J Community Health
DOI 10.1007/s10900-010-9346-2

ORIGINAL PAPER

The Racial Disparity in Breast Cancer Mortality

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Abstract Black women die of breast cancer at a much higher rate than white women. Recent studies have suggested that this racial disparity might be even greater in Chicago than the country as a whole. When data describing this racial disparity are presented they are sometimes attributed in part to racial differences in tumor biology. Vital records data were employed to calculate age-adjusted breast cancer mortality rates for women in Chicago, New York City and the United States from 1980–2005. Race-specific rate ratios were used to measure the disparity in breast cancer mortality. Breast cancer mortality rates by race are the main outcome. In all three geographies the rate ratios were approximately equal in 1980 and stayed that way until the early 1990s, when the white rates started to decline while the black rates remained rather constant. By 2005 the black:white rate ratio was 1.36 in NYC, 1.38 in the US, and 1.98 in Chicago. In any number of ways these data are inconsistent with the notion that the disparity in black:white breast cancer mortality rates is a function of differential biology. Three societal hypotheses are posited that may explain this disparity. All three are actionable, beginning today.

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Published online: 29 December 2010

Keywords Breast cancer · Joinpoint regression · Mammography access · Mammography quality · Mortality rates · Racial disparities · Treatment quality

Introduction

It is frequently noted that black women die from breast cancer at a higher rate than white women [1–3]. In addition, some analyses suggest that there are racial differences in biological characteristics of breast tumors [4, 5]. These two matters are often then conflated to suggest that differential biology may be a risk factor for the racial disparity in breast cancer mortality [6–8]. We believe that such a syllogism is faulty and is proven wrong by an examination of the data underlying this disparity. The purpose of this article is to present data relevant to this issue from the United States, New York and Chicago regarding the racial disparity in breast cancer mortality and to suggest systems-based hypotheses that might be examined in order to delineate the factors responsible for this disparity.

Methods

Deaths where the cause was malignant neoplasm of the breast (ICD-9 = 174, ICD-10 = C50) were included in this analysis. There was an ICD version change in 1999, however, we did not apply a comparability ratio to 1998 breast cancer deaths because there was no statistically significant difference in the number of cases captured between versions 9 and 10 based on coding changes (the comparability ratio for malignant neoplasm of breast = 1.01, 95%CI: 1.00–1.01) [9].

United States Data

All US numerators were abstracted from death files maintained by the National Center for Health Statistics. Population-based denominators for 1980, 1990, and 2000 were derived from Census data. Population-based denominators for non-Hispanic White in 2005 were gathered from the American Community Survey. Population-based denominators for the non-Hispanic Black population in 2005 were not readily available so we estimated this population using the same methodology employed to estimate the 2005 non-Hispanic Black population in Chicago. Population-based denominators for years other than 1980, 1990, 2000, and 2005 were estimated using exponential interpolation. For 1980 denominator data and 1980–1989 we utilized data on Black and White persons (which included Hispanics) because Hispanic origin data were not available for the US overall during that time period.

New York City Data

New York City numerator data for years 1980–1989 and 2005 were obtained through a special request to the New York Department of Mental Health and Hygiene. Numerator data for years 1990–2004 were abstracted from death files maintained by the National Center for Health Statistics. Population-based denominators for 1980, 1990, 2000, and 2005 were obtained via the same avenues as for Chicago (below). Population-based denominators for the non-Hispanic Black population in 2005 were not readily available and thus we used the same estimating techniques as for the non-Hispanic Black population in Chicago to estimate this population. Population-based denominators for years other than 1980, 1990, 2000, and 2005 were estimated using exponential interpolation.

Chicago Data

All Chicago numerators were abstracted from the vital records (birth and death) files maintained by the Illinois Department of Public Health and provided to us by the Chicago Department of Public Health. Denominators for population-based rates in Chicago in 1980, 1990, and 2000 were gathered from the Census. Denominators for non-Hispanic White (NHW) in 2005 were gathered from the American Community Survey [10]. Denominators for the non-Hispanic Black (NHB) population in 2005 were not readily available so we estimated the population using an age-specific ratio calculated by dividing the number of non-Hispanic Blacks by total Blacks in the 2000 Census and multiplying the proportion by the number of all blacks in 2005 from the American Community Survey for each age group. Denominators for years other than 1980, 1990, 2000, and 2005 were estimated using exponential interpolation.

