Effect of Race in Female-specific Cancer Mortality

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Abstract

INTRODUCTION: In 2020, cancer was the second leading cause of death in the US, claiming more than 600,000 lives (Schwartz, 2024). Globally, the highest incidence in women is cancer of the breast and cervix (Schwartz, 2024), and more broadly, cancers of the female genital tract - endometrial, cervical and ovarian - are among the leading causes of death in women (Parazzini et al., 1997). Racial residential segregation continues to threaten equal healthcare access (Williams and Collins, 2001), and geographic location plays an important role in these disparities (Lahiri and Pulungan, 2007 and Do and Frank, 2020). In light of recent research highlighting disparities for gender (Kent et al., 2012) and racial minorities (Chandran and Schulman, 2022), a deeper investigation of women's healthcare is of interest.

OBJECTIVE: This study aims to examine racial disparities in mortality rates for female-specific cancers. Specifically, it seeks to answer the following questions:

1. Is there an association between race and mortality for female-specific cancers?
2. Do Counties differ in mortality, after adjusting for population age structure?
3. Does the association between race and mortality vary across Counties? Is there variability in mortality between Counties?

METHODS: This study analysed a sample of 14938 subjects from the New York Hospital Inpatient Discharge Dataset (2023-2024), coming from 40 Counties with an average size of 373.2 patients ($SD = 638.02$). To answer question 1, associations between race (Black/African American, White, or Other) and mortality were measured using chi-square tests and odds ratios, both univariate and multivariate. To answer question 2, both direct and indirect standardisation were applied. Direct age-standardised mortality rates were computed using the overall age structure of the study population. Indirect standardisation was performed using risk-adjusted rates (RAR) derived from observed-to-expected mortality ratios based on a global logistic regression model. County-level comparisons were further explored using funnel plots. Questions 3 were assessed via multilevel logistic regression models. Comparisons were conducted using likelihood ratio tests and the AIC and BIC statistics. Model discrimination was evaluated using the area under the receiver operating characteristic curve (AUC), with the optimal classification threshold determined using the Youden index. Model robustness was also investigated through bootstrap estimation and cross-validation (CV).

RESULTS: An association between race and mortality for female-specific cancers is present ($p < 0.001$). Belonging to the Black/African American race is increasing the risk of mortality compared to White ($OR = 1.57$), as well as belonging to Other races ($OR = 1.41$). When considering other known risk factors (age, severity and risk at admission), these relative risks change to 1.47 and 1.94, respectively. These results are statistically significant for $\alpha = 0.05$. Direct age-standardised analyses revealed substantial variability in mortality across Counties after adjusting for age structure, with standardised mortality rates ranging from 0% to 31.27% (SD = 6.72). Indirect standardisation confirmed geographic variability across Counties, with risk-adjusted mortality rates showing similar dispersion (SD = 7.10), while estimates for smaller Counties remained less stable. After considering between-County variability with a random intercepts and slopes model, the association between race and mortality remains. For Black patients, the average conditional odds across counties are significantly lower ($OR = 0.71$) than for White people, while belonging to Other Races still acts as an average risk factor ($OR = 1.50$). However, these effects vary substantially across Counties ($MOR = 2.44$).

CONCLUSIONS: This study shows how race is significantly associated with mortality for female-specific cancers. These findings remain after adjusting for patient age, risk, severity, and County, confirming that observed racial differences persist. Furthermore, significant variability between counties plays a role in how this association manifests, suggesting that geographical context, such as local healthcare, has a significant impact on racial disparities.

Introduction

Over 3 million cancer cases occurred in 2020, with almost two-thirds accounted for by diagnosis in women (Vaccarella et al., 2021). Breast and cervical cancer, specifically, account for a substantial burden of cancer (Schwartz, 2024), which disproportionately affects women.

Although laws passed between 1954 and 1968 abolished unequal treatment in the US, structural sexism and racism persist, leading to unequal health status (Yearby, 2018). Widening socioeconomic disparities exacerbate the differences in healthcare access and outcomes (Dickman et al., 2017), especially in cancer treatment from diagnosis to care delivery (Loehrer et al., 2024).

