



Mortality from Breast Carcinoma Among US Women: The Role and Implications of Socio-Economics, Heterogeneous Insurance, Screening Mammography, and Geography

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Abstract. Despite rapid advances in medicine and beneficial lifestyle changes, the incidence and mortality rate of gynecologic carcinoma remains high worldwide. This paper presents the econometric model findings of the major drivers of breast cancer mortality among US women. The results have implications for public health policy formulation on disease incidence and the drivers of mortality risks. The research methodology is a fixed-effects GLS regression model of breast cancer mortality in US females age 25 and above, using 1990–1997 time-series data pooled across 50 US states and DC. The covariates are age, years schooled, family income, ‘screening’ mammography, insurance coverage types, race, and US census region. The regressions have strong explanatory powers. Finding education and income to be significantly and positively correlated with mortality supports the ‘life in the fast lanes’ hypothesis of Phelps. The policy of raising a woman’s education at a given income appears more beneficial than raising her income at a given education level. The relatively higher mortality rate for Blacks suggests implementing culturally appropriate set of disease prevention and health promotion programs and policies. Mortality differs across insurance types with Medicaid the worst suggesting need for program reform. Mortality is greater for women ages 25–44 years, females 40–49 years who have had screening mammography, smokers, and residents of some US states. These findings suggest imposing more effective tobacco use control policies (e.g., imposing a special tobacco tax on adult smokers), creating a more tractable screening mammography surveillance system, and designing region-specific programs to cut breast cancer mortality risks.

Keywords: breast cancer mortality, incidence, insurance types, race, socio-economic determinants

JEL code: I (health, education and welfare; health production)

1. Introduction

Despite general advances in medicine, beneficial lifestyle changes (e.g., reduced smoking and smoking cessation) and improved breast cancer management from early detection to treatment, the incidence and mortality rates from this gynecologic carcinoma continue to lead among all causes of death for US females [6]. The lifetime chance of a US woman at age 85 contracting breast cancer is roughly 10%. There are about 186,000 new cases annually and mortality is about 46,000 annually [72]. Breast cancer is the second most common cancer and the second leading cause of cancer deaths in women [76]. Environmental toxins and viruses [3], hormones (e.g., Hormone Replacement Therapies or HRTs) and genetic defects are high suspects [46]. Past research also indicate variations in breast cancer mortality due to genetics (e.g., the flawed genes BRCA1 and BRCA2), years of education, economic well-being, insurance status, access to care, and prevention, among others.

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Breast cancer risks and therefore, potential mortality also increase with age, high-risk family history of the disease, early menarche or late menopause, age at which the first child is born, never having had children, obesity, possibly estrogen HRT for post-menopausal women, and a preceding history of breast problems including mammary dysphasia [48,77]. During the late 1990s in the US, breast cancer mortality rates began to fall for the first time in many decades, but not for women 65 years or older and mortality actually rose among older black women [52].

The major goal of this study is to construct and estimate a multiple regression econometric model of the major factors driving breast cancer mortality among US women, using more recent aggregate data. This research is important because of the rising longevity and a growing stock of increasingly older, particularly susceptible females that are also the least likely to obtain timely prevention to improve survival. Moreover, many clinical trials are currently under way in the US and Europe testing new breast cancer vaccines and newer screening technologies (e.g., digital mammography and computer-assisted detection programs) other than film screen mammog-

raphy currently the imperfect gold standard. Measuring the health care outcome impacts of progress in the development and use of breast cancer vaccines and other treatment technologies necessitates new and updated baseline macro level studies.

Therefore, findings of this study are potentially useful for formulating public health policies and related research agenda on breast cancer incidence and associated markers of mortality risks in a defined population [25]. Our macro-data testing of some hypotheses that may have already been tested using disparate micro-data sets is also important. Macro-data research findings are relevant because they play unique roles in health care management economic research and public health care policy prescriptions. Justifications for conducting macro-data research such as this are many. First, there are no other known macro data studies of breast cancer mortality. Second, a macro data analysis may be useful for generalizing the wealth of microdata study findings of the past (i.e., for testing the principle of 'consistency of aggregation' in empirical econometrics). Third, macro data study findings that signal 'consistency of aggregation' of microdata results can confirm the usefulness of macro-econometric modeling methodologies for policy in this line of health care policy research. Fourth, our use of US-wide data extending to the late 1990s, and thus incorporating more fairly recent data series, adds value by capturing more and newer information on breast cancer mortality and its determinants. These, together with the additional contributions of this research, constitute major and timely strengths representing novel contributions to the important literature on factors determining breast cancer mortality among US women.

This paper proceeds as follows. Section 2 is literature review. Section 3 focuses on data and research methods. Section 4 reports the findings of a pooled data regression model. Due to sparse data, disease incidence is only incorporated as mortality driver in the cross-sectional regression model in section 5. Section 6 concludes with policy recommendations for reducing mortality and suggests an agenda for future public health studies of breast cancer mortality risks.

2. Literature review

Standard models of health production predict an improved health status with increased years of formal schooling [34]. Mortality, the reciprocal of health and a measure of health status, is both meaningful in public health and measured with sufficient precision [21,24]. Mortality is *a priori* expected to fall with years of schooling [36] and incomes. Fuchs, McClellan and Skinner [29] researched 313 US areas for variations in mortality risks from *non*-specific causes among white ages 65–84 in 1990, and confirmed the role of schooling, income, obesity, air pollution and percent black. Mortality related directly to smoking, obesity and percent black and was lowest among Florida residents.

There are competing theories of the positive correlation of schooling years and health status. Fuchs [28] reasoned that

individuals with low discount rates, or long time horizons, tend to invest both in education and health, or that schooling tends to effect a change in people's time preferences. One rationale is that a better educated, more informed population transforms health care inputs (e.g., medical, other market inputs such as information, own time) more efficiently into improved health using more innovative technologies. Auster et al. [1] econometrically tested this hypothesis using death rates across US states for 1960. Their model included education, income and other independent variables. Surprisingly, they found a positive education elasticity of mortality of +0.20. This suggests that mortality rises with education level.

