Infection Control & Hospital Epidemiology (2024), **45**, 1190–1196
doi:10.1017/ice.2024.86

Original Article

Leveraging multi-database linkages to assess racial and ethnic disparities among Carbapenem-resistant Enterobacterales cases in Tennessee, 2015–2019

Erika M. Kirtz MPH, Allison Chan MPH, Kristina McClanahan MPH and Rany Octaria PhD, MD, MPH
Tennessee Department of Health – Healthcare-Associated Infections and Antimicrobial Resistance Program, Nashville, TN, USA

Abstract

Background: Awareness of health disparities' impact on clinical outcomes is increasing. However, public health's ability to highlight these trends can be limited by data missingness, such as on race and ethnicity. To better understand race and ethnicity's impact, we compared all-cause 30-day mortality rates between non-Hispanic (NH) Black, NH White, and Hispanic/NH other racial and ethnic patients among cases of carbapenem-resistant Enterobacterales (CRE).

Methods: We performed data linkage using CRE statewide surveillance, Hospital Discharge Data System, and vital records data to obtain demographics and clinical outcomes on CRE cases in TN. We evaluated the association between race and ethnicity with all-cause 30-day mortality among CRE cases.

Results: Among 2,804 reported CRE cases from 2015 to 2019, 65% (n = 1,832) were missing race and ethnicity; data linkage methods reduced missingness to 10% (n = 285). 22%, 74%, and 3% of cases were among NH Black, NH White, and Hispanic/NH other patients, respectively. Thirty-day all-cause mortality among NH Black patients was 5.7 per 100,000 population, 1.9 and 5.7 times higher than NH White and Hispanic/NH other patients. We observed that the risk of dying within 30 days of CRE diagnosis was 35% higher for NH Black compared to NH White patients; unmeasured confounders may be present (adjusted risk ratio 1.35; 95% CI 1.00, 1.83).

Conclusion: Data linkage effectively reduced missingness of race and ethnicity. Among those with CRE, NH Blacks may have an increased risk of all-cause 30-day mortality. Data missingness creates barriers in identifying health disparities; data linkage is one approach to overcome this challenge.

(Received 23 January 2024; accepted 21 April 2024; electronically published 2 October 2024)

Background

Often faced by racial and ethnic minoritized groups in the United States, health disparities are differences in health due to sociological disadvantages that can build barriers to optimal health.[1,2] During the coronavirus disease 2019 (COVID-19) pandemic, the impact of these disparities intensified but was accompanied by heightened awareness, partly from public health surveillance.[3,4] Although public health surveillance data are increasingly reporting disease trends by racial and ethnic groups, there remains a persistent challenge in obtaining race and ethnicity data.[5,6]

Some public health surveillance fields, such as healthcare-associated infections (HAI) and antimicrobial resistance (AR), lack standardized race and ethnicity data reporting requirements. Data missingness hinders the assessment of health disparities among racial and ethnic minoritized groups, who often have higher rates of underlying acute and chronic conditions. HAI/AR health disparities are understudied despite these groups facing increased risks of adverse outcomes from multidrug-resistant organisms (MDROs).[4,5]

Carbapenem-resistant Enterobacterales (CRE) is a group of MDROs that have been routinely monitored by state and federal public health surveillance for years. CRE has been a Tennessee (TN) statewide reportable condition since 2011, with isolate submission required to the Public Health Laboratory (SPHL) for antibiotic resistance and carbapenemase (CP) testing. In 2018, carbapenemase-producing CRE (CP-CRE) became nationally notifiable, meaning cases are mandated for reporting to the Centers for Disease Control and Prevention (CDC).[7] Despite having over ten and five years of TN and national surveillance data, race and ethnicity breakdown are frequently missing due to no reporting requirements.

From 2015 to 2019, the TN Department of Health (TDH) detected over 2,800 CRE cases; approximately 35% of isolates were

Corresponding author: Erika M. Kirtz; Email: Erika.kirtz@tn.gov

Previous Abstract Submission: Kirtz, E.M., Octaria, R., Stover, et al. (2022, June). Comparison of Mortality Rates in Carbapenemase-Producing Enterobacterales Among Non-Hispanic Black and Non-Hispanic White Persons, Tennessee, 2015-2019. Presented at the CSTE 2022 Annual Conferences, Louisville, KY.

Cite this article: Kirtz EM, Chan A, McClanahan K, Octaria R. Leveraging multi-database linkages to assess racial and ethnic disparities among Carbapenem-resistant Enterobacterales cases in Tennessee, 2015–2019. *Infect Control Hosp Epidemiol* 2024. 45: 1190–1196, doi: 10.1017/ice.2024.86

© The Author(s), 2024. Published by Cambridge University Press on behalf of The Society for Healthcare Epidemiology of America. This is an Open Access article, distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives licence (https://creativecommons.org/licenses/by-nc-nd/4.0/), which permits non-commercial re-use, distribution, and reproduction in any medium, provided that no alterations are made and the original article is properly cited. The written permission of Cambridge University Press must be obtained prior to any commercial use and/or adaptation of the article.

confirmed CP producers.[8] Among these cases, 65% (n = 1,832) were missing race and ethnicity data. We aimed to demonstrate cross-database data linkage methods to mitigate the amount of missing race and ethnicity data. Secondarily, we aimed to better understand the association between race and ethnicity and mortality among CRE cases; we compared 30-day all-cause mortality rates among non-Hispanic (NH) Black patients, NH White patients, and Hispanic patients/NH other racial and ethnic groups.

