

Disparities in Breast Cancer Incidence across Racial/Ethnic Strata and Socioeconomic Status: A Systematic Review

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Objectives: A higher incidence of breast cancer has been reported both in white women and women of higher socioeconomic status (SES) compared to women of other races and lower SES, respectively. We explored whether differences in SES can account for disparities in breast cancer incidence between races.

Methods: We identified several studies published between 1990 and 2007 that addressed disparities in breast cancer incidence across racial and socioeconomic strata. For each study, we calculated incidence rate ratios (IRRs) for breast cancer incidence in the highest strata to lowest strata of SES for white, black, Hispanic and Asian/Pacific-Islander populations. We then used these IRRs to compare trends in SES and breast cancer incidence between races and across studies.

Results: The studies we identified revealed that the magnitude of the disparity in breast cancer incidence between races decreases with increasing SES. While individual census-tract based studies' methods of assessing the association between SES and breast cancer incidence did not identify consistent trends between races, adjustment for risk factors closely correlated with SES eliminated the statistical differences in breast cancer incidence between women of white, Hispanic and Asian/Pacific-Islander, but not black, ethnicity.

Conclusion: We found that racial differences in breast cancer incidence can largely be accounted for by ethnic differences in SES among white, Hispanic and Asian/Pacific-Islander women, but not between these populations and black women. We further highlight important differences in methodology between previously published studies that may account for their disparate findings.

Key words: breast cancer ■ race/ethnicity ■ socioeconomic status ■ cancer

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INTRODUCTION

Breast cancer is the most common cancer and the second most common cause of cancer fatality in women in the United States.¹ Several decades of epidemiological research have demonstrated widespread racial and socioeconomic disparities across a multitude of disease and public health aspects, including incidence, screening prevalence, stage at diagnosis, treatment, survival and mortality.² It has been well documented that the prevalence of breast cancer in the United States varies significantly among racial groups.¹ From 2000–2003, the average age-adjusted annual incidence was highest in white non-Hispanic women (141 cases per 100,000), with significantly lower rates in African-American women (118 per 100,000), Asian Americans/Pacific Islanders (89 per 100,000) and Hispanics/Latinas (89 per 100,000).¹ Furthermore, it has been well established that a correlation exists between a woman's socioeconomic status (SES) and her risk of developing breast cancer, with a higher incidence of breast cancer in women of higher SES than in those of lower SES.^{3–7} Numerous studies have investigated this relationship between SES and breast cancer incidence in various ethnic groups throughout the United States.^{2,5,6,8–13} We herein present the results of these studies and attempt to identify: 1) whether a consensus on the relationship between SES and breast cancer incidence exists and is consistent across races, and 2) whether this difference in SES and incidence could account for the observed disparity in breast cancer incidence between races.

MATERIALS AND METHODS

We searched PubMed to identify literature that described and analyzed population demographics of breast cancer patients both across socioeconomic strata and multiple racial/ethnic groups. The search strategy consisted of the search terms “breast cancer incidence” combined with the terms “disparity,” “socioeconomic status,” “racial,” “race” and/or “ethnicity.” The search results were limited to studies published between 1990 and 2006 that utilized data from national, state, regional or community cancer registries within the United States, includ-

ing the Surveillance, Epidemiology and End Results (SEER) Program of the National Cancer Institute, Women's Health Initiative (WHI), California Cancer Registry, Northern California Cancer Center's San Francisco/Oakland SEER cancer registry, Los Angeles Cancer Surveillance Program and Massachusetts Cancer Registry. To maximize the uniformity of comparison between studies, we limited our analysis to studies which provided comparative data on breast cancer incidence at different socioeconomic levels in women of white, black, Hispanic and Asian/Pacific Islanders. Therefore, studies that compared socioeconomic differences in the incidence of breast cancer between only two or among three racial groups, i.e., white and black women, were excluded from our present analysis, although those studies including more than these four ethnic groups were included. In order to compare the findings across studies identified by these criteria, we calculated incidence risk ratios (IRRs) for the rate of breast cancer incidence in the highest SES level of the population compared to the lowest SES level for each racial group in each study. We then compared the calculated IRRs to determine: a) whether the relationship between SES and breast cancer incidence *for each race* was consistent across studies, and b) whether the differences in this relationship *between races* were consistent across studies. We further compared the results of these studies to see whether their findings or the findings of any other studies could determine whether the differences in breast cancer incidence among white, black, Hispanic and Asian/Pacific-Islander women could be accounted for by differences in SES between these races.