Mortality production in the Medicare population [35] using 1970 US cross-section county data revealed the following 'gender-age' elasticity estimates for education: -0.128 (*statistically significant*, white males, 65+); -0.60 (black males, 65+), -0.025 (*statistically significant*, white females 65+) and -0.106 (black females, 65+). The study also reported statistically insignificant estimates of income elasticity of mortality for the respective population sub-groups, as follows: -0.020 , -0.012 , 0.00 , and -0.011 . Research on how higher living standards, a correlate of income and education, reduce mortality [27] and how the mothers' formal years of education affect neonatal mortality [8] also corroborate the positive impacts of education in health production. Heck et al.'s [37] recent study of whether highly educated women are at an elevated risk of breast cancer death found it to be highest among women with 12 and with 16 or more years of schooling for Hispanic and non-Hispanic Black and Asian women but not for non-Hispanic white women.

Disease-specific and all-causes mortality studies show that gender-specific differences occur across races, particularly for Blacks, Whites, Chinese, and Hispanics. These are attributable to many factors including, for instance the differences in physiology and variations in the biological pathways through which the disease mortalities are expressed [19]. Breast cancer mortality rates vary widely among US racial and ethnic groups, with the Hispanics, Chinese, Filipino and Japanese women having annual rates of 15 cases per 100,000 and blacks, whites and native Hawaii women with rates above 25 cases per 100,000. During the 1989–1993 period the age-adjusted breast cancer mortality rates declined about 6% in white women and rose about 1% for black females. Recent advances in cancer genetics, especially commercial testing and the Human Genome Project [38,45] identified Jewish women of Eastern European descent as particularly at an 85% elevated risk for contracting breast cancer from inheriting the defective BRAC1 and BRAC2 genes.

Flaws and Bush [23] hypothesized the increased risk of black women dying from breast cancer may relate to their inability to metabolize Tamoxifen™, a pharmaceutical agent widely used in breast cancer therapy. This drug is metabolized by the cytochrome P450 enzyme system, which white women metabolize better and so makes drug more effective for them. Black patients with breast cancer tend also to be diagnosed at a younger age but important differences per-

sist in tumor characteristics compared with whites, even after controlling for income, medical insurance status and method of tumor detection following screening mammography [15]. Race differences in breast cancer mortality are also attributed to differential risks of developing breast cancer, access to screening and early detection [12,13] treatment and follow-up and supportive. The median age at death for white breast cancer patients is 68 years and 62 years for black breast cancer patients. About 75% of racial differences in survival are due to differences in prognostic factors [16]. These, along with differences in clinical and biological prognostic factors for survival, also influence race-based mortality risks.

Contrary to diagnostic mammograms, screening mammograms are used to detect breast changes, particularly tumors that cannot be felt, in women with no signs of breast cancer [57]. Regular mammography use at recommended intervals by age-appropriate women could cut their breast cancer mortality risks by 30% [2], although radiologists are known to vary widely in their interpretations and recommendations for additional screens and biopsies [14]. The American Cancer Society guidelines stipulate annual clinical breast examination (CBE) for women 40 years and older, annual screening mammography for those 50 years and older, and a mammogram every one to two years for women 40–49 years. The US Preventive Services Task Force, however, recommends mammography every one to two years for 50–75 years old women and early screening for women with increased risk factors [50]. CBE and self-breast examinations (SBE) are specifically recommended for 40 years and older women.

Younger women ages 40–59 are most likely to take advantage of regular mammography [2,40,63], a finding largely invariant across race, income and education [2]. There is also overwhelming evidence from studies in the US, the UK, Netherlands, Sweden and Canada suggesting screening mammography's ineffectiveness for locating cancerous breast tissue tumors in younger age women whose breast tissues in general are denser and more fibrous [17,72,79]. However, screening reduced stage differentials among Black and Hispanic women in a study based on 1990 to 1998 metropolitan Colorado (US) mammography database of breast cancer incidence and tumor stage distribution [39]. In a study of white, educated middle class women, factors that differentiate between women who had undergone mammography and those who had not included age, health behavior, sense of well-being experience with breast cancer in relatives or friends, and the feeling that one can influence one's health outcome [20].

Coverage under health insurance plans effectively reduces out-of-pocket costs to patients and improves outcomes of preventive care. Results from the famed Rand Health Experiment (RHE) indicate that plans with the lower co-payment or out of pocket costs experienced greater use of health care facilities [53]. Therefore, enrollees in Health Maintenance Organizations (HMOs) obtained more preventive care than those in the relatively more costly fee-for-service plans. Since about 50% of the women in a 1989 survey reportedly would not pay \$150 per screening mammogram that required return visits [2], breast cancer mortality is hypothesized to vary directly

with a lack of insurance and inversely with the generosity and type of insurance coverage. Several microdata studies [62] using tumor registry data merged with discharge/payer records that observed insurance coverage and outcomes of incident breast cancer cases are illuminating. They found the conduit through which insurance coverage types influences breast cancer mortality includes variations in treatment modalities and stage of diagnosis, among others

Job loss or its fear may reduce access to preventive (e.g., screening) and therapeutic breast cancer care [5], the uninsured are less likely to obtain mammograms, and women in managed Medicaid received preventive gynecological care at the same rate as women in other insurance [41,65]. Currie and Gruber [9], using US Vital Statistics data on every birth for the 1987–1992 period, found treatment intensity for obstetrics care to rise among previously uninsured while falling for those switching from private insurance to Medicaid plan. This finding reinforces the hypothesis that potential variations exist in both access and (quality of care) outcomes among insurance types. Medicaid provides mammography coverage to over 3 million age-appropriate women. Griffin et al. [33] reported significant improvements in the adequacy of care utilization in a managed Medicaid. However, a lack of access to a regular physician in Medicaid, rather than insurance status of access to care, is reported as a stronger predictor of treatment outcomes [11,71]. Decker and Hempstead [10] reported HMO penetration as having positive effects on the probability of recent receipt of mammography, but no significant link of HMO penetration with the stage of breast cancer diagnosis or survival in women 55–64 years old.