Methods

Case definitions

A CRE case was defined as any organism within the Enterobacterales order that was phenotypically resistant to at least one carbapenem based on the Clinical and Laboratory Standards Institute breakpoints with a minimum inhibitory concentration of ≥4 μg/ml for doripenem, meropenem, or imipenem or ≥2 μg/ml for ertapenem.[7,9] Cases included clinical and colonization detections from infections and colonization screening (ie, rectal swabs), respectively. Cases with bacterial isolates that demonstrated the production of carbapenemase were classified as CP-CRE. Whereas cases with isolates that met the phenotypic definition based on clinical laboratory reports but tested negative for CP were classified as non-CP-CRE. In contrast, cases with no submitted isolates were classified as not tested. Among individuals with multiple CRE specimens collected, a new CRE case was counted only if a different organism or CP gene was identified. If the organism and CP gene were the same as previously reported, a new case was counted only if the specimen collection date was at least 12 months from the collection date of the last case.[7] Therefore, the same individual could have multiple cases counted throughout the study period.

Laboratory testing

For CP testing, the SPHL used the CarbaNP (June 5, 2015, to April 25, 2017) and the Modified Carbapenem Inactivation Method (mCIM) (April 25, 2017, to present).[10] Once an isolate was confirmed to produce CP, CDC-developed polymerase chain reaction testing was completed for five CP genes (eg, bla_{KPC}, bla_{NDM}, bla_{VIM}, bla_{IMP}, and $bla_{OXA\text{-}48\text{-}like}$). Specimens received from colonization screening swabs underwent CP gene testing using the Carba-R Cepheid (Cepheid, Sunnyvale, CA). Organism identification occurred using the MALDI-TOF (Bruker, Billerica, MA, USA).

Datasets and data cleaning

As part of statewide surveillance, CRE data were stored in the National Disease Surveillance System Base System (NBS), which included data on demographics, specimen source, specimen collection dates, and microbiological characteristics such as organism identification, antibiotic susceptibilities, and CP detection). Information on healthcare exposures, such as previous hospitalization stays and emergency department visits, and further detailed demographic and clinical characteristics, such as race and ethnicity, comorbidities, and health procedures, were obtained from the TN Hospital Discharge Data System (HDDS) using dates July 1, 2014, to December 31, 2019. Additional demographic data and mortality information, such as the date and causes of death, were obtained from TN vital records (VR) data, including data from death certificates from July 1, 2015, to December 31, 2019. Population-level case and mortality rates were calculated using the denominator from the TN population estimates from the US Census annual estimates of the resident population from April 1, 2010, to July 1, 2017.[11] Patient privacy has been protected by adhering to the criteria listed under the confidential information clause under Chapter 1200-7-3.[12]

Diagnosis and procedure codes obtained from HDDS were categorized using the method outlined in Quan et al.[13] Diagnostic codes were used to identify if records fell within one of the 17 Charlson index groups.[13] Deyo-Charlson comorbidity index (CCI) was calculated for each case to characterize patient morbidity. We used specimen collection dates and dates of death to determine if patients with CRE acquisitions (cases) died within 30 days after their CRE diagnosis.

Data linkage

To establish a comprehensive dataset for our analysis, we performed data linkage between statewide CRE surveillance data and HDDS based on full name and date of birth using the SOUNDEX function in SAS v9.4. (Cary, North Carolina).[14] English language phonetic similarities in character variables were used to account for misspellings of names. The SPEDIS function was subsequently used to address misspellings due to data entry mistakes.[15] Individuals that did not match on full name and date of birth were matched again using a combination of full name, date of birth, and zip code of residence. To determine the score threshold for inexact matching methods, we hand-reviewed the correctness of the matches across different SPEDIS and COMPGED scores and decided on a score that would result in the fewest incorrect matches informed by the reference suggested by the software (SAS) and our manual review. No multiple matches were identified. Lastly, we matched with VR data to complete remaining missing demographic information and to identify mortality information (Supplementary Figure 1). If any racial and ethnic data differed between HDDS and VR, the information obtained from VR was selected due to additional checks being performed by confirming demographics with next of kin on death certificates.[16] A total of 2,804 CRE cases were identified during the study period; linkage yielded a 90% (n = 2,519) match to HDDS and VR data, primarily representing exact matches between the datasets (82%). The remaining unmatched and those with missing race and ethnicity were excluded from subsequent analysis.

