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# Disparities in obesity-related cardiovascular-induced mortality trends in the U.S. of America over two decades

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**Background:** Cardiovascular disease (CVD) remains the leading cause of death in the U.S., and the growing prevalence of obesity has amplified this burden. Despite extensive research on their pathophysiologic interplay, long-term mortality trends among adults with both conditions remain insufficiently characterized.

**Methods:** Using national death certificate data from the CDC WONDER database (1999–2020), we examined age-adjusted mortality rates (AAMRs) for CVD with obesity listed as a contributing cause among adults aged  $\geq 25$  years. Temporal trends were analyzed using joinpoint regression to estimate annual percent change (APC) and average annual percent change (AAPC) with 95% confidence intervals, stratified by sex, ethnicity, census region, state, and urbanization level.

**Results:** From 1999 to 2020, the national AAMR increased steadily across all subgroups. Males had higher mortality and a steeper rise than females (AAPC 5.94% vs. 4.40%), with female rates accelerating after 2018. Non-Hispanic Black (NH Black) adults consistently experienced the highest mortality, while Hispanic and NH Black populations exhibited the sharpest recent increases. The South and Midwest regions showed the fastest growth (AAPC  $>5\%$ ), and nonmetropolitan areas surpassed metropolitan ones (AAPC 5.69% vs. 4.98%). Oklahoma displayed the most pronounced escalation nationwide (AAPC 10.52%).

**Conclusions:** Over two decades, obesity-related CVD mortality has risen markedly and unevenly across demographic and geographic lines in the U.S. These findings highlight the intensifying intersection of cardiometabolic disease and social inequity, underscoring the urgent need for equity-centered prevention and investment in vulnerable communities.

### KEYWORDS

cardiovascular mortality, CDC WONDER, global disease burden, joinpoint regression, obesity

## 1 Background

CVD remains the leading cause of mortality in the U.S., accounting for more deaths than any other chronic condition (1). Nearly 48.6% of U.S. adults are affected by some form of CVD, and the rising prevalence of obesity has further intensified this public health burden (2). Obesity, defined as a body mass index (BMI)  $\geq 30$  kg/m<sup>2</sup>, affects more than 42% of U.S. adults and represents a major modifiable risk factor for hypertension, dyslipidemia, type 2 diabetes, and coronary artery disease (3). The coexistence of obesity and CVD imposes a synergistic burden: obesity not only accelerates atherosclerotic progression but also contributes to adverse cardiovascular outcomes and premature mortality (4).

Prior research suggests that the association between obesity and CVD is mediated through complex metabolic, inflammatory, and neurohormonal pathways. Adipose tissue dysfunction promotes chronic low-grade inflammation, characterized by elevated levels of C-reactive protein, tumor necrosis factor- $\alpha$ , and interleukin-6, which are implicated in endothelial dysfunction and atherogenesis (5, 6). Obesity is also associated with insulin resistance and heightened sympathetic activity, both of which increase cardiac workload and contribute to structural remodeling (7, 8). However, the present study does not investigate these intermediate biological mechanisms; rather, it focuses on long-term, population-level mortality trends using nationally representative death certificate data.

Marked disparities in both obesity and CVD prevalence persist across demographic and geographic groups in the US. Non-Hispanic Black adults experience disproportionately higher rates of both conditions, reflecting structural inequities, socioeconomic barriers, and differential access to preventive care (9, 10). Although previous studies have described the pathophysiologic links between obesity and CVD, relatively few have systematically examined long-term mortality trends among individuals affected by both conditions. Therefore, this study aims to characterize disparities in CVD mortality among adults with obesity by sex, race, census region, state, and urbanization status over the past two decades. By delineating emerging epidemiologic patterns, this analysis seeks to inform more targeted and equitable public health interventions.

## 2 Methods

### 2.1 Data sources

The CDC's Wide-Ranging Online Data for Epidemiologic Research (WONDER) database was used to obtain death certificate data from 1999 to 2020 (11). CVD deaths were extracted using ICD-10 codes I00–I99 as the underlying cause of death, with obesity coded as a contributing cause (ICD-10: E66.0–E66.2, E66.8–E66.9) (12). CVD was operationalized as an aggregated underlying-cause ICD-10 category (I00–I99) in CDC WONDER; thus, findings represent group-level mortality surveillance rather than subtype-specific cardiovascular outcomes. Because the study used publicly available, de-identified data, Institutional Review Board approval was not required. This study is a retrospective population-based analysis using publicly available national mortality data. The study was conducted in

accordance with the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines (13).

### 2.2 Study variables

We examined obesity contributing- CVD mortality by sex, ethnicity (NH White, NH Black, Hispanic, and NH other), census region (14), state, and urbanization status (metropolitan vs. nonmetropolitan, based on the 2013 NCHS Urban–Rural Classification Scheme) (15).

### 2.3 Statistical analysis

AAMRs per 100,000 population were calculated using the 2000 U.S. standard population. Temporal trends were assessed using joinpoint regression (Joinpoint Regression Program, National Cancer Institute), which identifies statistically significant inflection points and estimates APC and AAPC with 95% confidence intervals (16). Statistical significance was defined as  $p < 0.05$ . Statistically significant values are marked with an asterisk (\*) in the results section. Analyses were stratified by geographic variables to evaluate disparities in mortality patterns.