More recently, the American Cancer Institute confirmed a declining trend for breast cancer mortality. Historical epidemiology contends that the major decline in mortality rates from most infectious diseases, such as respiratory tuberculosis, predated effective therapy although medical advances later speeded up the decline [19]. A similar phenomenon may operate through health promotion to account for the relative stability in incidence and reduce mortality rate over time. Geographical disparities also correlate with breast cancer mortality risks. Wells and Horn [80] demonstrated the utility of ecological variables constructed from the National Health Interview Survey, NHIS, for strategic targeting of health services for the underserved. Spatial patterns in the incidence of late-stage and in situ (or precancerous) breast cancer among white women aged 45 to 64 years been studied with a choropleth map (census tracts) to characterize areas of high versus low risk of breast cancer during the 1978–1982 period [68]. Policy targets in highest risk locations should be clearly identified to improve effectiveness of the appropriate interventions.

3. Research methodology: data and econometric modeling strategy

The data are annual observations of 50 US states and the District of Columbia for the 1990–1997 period of study. Descriptive statistics of the data are arrayed in table 1. Defi-

Table 1
Descriptive statistics, 1990–1997^a data.

Variable	Mean	Standard deviation
MORT	16.416	3.061
INCIDENC, 1989–1993	103.170	8.309
INCIDENC, 1990–1994	103.390	8.169
INC (\$000s)	21.044	3.970
EDUC	22.207	4.701
WHT	78.584	15.621
BLK	10.908	11.902
F 25–44	15.889	1.000
F 45–64	10.056	0.771
F > 65	7.477	1.279
MAM 40–49	63.595	6.428
MAM > 50	53.748	6.919
TOBA	24.769	4.010
IEMP	60.425	6.740
IMCID	10.799	3.405
IMCARE	13.035	2.287
INONE	13.983	4.142

^a Mortality and incidence data are per 100,000 of female population; all other variables are in percentages. ‘Data definitions and sources’ are arrayed in appendix, table 5.

inition of variables and the data sources are in appendix, table 5. The behavior of the regression disturbances over the cross-sectional units (i.e., states) is likely to differ from that of a given cross-sectional unit over time. Pooled data regression estimation methods include the Ordinary Least Squares (OLS) or the covariance (i.e., fixed-effects) model, and the Generalized Least Squares (GLS) or error-components (i.e., random-effects) model. The fixed-effects model, useful when cross-sectional attributes are correlated with the covariates, assumes that the cross-sectional and time-series units have specific intercepts. The attribute of a cross-section unit is a parameter in a covariance model, but is a normally distributed random variable in the error components model.

Thus, a pooled time-series and cross-sectional data (with total observations $n = T \times N$) model can be estimated based on particular specifications of the behaviors of the variance–covariance matrix of the residuals. Here, the typical assumption of cross-sectional heteroskedastic and time-wise autoregressive residuals underlie the estimation to drive our pooled data regression model:

$$\text{MORT}_{it} = \beta_1 X_{it,1} + \beta_2 X_{it,2} + \dots + \beta_\kappa X_{it,\kappa} + \xi_{it};$$

$$(i = 1, 2, \dots, \kappa = 51; t = 1990, \dots, 1997), \quad (1)$$

where MORT_{it} (breast cancer mortality) is the response, β_i 's are the regression parameters of the deterministic covariates X_i 's and ξ_{it} 's are the regression errors.

The specified model will be econometrically estimated here using the *SHAZAM* algorithm [81] based on a variant of Kmenta's [42] model in which ρ (rho) is estimated as a sample correlation coefficient between ξ_{it} and ξ_{it-1} , the estimated ρ ($-1 \leq \rho \leq 1$) differs for each cross-sectional unit, and the phi matrix is diagonal. This method yields a consistent estimator of ρ_i hence consistent estimates of the elements of variance–covariance matrix of the residuals when, as in this study, the time-series dimension of the pooled data

is rather short and N/T is *not* zero or an infinite number. The regression parameter estimates and their variances, after iterative convergence using this method, are maximum likelihood; they possess the desirable asymptotic properties. Compared to many other regression programs, the *SHAZAM* econometric program is used for estimation of the model, because it has a more efficient set of computational algorithms and outputs correct values of important summary statistics [58].

Our proposed model is a fixed-effects, GLS regression of breast cancer mortality among US females age 25 and above, using 1990–1997 time-series data pooled across 50 US states and the District of Columbia. The covariates include age (females 40–49, 50–65, and >65 years), years of formal schooling, family income, screening mammography (for 40–49 years old, for ≥ 50 years old) to capture prevention, insurance coverage types to measure potential access and quality of care, race indicators to capture racial peculiarities including biologic pathways, and US Census regions as ecological measure. Data for this research came from The Centers for Disease Control [50], the US Government [75], The American College of Radiology, National Alliance of Breast Cancer, National Cancer Institute [73,74], and others.

The inclusion of specific drivers of breast cancer mortality in this model derives from the literature in health economics and related disciplines. The hypothesized determinants include socio-economic variables such as income and education, along with insurance coverage that reduce the effective price of health care services to patients, play in health status production. Studies from public health epidemiology and other disciplines on the determinants of population health justify the inclusion of specific other variables including age, geographic location, incidence, and race dummies. These estimates yielded strong explanatory power for the model and they have policy implications, including for diagnosis and appropriate intervention, which vary by race, age, insurance types, regional location and other factors.

4. The empirical results

The pooled time-series and cross-sectional data require estimation and testing of three alternative models for specification bias if either or both dimensions are omitted. The model incorporating both time dummies (year 1990 = base) and regional dummies ('South Atlantic' Census region = base) is unrestricted. The model without time and cross-sectional dummies, and that with only time dummies, each becomes restricted. The likelihood ratio (LR) test results evaluated at the $\alpha = 0.05$ level of the critical χ^2 distribution rejected each restricted model. The generalized Box–Cox power family of transformations model was further used to test hypotheses on the adequacy of specific a priori limited functional forms against the more general model. Rejection of these a priori limited or biased functional form models occurred uniformly at the $\alpha = 0.05$ critical values of the χ^2 test distribution. (Detailed computational results are obtainable from the authors.)