Statistical analysis

We conducted a retrospective cohort study among 2,508 CRE cases to evaluate the association between race and ethnicity with all-cause-30-day mortality since the case's first positive specimen. Race and ethnicity were grouped by NH White, NH Black, and Hispanic and NH other patients. For continuous and categorical variables, descriptive statistics for characteristics were calculated using median and interquartile range (IQR) and frequency count (percentage). A directed acyclic graph (DAG) was created to evaluate covariates as potential confounders using a *priori* knowledge. Based on the DAG, covariates associated with both the exposure and outcome were considered confounders for the model; covariates associated only with the outcome were considered for model adjustment as precision variables (Supplementary Table 1). A P value of <.15 for the interaction term between the potential modifier and race variable was determined as effect measure modification (EMM).

Lastly, to evaluate the association between race and ethnicity and mortality, we generated a multivariable generalized linear

https://doi.org/10.1017/ice.2024.86 Published online by Cambridge University Press

model with a Poisson distribution and a logarithmic link to generate risk ratios. To determine our final model, we conducted a bias-precision analysis to compare the confounder-adjusted and confounder-adjusted including precision variables models. Statistical analyses were performed with SAS v9.4. (Cary, North Carolina).

Result

Case characteristics

Thirty-five percent of TN CRE surveillance data from 2015 to 2019 had completed race and ethnicity reporting, which increased to 90% following data linkage (Figure 1). A total of 2,508 CRE cases were included in the study, where 74% (n = 1,865) were NH White patients, 22% were NH Black patients (n = 562), and 3% were Hispanic and NH other (n = 81) patients (Table 1). The median age was 67 (IQR: 58–79), 57 (IQR: 45–71), and 46 (IQR: 30–66) for NH White, NH Black, and Hispanic and NH other patients, respectively. Among NH White and NH Black patients, the proportions of nursing home residents (52% vs 49%) and history of previous hospitalizations (62% vs 64%) were similar. Most NH Black and Hispanic/NH other patients resided in urban counties at 64% and 57% compared to NH White patients at 30%. A higher proportion of NH Black patients received dialysis (9.1% vs 2.5%) and had renal disease (69% vs 52%) compared to NH White patients. None of the 81 Hispanic/ NH other patients had prior cancer, dementia, chronic obstructive pulmonary disease (COPD), or diabetes (Figure 2). The median time from specimen collection date to death date was shorter for NH Black patients at 98 as opposed to 157 days for NH White patients and 139 for Hispanic and NH other patients. The difference in the proportions for dementia and COPD between NH White and NH Black patients were small, all under 5%. We observed larger differences in diabetes (58% of NH Black patients vs 35% in NH White patients) and renal disease (69% in NH Black patients vs 52% in NH White patients).

Characterization of case and mortality rates

Based on our DAG, a crude model was sufficient to show the causal total effect of race and ethnicity and 30-day all-cause-mortality, given the available data source (Supplementary Figure 2). Our bias-precision tradeoff analysis comparing two models (crude vs. adjusted for CP, specimen site, and age) suggested that the precision gained in the crude model exceeds the validity that is lost. However, final effect estimates and confidence intervals are very similar, therefore we displayed both. We highlighted the adjusted effect throughout our discussion because of discussion regarding the precision variables (Supplementary Table 1). Based on our DAG, our total effect estimate does not have any variables that serve as confounders (associated with race/ethnicity AND 30-day mortality). However, our DAG does have a handful of precision variables that are associated with the outcome only. Therefore, precision variables were included in our adjusted model. Because CP production and site of infection (invasive vs noninvasive) have been shown to increase the risk for mortality, we assessed EMM from bloodstream infections (BSI) and CP status, and results showed no evidence of effect modification.[17,18]

The proportion of NH Black patients who died within 30 days was 11.6% (n = 65), 3.2% (n = 157) higher than NH White patients, and 4.2% (n = 6) higher than Hispanic/NH other patients. Of the 228 patients who died within 30-days of their CRE infection, the proportion of deaths was higher among patients with more invasive infections (blood and other normally sterile sites) across all three racial and ethnic groups (Supplementary Table 2). The case rate of CRE among NH Black patients was 48.9 per 100,000 population, 1.4 and 4.0 times higher than NH White patients (35.3 per 100,000) and Hispanic and NH other patients (12.3 per 100,000) (Figure 3). Similarly, the all-cause-30-day mortality among NH Black patients was 5.7 per 100,000 population, 1.9 and 5.7 times higher than NH White patients (3.0 per 100,000) and Hispanic NH other patients (1.0 per 100,000) (Figure 3). We observed that the risk of dying within 30 days of their CRE diagnosis was 35% higher for NH Black patients compared to NH White patients [adjusted risk ratio 1.35; 95% CI, (1.00, 1.83)] (Table 2).