As women compose 70% of the healthcare workforce (Vaccarella et al., 2021) and are the primary unpaid caregivers in most households, empowering all women to be proactive in their health is of primary importance. Moreover, encouraging policymakers to take into account these systemic inequalities in both research and clinical settings is fundamental to providing brighter outcomes worldwide.

Objectives

In an effort to investigate the racial disparities in mortality rates for female-specific cancers, this study seeks to answer the following questions:

1. Is there an association between race and mortality for female-specific cancers?
2. Do Counties differ in mortality, after adjusting for population age structure?
3. Does the association between race and mortality vary across Counties? Is there variability in mortality between Counties?

Answering these research questions allows for a deeper understanding of the healthcare inequality among women, possibly leading to more information about the underlying phenomena that result in mortality differences.

Materials and Methods

The data for this study comes from the union of the 2023 and 2024 New York Hospital Inpatient Discharge Datasets, provided by the New York State Department of Health. Records from the 2023 and 2024 datasets were merged after harmonising variable names and coding schemes across years. In particular, geographic information was checked for consistency across datasets.

After the merging of the two datasets, 14976 cases were identified using the Clinical Classifications Software Refined (CCSR) diagnosis categories corresponding to breast cancer and female reproductive system cancers. These included malignant neoplasms of the breast, ovary, cervix, endometrium, uterus, vulva, vagina and fallopian tube, as well as other specified female reproductive system cancers. Only primary malignant cancer diagnoses were considered, excluding secondary neoplasms, benign tumours and treatment-related complications.

The outcome variable was hospital mortality, defined as death occurring during the hospital admission, and County was used as the clustering variable for the multilevel analysis. Several variables were recoded to improve comparability and model stability. Age was grouped into four classes, with the original categories 0–17 and 18–29 merged into a single 0–29 group, as no deaths were observed in the youngest class. Race was analysed both in its original three-level form (White, Black/African American, Other) and, when required for descriptive comparisons, in a binary White vs Non-White form. The All Patient-Related (APR) severity of illness and the APR risk of mortality variables were treated as ordered categories ranging from Minor to Extreme.

From an initial dataset of 14976 patients from 53 Counties, Counties with a population below the 25th percentile of the County size distribution (corresponding to 6 patients) were excluded in an effort to improve findings consistency, model estimations, and to reduce instability in standardised estimates. This resulted in a final analytical sample of 14938 patients from 40 Counties.

All analyses were performed using the `R` software in version 4.5.2 (https://cran.r-project.org/src/base/R-4). The level of significance α was set to 0.05.

Descriptive analysis was performed both univariately and bivariately. As the investigation of the association between race and mortality is of interest for Question 1, the chi-square statistic was employed:

$$X^2 = \sum_i \frac{(O_i - E_i)^2}{E_i}, \tag{1}$$

where i is the identifier for a specific cell in a $r \times c$ two-way table, and the resulting statistic follows a $\chi^2_{(r-1)(c-1)}$ distribution. To estimate odds ratios (ORs), logistic modelisation with the canonical link function was used, following the formula:

$$\text{logit}(P(\text{Dead}_j)) = \beta_0 + \beta_1 \cdot \text{Race}_j, \tag{2}$$

where the variable Dead_j can be 1 in case of patient j presenting an expired state or 0 otherwise, and Race_j was a factor variable with baseline level being White. Race-specific odds ratios were then estimated as $OR = \exp(\beta_1)$. Analogous procedures were followed to estimate ORs for the other relevant variables that act as known outcome predictors.

Age-adjusted comparisons between racial groups were further explored through indirect standardisation using the Standardised Mortality Ratio (SMR), defined as:

$$SMR = \frac{O}{E} = \frac{\sum_i O_i}{\sum_i r_i N_i} \tag{3}$$

where O_i denotes the observed number of deaths in age group i, r_i is the age-specific mortality rate of the reference population (White patients), and N_i represents the number of individuals in age group i in the study population. The expected number of deaths E is obtained by applying the reference age-specific rates to the age distribution of the study population.