Regression estimates of the full (or unrestricted) model in table 2 form the basis of research findings and policy sugges-

Table 2

GLS regression estimates, 1990–1997 pooled data. Dependent variable MORT, is age-adjusted mortality rate per 100,000 of the US female population.

Regressor	Coefficient ^a	<i>t</i> -ratio	Elasticity at means
CONSTANT	−19.8970*	−9.7240	
INC ^b	0.2870*	3.8160	0.3680
EDUC ^b	0.1820*	3.4010	0.2461
INC*EDUC	−0.0066*	−2.8120	−0.1938
WHT	0.0571*	11.4000	0.2734
BLK	0.0839*	11.0400	0.0558
F 25–44	0.5033*	4.5550	0.4872
F 45–64	0.0250	0.2301	0.0153
F > 65	1.8619*	22.6000	0.8480
MAM 40–49	0.0344*	3.8960	0.1332
MAM > 50	−0.0176*	−1.7970	−0.0575
TOBA	0.0455*	3.1760	0.0687
IEMP	0.0002	0.0147	0.0006
IMCID	0.0715*	3.9070	0.0470
IMCARE	−0.1009*	−2.9920	−0.0801
INONE	0.0443*	2.4030	0.0378
Y1991	−0.2520	−1.4310	−0.0019
Y1992	−0.6882*	−3.4530	−0.0052
Y1993	−0.9509*	−4.4600	−0.0072
Y1994	−1.3990*	−6.1090	−0.0107
Y1995	−1.3482*	−4.9890	−0.0103
Y1996	−1.7867*	−5.7650	−0.0136
Y1997	−2.1191*	−5.6270	−0.0161
NE	0.5937*	2.7100	0.0043
MA	1.9210*	7.3410	0.0069
MW	1.0205*	5.2660	0.0061
WNC	0.2674	1.1360	0.0022
ESC	−0.5548*	−3.2010	−0.0027
WSC	0.3703*	1.6790	0.0018
MO	0.8719*	3.5920	0.0083
PA	−0.2258	−0.9450	−0.0013
<i>Summary statistics</i>			
Base R^2	0.9414	Akaike information criterion	1.1250
<i>F</i> -statistic (ANOVA from μ)	202.061	Durbin–Watson Stat.	1.7759
Log likelihood footnote value	−514.052	Runs test (normal statistic)	−2.4716

^a Statistical significance at the 0.05 and 0.10 level are denoted with *, and **, respectively.

^b ‘Full’ income effect at the data means, $\partial \text{MORT} / \partial \text{INC} = 0.2870 - (0.00066 * \text{EDUC})$, is +0.1404 with the estimated *t*-ratio of 1.32. The ‘full’ education effect at the data means, $\partial \text{MORT} / \partial \text{EDUC} = 0.1820 - (0.0066 * \text{INC})$, is +0.0431 with an estimated *t*-ratio of 0.36. The estimated *t*-ratios were obtained using the variance formula in Pindyck and Rubinfeld (1991).

tions in this paper. The parameter estimates of the full model in table 2 are largely significant, and the covariates together explain roughly 94% of the variance in MORT, the response variable. The explained variance is highly significant at the $\alpha = 0.001$ level as reflected in the model’s corresponding *F*-statistic (i.e., ANOVA from mean) value of 190.355. The hypothesis of independent residuals could not be rejected by the Durbin–Watson test statistic criterion. Finally, lack of data on breast cancer ‘incidence’ for the entire 1990–1997 period of study precluded its inclusion in the pooled model estima-

tion. Nonetheless, a set of annual regressions incorporating the averaged (e.g., 1990–1994) incidence rates yields useful insights on the delayed or lagged effects of period-specific incidence rates on downstream breast cancer mortality rates.

4.1. Do income and education affect breast cancer mortality?

Table 2 indicates the independent effects of income (INC) and education (EDUC) variables as positively and significantly correlated with breast cancer mortality. This contradicts the theory relating schooling and higher living standards to a better health status, but it is highly suggestive of the possible scenarios that Phelps [60] labeled ‘life in the fast lanes’ effects in which rising society incomes comes at the expense of health hazardous industrial processes causing declines in health status. Continuous attrition of health status eventually causes fully depreciated health or mortality. Since previous studies may suffer from misspecification of the omitted variables type, we tested for the potential interaction of both variables (INC*EDUC) to strengthen or weaken the observed positive main effects of education or income on mortality. The comparatively small numerical magnitude of the INC*EDUC interaction is negative and highly statistically significant.

The full impact of INC on mortality, evaluated at the mean level of education data, becomes $\partial \text{MORT} / \partial \text{INC} = 0.2870 - (0.0066 * \text{EDUC}) = 0.1404$ (estimated *t*-ratio = 1.32) and is not significant. Similarly, the full effect of EDUC on mortality at the sample data mean income level becomes $\partial \text{MORT} / \partial \text{EDUC} = 0.182 - (0.073 * \text{INC}) = 0.043$ (estimated *t*-ratio = 0.36). That is, given higher income, education at the Bachelors degree or higher thus significantly weakens the positive main effect of higher education on breast cancer mortality. Similarly, given a higher education level a rise in per capita income (main effect) tends to weaken the strong and positive linkage of income to breast cancer mortality. However, these effects (i.e., higher income given the level of schooling versus higher education given an income level) on breast cancer mortality are asymmetrical. They suggests that in order to reduce breast cancer mortality rates, it appears comparatively more beneficial to raise schooling years or education at the average income level than the reverse.

McDonough et al. [54], after adjusting for education and initial health status, reported that income *instability* among middle-income individuals is consequential to all-cause mortality. This conclusion is echoed in Lynch et al. [49]; they attribute higher income inequality to higher mortality rates at all per capita income levels in 282 US metropolitan areas. Gerdtham and Ruhm [31] and Ruhm [64] each found that most disease-specific death rates rise during periods of economic prosperity when incomes rise, and that the time away from working (i.e., from ‘living life in fast lanes’) is re-invested in more healthy behaviors (e.g., relaxation, non-sedentary life style) during economic recessions when reduced incomes and work hours cut mortality rates. This suggests that mortality tends to rise (*fall*) during economic progression (*recession*). The recent study by Krieger [44], echoes the similar finding that breast cancer mortality is higher in

high- than in low-income nations. This means that breast cancer deaths rise with rising incomes, in both the US (our study and others) and worldwide. These recent research results, in addition to the earlier findings of Auster et al. [1], strengthen the empirical evidence consistent with 'life in the fast lanes' hypothesis in support of the positive but statistically insignificant association of breast cancer mortality with income detected in this present study.