Discussion

Using TN statewide surveillance data to evaluate mortality among CRE cases, we observed a notable increased risk in all-cause-30-day mortality rates for NH Black compared to NH White and Hispanic/NH other patients. NH Black and NH White patients had similar risk factors for 30-day mortality, including previous inpatient stays and residing in a nursing home. NH White patients had a higher proportion of previous LTACH stays, and NH Black patients died sooner than NH White patients after their CRE specimen was collected (median 98 days and 157 days, respectively). Despite NH Black patients having a younger mean age, we observed that they had a 35% increased risk of dying within 30 days of their CRE diagnosis. These findings signal a disparity that warrants further investigation into underlying root causes.

While race and ethnicity are considered social constructs, they have consistently been used as indicators for health outcomes.[19] Existing literature highlights the disparity of adverse health outcomes and lower quality of care among racial and ethnic minorities compared to their non-minority counterparts.[19] Among racial minority groups such as NH Black individuals, other non-White races, and Asian individuals, higher rates of central-line bloodstream infections (CLABSI) and catheter-associated urinary tract infections have been documented compared to NH White individuals.[20] Additionally, increased risks of recurrent *Clostridioides difficile* infections and hospital-acquired methicillin-resistant *Staphylococcus aureus* among Black individuals have been observed compared to White individuals.[21,22]

Our observed results reflect trends similar to those in the existing literature. Literature has highlighted that a multifaceted intersection of factors, including but not limited to socio-ecological influences such as the unconscious bias of healthcare workers, inequities in social and economic determinants of health, structural racism, and research gaps can contribute to racial/ethnic disparities.[4,20,23] Due to a history of medical distrust in the Black community, the results of this study are not surprising. Racial and ethnic minorities are less likely to receive routine medical procedures and experience lower-quality health care than NH White individuals.[19,24] The COVID-19 pandemic showed minorities to have an increased risk of hospitalization, a known risk factor for MDROs; however, our data did not indicate differences between hospitalizations among NH Black and NH White patients.[3] It can be challenging to separate these influences when evaluating the root causes. Therefore, expanding the literature is essential to improve our understanding of the underlying causes and evaluate evidence-based interventions.

https://doi.org/10.1017/ice.2024.86 Published online by Cambridge University Press