Analysis of Trends

To measure disparity we calculated the rate ratio between the NHB and NHW rates. The rate ratio is greater than 1.00 if the NHB rate is higher than the NHW rate and less than 1.00 if the NHW rate is higher than the NHB rate.

Statistical Analyses

To determine if a disparity widened or narrowed significantly between 1980 and 2005 we calculated a two-sided z-score using a bootstrap technique developed by Keppel and colleagues [11] and examined the corresponding P-value for the z-score. A P-value of < 0.05 was considered significant for all analyses. The significance of trends was tested using joinpoint analysis [12]. Each joinpoint represents a significant change in the trend, denoted as a straight line on a log scale. The overall significance was set at $P = 0.05$. No more than three joinpoints were allowed.

Results

Figure 1a presents results for the United States. As Table 1 indicates, the graph for white women contains 4 segments, the last 3 of which indicate significant declines (the first corresponds to a significant increase). For black women there is a significant upward slope for 1980–1993 and a significant downward slope after that. The black breast cancer mortality rate was 31.8 in 1980 and the NHB rate was 35.6 in 2005, a statistically significant increase ($P < 0.001$). The white rate was 32.6 in 1980 and the NHW rate was 25.8 in 2005, a statistically significant decrease ($P < 0.001$). Since 1982 the black/NHB rates have been higher than the white/NHW rates.

Figure 1b contains NHW and NHB rates for NYC. There are 3 segments for NHW women, two of which show significant declines. There was a statistically significant decrease in the NHW rates between 1980 (39.5) and 2005 (24.5) ($P < 0.001$). There was only 1 segment for NHB women and it showed no significant change over the 25 years (Table 1). The rate changed from 37.1 in 1980 to 33.5 in 2005, but the difference was not statistically significant ($P > 0.05$).

Figure 1c presents the breast cancer mortality rates for Chicago from 1980–2005. In 1980 the rates for NHB and NHW women were essentially equal at about 38. The rates remained more or less constant until the early 1990s when the NHW rate began to decline. By 2005 the NHW rate was 21.8, a decline of 42% ($P < 0.001$) while the NHB rate had increased (a non-significant amount) to 43.2.