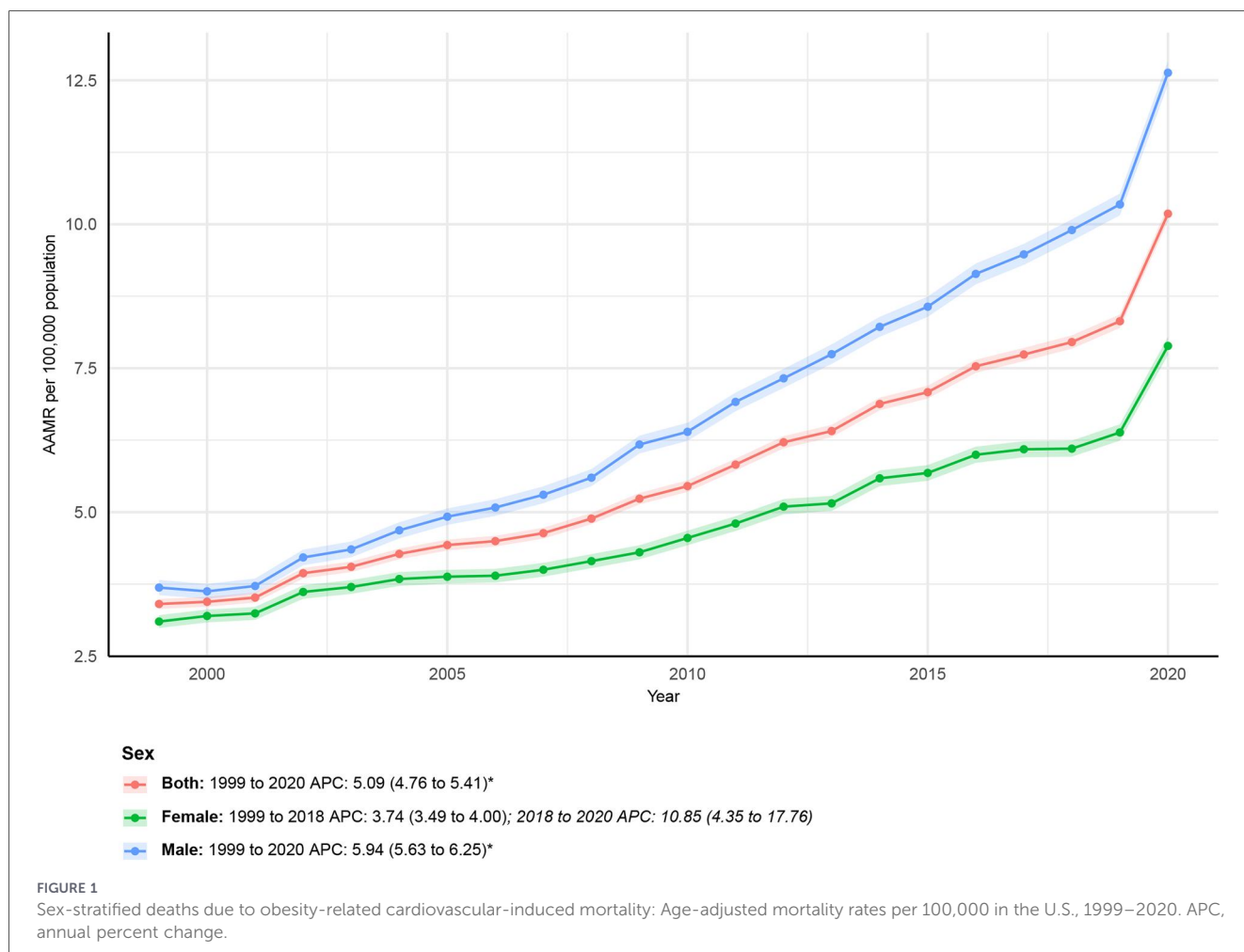
## 3 Results

### 3.1 Trends by sex

From 1999 to 2020, the AAMR in males increased from 3.69 (95% CI: 3.56–3.82) in 1999 to 12.63 (95% CI: 12.42–12.84) in 2020, with an AAPC of 5.94 (95% CI: 5.63–6.25)\*. The APC for males varied across time periods: 1999–2020: APC = 5.94 (95% CI: 5.63–6.25)\*. In female patients, the AAMR also increased over the study period, from 3.10 (95% CI: 2.99–3.21) in 1999 to 7.89 (95% CI: 7.73–8.04) in 2020. The AAPC was 4.40 (95% CI: 3.80–5.00). The APC by time period for females was: 1999–2018: APC = 3.74 (95% CI: 3.49–4.00); 2018–2020: APC = 10.85 (95% CI: 4.35–17.76). All the above changes are visualized in [Figure 1](#) and further detailed in [Supplementary Table S1](#).

### 3.2 Trends by ethnicity

NH Other consistently had the lowest AAMR among racial groups, increasing from 0.78 (95% CI: 0.58–1.03) in 1999 to 3.78 (95% CI: 3.48–4.08) in 2020, with an AAPC of 6.09 (95% CI: 5.29–6.90)\*. APC by time period for NH Other: 1999–2020: APC = 6.09 (95% CI: 5.29–6.90)\*. Among NH White, AAMR decreased from 3.29 (95% CI: 3.19–3.39) in 1999 to 9.91 (95% CI: 9.75–10.06) in 2020. The AAPC was 5.28 (95% CI: 4.99–5.58)\*. APC by time period: 1999–2020: APC = 5.28 (95% CI: 4.99–5.58)\*. The AAMR for NH Black increased from 6.45 (95% CI: 6.07–6.83) in 1999 to 18.59 (95% CI: 18.08–19.11) in 2020. The AAPC was 5.09 (95% CI: 4.42–5.76)\*. APC by time period: 1999–2018: APC = 4.12 (95% CI: 3.82–4.42); 2018–2020: APC = 14.72 (95% CI: 7.37–22.57). Among Hispanic patients, the AAMR increased from 1.96 (95% CI: 1.70–2.22) in 1999 to



6.75 (95% CI: 6.46–7.04) in 2020. The AAPC was 5.48 (95% CI: 4.62–6.36)\*. APC by time period: 1999–2018: APC = 4.72 (95% CI: 4.26–5.18); 2018–2020: APC = 13.02 (95% CI: 4.00–22.83). See Figure 2 and Supplementary Table S1 for detailed trends for all racial groups.