4.2. Do race differences, female age range, and tobacco use matter?

Recent debates question the appropriateness of using race as a variable in public health and policy research [30]. Nonetheless, the regression coefficients in table 2, for the separately measured percentages of white (WHT) and black (BLK) females in the total US female population, are positive and highly significant. The BLK coefficient roughly twice that of WHT is consistent with earlier results and reinforces the need for culturally appropriate health promotion and disease prevention programs to improve awareness of breast cancer care programs [59,83]. Perhaps, more interesting are the race-population mortality elasticity estimates, computed as ratio of the percentage change in breast cancer deaths due to a given percentage change in the respective race population. The estimates of 0.247 for WHT and 0.0558 for BLK suggest that, all else equal, a 100% rise in the Black female population is on average likely to raise breast cancer deaths by 6% compared with 25% for White females.

Mortality from breast carcinoma is rare in women under 25 years of age [77]. Susceptibility to disease mortality tends to be greater for younger than older age range females and the regression estimates confirm this as significant in the 25–44 years of age group (F 25–44). While positive but statistically insignificant in the 45–64 year olds (F 45–64), it is highly significant and positive for the geriatric age (F > 65) females. Controlling for some other potentially confounding effects (e.g., insurance status, mammography, etc.) in our model, the literature hypothesized several reasons could account for the differential mortality across age groups. For example, the denser breast tissues in younger females make cancerous tumors less detectable in preventive screening or diagnosis and thus result in late medical attention and increased risk of mortality.

Unhealthy life-style habits are known to increase the probability of certain chronic illnesses [26] and potential mortality from them. Smoking, ingestion of high cholesterol foods, physical inactivity and even moderate alcohol intake have been linked to breast cancer mortality and the findings here support the assertion for tobacco use [26,48]. The regression coefficient of tobacco use among women of reproductive age range 18–44 (TOBA) is positive as expected and highly statistically significant in the breast cancer mortality model. This finding reinforces the recent evidence [82] that smoking-related heart disease and cancers caused the most deaths and account for a large portion of the socio-economic and racial disparities in health. Public health policies on smoking ces-

sation, largely in reproductive-age females, could be effective in reducing breast cancer mortality rate in sub-populations. One interesting policy lesson from the European Union (EU) countries appears instructive for the US. Raising a special tobacco tax by 10% for adult smokers in twelve EU countries translated into reducing lung cancer mortality rate by 8.81% in the long run [18]. According to the authors, this would save 1707 lives during the first year, 4491 lives in the fifth year and 12,366 lives overall after the smoking population has been completely renewed. Since tobacco use is a known major driver of breast and other forms of cancer mortality in the US, a similar policy experiment could reduce breast cancer incidence and deaths in US women.

4.3. How effective is age-specific screening mammography?

MAM 40–49 and MAM \geq 50 are separate variables in the regression model (table 2) capturing screening mammography use among women 40–49 years old and women 50 years and older. The estimates indicate positive and highly significant mortality among the 40–49 years olds who have undergone mammography. This result agrees with the pronouncements of the National Cancer Institute [56] that "randomized clinical trials have not shown a statistically significant reduction in mortality for women under age 50 associated with the use of routine mammography screening". Despite advancing age, there is a statistically significant reduction in the odds of the 50 years and older group dying of breast cancer. Since low income, Hispanic ethnicity and other race, low educational attainment, age greater than 65 [48], rural residence [47] and an absence of a regular source of care [51] predict mammography underuse [4], preventive public health care agenda should focus on alleviating burdens associated with these barriers through publicly funded health centers and recommendation of CBE and SBE as adjuvant screening devices for younger women to supplement screening mammography. This policy suggestion agrees with Figueroa and Breen [22] attributing late-stage cervical and breast cancer screening and diagnosis in residents of underclass neighborhoods to geographic-specific market failures [70].

4.4. Do types of health insurance coverage influence mortality?

Public insurance plans are Medicare (IMCARE) for older women and Medicaid (IMAD) for the indigent women. The effects of insurance status on medical treatment and the implications of insurance-induced treatment variations in health outcomes are key policy issues [9]. Regression estimates of the health insurance coverage effects on breast cancer mortality in our study reveals no statistically significant impacts between 'other insurance' (the base) and employer-sponsored plans (IEMP), significantly increased mortality under Medicaid (IMAD) and for the uninsured (INONE), and a statistically significant reduction in mortality for age-appropriate women enrolled in Medicare (IMCARE). These findings corroborate earlier studies and underscore the need for improving

the quality of Medicaid care for women and creation of effective schemes that ensures the provision of timely and age-appropriate gynecological health care to the uninsured. (Past microdata studies attribute variations in the effects of insurance types on mortality to the treatment modalities and stage of diagnosis, measures not explicitly included our study due to paucity of data.) Muntz [55] recently found that curbside consultations frequently occur in the practice of gynecologic oncologist, particularly for invasive gynecologic malignancy. Thus, there appears to be a greater need for preventive gynecological cancer care of the breast.

4.5. Time trend effects and the role of geographic location

Relative to the year 1990 dummy, and with the exception of 1991, breast cancer deaths fell consistently and statistically significantly. This effect may be ascribed to a number of factors including prevention, public health measures, improved health information, self-care and a compendium of other factors that perhaps interact. The possibility that a set of unobserved and unmeasured attributes peculiar to a geographic area is correlated with breast cancer mortality cluster is here captured with dummies in which the states are classified into their US Bureau of the Census regions, with the South Atlantic region as the base.