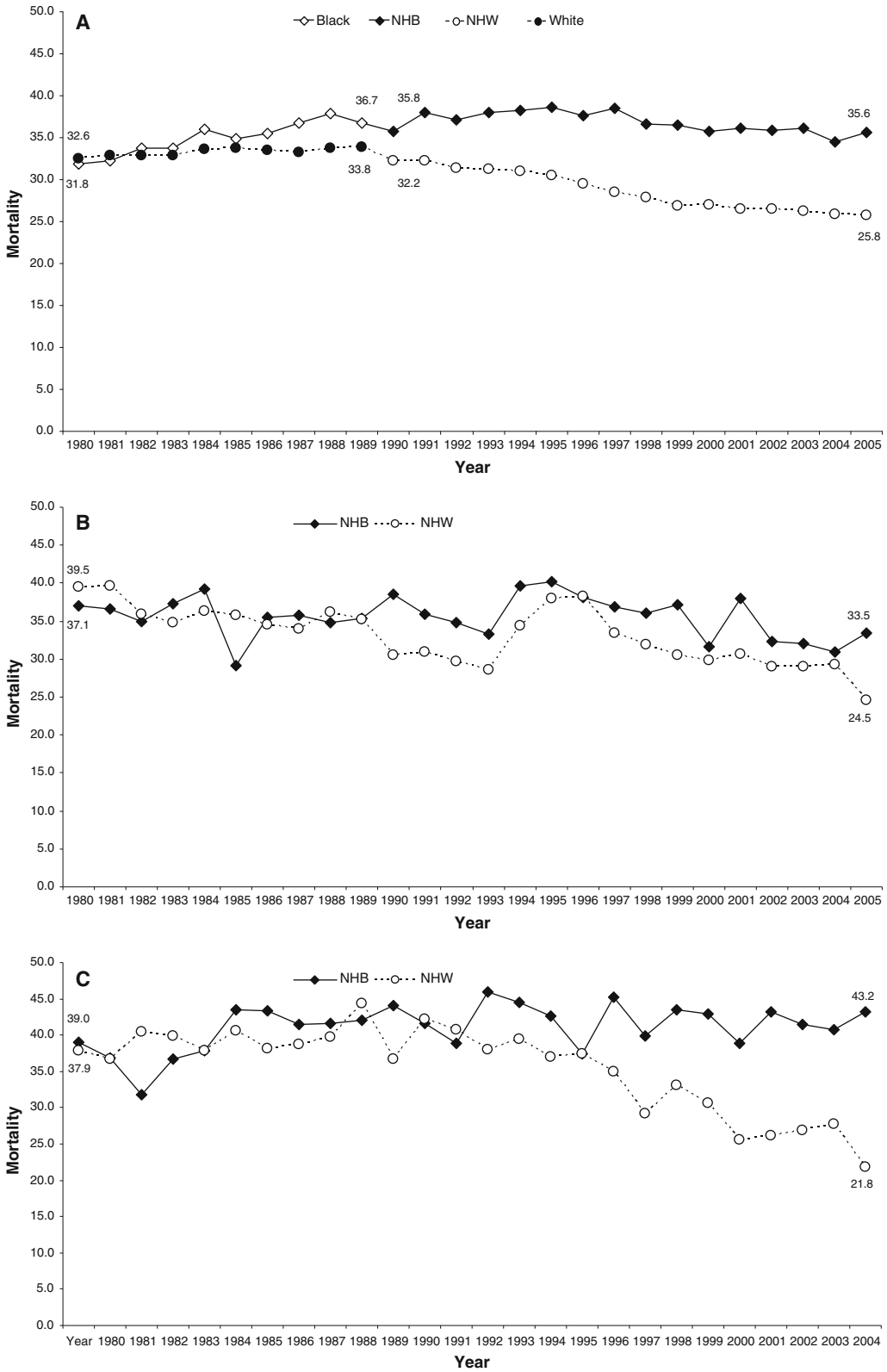

Fig. 1 Age adjusted female breast cancer mortality rates, **a** United States, By Race*, 1980-2005, **b** New York City, By Race, 1980-2005, **c** Chicago, By Race, 1980-2005. * *Open diamonds* represent Black data that includes Hispanics and *closed circles* represent White data that includes Hispanics (1980–1989). *Closed Diamonds* represents Non-Hispanic Blacks and open circles represents Non-Hispanic Whites (1990–2005)

Table 1 Joinpoint analysis of trends in breast cancer mortality rates for the United States, New York and Chicago, 1980–2005

	Segment 1			Segment 2			Segment 3			Segment 4		
Race/place	Years	APC	95%CI	Years	APC	95%CI	Years	APC	95%CI	Years	APC	95%CI
US White	1980–88	0.4	(0.0, 0.7)*	1988–95	−1.5	(−2.0, −1.0)*	1995–99	−2.9	(−4.3, −1.4)*	1999–05	−0.8	(−1.3, −0.3)*
US Black	1980–93	1.3	(0.9, 1.7)*	1993–05	−0.8	(−1.2, −0.5)*						
NYC White	1980–92	−2.2	(−2.9, −1.4)*	1992–95	6.6	(−8.3, 24.0)	1995–03	−3.3	(−4.5, −2.1)*			
NYC Black	1980–05	−0.3	(−0.7, 0.01)									
Chicago White	1980–92	0.7	(−0.2, 1.7)	1992–05	−4.1	(−5.1, −3.1)*						
Chicago Black	1980–82	−9.2	(−24.4, 9.0)	1982–85	9.4	(−8.4, 30.6)	1985–05	−0.1	(−0.05, 0.3)			

* Statistically significant annual percent change, $P < 0.05$

The associated joinpoint analysis (Table 1) locates three trend lines for NHB mortality, none of them with a significant slope, indicating that there has been no change in the breast cancer mortality rates for Black women in Chicago over the past 25 years. Table 1 also indicates a constant trend for NHW women in Chicago from 1980–1992 and a significant downward trend after that associated with an annual change of –4.1% ($P < 0.05$).

Table 2 presents selected data points from these three graphs. All three begin in 1980 with the NHB and NHW breast cancer mortality rates being approximately equal and each ends in 2005 with the NHB rate being much higher than the NHW rate. Over time, the NHB:NHW relative risk (RR) in the US increased from 0.98 to 1.38; in NYC from 0.94 to 1.36; and in Chicago from 1.03 to 1.98.