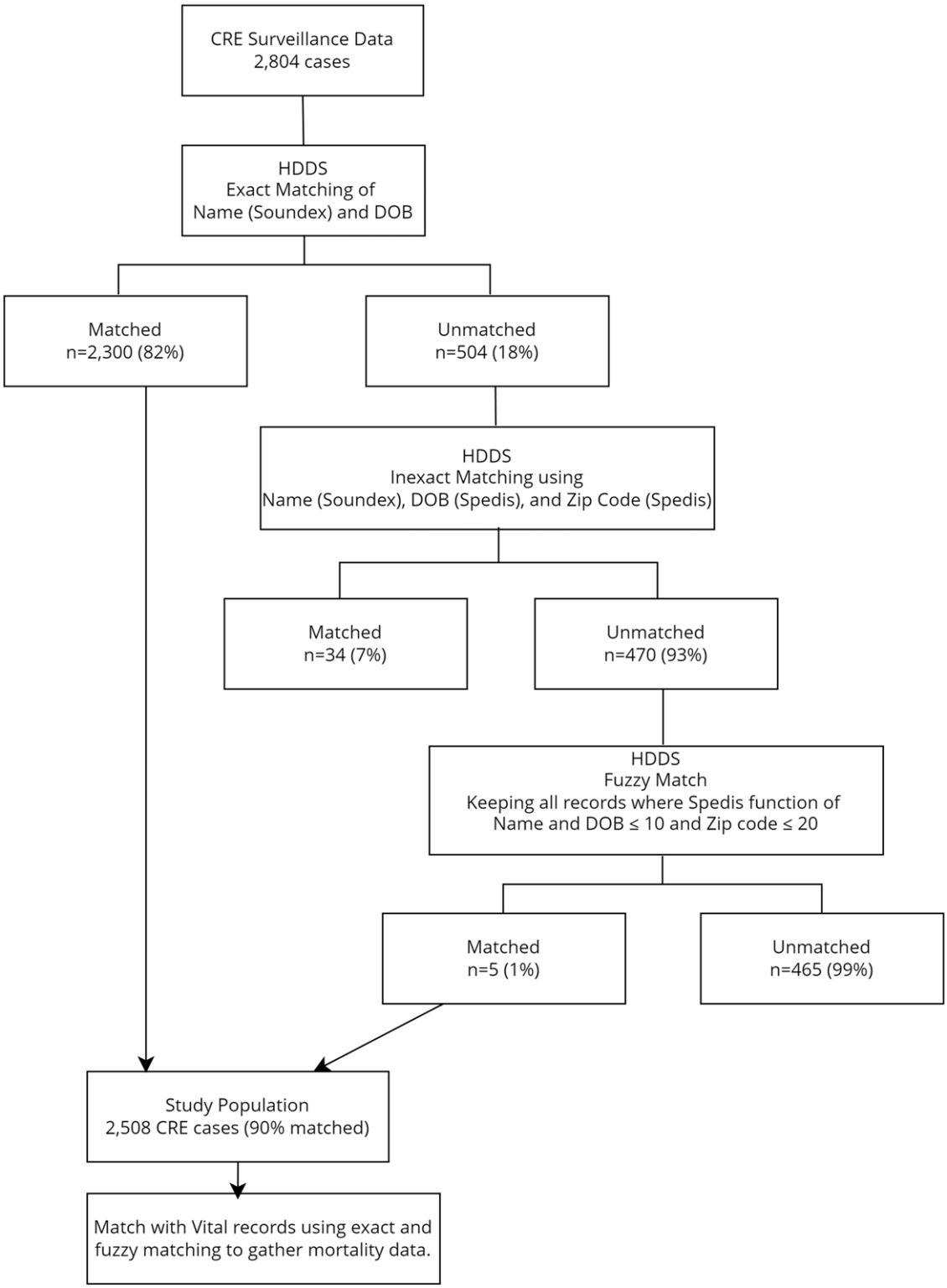

Figure 1. Results of multi-database linkage between Carbapenem-resistant Enterobacterales surveillance data stored in the National Disease Surveillance System (NEDSS) Base System (NBS) and Hospital Discharge Database Surveillance data using exact, inexact, and fuzzy matching.

In our study, for those with an active infection, NH Black patients likely presented at healthcare settings with a more progressed CRE infection than their NH white counterparts, leading to an increased mortality risk. BSIs caused by CRE are related to high mortality rates ranging from 30% to 80%.[17] In our study, BSIs occurred in 4% of NH White patients and 7% of NH Black patients. While the 30-day mortality rate among cases with a BSI was comparable between NH Black and NH White patients (55% vs 53%), the rate of BSI among NH Black patients was nearly double that of NH White patients (7% vs 4%). Therefore, we assessed CP and BSIs as effect modifiers, and our results did not suggest evidence of effect modification.

https://doi.org/10.1017/ice.2024.86 Published online by Cambridge University Press

Table 1. Demographic and clinical characteristics of carbapenem-resistant Enterobacterales cases by race and ethnicity from July 2015 to September 2019 in Tennessee

	Non-Hispanic White	Non-Hispanic Black	Hispanic and Non-Hispanic Other Races
N (%)	1865 (74.4)	562 (22.4)	81 (3.2)
Sex			
Female	1186 (63.6)	317 (56.4)	54 (66.7)
Age, median (Q1–Q3)	67 (58–79)	57 (45–71)	46 (30–66)
CRE Classification			
Non-CP-CRE	908 (48.7)	251 (44.7)	37 (45.7)
CP-CRE	461 (24.7)	173 (30.8)	27 (33.3)
Not Tested/Confirmed	496 (26.6)	138 (24.6)	17 (21.0)
Bacterial Genus			
Enterobacter	655 (35.1)	266 (47.3)	25 (30.9)
Escherichia	462 (24.8)	116 (20.6)	25 (30.9)
Klebsiella	547 (29.3)	135 (24.0)	26 (32.1)
Other Genera	201 (10.8)	45 (8.0)	5 (6.2)
Nursing Home Residence	973 (52.2)	277 (49.3)	17 (21.0)
Residence in Urban Counties	559 (30.0)	361 (64.2)	46 (56.8)
Previous LTACH Stay	105 (5.6)	22 (3.9)	5 (6.2)
Previous Inpatient Stay	1151 (61.7)	357 (63.5)	37 (46.9)
Nonsurgical Endoscopic	128 (6.9)	49 (8.7)	6 (7.4)
Dialysis	47 (2.5)	51 (9.1)	1 (1.2)
Assisted Ventilation	217 (11.6)	75 (13.4)	10 (12.4)
Died within 1 year	519 (27.8)	165 (29.4)	17 (21.0)
Died within 6 months	395 (21.2)	133 (23.7)	14 (17.3)
Died within 30 days	157 (8.4)	65 (11.6)	6 (7.4)
Died within 14 days	91 (4.9)	41 (7.3)	5 (6.2)
Median time (days) from specimen collection to date of death	157	98	139
Charlson Comorbidity Index Score			
Median (Q1–Q3)	5 (2–7)	5 (2–8)	4 (0–6)

Note. All data are expressed as no. (%) unless otherwise indicated. CP, carbapenemase-producing; CRE, carbapenem-resistant Enterobacterales; LTACH, long-term acute care hospital.

Additionally, the routine missingness of demographic information in public health surveillance data creates a challenge to identify racial and ethnic trends, particularly among AR data, since much is reported on the clinical laboratory level.[25] A big strength of this study is that we reduced these data gaps with a multi-database linkage strategy. Our data linkage helped complete missingness on healthcare exposures, comorbidities, race and ethnicity, and death statistics.