RESULTS

We identified five studies that examined the association between breast cancer incidence and SES in white, black, Hispanic and Asian/Pacific-Islander women. Four studies used census-tract level data to stratify patients into quintiles or terciles of SES, based on algorithms that employed neighborhood-level parameters such as median household income, poverty rate, average education level, household crowding, employment rate and Townsend deprivation index, among others.^{9-11,13} We calculated IRRs for each of these studies, subdivided into locale and study time-period-specific data sets (Table 1). The fifth study, by Chlebowski and colleagues, utilized individual patient demographic data from the WHI to calculate hazard ratios for breast cancer incidence for each racial group.⁸ In contrast to the first four studies analyzed, Chlebowski's analysis did not report discrete SES-level breast cancer incidence data, but rather used logistic and multivariate regression models to control for several SES-related breast cancer risk factors in order to determine the contribution of SES on racial differences in breast cancer incidence.⁸ Due to its vastly different methodology, this study could not be directly compared to the previous studies; hence, it is excluded from the comparison of Table 1 and is rather summarized in Table 2.

All of the studies we analyzed supported the observation that white non-Hispanic women are at the highest nonadjusted and age-adjusted risk for breast cancer, followed by black, Hispanic and Asian women.^{8-11,13} A closer examination of Table 1 reveals several interesting find-

Table 1. Breast cancer incidence rate ratios (IRRs) and 95% confidence intervals (in italics) in highest SES level compared to lowest SES level by race in several comparative studies.⁵⁻⁸

Study/Data Subset	N	White Non-Hispanic	Black	Hispanic	Asian/Pacific Islander
1. Liu et al. ¹¹ (LA: 1972-1992)	81,737	1.59* <i>(1.20-2.12)</i>	1.39* <i>(1.04-1.90)</i>	1.37 <i>(0.99-1.93)</i>	1.36 <i>(0.89-2.12)</i>
2. Krieger et al. ¹⁰ (SF: 1988-1992)	70,899	0.86 <i>(0.68-1.10)</i>	1.01 <i>(0.77-1.36)</i>	1.98* <i>(1.41-2.79)</i>	1.27 <i>(0.87-1.81)</i>
3. Yost et al. ¹³ (CA: 1988-1992)	97,227	1.27* <i>(1.04-1.56)</i>	1.22 <i>(0.98-1.52)</i>	1.83* <i>(1.41-2.40)</i>	1.65* <i>(1.23-2.21)</i>
4. Krieger et al. ⁹ (LA: 1988-1992)	23,526	1.16* <i>(1.08-1.25)</i>	1.15 <i>(0.82-1.6)</i>	1.77* <i>(1.45-2.15)</i>	1.36* <i>(1.10-1.68)</i>
5. Krieger et al. ⁹ (SF: 1988-1992)	12,610	0.96 <i>(0.86-1.08)</i>	1.19 <i>(0.84-1.68)</i>	2.07* <i>(1.52-2.81)</i>	2.03* <i>(1.54-2.66)</i>
6. Krieger et al. ⁹ (LA: 1998-2002)	26,323	1.19* <i>(1.11-1.26)</i>	1.21 <i>(0.98-1.51)</i>	1.81* <i>(1.51-2.18)</i>	1.34* <i>(1.15-1.57)</i>
7. Krieger et al. ⁹ (SF: 1998-2002)	14,622	1.21* <i>(1.06-1.38)</i>	0.90 <i>(0.67-1.20)</i>	1.87* <i>(1.44-2.44)</i>	1.46* <i>(1.21-1.77)</i>
8. Krieger et al. ⁹ (MA: 1998-2002)	23,896	1.14* <i>(1.07-1.21)</i>	0.68 <i>(0.42-1.11)</i>	1.55 <i>(0.93-2.57)</i>	1.69* <i>(1.10-2.59)</i>