Table 2 Breast cancer mortality rates for the United States, New York City and Chicago, non-hispanic black and non-hispanic white women, selected years

Year	Location	NHB	NHW	Rate ratio (95% CI)	Change in RR**
1980*	US	31.8	32.6	0.98 (0.94–1.01)	–
1990	US	35.8	32.2	1.11 (1.08–1.15)	
2000	US	35.7	27.0	1.32 (1.28–1.36)	
2005	US	35.6	25.8	1.38 (1.34–1.42)	(<0.001)
1980	NYC	37.1	39.5	0.94 (0.82–1.08)	–
1990	NYC	38.5	30.6	1.26 (1.10–1.44)	
2000	NYC	31.6	29.8	1.06 (0.93–1.21)	
2005	NYC	33.5	24.5	1.36 (1.19–1.56)	(<0.001)
1980	Chicago	39.0	37.9	1.03 (0.85–1.25)	–
1990	Chicago	44.0	36.7	1.20 (1.00–1.44)	
2000	Chicago	42.9	30.6	1.40 (1.15–1.70)	
2005	Chicago	43.2	21.8	1.98 (1.58–2.49)	(<0.001)

* Data for 1980 for the US are for Black and White women regardless of Hispanic ethnicity

** Values in parentheses indicate whether RR in 2005 is significantly different from the one in 1980

Each of the 2005 RRs within each location is significantly different than the 1980 RR ($P < 0.001$). For the US as a whole the NHB:NHW RRs have remained rather constant in recent years (2000 and 2005) but are significantly different than 1.00 ($P < 0.001$). Changes in New York City NHB:NHW RRs have varied over time with only the RRs for 1990 and 2005 being significantly different from 1.00 ($P < 0.001$). In Chicago, the NHB:NHW RRs have increased steadily over time, with the RRs for 2000 and 2005 being significantly different than 1.00 ($P < 0.001$).

Discussion

The image portrayed by the three graphs could not be explained by biological differences. In all three locations the black and white rates were similar in the 1980s and then started to diverge, just as the benefits from early detection via mammography [13] and treatment [14] were manifesting themselves. In all cases this divergence took place because the white rate started improving and the black rate did not. For the US and Chicago the black rate is higher than it was 25 years ago. In NYC it is lower but only by a small, non-significant amount. Although there may be differences between the races in tumor biology, these explanations would be inadequate to explain why the mortality disparity has been growing rapidly in Chicago but remaining rather constant in NYC and the US. Biology also cannot explain the variability in the disparities in the three areas.

A recent article addressing this issue has reached the same conclusion based on a specific database from Louisiana [15]. Another recent article has found that for many cancers, including breast cancer, disparities in survival actually increase as "amenability to medical interventions" increase [16]. That is, as we become more able to improve cancer outcomes, racial disparities widen because more privileged groups are able to gain access to these interventions.

This is precisely what has happened with breast cancer mortality in the three geographies analyzed above.

Since differential biology can't explain these racial disparities, what might? We have been able to identify three hypotheses [2].

Differential Access to Mammography

Most surveys of self-reported mammography utilization have shown that Black and White women have equal screening rates at about the national goals [17, 18]. However, several studies of medical records and chart reviews demonstrate that self-report of mammography utilization is substantially inaccurate because many women over-report utilization [19, 20]. A recent comprehensive meta-analysis indicates that poor women, and thus black women, over-report more than other (white) women, rendering the equality of self-reported mammography use a misleading measure and leaving a substantial racial gap [21]. If mammography is an effective screening tool then differential access favoring white women would contribute to the disparity in breast cancer mortality. For example, we know that breast cancers detected by screening are smaller, less likely to be estrogen receptor negative, and less likely to be undifferentiated than unscreened cancers [22].

Differential Quality of Mammography

There are a number of different measures of mammography quality [23, 24], but we focus on just one here as an example of how such thinking might proceed. The literature suggests that for every 1,000 screening mammograms we should expect to find about 6 breast cancers. This rate of 0.006 is an average that is based on millions of mammogram exams worldwide [25–27]. The detection rate will be lower for women who are screened regularly (as low as 2 breast cancers per 1,000) and higher for women who are rarely screened (10 per 1,000) [15]. For example, the National Breast and Cervical Cancer Early Detection Program, which provides mammograms to poor women who tend not to receive regular screening, found a breast cancer detection rate of 0.0094 based upon the experiences of about 1.2 million women between 1991 and 2002 [28]. Breast cancer screening programs that find cancer detection rates well below 0.006 may suffer from quality problems.