### 3.3 Trends by census regions

All census regions showed an increase in AAMR over time. The AAMR for Midwest increased from 3.45 (95% CI: 3.27–3.63) in 1999 to 10.92 (95% CI: 10.63–11.21) in 2020. The AAPC was 5.35 (95% CI: 3.09–7.66)\*. APC by time period: 1999–2009: APC = 4.16 (95% CI: 2.63–5.72); 2009–2012: APC = 9.62 (95% CI: –6.06–27.92); 2012–2020: APC = 5.27 (95% CI: 3.85–6.71). Among Northeast, the AAMR increased from 3.22 (95% CI: 3.03–3.40) in 1999 to 9.22 (95% CI: 8.93–9.51) in 2020. The AAPC was 5.29 (95% CI: 4.91–5.68)\*. APC by time period: 1999–2020: APC = 5.29 (95% CI: 4.91–5.68)\*. For South, the AAMR also increased over the study period, from 3.06 (95% CI: 2.93–3.20) in 1999 to 10.51 (95% CI: 10.30–10.72) in 2020. The AAPC was 5.73 (95% CI: 5.27–6.19)\*. The APC by time period for South was: 1999–2018: APC = 4.97 (95% CI: 4.75–5.18); 2018–2020: APC = 13.26 (95% CI: 8.26–18.49). Among West, the AAMR increased from 4.19 (95% CI: 3.98–4.40) in 1999 to 9.70

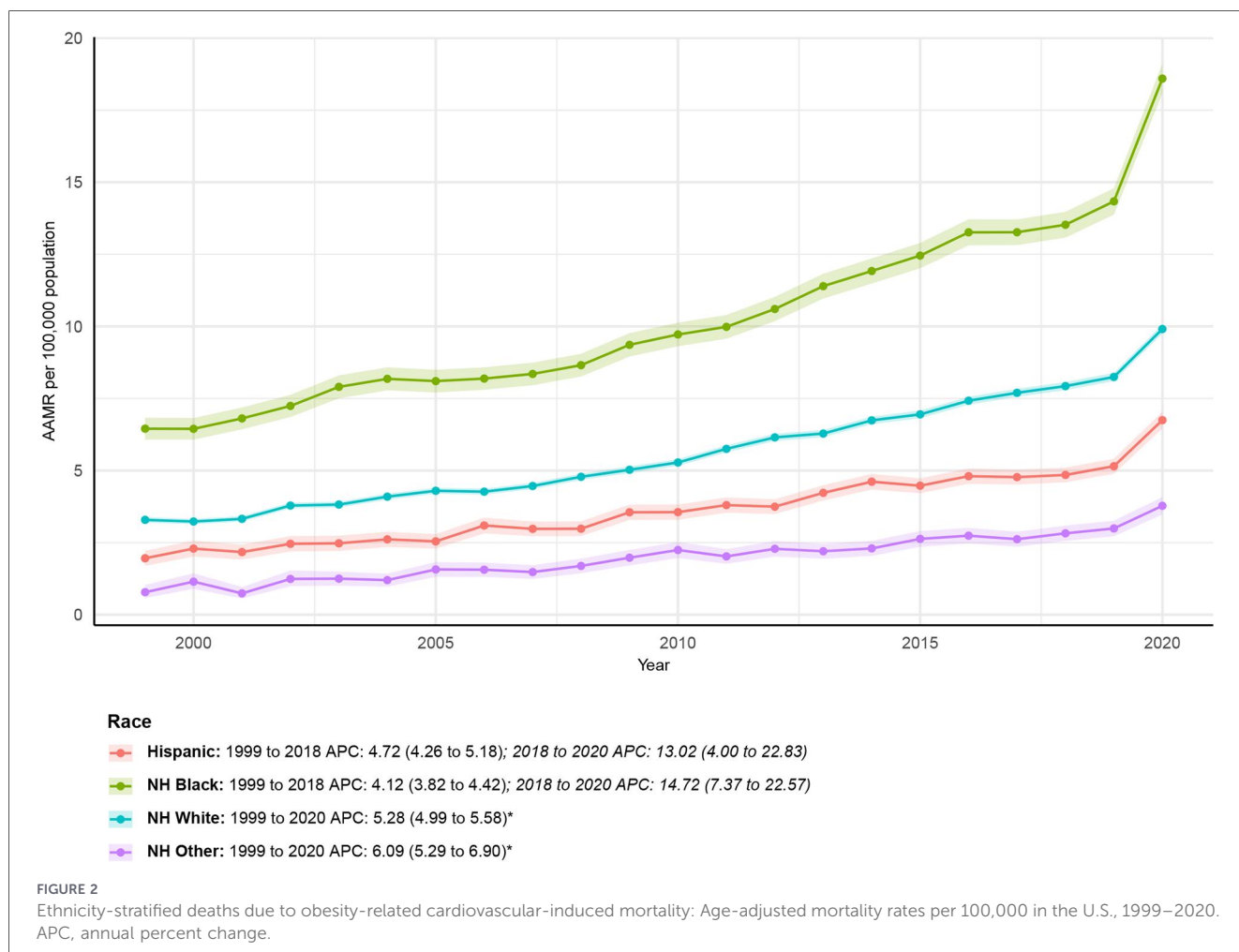
(95% CI: 9.45–9.96) in 2020. The AAPC was 3.75 (95% CI: 3.38–4.13)\*. APC by time period: 1999–2020: APC = 3.75 (95% CI: 3.38–4.13)\*. Regional differences are illustrated in Figure 3 and further detailed in Supplementary Table S1.