Compared with the base states (*DE, MD, DC, VA, WV, NC, SC, GA, FL*) only the ESC region (*KY, TN, AL, MS*) offers a significantly reduced risk of mortality and the remaining regions display fairly greater mortality tendencies. Due to location-specific differences in cancer treatment practices and public health policies affecting survival, there are geographic variations in breast cancer mortality and it is particularly higher among women 65 years and older in northeastern US than in the South or West [32]. Higher mortalities in some regions could mean higher incidence, poor survival, or both. Chandra and Skinner [7] recently tested the alternative hypothesis that the black–white health disparities and treatment outcomes to arise from geography. They found that African Americans tend to reside in areas or seek medical care in regions in which quality levels for all patients, regardless of race, are lower. Therefore, reducing geographical disparities in both the quality of care and the quality of health care decisions of patients and providers can help ameliorate racial disparities in medical treatment outcomes. This likely applies to breast cancer mortality rates as well.

5. Findings of annual cross-sectional regressions with ‘incidence rate’ as regressor

Disease incidence rate, an epidemiological measure, reflects the incidence and prevalence of a disease and is defined as the number of new cases of illness over a time period divided by the person time-at-risk [67]. Mortality is positively related to disease incidence although not all incidence results in death. Since the case fatality proportion of breast cancer is not one, incidence is a determinant of the disease mortality. Consequently, breast cancer mortality rate depends on ‘incidence’,

the number of new cases that arise in a defined population, and ‘case fatality’, the proportion of diseased individuals who die [67].

Cancer incidence data are not consistently published annually; but are available as averaged 1989–1993 data ($\mu_{\text{incidence}} = 103.17$ per 10,000 female population, $\sigma = 8.309$) and 1990–1994 data ($\mu_{\text{incidence}} = 103.39$ per 100,000 age-appropriate females, $\sigma = 8.169$). The data limitation precluded the inclusion of annual incidence rate as regressor in the panel data model. Nonetheless, the role that ‘incidence’ plays as a potential driver of breast cancer mortality is tested with cross-sectional regression model for each post-incidence year and 1989–1993 or 1990–1994 averaged incidence data. Given complete 1990–1997 data on the remaining variables, five separate annual (1993, 1994, 1995, 1996, 1997) regressions are estimated and presented in table 3 when 1989–1993 incidence data are used. Similarly, table 3 contains the regression results for 1990–1994 based on 1990–1994 incidence data. Several diagnostic tests generally indicate that the estimated regression parameters in tables 3 and 4 are econometrically solid and for each year, their covariates explained a large and significant portion of mortality variance as measured by the *adjusted R²* and the overall model *F*-statistic. Specification of the annual regression is similar to the pooled data model, except that the cross-sectional regressions tested for the distributed lag effects of ‘incidence’ on mortality. So, the ensuing discussions pertain only to this additional determinant.

Epidemiological studies of breast carcinoma typically test for five-year survivals following incidence. Wang et al. [78] studied incidence, mortality and survival of breast cancer patients in Norway from 1970 to 1993. They found age-adjusted incidence rate increased significantly in the 40 years and younger age women although the age-adjusted mortality rate remained almost unchanged and the 5-year survival rate has increased among cases with axillary lymph node metastases at the time of diagnosis. This suggests that incidence does not necessarily result in mortality within 5 years. In our paper, incidence uniformly increased death probability marginally from 1993 to 1997 but mortality was impacted statistically significantly only in 1997. This suggests a mean post-incidence survival of 7 years if 1989–1993 mortality rates are used. Contrary to this finding, using the 1990–1994 incidence rates data in the annual regressions shows significant effects on mortality for 1995 and 1996. The mean post-incidence survival is 3.5 years for the significant 1995 mortality and 5.33 years for the significant mortality that occurred in 1996, to average 4.5 years. The findings on the role incidence rates play in breast cancer deaths are illuminating but cautionary. The degree to which findings may be influenced by lack of year-specific, rather than the 5-year average, incidence rate is a testable hypothesis for future studies.

Despite the fairly similar incidence rates in the 1989–1993 and 1990–1994 periods, the surprising result that more recent 1990–1994 incidence is correlated with reduced survival rates is interesting. First, due to metastatic spread [77] perhaps arising from late detection [66] the more recently de-

Table 3
Cross-sectional annual OLS regression model estimates with averaged 1989–1993 breast cancer incidence rate as an additional regressor.

	1993		1994		1995		1996		1997	
	Coefficient	<i>t</i> -ratio	Coefficient	<i>t</i> -ratio	Coefficient	<i>t</i> -ratio	Coefficient	<i>t</i> -ratio	Coefficient	<i>t</i> -ratio
CONSTANT	-62.721	-2.939	-44.632	-1.673	13.469	0.283	-31.467	-0.912	23.330	1.737
INCIDENC	0.102	1.421	0.034	0.566	0.116	1.188	0.047	0.367	0.163	2.401 ^b
INC ^a	1.384	1.423	0.970	1.496	-0.272	-0.341	-0.607	-0.744	0.807	1.528
EDUC ^a	1.810	2.218	1.222	1.668	-0.240	-0.281	0.164	0.181	0.236	0.547
INC*EDUC	-0.073	-2.122	-0.052	-1.809	0.013	0.397	0.014	0.387	-0.016	-0.873
WHT	0.099	2.965	0.032	1.034	0.036	0.570	-0.018	-0.519	0.050	1.967
BLK	0.103	0.656	0.248	4.572	0.103	0.833	-0.006	-0.029	0.099	1.542
F 25–44	-0.695	-0.753	-0.016	-0.037	-0.375	-0.385	1.026	0.619	-1.207	-3.022
F 45–64	4.156	2.974	0.519	0.489	-0.961	-0.513	-0.495	-0.617	-1.017	-2.664
F > 65	0.181	0.162	1.938	6.775	0.244	0.360	0.480	0.546	1.264	3.276
MAM 40–49	-0.175	-1.165	0.164	3.213	0.124	1.051	0.003	0.017	0.184	4.392
MAM > 50	0.058	0.586	-0.027	-0.418	0.025	0.324	-0.170	-1.326	-0.244	-2.444
TOBA	0.037	0.371	-0.004	-0.032	0.211	0.947	0.259	1.251	0.257	4.550
IEMP	0.018	0.139	-0.017	-0.111	-0.180	-0.605	0.172	0.997	-0.219	-1.753
IMCID	0.115	0.400	-0.230	-1.692	0.138	0.596	0.292	1.503	0.222	2.700
IMCARE	-0.543	-1.292	-0.145	-1.311	0.199	1.008	1.308	3.046	-0.807	-2.524
INONE	0.390	2.030	0.202	1.111	-0.111	-0.256	0.281	0.997	-0.288	-1.597
NE	5.749	2.678	5.820	4.880	-0.032	-0.011	-1.784	-0.401	-1.969	-1.434
MA	3.355	1.847	6.948	7.531	3.275	1.270	0.761	0.454	-2.040	-1.022
MW	1.421	0.649	5.126	5.347	1.771	1.096	1.613	1.017	-3.284	-1.943
WNC	2.208	0.991	4.981	4.914	0.756	0.346	3.624	1.126	-7.348	-2.700
ESC	-1.133	-0.270	6.196	5.282	-0.450	-0.214	-3.738	-1.340	-3.716	-3.433
WSC	-2.161	-1.172	1.749	1.484	-2.179	-0.925	0.417	0.135	-0.005	-0.004
MO	-2.379	-0.734	3.509	3.395	-0.394	-0.174	-0.215	-0.084	-3.489	-1.989
PA	0.299	0.073	4.343	3.090	-4.146	-1.098	-0.557	-0.155	-3.997	-1.368