In this study, we were unable to confidently compare the mortality risks among Hispanic and NH other patients. The sample size for the Hispanic and NH other populations (n = 81) was small; therefore, we primarily used the data from NH Black and NH White patients to establish our final models during the bias-precision tradeoff analysis. Since race and ethnicity are social constructs, we recognize that obtaining accuracy can be challenging. Agreement between self-reports and death certificate proxy reports was shown to be strong for White and Black populations but considerably low for other races, which suggests the use of vital records data to obtain missing race and ethnicity data is appropriate for only NH White and Black individuals.[26] A limitation of our study is the use of surveillance data instead of data from medical records. We classified clinical and colonization cases based on the source of specimens that were tested, which we observed that the proportion of deaths within 30-days were higher among patients with more invasive infections although majority of cases were isolated from normally nonsterile sites. However, we did not have sufficient data on patient symptoms to confirm. Another limitation of our study is the use of all-cause mortality since we were unable to confirm that the patient's death was impacted/caused by CRE or CRE-related complications. This type of data is common among public health surveillance, and cause of death can be challenging in general. However, our findings are important to highlight what is commonly seen in surveillance data.

Recognition of health disparities among racial and ethnic groups can allow public health to enhance surveillance to aid with the identification of root causes and establishment of targeted interventions. We foresee initial interventions to start with education about identified trends that can increase awareness among healthcare workers when caring for patients of a racial minority. This work also highlights the importance of health equity

https://doi.org/10.1017/ice.2024.86 Published online by Cambridge University Press

Table 2. 30-day all-cause mortality risk estimated of Carbapenem-resistant Enterobacterales cases by race/ethnicity

	Unadjusted Risk Ratio (95% CI)	Adjusted Risk Ratio (95% CI)
NH White	Referent	Referent
NH Black	1.37 (1.03, 1.83)	1.35 (1.00, 1.83)
Hispanic/NH Other	0.88 (0.39, 1.99)	1.00 (0.44, 2.27)

Note. CI, Confidence Interval; NH, Non-Hispanic. Adjusted risk ratio is adjusted for precision variables carbapenemase production, specimen source, and age. Definition: Given our DAG did not have identified confounders, the adjusted model included precision variables, which are variables that are known to be associated with our outcome of interest, 30-day mortality.

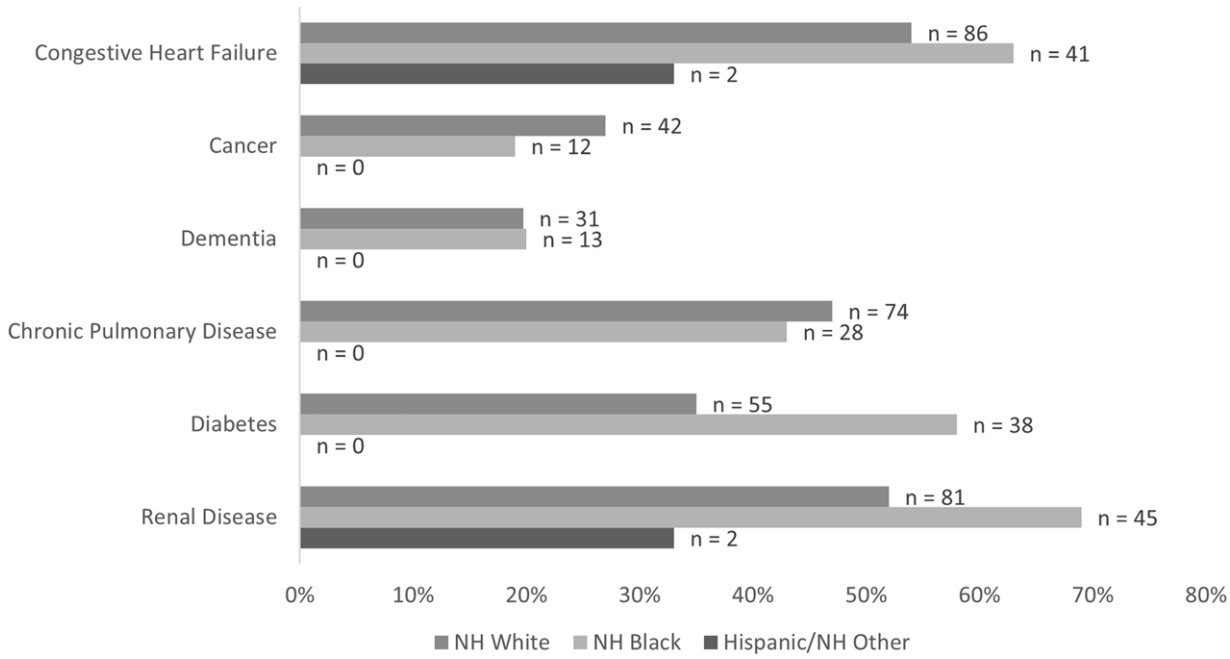

Figure 2. Proportion of prior comorbidities for patients who died within 30 days of their carbapenem-resistant Enterobacterales diagnosis, July 2015 to September 2019. *NH, non-Hispanic.