A well-publicized example may be suggestive of our hypothesis. In October of 2002 the New York Times ran a very long front page story about a woman who obtained a mammogram at a city clinic, was told she was fine and 8 months later was diagnosed with breast cancer. The clinic that missed the cancer was investigated and found to be detecting breast cancers at a rate of only 1 per 1,000 screening mammograms. As a result, the State Department of Health offered free mammograms to women who had been seen recently by the clinic. Over 4,500 women returned and were re-screened and 25 cancers that had been missed were detected [29, 30].

Substantial information suggests that there is variation in the quality of the mammography process [31]. If this quality tended to be inferior at institutions that serve poorer women then this would contribute to the racial disparity in breast cancer mortality. Despite the logic to this argument we have been able to locate only one paper that investigated this hypothesis and it found negative results [32]. We thus discuss this topic in somewhat greater detail.

Some reports have found that radiologists who spend more time (variously defined) reading mammograms tend to find more tumors and to find them smaller [33]. However, this relationship between volume and quality is not uniformly agreed upon [34, 35]. It has also been found that breast imaging specialists find more cancers than general radiologists. For example, Sickles and his colleagues reported that specialists found breast cancers at a rate almost twice as high as general radiologists when reading screening mammograms (6.0/1,000 compared with 3.4—our calculations) [27]. Breast imagers tend to do better when seeking to resolve diagnostic mammograms as well [27, 36]. Again, if such imagers tended to work at institutions that served wealthier women, then this too would serve to increase disparities in mammography interpretation, cancer detection and ultimately mortality.

But the reading is just one part of the mammography process. Another is recalling women who have had abnormal mammograms that require follow-up. In our experience it is not uncommon for the lost to follow-up rate at community institutions for this group to be as high as 33% [37]. Reports in the literature have found similar results with varying definitions, resulting in 28% without diagnostic resolution within 6 months [38] and 16% for which a final diagnosis was not recorded [25]. Such loss to follow-up and/or an incomplete diagnostic process would also decrease the cancer detection rates found by screening mammography.

Furthermore, it has been documented that black women experience longer delays between an initial abnormal finding on a mammogram and obtaining a diagnosis [39]. There are other issues as well. For example, a recent study found that black women were twice as likely not to be notified about an abnormal result or to not correctly be able to interpret the information they received [40].

Differential Access to Quality Treatment

Multiple studies have demonstrated that Black people receive inferior medical care for almost every medical condition [41–43]. Breast cancer is no exception [39, 44,

45]. The disparities in breast cancer treatment occur for various reasons such as delays in treatment, inadequate access to adjuvant therapy, non-receipt of designated care, co-morbidities, financial barriers, etc. [46–49]. Certainly such disparate treatment would contribute to the racial disparity in breast cancer mortality.

The Biological Explanation

Taken together, these three hypotheses might be enough to explain the racial disparity in breast cancer mortality without invoking genetic etiologies. This is certainly the view of the Metropolitan Chicago Breast Cancer Task Force which has been organizing from this point of view [50]. It is nonetheless instructive to review the biological explanation. The fabric of this argument has two main threads that are sometimes presented simultaneously.

Racial Differences in Tumor Biology

Studies have revealed racial differences in diagnosed tumor size, stage, lymph node involvement, grade, estrogen receptor status, etc. [7, 51, 52]. However, in some cases these differences might be generated by the later detection of tumors in black women and thus be a function of the detection process and not the race of the women. A recent prominent study lends support to this view.

Smith-Bindman and her colleagues examined data on over 1,000,000 women. Unadjusted data revealed statically significant black:white differences in tumor size, stage, grade, and lymph node involvement. However, these observed differences "were attenuated or eliminated after the cohort was stratified by screening history." (p 541) Specifically, after mammography history was taken into account racial differences in tumor size, stage and lymph node involvement disappeared. Only tumor grade remained significant, though the differences varied in inexplicable ways (some significant, some not significant) across mammography history [19].

The Independent Predictor Explanation

Even if there were innate racial differences in breast cancer biology it would still be necessary to tie these to disparities in breast cancer mortality to complete the syllogism. This is the task that generally falls to various versions of regression analysis. In such analyses researchers gather data for breast cancer mortality, race and other variables (confounders), notably some measure of socioeconomic status. Initially racial differences in breast cancer mortality are observed. If, after "adjustment" or "correction" for these other confounders race remains statistically significant, then these researchers conclude that race is an independent (i.e., innate) risk factor for breast cancer mortality. Consider a recent example, simply one among many.