### 3.4 Trends by urbanization

From 1999 to 2020, the AAMR in metropolitan areas increased from 3.43 (95% CI: 3.33–3.52) in 1999 to 9.90 (95% CI: 9.76–10.03) in 2020, with an AAPC of 4.98 (95% CI: 4.63–5.33)\*. APC for metropolitan areas: 1999–2020: APC = 4.98 (95% CI: 4.63–5.33)\*. In nonmetropolitan areas, the AAMR also increased, from 3.38 (95% CI: 3.17–3.58) in 1999 to 11.88 (95% CI: 11.52–12.25) in 2020. The AAPC was 5.69 (95% CI: 5.37–6.01)\*. APC by time period: 1999–2020: APC = 5.69 (95% CI: 5.37–6.01)\*. See Figure 4 and Supplementary Table S1 for details.

### 3.5 Distribution of state-level mortality and trends in the U.S

In the spatial analysis, we mapped state-level total deaths (Figure 5A) and AAMR (Figure 5B) in 2020, as well as the percentage change in deaths (Figure 5C) and AAPC (Figure 5D)



from 1999 to 2020. State-level distributions of deaths and AAMR were displayed using discrete classification with fixed legends, ensuring comparability across states. While some states reported the highest absolute number of deaths in 2020, their corresponding AAMR values were not always the highest, underscoring the influence of population size on absolute mortality counts (Supplementary Table S1). In addition, Oklahoma exhibited one of the steepest increases in age-adjusted mortality rate (AAMR) nationwide, rising from 3.01 (95% CI: 2.33–3.82) in 1999 to 27.55 (95% CI: 25.58–29.52) in 2020, with an AAPC of 10.52% (95% CI: 7.87–13.25). The percentage change in deaths over 1999–2020 was visualized with a warm color scale, highlighting substantial heterogeneity in growth magnitude across states. Most states experienced an increase, but the extent of change varied considerably. Finally, the distribution of AAPC was represented on a blue-to-red gradient, with positive values indicating an increase. Most states exhibited positive AAPC, consistent with a long-term upward trend (Supplementary Table S1).

## 4 Discussion

From 1999 to 2020, AAMR increased substantially across all examined sex, racial/ethnic, regional, urbanization, and state subgroups. The consistent upward trajectory, with notable

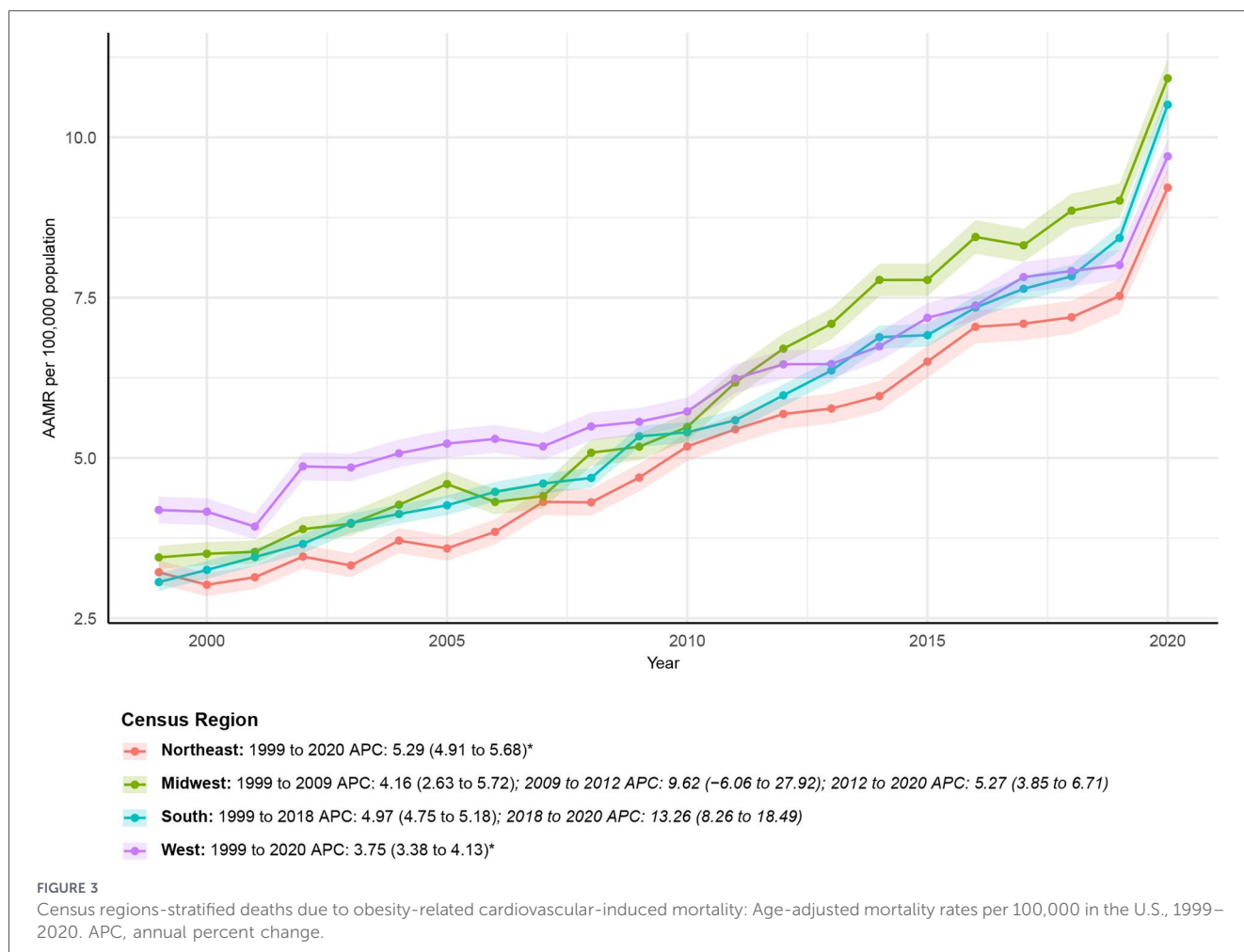
acceleration after 2018, indicates an expanding mortality burden rather than short-term fluctuation.