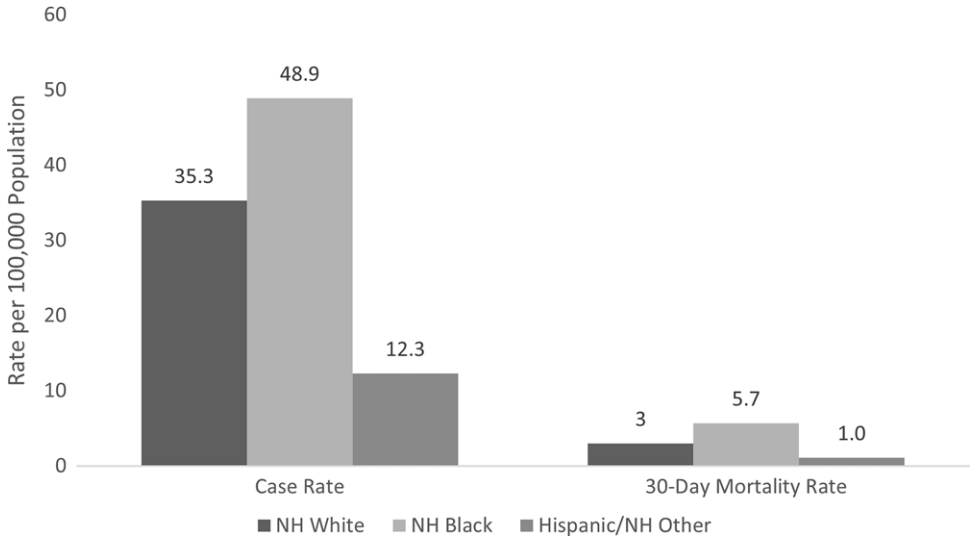

Figure 3. Carbapenem-resistant Enterobacterales case rates and all-cause-30-day mortality rates per 100,000 population by race and ethnicity from July 2015 to September 2019. Population denominators were obtained from the Tennessee population estimates from the United States Census annual estimates of the resident population from April 1, 2010, to July 1, 2017. Note: Hispanic/NH other had a rate of 0% due to a small sample size. *NH, non-Hispanic.

https://doi.org/10.1017/ice.2024.86 Published online by Cambridge University Press

practices in infectious disease, specifically MDRO surveillance. More studies are needed to explore how societal influences impact the acquisition of MDROs, subsequent health outcomes, and how public health can play a role. Our data linkage strategy was complex, highlighting the need to modify the current surveillance systems to better collect demographic data. However, data linkage allowed us to assess racial and ethnic disparities among patients who met the case definition for CRE despite our surveillance system not having the necessary demographic data. There is space to implement systemic changes in our data collection processes that will allow for easier detection of disparities.

In conclusion, improving surveillance systems and mandating reporting takes time, which delays identifying and understanding disparities. Our study exemplifies solutions that allow public health to address inequities in MDROs now.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/ice.2024.86.

Acknowledgements. We are thankful to the laboratory staff at the Tennessee SPHL for their assistance in testing all submitted isolates. The authors thank the following individuals for their contributions: Matthew Estes, Albert Burks, and Nikhil Khankari.

Financial support. No direct funding for this project. This work was supported by the CDC (Epidemiology Laboratory Capacity Funding, sections G1 and G2).

Competing interests. No reported conflicts. All authors have completed and submitted the International Committee of Medical Journal Editors form of disclosure of potential conflict of interests. Competing Interests: The authors declare none.

Disclaimer. The findings and conclusions in this article are those of the authors and do not necessarily represent the views of the Tennessee Department of Health.