CA: California; LA: Los Angeles, CA; MA: Massachusetts; SF: San Francisco, CA; * Denotes statistically significant IRR values (i.e., 95% confidence interval does not include the null value, 1)

ings on disparity trends across both the studies and ethnic groups (Table 1). Six of the eight datasets analyzed in Table 1 demonstrated a statistically significant difference in breast cancer incidence between the highest and lowest SES levels in the Hispanic (datasets 2–7: IRRs 1.77–2.07, lowest limit of 95% CIs=1.41), Asian/Pacific Islander (datasets 3–8: IRRs 1.36–2.03, lowest limit of 95% CIs=1.10) and white (datasets 1, 3, 4, 6–8: IRRs 1.14–1.59, lowest limit of 95% CIs=1.04) populations. In contrast, for the black population, seven of the eight analyzed datasets showed no difference in the incidence of breast cancer at highest level of SES compared to the lowest level, with only one dataset demonstrating a statistically significant disparity (data set 1: IRR 1.39, lower limit of 95% CI=1.04). This suggests that, as opposed to the apparent contributory effect of high SES on breast cancer risk in white, Hispanic and Asian/Pacific-Islander women, the risk of breast cancer in black women may not be significantly modified by SES.

To assess whether adjusting for SES could account for the widely observed racial disparities in breast cancer incidence, we further analyzed the studies in Table 1 to look for trends that may support or reject this hypothesis. However, the wide methodological variability between the studies significantly limited the analyses we could perform. Different studies often defined SES levels by inconsistent criteria from one another, making quantitative comparisons between SES levels in different studies implausible. Instead of attempting a fundamentally flawed aggregated statistical analysis, we qualitatively assessed the trends in the relationship between SES, race and breast cancer incidence across studies. When matched for SES, breast cancer incidence

remained highest in white women, followed by black, Hispanic and Asian/Pacific-Islander women. The racial differences in breast cancer incidence were greatest in the lowest socioeconomic group and decreased progressively as SES increased. This effect was especially pronounced in Hispanic and Asian women, suggesting that increased SES may be responsible for introducing risk factors, with the most significant effects on demographic populations with the lowest overall risk of breast cancer.^{9–11} This narrowing of the disparity gap that was observed in Hispanic and Asian women at higher levels of SES was notably absent or present only minimally in the black populations studied, supporting the evidence for the role of SES in modifying breast cancer risk presented in Table 1.^{9–11}

Ultimately, we deemed this qualitative analysis inadequate to determine whether differences in SES between races can account for racial disparities in the incidence of breast cancer. Therefore, we examined the final study identified by our search, by Chlebowski and colleagues, to address our hypothesis.⁸ Utilizing observational and randomized data from the WHI, Chlebowski and colleagues identified a study population of 156,570 postmenopausal women, which they divided by ethnicity into white, black, Hispanic and Asian/Pacific-Islander cohorts.⁸ During the follow-up period (median 6.3 years), 3,938 new invasive breast cancers were identified in this population, from which hazard ratios for breast cancer risk were calculated (using white women as a reference group) and then adjusted by three multivariate analysis models to control for a variety of breast cancer risk factors (Table 2).⁸ The most comprehensive “final model” employed by the authors adjusted for a

Table 2. Hazard ratios and P values of invasive breast cancer incidence by race after adjusting for breast cancer risk factors and other covariates (white women as reference).⁹