Males consistently demonstrated higher AAMR than females, with a steeper overall increase (AAPC 5.94% vs. 4.40%). This persistent excess male mortality parallels national patterns in cardiometabolic and cardiovascular disease, where male mortality is typically 1.3–1.8 times higher than female mortality (17, 18). The male-to-female ratio remained stable over time, indicating sustained disparity.

In contrast, females experienced a marked acceleration after 2018 (APC 10.85%), representing a shift from prior gradual growth. National data documenting rising obesity, metabolic dysfunction, and psychosocial stressors among women during the late 2010s temporally align with this inflection (19, 20). Although causal inference cannot be drawn from aggregate data, the timing suggests evolving risk dynamics.

NH Black individuals had the highest AAMR throughout the study period, whereas NH Other groups had the lowest rates. The magnitude of disparity is consistent with prior national analyses demonstrating disproportionate chronic disease mortality among NH Black populations (21, 22). Notably, acceleration after 2018 was most pronounced among Hispanic and NH Black populations, indicating widening recent disparities.

These patterns align with extensive evidence linking structural inequities—including differential access to preventive care, higher comorbidity burden, and neighborhood-level socioeconomic



disadvantage—to racialized mortality gradients (23, 24). While mechanisms cannot be directly assessed in this ecological framework, the consistency with established literature supports the robustness of these observations.

Geographic heterogeneity was substantial. The South and Midwest exhibited the highest AAPC values, with the South showing a sharp post-2018 acceleration (APC 13.26%) exceeding national growth rates. These regions have historically carried higher burdens of obesity, diabetes, and limited healthcare access (25, 26), suggesting persistence and intensification of preexisting vulnerabilities.

Mortality increased across all urbanization levels; however, nonmetropolitan areas experienced faster growth than metropolitan areas (AAPC 5.69% vs. 4.98%). This widening urban–rural gap mirrors national reports of accelerated mortality growth in rural communities across multiple causes (27, 28). Structural factors—including aging populations, hospital closures, and limited specialty care—likely contribute to this divergence.