To address Question 2, mortality differences across Counties were first investigated through age standardisation. Direct standardisation was performed by computing age-specific mortality rates within each County and applying them to the overall age distribution of the study population, used as the standard population.

$$SR_i = \sum_a w_a r_{ia} \tag{4}$$

where r_{ia} represents the age-specific mortality rate for age group a in County i, and w_a represents the proportion of individuals in age group a in the standard population. The resulting quantity SR_i is the directly age-standardised mortality rate for County i. This procedure yielded age-standardised County-level mortality rates, allowing comparison across Counties after adjusting for differences in age structure.

Because direct standardisation may produce unstable estimates in the presence of small cluster sizes, indirect standardisation was also performed. Expected deaths for each County were estimated from a global logistic regression model including age class as a predictor. County-level observed-to-expected ratios were then used to derive risk-adjusted mortality rates (RAR).

$$RAR_i = \frac{O_i}{E_i} \cdot \bar{Y} \tag{5}$$

where O_i denotes the observed number of deaths in County i, E_i represents the expected number of deaths estimated from the global model, and $\bar{Y}$ is the overall mortality rate in the study population. The resulting RAR_i represents the risk-adjusted mortality rate for County i.

To further explore geographic variability in mortality, funnel plots were constructed by plotting County-level RAR values against the number of patients in each County. Control limits were derived assuming binomial variability around the overall mortality rate and represent the expected range of random variation.

To answer Question 3, more advanced modelling techniques were required. The (generalised) random linear models were of primary interest. 10 patients were discarded when computing such models, as they did not have any hospital County recorded. The first model fitted was the random intercept logistic model:

$$\text{logit}(P(\text{Dead}_{ij})) = \beta_0 + \gamma_{0i},\ \gamma_{0i} \sim N(0, \tau_0^2) \tag{6}$$

where i indicates the County (cluster) to which patient j belongs. The random intercept γ_{0i} represents the deviation of County i, while its variance τ_0^2 is the between-County variance. This model was then compared to the one containing also the variables of interest:

$$\text{logit}(P(\text{Dead}_{ij})) = \beta_0 + \beta_1 \cdot \text{Race}_j + \beta_2 \cdot \text{AgeClass}_j + \beta_3 \cdot \text{Severity}_j + \beta_4 \cdot \text{Risk}_j + \gamma_{0i}. \quad (7)$$

Lastly, an additional model considering random slopes was also considered:

$$\text{logit}(P(\text{Dead}_{ij})) = \beta_0 + \beta_1 \cdot \text{Race}_j + \beta_2 \cdot \text{AgeClass}_j + \beta_3 \cdot \text{Severity}_j + \beta_4 \cdot \text{Risk}_j + \gamma_{0i} + \gamma_{1i} \cdot \text{Race}_{ij}. \quad (8)$$

Here, the random component is a vector where $\begin{pmatrix}\gamma_{0i}\\ \gamma_{1i}\end{pmatrix} \sim N(\begin{pmatrix}0\\0\end{pmatrix}, \begin{bmatrix}\tau_0^2 & \tau_{01}\\ \tau_{01} & \tau_1^2\end{bmatrix})$.

Odds ratios for race could be estimated similarly as described above, paying attention to the fact that in the latter model their variation depends on the County variability as described by τ_1^2. The Median Odds Ratio (MOR) was also considered, as it translates such variability in the OR scale:

$$MOR = \exp(\sqrt{2\tau_0^2 \cdot \Phi^{-1}(0.75)}). \quad (9)$$

It describes the median variation in OR when moving to a higher risk cluster, when comparing two virtually identical patients. To summarise the effect of clustering, the Intra-Class Correlation (ICC) index was used:

$$ICC = \frac{\tau_0^2}{\tau_0^2 + \pi^2/3}, \quad (10)$$

where τ_0^2 represents the variance of the random intercept in the mixed-effect models. The ICC represents the portion of latent variance that is attributable to County differences. In more complex models, such as the one in Equation 8, such an indicator represents the baseline effect of the clustering, which ignores the contribution of random slopes.