Woodward and her colleagues compared two independent cohorts (consisting of NHB and NHW women, among others) attending the same university hospital. The two cohorts were defined based upon their treatment needs. After adjustment for many biological variables black race remained an independent predictor of lower overall survival in both cohorts. The authors note that "Such differences in tumor biology, as well as previously described socioeconomic factors, likely contribute to the lower rate of survival in the AA breast cancer population." They then reflect on the following: "It is clear that, as with any cohort grouped by self-reported race, those who self-report their race as AA or black represent a genetically and culturally diverse group. *Therefore, explaining how AA race is associated with biologically more aggressive breast cancer will likely be difficult*" [6] (our italics). Similar analyses are employed by other authors [8].

Two major factors suggest that the analytical structure used in these studies is faulty. First, it is not clear that there exists a biological black or African-American race/grouping. Such a construct is heatedly debated in the medical literature [53, 54] while the social science literature has virtually unanimously agreed that this race is a social (not biological) construct [55, 56]. If such a genetic group does not exist, then of course genetics could not be responsible for the mortality disparity.

Second, the mathematical assumptions underlying this type of "independence after regression analysis" and the "problem of residual confounding" are troublesome to say the least, involving issues such as difficulties with categorization, measurement, aggregation and non-commensurate indicators, in addition to contributors to racial differences not even touched on by measures of socioeconomic status (e.g., racism) [57]. We have not been able to locate even one paper that has used this "independence after regression" model that has even noted these issues, let alone tried to explain how they might affect their findings as they cited biology as a risk factor for the mortality disparity.

The causes of the black:white disparity in breast cancer mortality is a complex matter that deserves serious analysis. We would like to make it clear that we are not suggesting that there are no racial biological differences in tumor characteristics. Biological risk factors other than tumor histology which could affect the observed mortality disparity might involve issues like obesity and estrogen status. Another possible factor involved in the racial disparity in breast cancer mortality is the use of hormone replacement therapy (HRT). It is established that white women use HRT more. In fact, since the Women's Health Initiative's report the use of HRT has declined a great deal

and following this so has the incidence of breast cancer among white (but not black) women [58]. This will decrease the breast cancer mortality rate among white women and thus increase the racial disparity.

However, we cannot see how these could generate the racial disparities in mortality that are the subject of this report, most of which have appeared recently and which vary substantially by the three analyzed geographies. Some authors discuss the possibility of a gene-environment interaction [59]. This may be a possibility but here too we cannot envision how such an interaction would produce the nature of the racial disparities delineated in this paper. Rather, we hypothesize that these disparities are a function of racial disparities in screening and treatment. Such hypotheses are, of course, subject to empirical analysis. Until empirical verification these hypotheses remain just that.

Conclusion

Racial disparities in breast cancer mortality cannot be viewed outside the context of racism in the US. Misdirected focus on biological causes places the burden of this disparity on the innate genetic characteristics of the woman and not on external modifiable factors such as access and quality. As Sankar and her colleagues have written, "Overemphasis on genetics as a major explanatory factor in health disparities could lead researchers to miss factors that contribute to disparities more substantially and may also reinforce racial stereotypes, which may contribute to disparities in the first place" [60].

In a more general sense Cooper and his colleagues have noted that: "Minority groups, particularly Blacks in the United States, are assumed to be genetically predisposed to virtually all common chronic diseases. The correlation between the use of unsupported genetic inferences and the social standing of a group is glaring evidence of bias and demonstrates how race is used both to categorize and to rank order subpopulations"[61].

It is easy for us to look back now and see the folly—and racism—of the claim that differential racial biology was responsible for the elevated prevalence of syphilis among black people in Alabama—so much so that it had to be studied as a unique entity [62]. We fear that current research claims about race, biology and breast cancer mortality similarly perpetuate racial stereotypes about disease and have the potential to harm black people still once again. This is why we must get this correct. As Brawley and Freeman have pointed out: "Deep ethical and moral questions [are raised] concerning how the research community, the American health care system, and society as a whole will move toward providing remedies for this unacceptable reality [of disparities in health]" [63]. It is our hope that the analyses presented in the current report will help move this pursuit ahead.

Acknowledgment The authors would like to thank the Avon Breast Cancer Foundation (grant #05-2010-075) for its generous support of this research and other efforts to eliminate disparities in breast cancer mortality in Chicago.

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