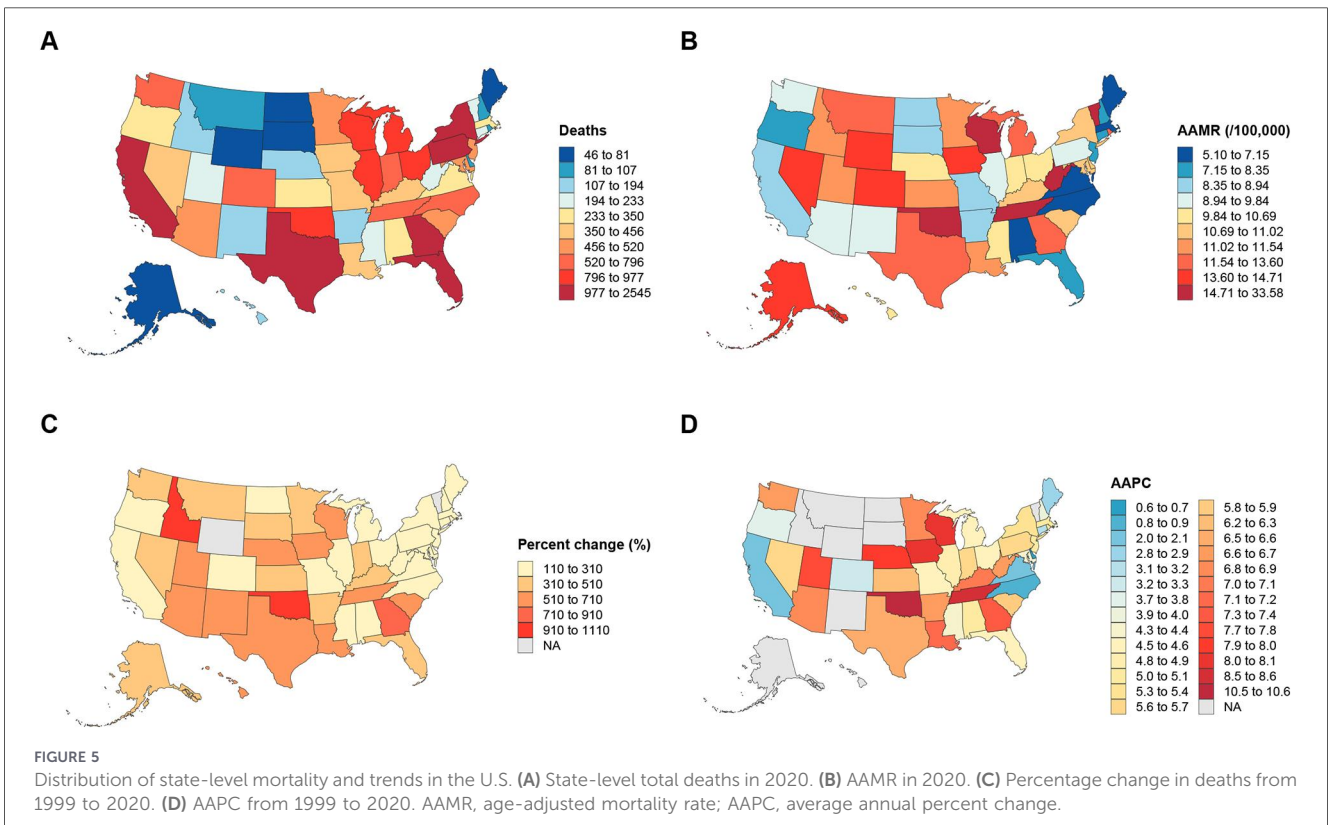
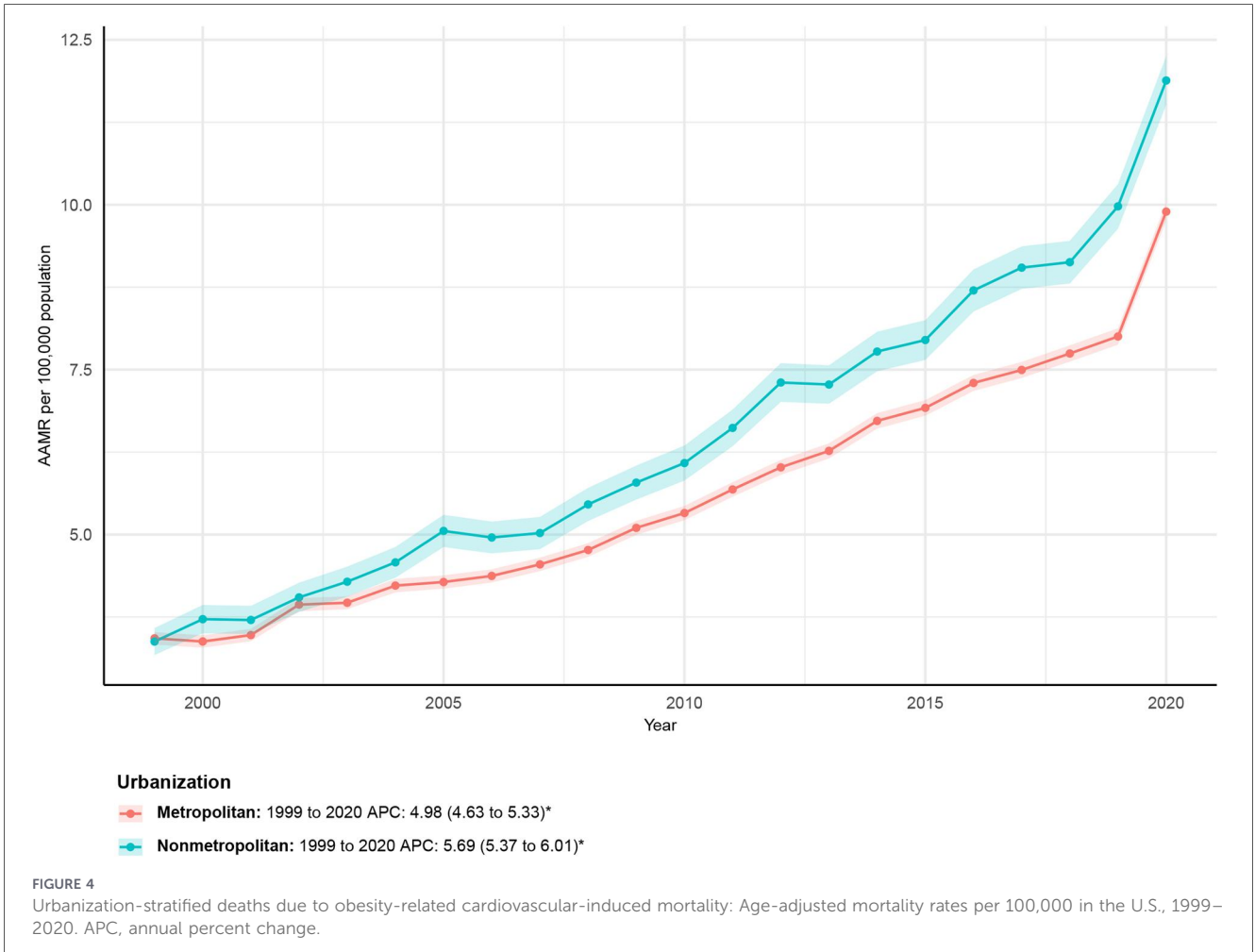
Oklahoma exhibited one of the steepest increases nationwide, with an AAPC nearly fourfold higher than the national average. Public health surveillance consistently reports higher prevalence of obesity, smoking, uninsured status, and cardiometabolic disease in Oklahoma compared with national averages (29–31). The convergence of elevated risk factor burden and accelerated mortality growth highlights substantial state-level vulnerability.