Due to the highly varying cluster size and event occurrence, Generalised Estimating Equations (GEE) modelisation showed numerical instability and could not be performed with reliable results.

The described models were then compared using the likelihood ratio test (LRT):

$$LRT = -2(l_1 - l_2), \quad (11)$$

which compares the likelihoods of two nested models with p_1 and p_2 parameters, and follows a $\chi^2_{p_1-p_2}$ distribution. If significant, it indicates that the more complex model is a better fit. In addition, the Akaike information criterion (AIC) and the Bayesian information criterion (BIC) were employed, respectively:

$$AIC = -2l + 2 \cdot p, \text{ and } BIC = -2l + \log(N) \cdot p. \quad (12)$$

These allow for comparison of models that are not necessarily nested. The one which presents lower values for these statistics is considered better at the parsimony-fitting tradeoff.

The models were also compared in terms of discriminatory power through the area under the ROC curve (AUC), which for every possible cutoff point shows on the x axis the $1 - Specificity$ (False Positive Rate), and on the y axis the $Sensitivity$ (True Positive Rate). The diagonal in the diagram represents a method which gives random guesses, while a perfect method would be as close as possible to the top left corner of the diagram. The optimal cutoff was determined using the maximisation of the Youden index, namely $Specificity + Sensitivity - 1$.

The robustness of the best-performing model was assessed using bootstrap estimation (on 200 repetitions) of coefficients and MOR, and using a 10-fold cross-validation (CV) for the AUC value.

Results

Among the selected 14938 patients, 47.28% were White, 21.07% were Black, and 31.65% were Mixed or Other. 873 of all admitted patients (5.8%) had death as the outcome. The data was collected in 40 Counties with an average size of 373.2 patients ($SD = 638.02$), indicating high County-level variability.

Significant association between female-specific cancer mortality and race was highlighted ($X^2 =$ 31.09, p<0.001), leading to crude odds ratios of 1.57 (CI 1.32, 1.87) for black patients compared to

white and 1.41 (CI 1.20, 1.65) for other races compared to white. This indicates that, compared to white people, patients belonging to other races have higher odds of dying following hospital admission for female-specific cancers. 78.81% of patients are aged 50 or more, and they also account for 90.95% of deaths. This is a typical pattern of non-communicable diseases, such as cancer, where patient age plays a crucial role in disease presence and outcome. The association between death and age class is therefore significant ($p<0.001$), where moving from one class to the next leads to an increase in odds ratios of 2.05 (CI 1.85, 2.28). Severity at admission and risk are also associated with mortality (both with $p<0.001$), and the increase of their level leads to significant increases in odds ratios, respectively of 3.43 (CI 3.15, 3.75) and 3.77 (CI 3.47, 4.10). When considering these variables in a logistic model, the odds ratio for Black people becomes 1.47 (CI 1.22, 1.76), while the one for race Mixed or Other becomes 1.94 (CI 1.63, 2.30).

The distribution of clinical severity and risk at admission was similar across racial groups. Mortality differences between White and Non-White patients therefore, do not appear to be explained by worse clinical conditions at admission. Age-adjusted comparisons between racial groups further confirmed this pattern. When applying the age-specific mortality rates of White patients as the reference population, Non-White patients showed an excess mortality with a standardised mortality ratio (SMR) of 1.61. This indicates that Non-White patients experience approximately 61% more deaths than expected if they had the same age-specific mortality rates as White patients.

Direct age-standardised analyses revealed substantial variability in mortality across Counties after adjusting for age structure, with standardised mortality rates ranging from 0% to 31.27% ($SD = 6.72$).