References

1. Braveman P. What are health disparities and health equity? We need to be clear. *Public Health Rep* 2014;129:5–8.
2. Fiscella K, Williams DR. Health disparities based on socioeconomic inequities: implications for urban health care. *Acad Med* 2004;79:1139–1147.
3. Mackey K, Ayers CK, Kondo KK, *et al.* Racial and ethnic disparities in COVID-19-related infections, hospitalizations, and deaths: a systematic review. *Ann Intern Med* 2021;174:362–373.
4. Chen J, Khazanchi R, Bearman G, Marcelin J. Racial/ethnic inequities in healthcare-associated infections under the shadow of structural racism: Narrative review and call to action. *Curr Infect Dis Rep* 2021;23. doi: 10.1007/s11908-021-00758-x.
5. Gualandi N, Mu Y, Bamberg WM, *et al.* Racial disparities in invasive methicillin-resistant Staphylococcus aureus infections, 2005-2014 [published correction appears in Clin Infect Dis. 2019 Mar 5;68(6):1070-1071]. *Clin Infect Dis* 2018;67:1175–1181.
6. See I, Wesson P, Gualandi N, *et al.* Socioeconomic factors explain racial disparities in invasive community-associated methicillin-resistant Staphylococcus aureus disease rates. *Clin Infect Dis* 2017;64:597–604.
7. CSTE position statement 17-ID-04: Public health reporting and national notification of Carbapenemase producing Carbapenem-resistant Enterobacteriaceae (CP-CRE) for *E. coli*, *Klebsiella* spp. and *Enterobacter* spp., 2017. https://cdn.ymaws.com/www.cste.org/resource/resmgr/2017PS/2017PSFinal/17-ID-04.pdf. Accessed October 17, 2023.
8. Enterobacterales, Carbapenem-Resistant (all genera). Tennessee Department of Health, Healthcare-associated infections and antimicrobial resistance program, 2022. https://www.tn.gov/health/cedep/reportable-diseases/enterobacteriaceae–carbapenem-resistant-all-genera.html. Accessed October 18, 2023.
9. CLSI. *Performance Standards for Antimicrobial Susceptibility Testing. CLSI supplement M100*. 27th ed., Wayne, PA: Clinical and Laboratory Standards Institute; 2017.
10. Pierce VM, Simner PJ, Lonsway DR, *et al.* Modified carbapenem inactivation method for phenotypic detection of carbapenemase production among Enterobacteriaceae. *J Clin Microbiol* 2017;55:2321–2333.
11. General Health Data: Population. Population Counts by Age Group, Sex, Race and Ethnicity, Estimates 2017. Tennessee Department of Health, Division of Policy, Planning and Assessment, 2017. https://www.tn.gov/health/health-program-areas/statistics/health-data/population.html. Accessed October 18, 2023.
12. Tennessee Department of Health. Rules of Tennessee Department of Health, Health Statistics: Chapter 1200-7-3 Hospital Discharge Data System, 2006. https://www.tn.gov/content/dam/tn/health/documents/HDDS_Rules_1200-07-03.pdf. Accessed October 18, 2023.
13. Quan H., Sundararajan V., Halfon P., *et al.* Coding algorithms for defining comorbidities in ICD-9-CM and ICD-10 administrative data. *Med Care* 2005;43:1130–1139. http://www.jstor.org/stable/3768193
14. Fam, Z. Matching character variables by sound: A closer look at SOUNDEX function and Sounds-Like Operator (=*) [PDF file], 2002. https://support.sas.com/resources/papers/proceedings/proceedings/sugi29/072-29.pdf
15. Gershteyn Y. *Use of SPEDIS Function in Finding Specific Values*. Chicago: SCIREX Corporation, pp. 86–25, n.d. https://support.sas.com/resources/papers/proceedings/proceedings/sugi25/25/cc/25p086.pdf
16. General Health Data: Death Statistics. Tennessee Department of Health Bureau of Policy, Planning and Assessment, Division of Health Statistics. https://www.tn.gov/health/health-program-areas/statistics/health-data/death-statistics.html. Published 2014. Accessed October 18, 2023.
17. Tamma PD, Goodman KE, Harris AD, *et al.* Comparing the outcomes of patients with Carbapenemase-producing and non-Carbapenemase-producing Carbapenem-resistant Enterobacteriaceae bacteremia. *Clin Infect Dis* 2017;64:257–264.
18. Zhou C, Jin L, Wang Q, *et al.* Bloodstream infections caused by Carbapenem-resistant Enterobacterales: Risk factors for mortality, antimicrobial therapy and treatment outcomes from a prospective multicenter study. *Infect Drug Resist* 2021;14:731–742.
19. Institute of Medicine (US) Committee on understanding and eliminating racial and ethnic disparities in health care. In Smedley BD, Stith AY, Nelson AR, eds. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. Washington (DC): National Academies Press (US); 2003.
20. Gettler EB, Kalu IC, Okeke NL, *et al.* Disparities in central line-associated bloodstream infection and catheter-associated urinary tract infection rates: An exploratory analysis. *Infect Control Hosp Epidemiol* 2023;44(11):1857–1860.
21. Freedberg DE, Salmasian H, Friedman C, Abrams JA. Proton pump inhibitors and risk for recurrent Clostridium difficile infection among inpatients. *Am J Gastroenterol* 2013;108:1794–1801.
22. Klevens RM, Morrison MA, Nadle J, *et al.* Invasive methicillin-resistant Staphylococcus aureus infections in the United States. *JAMA* 2007;298:1763–1771.
23. Yang S, Rider BB, Baehr A, Ducoffe AR, Hu DJ. Racial and ethnic disparities in health care-associated Clostridium difficile infections in the United States: State of the science. *Am J Infect Control* 2016;44:91–96.
24. Bambra C. Pandemic inequalities: emerging infectious diseases and health equity. *Int J Equity Health* 2022;21:6.
25. Naumova EN. Public health inequalities, structural missingness, and digital revolution: time to question assumptions. *J Public Health Pol* 2021;42:531–535.
26. Arias E, Schauman WS, Eschbach K, Sorlie PD, Backlund E. The validity of race and Hispanic origin reporting on death certificates in the United States. National Center for Health Statistics. *Vital Health Stat 2* 2008;1–21.

https://doi.org/10.1017/ice.2024.86 Published online by Cambridge University Press