Although causation cannot be inferred, these findings underscore the importance of locally tailored interventions.

The uniform upward trend across demographic and geographic strata suggests systemic drivers rather than isolated population effects. The post-2018 acceleration across multiple subgroups warrants focused investigation, including potential contributions from healthcare disruptions, shifts in preventive care utilization, and early indirect effects of the COVID-19 pandemic (32). Future studies integrating individual-level risk factor and healthcare utilization data are needed to clarify causal pathways.

## Limitations

This study has several limitations. First, the analysis is ecological and based on aggregated mortality data; therefore, causal inferences at the individual level cannot be established. Second, mortality estimates rely on death certificate data, which may be subject to misclassification and potential temporal changes in coding practices. Third, the absence of individual-level clinical, socioeconomic, and behavioral information precludes adjustment for important confounders and limits mechanistic interpretation of the observed disparities. Fourth, while joinpoint regression identifies statistically significant



inflection points in temporal trends, it does not provide insight into the underlying drivers of these changes. Fifth, data were available only through 2020; thus, more recent trends require continued surveillance. Additionally, because ICD-10 codes I00–I99 encompass a broad spectrum of cardiovascular conditions with heterogeneous etiologies, the observed trends likely reflect varying contributions from different CVD subtypes. Our analysis was not designed to disentangle subtype-specific mechanisms or attribute changes to particular cardiovascular diagnoses. Despite these limitations, the use of nationally representative data with standardized methodology over a prolonged period offers robust and valuable insight into long-term mortality patterns in the U.S.

## Conclusion

Between 1999 and 2020, AAMR increased markedly across the U.S., with persistent and in some cases widening disparities by sex, ethnicity, region, urbanization, and state. The acceleration observed after 2018 suggests intensifying underlying risk dynamics. These findings highlight the need for equity-focused prevention strategies and sustained investment in healthcare access and chronic disease mitigation. Without targeted structural interventions, existing mortality gradients are likely to persist or expand.

## Data availability statement

The original contributions presented in the study are included in the article/[Supplementary Material](#), further inquiries can be directed to the corresponding author.

## Author contributions

LH: Software, Writing – original draft, Validation, Supervision, Writing – review & editing. LM: Visualization, Writing – original draft, Writing – review & editing. YW: Writing – original draft, Data curation, Writing – review & editing, Conceptualization. Z: Formal analysis, Writing – original draft, Methodology, Writing – review & editing, Project administration.

## References

- Virani SS, Alonso A, Aparicio HJ, Benjamin EJ, Bittencourt MS, Callaway CW, et al. American Heart Association Council on epidemiology and prevention statistics committee and stroke statistics subcommittee: heart disease and stroke statistics-2021 update: a report from the American Heart Association. *Circulation*. (2021) 143:e254–743. doi: 10.1161/CIR.0000000000000950
- Benjamin EJ, Muntner P, Alonso A, Bittencourt MS, Callaway CW, Carson AP, et al. American Heart Association Council on epidemiology and prevention statistics committee and stroke statistics subcommittee: heart disease and stroke statistics-2019 update: a report from the American Heart Association. *Circulation*. (2019) 139:e56–528. doi: 10.1161/CIR.0000000000000659
- Lavie CJ, McAuley PA, Church TS, Milani RV, Blair SN. Obesity and cardiovascular diseases: implications regarding fitness, fatness, and severity in the obesity paradox. *J Am Coll Cardiol*. (2014) 63:1345–54. doi: 10.1016/j.jacc.2014.01.022
- Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ, et al. American Heart Association Council on lifestyle and cardiometabolic health; council on cardiovascular and stroke nursing; council on clinical cardiology; council on epidemiology and prevention; and stroke council: obesity and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation*. (2021) 143:e984–1010. doi: 10.1161/CIR.0000000000000973
- Hotamisligil GS. Inflammation, metaflammation and immunometabolic disorders. *Nature*. (2017) 542:177–85. doi: 10.1038/nature21363
- Libby P, Ridker PM, Hansson GK. Leducq transatlantic network on atherothrombosis: inflammation in atherosclerosis: from pathophysiology to practice. *J Am Coll Cardiol*. (2009) 54:2129–38. doi: 10.1016/j.jacc.2009.09.009
- Lopez-Jimenez F, Almahmeed W, Bays H, Cuevas A, Di Angelantonio E, le Roux CW, et al. Obesity and cardiovascular disease: mechanistic insights and management

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## Supplementary material

The Supplementary Material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fcvm.2026.1736290/full#supplementary-material>