	Hazard Ratio	95% CI	P Value
Age Adjusted Model			
Black	0.69*	0.60–0.78	<0.001
Hispanic	0.70*	0.57–0.85	<0.001
Asian/Pacific Islander	0.85	0.69–1.05	0.13
“Gail” Adjusted Model			
Black	0.85*	0.73–1.00	0.05
Hispanic	0.80*	0.63–1.01	0.07
Asian/Pacific Islander	0.89	0.71–1.11	0.30
Final Model			
Black	0.75*	0.61–0.92	0.006
Hispanic	0.98	0.74–1.30	0.90
Asian/Pacific Islander	0.94	0.72–1.22	0.62

Age-adjusted model adjusted for age only; “Gail”-adjusted model adjusted for age, number of first-degree relatives with breast cancer, age at menarche, age at first birth and prior breast biopsy for benign disease; Final model adjusted for the covariates in “Gail” model plus education, BMI, physical activity, number of second-degree relatives with breast cancer, parity, hormone therapy (HT) use, prior contraceptive use, alcohol, smoking, dietary intake, HT × BMI interaction and mammography (as a time-dependent covariate). P values indicate a global test of whether breast cancer incidence differs by race/ethnicity.* Denotes statistically significant ($p \leq 0.05$) hazard ratios.

total of 17 risk factors, many of which are closely related to SES, including age at primiparity, total parity, education, body mass index (BMI), physical activity, hormone replacement therapy (HRT) use, prior oral contraceptive use, alcohol, smoking, dietary intake, HRT and BMI interaction, mammography utilization, and prior breast biopsy for benign disease.⁸ This final model revealed no difference in breast cancer incidence among Hispanic (hazard ratio=0.98; $p=0.90$), Asian/Pacific Islander (hazard ratio=0.94; $p=0.62$) and white women (statistical reference group).⁸ Additionally, after controlling for SES-related risk factors, Chlebowski found strong evidence for a significantly lower risk of breast cancer in the black population relative to whites, Hispanics and Asian/Pacific Islanders (hazard ratio=0.75, $p=0.006$).⁸ These findings represent a novel and important conclusion regarding the role of race as an independent risk factor for the development of breast cancer in white, Hispanic and Asian/Pacific-Islander women, and as a potentially protective factor against breast cancer in the black population.

DISCUSSION

Our review of the literature on the association between breast cancer incidence and SES across ethnicities revealed that while there was generally a positive correlation between the two variables, the strength of that trend for each ethnic group varied widely from study to study. Much of this lack of consistency between studies can likely be attributed to the lack of standard determination of SES. Patients in the studies analyzed in Table 1 were assigned to SES strata based on calculations using neighborhood-level variables from census-tract data, such as median household income, poverty rate, employment rate and others, as described above. As these factors were inconsistently employed and weighted in each of these studies, the data and findings from the studies cannot be meaningfully compared to one another. Since data from multiple census-tract-based studies cannot be reliably aggregated, it is therefore not possible to perform a statistically valid meta-analysis on the racial disparity in breast cancer risk modification by SES using the studies in Table 1.

The approach taken by Chlebowski and colleagues, however, fundamentally differed from these census-tract based studies. Chlebowski's methodology adjusted for a multitude of risk factors for breast cancer that are closely linked to and correlate with SES.^{8,14-34} Thus, the Chlebowski group was able to control for SES in a surrogate, but more precise, manner than comparable census-tract-based studies. As a result, the findings of Chlebowski's study are less likely to suffer from many of the flaws inherent to the census-tract-based studies considered in this review, and thus make their conclusions more broadly applicable than the results of an attempted meta-analysis of multiple census-tract-based studies.