Summary statistics

Adjusted R^2	0.930	0.983	0.900	0.926	0.975
F -statistic	16.001	65.877	11.133	15.004	45.977
D - W test	1.213	1.804	2.275	2.324	2.069
Runs test	-0.747	-1.134	0.506	2.351	0.028

^a The 'full' effect of income on mortality, $\partial \text{MORT} / \partial \text{INC} = \beta_1 + (\beta_3 * \text{EDUC})$, evaluated at data means are, respectively, -0.237, -0.185, 0.0017, -0.296, 0.452. The estimated t -ratios, using the variance formula in Pindyck and Rubinfeld [61] for standard errors, are -0.164, -0.168, 0.012, -0.217, 0.574, respectively, for the years 1993–1997. The 'full' education effect, $\partial \text{MORT} / \partial \text{EDUC} = \beta_2 + (\beta_3 * \text{INC})$, are 0.274, 0.128, 0.034, 0.459, -0.101 with t -ratios of 0.156, 0.086, 0.019, 0.250, -0.017, respectively, for the years 1993–1997.

^b Indicates the cross-sectional 'incidence' effects on mortality is statistically significant at the 0.10 level or better.

tected lobular or ductal malignant cancer tumors may be at the more fatal, advanced stages III and IV. Second, there may be more virulent co-morbidities associated with more recent breast cancer incidence. Policy suggestions could include reducing the start age of screening from the level currently recommended for 'early detection', a more effective intervention, and an improved understanding of the highly complex precursors and effects of breast cancer co-morbidities, including the rising epidemics of obesity and diabetes in defined sub-populations.

6. Summary conclusion and policy recommendations

Breast cancer mortality of US women from 1990 to 1997 was investigated in this paper, drawing from economic and epidemiological literature on the determinants of subpopulation health status. The regression estimates yielded many useful insights for policy. There is a significant linkage of mortality to incidence rate, with tendency for reduced survivals from more recent incidence. Socio-economic factors, e.g., education and income with interaction effects, raise mor-

tality rates but not significantly in the estimated annual and pooled data models. Third, race matters, as the mortality rate of African-American women is twice the Caucasians – statistically significant result with both meaningful policy implications and clinical relevance [43]. Racial disparities in access to care, timeliness of treatment, and differences in treatment intensities if racial access is similar, are important mechanism through which differential mortality might operate. Choice-based lifestyle habits, such as, tobacco use, significantly elevate cancer mortality risks. The age range within which women received screening mammograms is an important determinant of mortality, and US geographic location controlling for unmeasured ecological attributes does matter for breast cancer mortality.

Finally, and perhaps most importantly, insurance status and the types of plan are strong and statistically significant determinants of the probability of breast cancer deaths. Women who are uninsured or enrolled in Medicaid managed care insurance, particularly fare worse on mortality outcomes. Past studies based on microdata have attributed this to the treatment modalities and stage of diagnosis, which vary across

Table 4
Cross-sectional annual OLS regression model estimates with 1990–1994 averaged breast cancer incidence rates as an additional regressor variable.

	1994		1995		1996		1997	
	Coefficient	<i>t</i> -ratio	Coefficient	<i>t</i> -ratio	Coefficient	<i>t</i> -ratio	Coefficient	<i>t</i> -ratio
CONSTANT	−30.593	−1.463	−40.384	−2.213	−37.419	−2.181	34.129	1.262
INCIDENC	−0.008	−0.243	0.096 ^b	2.416	0.082	1.787	0.053	0.761 ^b
INC ^a	0.053	0.129	0.850	2.103	0.288	0.736	−0.286	−0.363
EDUC ^a	0.260	0.527	0.717	1.764	0.951	2.206	−0.645	−0.754
INC*EDUC	−0.012	−0.649	−0.028	−1.747	−0.021	−1.367	0.020	0.687
WHT	0.046	2.144	0.074	3.061	−0.016	−0.653	0.050	2.028
BLK	0.143	2.425	0.069	1.538	0.097	2.201	0.086	1.303
F 25–44	0.785	1.229	−0.173	−0.324	0.273	0.402	0.045	0.051
F 45–64	0.527	0.657	0.585	0.813	−0.354	−0.606	−0.734	−0.964
F > 65	2.119	5.946	1.131	2.945	0.852	1.670	1.036	1.796
MAM 40–49	0.129	2.713	0.064	1.363	−0.027	−0.361	0.061	1.085
MAM > 50	−0.047	−0.749	−0.059	−1.716	−0.150	−3.269	−0.097	−0.762
TOBA	−0.063	−0.720	−0.094	−1.004	0.239	2.506	0.157	1.833
IEMP	0.040	0.275	0.026	0.218	0.120	1.041	−0.202	−1.077
IMCID	−0.098	−0.768	0.341	3.063	0.312	3.261	0.036	0.198
IMCARE	−0.146	−1.026	0.080	0.582	0.791	3.395	−0.343	−1.229
INONE	0.171	0.809	0.198	1.207	0.231	1.361	−0.210	−0.907
NE	3.105	2.158	0.505	0.332	1.417	0.964	−1.404	−0.675
MA	5.048	5.186	0.090	0.098	1.524	1.901	0.721	0.449
MW	3.295	3.255	0.389	0.418	0.906	1.051	−1.502	−1.044
WNC	2.773	2.108	0.169	0.141	3.183	2.289	−3.607	−1.768
ESC	2.561	2.660	0.378	0.362	−1.376	−1.332	−3.212	−1.921
WSC	1.233	1.171	−0.159	−0.139	−0.809	−0.649	−1.345	−0.777
MO	1.760	2.126	−0.553	−0.537	−0.009	−0.008	−2.020	−1.136
PA	2.759	1.768	−2.265	−1.498	−0.715	−0.487	−3.057	−1.335