County	**N**	**CR (%)**	**SR (%)**	**Lower95**	**Upper95**
Manhattan	2247	1.34	1.48	0.98	1.98
New York	2207	1.90	2.02	1.43	2.60
Queens	1742	1.95	1.93	1.29	2.58
Bronx	1567	25.65	24.18	22.06	26.30
Kings	1394	10.62	10.32	8.72	11.91
Nassau	1104	2.17	2.28	1.40	3.16
Suffolk	1024	1.86	1.70	0.91	2.49
Westchester	621	3.38	3.33	1.92	4.74
Erie	519	5.97	5.71	3.71	7.71
Monroe	487	3.29	3.30	1.72	4.89
Albany	434	2.76	2.67	1.16	4.19
Onondaga	332	6.63	6.61	3.94	9.29
Richmond	234	5.56	5.45	2.54	8.36
Dutchess	186	1.61	1.35	0.00	3.01
Rockland	108	4.63	4.46	0.57	8.35
Orange	102	8.82	8.79	3.29	14.28
Saratoga	95	4.21	4.12	0.12	8.12
Oneida	77	3.90	3.28	-0.70	7.27
Warren	62	3.23	2.68	-1.34	6.70
Broome	58	10.34	10.42	2.56	18.28

Table 1: Crude mortality rates (CR) and directly age-standardised mortality rates (SR) for the fifteen Counties with the largest number of patients.

Table 1 reports crude mortality rates (CR) and directly age-standardised mortality rates (SR) for the twenty Counties with the largest number of patients, using the overall study population age distribution as the standard population. Substantial variability in mortality persists even after adjusting for differences in age structure. In particular, some large Counties such as Bronx and Kings show markedly higher standardised mortality rates compared to other urban Counties such as Manhattan, Queens and New York. As expected, the stability of the estimated rates is strongly influenced by the number of patients observed in each County. Counties with smaller sample sizes show substantially wider confidence intervals and greater variability in the standardised rates. This pattern reflects the well-known small numbers problem in geographic comparisons of health outcomes, where estimates based on limited observations may fluctuate considerably due to random variation. For this reason, caution is required when interpreting estimates for Counties with limited patient populations. In general we can observe that SR and CR are similar for the same County, this result suggests that geographic variability cannot be explained solely by differences in population age composition.

Indirect standardisation confirmed geographic variability across Counties, with risk-adjusted mortality rates showing similar dispersion ($SD = 7.10$). As expected, estimates for Counties with smaller

patient populations appeared less stable. The similarity between the variability observed in the directly standardised rates and in the risk-adjusted rates obtained through indirect standardisation suggests consistent evidence of geographic heterogeneity in mortality across Counties.

To further investigate geographic variability in mortality, indirectly standardised County-level rates were visualised through funnel plots. This graphical approach allows comparison of County mortality while accounting for differences in patient volume. Figure 1 displays the funnel plot for County-level