In the census-tract-based studies we analyzed, population data were derived largely from urban locales in California and Massachusetts. Therefore, in interpreting the comparison of these studies in Table 1, we cannot rule out the possibility that the variability observed in the findings of the studies may have resulted from variation in population risk factors in the specific areas sampled. Specifically, potential confounding factors not controlled for in these studies, such as environmental exposures, dietary habits and other breast cancer risk factors, defined or otherwise, that may be coincident with SES in one locale but not in another may contribute to differences in the incidence of breast cancer across both racial and socioeconomic strata in two different locales. Therefore, the findings presented in Table 1 may be of only limited applicability to different populations from those in which the studies we reviewed were conducted. This limitation stems from the lack of well-conducted studies on the effects of SES and race on breast cancer risk in other populations than those discussed above, which significantly limited the number of studies we could include in our comparison. In contrast, the conclusions of Chlebowski's study are based on data sampled from a diverse and widely representative sample population, encompassing >150,000 patients from 40 clinical centers nationally. The benefit of Chlebowski's methodological design is clear; their results can be widely applied to the general population with confidence, whereas the results of the census-tract-based studies cannot.

Another question raised by the observations of this review involves the inconsistency in the effect of SES on breast cancer incidence between the studies in Table 1. As could be expected in any comparison of cancer demographics between different locales, specific incidence rate ratios (IRRs) in our review varied from study to study. Despite this expected degree of variation, the finding from the study by Liu and colleagues (Table 1, dataset 1), that higher SES levels were correlated with increased risk of breast cancer in white and black women, but not in Hispanic or Asian/Pacific-Islander women, is an intriguing departure from the results of other studies we reviewed.¹¹ The racial classification scheme used in the study by Liu was inherently limited by the availability and accuracy of retrospectively attained demographic information, in which patients were assigned to a race based on sometime-questionable criteria, i.e., last name. This methodological detail could have accounted for some of the inconsistency between this study and the other studies in Table 1. More importantly, this methodological flaw highlights the need for standardized reporting of patient demographic data in order to maintain the external validity of the studies that utilize this data. Similar flaws likely exist in all epidemiological studies based on retrospectively collected demographic data; however, awareness of these flaws and efforts to limit them is important for future studies in order to facilitate

valid intrastudy comparisons for the sake of meta- and other cumulative analyses.

Several previous studies have explored the association of higher SES with breast cancer risk factors. Later age of primiparity and low total parity are commonly associated with higher levels of education and higher SES, due to postponement of childbearing until after the completion of education and the consequent increase in maternal age.¹⁷⁻²⁰ However, in the study by Liu and colleagues, adjustment for fertility, marital status, education and late primiparity was not sufficient to eliminate a strong positive association between SES and breast cancer risk.¹¹ These authors further proposed that diet and physical activity, which may also vary by income and SES, may play a role in the regulation of the internal hormonal environment, thereby further influencing risk of breast cancer in association with SES.¹¹ An analysis of data from the WHI revealed nonsignificant trends which suggest that adherence to lower-fat diets may reduce a woman's risk of developing breast cancer. Furthermore, subgroup analysis revealed a significant risk reduction in women with the highest level of baseline percentage energy intake from fat prior to intervention ($p=0.04$).³⁵ The Chlebowski study controlled for these risk factors, among others, which eliminated the differences in breast cancer incidence for all groups except black women. However, given the large number of factors for which Chlebowski and colleagues simultaneously controlled, the individual contributions of specific risk factors on the development of breast cancer are difficult to compare without further multivariate analysis.

Another factor that requires specific attention in future breast cancer studies is the disparity in HRT utilization between socioeconomic groups. Anecdotal inference has causatively implicated the decreased use of HRT by postmenopausal women following the first report of the WHI in July 2002 in the decreased incidence of hormone-receptor-positive breast cancers in postmenopausal women in 2003.^{36,37} Most studies on the use of hormone replacement therapy, conducted both before and after the premature termination of the WHI Estrogen Plus Progestin Trial (WHI-EPT), analyzed differences in HRT usage by racial group. These studies unequivocally demonstrated significantly greater use of HRT by women of white ethnicity and higher SES, both prior to and after the WHI-EPT.³⁸⁻⁴³ Interestingly, these analyses failed to demonstrate a difference in the rates of discontinuation and initiation of HRT between different racial and socioeconomic groups after the WHI-EPT, suggesting that the findings from this seminal study were disseminated equivalently to HRT users regardless of race or SES.⁴³ These studies, however, vary in their determination of SES, using income or education as surrogate indicators of SES, and thereby suffer from the same drawbacks as the studies in Table 1. Furthermore, these studies of the WHI-EPT failed to subclassify SES