- strategies. A joint position paper by the world heart federation and world obesity federation. *Eur J Prev Cardiol.* (2022) 29:2218–37. doi: 10.1093/eurjpc/zwac187
8. Peterson LR, Herrero P, Schechtman KB, Racette SB, Waggoner AD, Kirsieva-Ware Z, et al. Effect of obesity and insulin resistance on myocardial substrate metabolism and efficiency in young women. *Circulation.* (2004) 109:2191–6. doi: 10.1161/01.CIR.0000127959.28627.F8
9. Wang Y, Beydoun MA. The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiol Rev.* (2007) 29:6–28. doi: 10.1093/epirev/mxm007
10. Ashraf H, Ashfaq H, Ashraf A. Gender and racial disparities in obesity-related cardiovascular-induced mortality in the USA, 1999–2020. *Curr Probl Cardiol.* (2024) 49:102178. doi: 10.1016/j.cpcardiol.2023.102178
11. Centers for Disease Control and Prevention (CDC). National Center for Health Statistics mortality data on CDC WONDER (2023). Available online at: <https://wonder.cdc.gov/mcd.html> (Accessed March 12, 2026).
12. ICD10Data.com. ICD10Data.com: The web's free 2019 ICD-10-CM/PCS medical coding reference (2019). Available online at: <https://www.icd10data.com/> (Accessed March 12, 2026).
13. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. STROBE Initiative: the strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *J Clin Epidemiol.* (2008) 61:344–9. doi: 10.1016/j.jclinepi.2007.11.008
14. National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention. Geographic division or region—Health, United States (2024). Available online at: <https://www.cdc.gov/nchs/hs/sources-definitions/geographic-region.htm> (Accessed March 12, 2026).
15. Ingram DD, Franco SJ. 2013 NCHS urban–rural classification scheme for counties. *Vital Health Stat.* (2014) 2:1–73.
16. Statistical Research and Applications Branch, National Cancer Institute. *Joinpoint Regression Program [Computer Software], Version 5.1.0.0* (2024). Available online at: <https://surveillance.cancer.gov/joinpoint/download> (Accessed March 13, 2026).
17. You CH, Kwon YD, Kang S. Sex differences in factors affecting hospital outpatient department visits: Korea health panel survey data from 2009 to 2016. *Int J Environ Res Public Health.* (2019) 16:5028. doi: 10.3390/ijerph16245028
18. Case A, Paxson C. Sex differences in morbidity and mortality. *Demography.* (2005) 42:189–214. doi: 10.1353/dem.2005.0011
19. Regitz-Zagrosek V. Sex and gender differences in health. *EMBO Rep.* (2012) 13:596–603. doi: 10.1038/embor.2012.87
20. Mehta LS, Beckie TM, DeVon HA, Grines CL, Krumholz HM, Johnson MN, et al. American Heart Association Cardiovascular disease in women and special populations committee of the council on clinical cardiology, council on epidemiology and prevention, council on cardiovascular and stroke nursing, and council on quality of care and outcomes research: acute myocardial infarction in women: a scientific statement from the American Heart Association. *Circulation.* (2016) 133:916–47. doi: 10.1161/CIR.0000000000000351
21. Williams DR, Lawrence JA, Davis BA, Vu C. Understanding how discrimination can affect health. *Health Serv Res.* (2019) 54 Suppl 2:1374–88. doi: 10.1111/1475-6773.13222
22. Gee GC, Ford CL. Structural racism and health inequities: old issues, new directions. *Du Bois Rev.* (2011) 8:115–32. doi: 10.1017/S1742058X11000130
23. Bailey ZD, Feldman JM, Bassett MT. How structural racism works—racist policies as a root cause of U.S. Racial health inequities. *N Engl J Med.* (2021) 384:768–73. doi: 10.1056/NEJMms2025396
24. Dwyer-Lindgren L, Bertozzi-Villa A, Stubbs RW, Morozoff C, Shirude S, Unützer J, et al. Trends and patterns of geographic variation in mortality from substance use disorders and intentional injuries among US counties, 1980–2014. *JAMA.* (2018) 319:1013–23. doi: 10.1001/jama.2018.0900
25. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in obesity among adults in the United States, 2005–2014. *JAMA.* (2016) 315:2284–91. doi: 10.1001/jama.2016.6458
26. Singh GK, Siahpush M. Widening rural–urban disparities in all-cause mortality and mortality from major causes of death in the USA, 1969–2009. *J Urban Health.* (2014) 91:272–92. doi: 10.1007/s11524-013-9847-2
27. Weeks WB, Wallace AE, Wang S, Lee A, Kazis LE. Rural–urban disparities in health-related quality of life within disease categories of veterans. *J Rural Health.* (2006) 22:204–11. doi: 10.1111/j.1748-0361.2006.00033.x
28. Koly KN, Islam MS, Reidpath DD, Saba J, Shafique S, Chowdhury MR, et al. Health-related quality of life among rural–urban migrants living in Dhaka slums: a cross-sectional survey in Bangladesh. *Int J Environ Res Public Health.* (2021) 18:10507. doi: 10.3390/ijerph181910507
29. Singh GK, Azuine RE, Siahpush M, Williams SD. Widening geographical disparities in cardiovascular disease mortality in the United States, 1969–2011. *Int J MCH AIDS.* (2015) 3:134–49.
30. Woolf SH, Aron L. *National Research Council (US) and Institute of Medicine (US): U.S. Health in International Perspective: Shorter Lives, Poorer Health.* Washington (DC): National Academies Press (US) (2013).
31. Moy E, Garcia MC, Bastian B, Rossen LM, Ingram DD, Faul M, et al. Leading causes of death in nonmetropolitan and metropolitan areas—United States, 1999–2014. *MMWR Surveill Summ.* (2017) 66:1–8. doi: 10.15585/mmwr.ss6601a1
32. Woolf SH, Chapman DA, Sabo RT, Zimmerman EB. Excess deaths from COVID-19 and other causes in the US, March 1, 2020, to January 2, 2021. *JAMA.* (2021) 325:1786–9. doi: 10.1001/jama.2021.5199