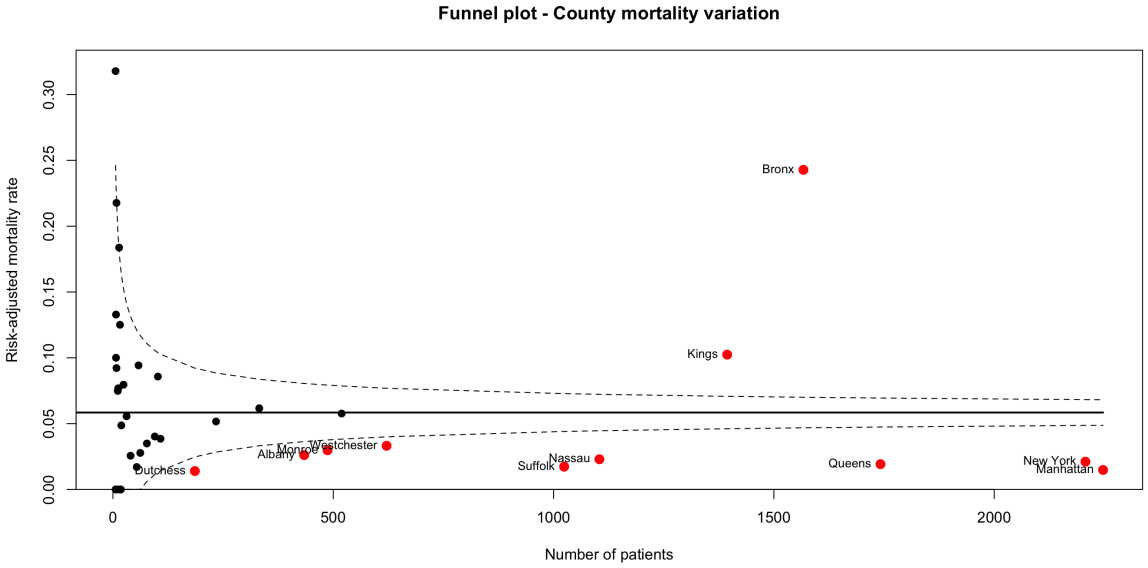

Figure 1: Funnel plot of County-level risk-adjusted mortality rates (RAR) against the number of patients per County. The solid horizontal line represents the overall mortality rate, while the control limits represent the expected range of variation due to random fluctuation.

risk-adjusted mortality rates. Most Counties lie within the expected control limits, indicating variability compatible with random fluctuation. However, some Counties fall outside the limits, suggesting mortality levels higher or lower than expected given their patient volume. As expected, Counties with smaller sample sizes show greater dispersion around the overall rate. In particular, some small Counties such as Chemung and Niagara appear as high outliers in the funnel plot. However, these Counties have very small patient populations, and their elevated mortality estimates are therefore likely driven by statistical instability rather than systematic differences in healthcare outcomes; these Counties were not highlighted in the plot like the other outliers, in red. Among the larger Counties (Kings, Bronx, Queens, New York, Manhattan), differences in mortality patterns may also reflect differences in population composition. The largest Counties in the dataset are the only ones where Non-White patients represent more than half of the population, whereas most other Counties remain predominantly White. This indicates that the largest clusters in the dataset also correspond to areas with greater demographic diversity. For instance, Bronx and Kings have particularly high proportions of Non-White patients (81% and 78%, respectively). However, this pattern does not translate uniformly into higher mortality levels. Queens, for example, also has a large Non-White population (72%) but shows mortality rates closer to the overall average. This suggests that differences in demographic composition alone may not fully account for the observed variation in mortality across Counties.

These findings motivated the use of multilevel models to further investigate whether the observed geographic variability persists after accounting for individual-level predictors.

A first model, including random intercepts for the considered Counties, was fitted according to Equation 6. The additional full model that also considered the relevant variables - race, age class, severity and risk - was also fitted according to Equation 7. Finally, the model including all variables and random slopes for race levels, as per Equation 8, was fitted. Results for all models are reported in Table 2. Comparing the AIC and BIC values, the final full model proves to be the best. The same conclusion happens when it is compared through the likelihood ratio test to the random intercept model only (LRT=1351.7, $p<0.001$) and the model with random intercept and covariates (LRT=20.607,