Summary statistics

Adjusted R^2	0.965	0.934	0.941	0.887
<i>F</i> -statistic	37.695	19.986	22.246	11.486
<i>D</i> – <i>W</i> test	1.980	1.699	1.676	2.334
Runs test	0.941	−1.914	0.182	1.598

^a The ‘full’ effects of income on mortality rates, $\partial \text{MORT} / \partial \text{INC} = \beta_1 + (\beta_3 * \text{EDUC})$, evaluated at mean education level are: −0.213, 0.228, −0.178, 0.158. Their estimated *t*-ratios, using the variance–covariance formula in Pindyck and Rubinfeld [61] for standard errors, are: −0.299, 0.356, −0.287, 0.129 for the years 1994–1997. The ‘full’ effects of education on mortality rates, $\partial \text{MORT} / \partial \text{EDUC} = \beta_2 + (\beta_3 * \text{INC})$, evaluated at the mean income levels, are: 0.007, 0.128, 0.509, −0.224 with *t*-ratios of 0.008, 0.154, 0.617, −0.140, respectively, for the years 1994–1997.

^b Indicates the cross-sectional ‘Incidence’ effects on mortality is statistically significant at the 0.05 level or better.

insurance types, and not explicitly included in this current study due to data paucity. Policy implications of the study findings have been integrated into the discussions of the research results. Suggestions for future studies include the use of more recent data series and hypotheses tests based on psychodynamics – e.g., the role of religion and family or social functioning and support systems, particularly among the community dwelling elderly women – on survival odds or disease mortality, e.g., breast cancer. Screening mammography, a useful technology, has limitations and there are wide variances in interpretation of the results of film screen impressions. Consequently, public health research efforts should investigate adjuvant routine preventive strategies, including the simultaneous use of screening mammography and ultrasound for detecting tissue anomalies (e.g., calcification in breast ducts) in younger women with denser breast tissues. Socio-demographic variations exist in breast cancer screening among women [69], and the absence of effective screening has been shown in this current study to raise the odds of breast cancer mortality. Therefore, developing more effective protocols for raising tractable participation in screening mammog-

raphy and implementing regimens for improving the lifestyle choice habits of indigent rural and urban women, uninsured or on Medicaid insurance, should be implemented.

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Appendix

Table 5
Definition of variables and data sources.

Data		Definition	Source
Breast cancer mortality	MORT	Breast cancer mortality rate per 100,000 female population, age-adjusted to the 1970 US standard population	National Center for Chronic Disease Prevention and Health Promotion, Behavioral Risk Factor Surveillance System (BRFSS)
Incidence	INCIDENC	Breast cancer incidence rate per 100,000 female population, age-adjusted to the 1970 US standard population	National Cancer Institute, Surveillance, Epidemiology and End Results Program
Education	EDUC	% of female population with a Bachelors degree or higher	US Bureau of Census
Income	INC	Per capita income (in \$000)	US Bureau of Economic Analysis
<i>Race</i>			
White	WHT	% of white female in the total female population	US Bureau of Census
Black	BLK	% of black female in the total female population	US Bureau of Census
<i>Age category</i>			
Female ages between 25 and 44	F 25–44	% of female in the total female population between the ages 25–44	US Bureau of Census
Female ages between 45 and 64	F 45–64	% of female in the total female population between the ages 45–64	US Bureau of Census
Female older than 65	F > 65	% of female in the total female population older than 65	US Bureau of Census
<i>Screening mammography</i>			
Female ages between 40 and 49	MAM 40–49	% of women 40–49 who had mammogram within last two years	Center for Disease Control
Female older than 50	MAM > 50	% of women older than 50 who had mammogram within the last year	Center for Disease Control
Tobacco use	TOBA	% of women of reproductive age 18–44	National Center for Chronic Disease Prevention and Health Promotion, BRFSS
<i>Insurance type</i>			
Employment based	IEMP	% of population with employment based health insurance coverage	US Bureau of Census
Medicaid	IMAID	% of population in Medicaid program	US Bureau of Census
Medicare	IMCARE	% of population in Medicare program	US Bureau of Census
Uninsured	INONE	% of population without health insurance coverage	US Bureau of Census
<i>US Census Bureau Geographic Divisions</i>			
Northeast	NE	New England, Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut	US Bureau of Census
Middle Atlantic	MA	New York, New Jersey, Pennsylvania	US Bureau of Census
Midwest	MW	Ohio, Indiana, Illinois, Michigan, Wisconsin	US Bureau of Census
West North Central	WNC	Minnesota, Iowa, Missouri, North Dakota, South Dakota, Nebraska, Kansas	US Bureau of Census
South Atlantic	SA ^a	Delaware, Maryland, District of Columbia, Virginia, West Virginia, North Carolina, South Carolina, Georgia, Florida	US Bureau of Census
East South Central	ESC	Kentucky, Tennessee, Alabama, Mississippi	US Bureau of Census
West South Central	WSC	Arkansas, Louisiana, Oklahoma, Texas	US Bureau of Census
Mountain	MO	Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada	US Bureau of Census
Pacific	PA	Washington, Oregon, California, Alaska, Hawaii	US Bureau of Census

^a Used as the “base region” (dummy) in the regression models that include regional controls.

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