration of possible breast cancer risk factors has also been demonstrated in an Italian study (Tavani *et al.*, 1997). It is thus encouraging that consideration of additional potential risk factors might lead to greater PARs than the 50% figure generally attributed to identified predictors, of importance given that these more speculative factors are ones that should be subject to personal modification.

In our study, only one risk factor appeared to have a larger PAR in older blacks than whites, namely years of use of oral contraceptives. Whites actually had a slightly higher prevalence of use than

TABLE V – POPULATION ATTRIBUTABLE RISKS (PARs) (%) FOR SELECTED COMBINATIONS OF BREAST CANCER RISK FACTORS BY AGE AND ETHNICITY, ATLANTA AND NEW JERSEY (1990–1992)¹

	20–39 years of age				40–54 years of age			
	Whites		Blacks		Whites		Blacks	
	PAR	(95% CI)	PAR	(95% CI)	PAR	(95% CI)	PAR	(95% CI)
Reproductive variables								
No. of births, ages at first birth, ages at menarche	52	(25, 80)	–22	(–134, 91)	50	(35, 65)	21	(–17, 59)
Life style factors								
Years of use of oral contraceptives, body mass, alcoholic drinks	36	(5, 67)	18	(–38, 74)	12	(–14, 37)	31	(5, 57)
Well-established risk factors								
No. of births, ages at first birth, ages at menarche, breast biopsy, family history of breast cancer	55	(28, 82)	–8	(–109, 93)	57	(43, 70)	30	(–4, 64)
Multiple risk factors								
Reproductive and life style factors, ² breast biopsy, family history of breast cancer	69	(46, 92)	21	(–67, 109)	62	(45, 79)	54	(24, 84)

¹Calculations based on categories, reference levels and results as shown in Tables I to III. PARs were adjusted for age, for study site and where appropriate for a combined variable of numbers of births and ages at first birth. ²Reproductive and life style factors are defined in the first part of the Table.

blacks, so that the larger PAR was due to the increased magnitude of association in blacks. Although chance must be considered as a possible explanation for the higher PAR in blacks, especially given the wide associated confidence interval, it is of interest that 2 other investigations found slightly higher relative risks associated with oral contraceptive use in blacks than whites (Palmer *et al.*, 1995; Mayberry, 1994). Whether such a difference is attributable to unique host factors or to differences in use patterns between blacks and whites may be worthy of further investigation.

Given the observation that blacks have higher incidence rates than whites at young ages (before age 40), we had hoped that risk factor differences might be associated with this trend, *i.e.*, that younger blacks would have higher PARs than whites. Based on previous speculations, we were particularly interested in evaluating whether the higher rates of breast cancer in young black women might be due to younger ages at menarche (Mayberry, 1994) or higher frequencies of induced abortions and oral contraceptive use at early ages (Krieger, 1990). Although our analyses must be cautiously interpreted due to the low precision of many of the point estimates, particularly in younger black women, our results do not seem to indicate that the risk factors that we studied can be attributed to the ethnic patterns among younger women. There was no difference in the prevalence of or risk associated with induced abortion between the 2 ethnic groups. Although there was some indication that blacks had earlier ages at menarche, less extended periods of breastfeeding and more frequent use of oral contraceptives at early ages, ethnic differences did not persist when PARs were examined. In fact, among young blacks, very little disease occurrence was associated with either established or speculative risk factors (PAR = 21%). This reflected the fact that reproductive factors did not contribute to disease occurrence among younger blacks as expected, since multiparity was associated with increased risk among this subgroup.

Our analyses were limited by relatively small numbers of young black women, necessitating that further studies focus on the role of breast cancer risk factors in this ethnic subgroup. However,

tentative results from our study suggest that the difference in breast cancer incidence rates between younger whites and blacks is not due to established risk factors, but possibly to some yet unidentified predictors. Other analyses and publications from our study addressed many new etiologic hypotheses, including effects of induced abortions, *in utero* exposures, physical activity, adolescent and adult diet as well as cigarette smoking. Although analyses to date of our data do not indicate that these factors are strong predictors of risk, future analyses will continue to explore ethnic differences in effects of these speculative factors. However, our study was initiated before there was widespread interest in the role of general environmental agents. Thus, it would appear worthwhile for future investigations to focus on these exposures as possible explanations for differing incidence patterns in whites vs. blacks, particularly among younger women. The evaluation of pesticides might be particularly informative, given their biologic plausibility as risk factors (Houghton and Ritter, 1995) and the fact that one previous study has shown ethnic differences in both the prevalence of exposure to organochlorines and associated breast cancer risk (Krieger *et al.*, 1994).

Although our results must be cautiously interpreted because of relatively small numbers and the potential effects of selection and reporting biases by ethnicity, the results are provocative in suggesting that the excess incidence of breast cancer among older white compared with black women is associated with a relatively limited number of factors relating to reproductive and menstrual behaviors. The similarity of our PARs to those published previously lends credence to their validity. It is also encouraging that an increase in PARs was observed when more speculative risk factors were taken into account. Less conclusive were our attempts to understand the high incidence rates of breast cancer in young black women. Further efforts to address this issue will need to focus on larger numbers of women; in addition, consideration should be given to addressing factors not considered in our study, including effects of a variety of potential environmental carcinogens.

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