Covariates	**Random intercepts**	**+ Covariates**		**+ Random slopes**	
Intercept	-3.0609	-7.2300		-7.3815	
		OR (95% CI)	**p-val**	**OR (95% CI)**	**p-val**
Race					
White		Ref.		Ref.	
Black		0.6423 (0.5130, 0.8041)	0.0001	0.7058 (0.5636, 0.8840)	0.0024
Other		1.1201 (0.9110, 1.3772)	0.2821	1.4978 (1.2035, 1.8642)	0.0003
Age class					
0-29		Ref.		Ref.	
30-49		1.6827 (1.484864, 1.9070)	< 0.0001	1.6876 (1.4888, 1.9130)	< 0.0001
50-69		2.8317 (2.2048, 3.6368)	< 0.0001	2.8480 (2.2164, 3.6595)	< 0.0001
70+		4.7651 (3.2739, 6.9355)	< 0.0001	4.8062 (3.2997, 7.0007)	< 0.0001
Severity					
Minor		Ref.		Ref.	
Moderate		1.3679 (1.1667, 1.6039)	0.0001	1.3728 (1.1703, 1.6102)	0.0001
Major		1.8712 (1.3611, 2.5726)	0.0001	1.8845 (1.3697, 2.5928)	0.0001
Extreme		2.5597 (1.5879, 4.1263)	0.0001	2.5870 (1.6030, 4.1749)	0.0001
Risk					
Minor		Ref.		Ref.	
Moderate		3.6147 (3.0920, 4.2258)	< 0.0001	3.6007 (3.0793, 4.2104)	< 0.0001
Major		13.0661 (9.5602, 17.8576)	< 0.0001	12.9650 (9.4820, 17.7274)	< 0.0001
Extreme		47.2302 (29.5600, 75.4633)	< 0.0001	46.6829 (29.1977, 74.6392)	< 0.0001
Variance of random effects					
τ_0^2	0.6203	0.6634		0.8719	
τ_1^2	/	/		0.0182	
τ_{01}	/	/		-0.1260	
ICC	0.1586	0.1678		0.2095	
MOR	2.1196	2.1748		2.4368	
AIC	5624.0	4302.9		4286.3	
BIC	5639.3	4356.2		4354.8	

Table 2: Results for the fitted models.

p<0.001). The final model also leads to an increase in both intra-class correlation (ICC, based on the random intercept variance) and median odds ratio (MOR). A baseline ICC of around 21% indicates moderate clustering of mortality within Counties, and a value of 2.44 for the MOR, higher than several individual-level effects, is compatible with strong contextual factors driving the mortality differences. Intuitively put, when comparing two identical patients from randomly selected counties, the median increase in the odds of death when moving to the higher-risk county is about 2.44. However, even when taking into account the strong effect of County, the race variable still shows significance: black women seem to be protected against mortality for female-specific cancers compared to white women (OR=0.71), while belonging to other races still acts as a risk factor (OR=1.50). Such findings clash with the previous odds ratio estimations, suggesting the presence of cluster-level confounders in the relationship between mortality for female-specific cancers and race.

The predictive power of the three fitted models was assessed using the Receiver Operating Characteristic (ROC) and the area under it (AUC). The model with random intercepts only achieved an AUC of 79.1% (77.29%, 80.93%). The added covariates achieved an AUC of 92.7% (91.53%, 93.94%), which rose to 92.8% (91.64%, 94.03%) with the addition of random slopes. Once again, the more complex model is preferable, although the difference it has with the model without the random slopes is not as evident as the other statistical indices suggested. The optimal cutpoint for the best-performing method is 0.0570, as determined by the Youden index with a value of 0.7337. Such a cutpoint achieves 90.54% sensitivity and 82.83% specificity.

The robustness of the method with the best performance was assessed through bootstrapping with 200 repetitions of the odds ratios it produced. The full results can be found in Table 3 of the Appendix. The bootstrap results do not differ significantly from the Wald results reported in Table 2. This indicates the model's robustness to sampling variability. To further investigate between-county variability, bootstrapping was also performed on the mean odds ratio (MOR). After 200 repetitions, the MOR was found to be 2.76, with a 95% confidence interval of (2.06, 4.04). While the average value is greater than the 2.44 obtained without bootstrapping, the confidence interval does allow for this value. Most notably, however, the interval does not contain 1, which suggests that the variability between counties is statistically significant. Finally, the robustness of the AUC produced by the model