groups by race (or vice versa), making an analysis of the associations among race, SES, HRT usage and breast cancer incidence difficult to perform at this time.^{41,43} An analysis that closely examines these factors would serve to determine whether all racial and socioeconomic groups benefit equally from HRT discontinuation, and would further the understanding of the breast cancer risk conferred by HRT and its interaction with other risk factors that may vary by race or SES.

Our review highlights a common, and ultimately flawed, implication of several recent census-tract-based studies of breast cancer epidemiology.^{9-11,13} These studies conclude that certain risk factors exist in white women that predispose them to a higher risk of breast cancer than black, Hispanic or Asian/Pacific-Islander women, and that these risk factors are not modified by adjustment for SES.^{9-11,13} However, the novel methodological approach taken in the Chlebowski study appears to identify many of these risk factors, several of which indeed do correlate with SES, including age at primiparity, total parity, education, BMI, physical activity, hormone therapy use, prior oral contraceptive use, alcohol, smoking, dietary intake and mammography utilization.^{8,14-34} The correlation of these identified risk factors with SES suggests that the disparity in breast cancer risk between white, Hispanic and Asian/Pacific-Islander women can be accounted for by differences in the SES of those ethnic groups, and is not attributable to racial factors. As for black women, the overall lack of statistically significant incidence rate ratios in Table 1 suggests that SES effects do not play an important role in modifying the risk of breast cancer in this population. This suggestion is supported by Chlebowski's analysis, in which adjustment for SES-correlated breast cancer risk factors failed to eliminate the difference in hazard ratio of between black women and women of other races. Taken together, these findings imply the presence of a protective factor, which is not modified by SES, against breast cancer in black women.

The identification of this protective factor, or set of factors, has thus far proven elusive. In their discussion, Chlebowski et al. postulate that this factor may be the lower mammographic breast density in black women compared to the other groups, as mammographic breast density is considered a strong predictor of risk in all racial groups.^{8,9,44,45} Indeed, black women overall do have the lowest mammographic breast densities of the racial groups discussed in our review.⁴⁶ However, adjustment of breast density for age and BMI eliminates the differences in breast density between black and white women, while a higher average breast density persists in Asian women despite this adjustment.^{47,48} Given that Chlebowski's analysis controlled for both age and BMI in their "final model," it is highly unlikely that the lower hazard ratio observed for breast cancer in black women could be attributed to racial differences in breast density. The protective effects of other genetic or environmental

factors against breast cancer in black women are as of yet undetermined, and warrant further investigation.

It is important to note that despite the lower overall incidence of breast cancer in black women, black women on average have more poorly differentiated, higher grade and 35% more advanced-stage breast cancers than do their white counterparts.⁴⁹ Black women also have a higher frequency of estrogen-receptor-negative tumors (a poor prognostic indicator) than do white, Hispanic or Asian women.⁴⁹ Additionally, the mortality rate due to breast cancer in black women is declining at a lower rate than it is in white women.¹ However, it is at present unclear whether these disparities, which appear to favor white women, are due primarily to the lower rates of mammography screening and postmammogram follow-up in black women, or are a product of a much more complex host of differences between these populations.

ACKNOWLEDGEMENTS

I would like to thank Diane McKee, Clyde Schechter, and Rachel Bartash of the Albert Einstein College of Medicine for their invaluable help in the preparation and editing of this manuscript.

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The NCCC is a component of the AIDS Education and Training Centers (AETC) Program funded by the Health Resources and Services Administration (HRSA) HIV/AIDS Bureau in partnership with the Centers for Disease Control and Prevention (CDC).