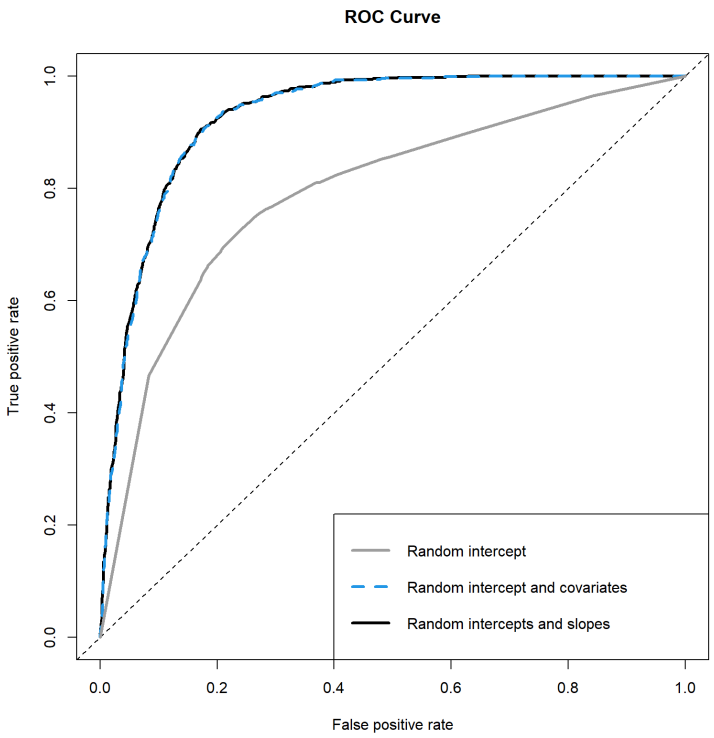

Figure 2: ROC curve for the model with random intercept (in solid grey), the model with covariates (in dashed blue), and the full model with covariates, random intercept and slope (in solid black).

was assessed through 10-fold cross-validation, yielding a mean value of 92.13%, which further indicates the good level of model stability.

Conclusions

Key findings. The main results of this study can be summarised as follows:

- A significant association between race and mortality for female-specific cancers was observed. Even after controlling clinical conditions at admission, and adjusting for age, Non-White patients showed 61% higher mortality compared with the reference population (SMR = 1.61).
- Mortality varies substantially across Counties even after adjusting for differences in age structure. Both direct and indirect standardisation highlighted geographic heterogeneity in mortality rates.
- Multilevel modelling confirmed the presence of meaningful between-County variability. The estimated intra-class correlation (ICC) indicates that approximately 21% of the variability in mortality can be attributed to differences between Counties, while the median odds ratio (MOR = 2.44) suggests that geographic context has a substantial effect on mortality risk.

Interpretation. Taken together, these results suggest that racial disparities in mortality cannot be explained solely by differences in age structure or clinical conditions at admission. The persistence of these disparities after adjustment for several individual-level predictors indicates that broader contextual mechanisms may contribute to unequal outcomes. The multilevel analysis further highlights the importance of geographic context. The magnitude of the MOR implies that two otherwise identical patients admitted in different Counties may experience substantially different mortality risks depending on the County in which they receive care. This finding is consistent with sociological and public health literature (Lahiri and Pulungan, 2007, Loehrer et al., 2024, and Yearby, 2018), showing that structural factors such as healthcare accessibility, socioeconomic inequalities, and residential segregation may influence health outcomes. From a broader perspective, the results support the view that cancer disparities are shaped not only by individual-level risk factors but also by contextual and

structural determinants of health. Differences in healthcare infrastructure, access to screening and treatment, the historical underrepresentation of minority populations in medical research, and local social conditions may contribute to the geographic heterogeneity observed in this study.

Limitations. Study limitations concern the absence of information about patients who die outside the hospital setting, such as at home or in hospice care. In addition, the lack of patient identifiers prevented longitudinal tracking of individuals across hospitalisations, making it impossible to analyse readmissions or long-term survival patterns, especially when combining data from two consecutive years (2023 and 2024). Future research could benefit from integrating administrative hospital data with additional information on County-level healthcare access and socioeconomic conditions to better understand the mechanisms underlying the observed disparities.

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Appendix

	2.5% Quantile	**97.5% Quantile**
Intercept	0.0004	0.0013
Race		
Black	0.5537	0.8829
Other	1.1674	1.8356
Age Class	1.4677	1.8543
Severity	1.1863	1.5868
Risk	3.0940	4.0624

Table 3: Bootstrap confidence intervals for the odds ratios produced by the model of Equation 8. The ORs for age class, severity and risk are reported for the increase of one level for simplicity.