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Trends and Characteristics of Cardiovascular Disease Co- Reported with Benign Prostatic Hyperplasia Among U.S. Male Decedents: A CDC WONDER Analysis, 1999–2024

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Abstract

Background

Benign prostatic hyperplasia (BPH) is common among older men, yet national patterns of cardiovascular disease (CVD) co-reported with BPH on death certificates have not been fully characterized.

Objective

This study explored temporal trends and disparities in deaths involving cardiovascular disease co-reported with benign prostatic hyperplasia among U.S. male decedents from 1999 to 2024.

Methods

We conducted a retrospective, population-based multiple-cause-of-death analysis of male U.S. decedents using CDC WONDER mortality data from 1999 through 2024. Deaths were included when BPH (ICD-10 code N40) and diseases of the circulatory system (ICD-10 codes I00–I99) were both reported on the death certificate. Analyses examined annual death counts and age-adjusted mortality rates (AAMRs) per 100,000 population overall and by age group, race and ethnicity, U.S. Census region, urbanization, state, and place of death. Age groups were 55–64, 65–74, 75–84, and ≥ 85 years. Temporal trends were evaluated using joinpoint regression, with annual percent changes (APCs) and average annual percent changes (AAPCs).

Results

From 1999 through 2024, 88,076 cardiovascular disease deaths were co-reported with BPH among male U.S. decedents. The annual number of deaths increased from 2,035 in 1999 to 6,221 in 2024, and the overall AAMR increased from 10.05 to 16.41 per 100,000 population. The AAMR increased significantly from 1999 to 2017 (APC, 0.79% per year) and more sharply from 2017 to 2021 (APC, 10.79% per year), followed by a nonsignificant decline from 2021 to 2024 (APC, − 0.79% per year); the overall AAPC was 2.13% per year. In 2024, crude mortality rates increased with age, from 1.03 per 100,000 among individuals aged 55–64 years to 123.38 per 100,000 among those aged ≥ 85 years. AAPCs indicated significant overall increases across all analyzed racial and ethnic groups with the highest AAPC among Hispanic or Latino individuals (3.42% per year), followed by Asian or Pacific Islander (2.32%), Black or African American (2.28%), and White individuals (2.15%). The highest mean regional AAMR was observed in the West (14.24), followed by the Northeast (12.06), Midwest (11.62), and South (9.57). The Midwest had no significant net change over the study period. From 1999 through 2020, the mean AAMR was higher in nonmetropolitan than metropolitan areas (12.85 vs. 10.24). Most deaths occurred in

nursing home or long-term care facilities (33.1%), followed by decedents' homes (28.6%) and inpatient medical facilities (24.3%).

Conclusions

CVD mortality co-reported with BPH increased substantially among U.S. male decedents from 1999 through 2024, particularly among men ≥ 85 years with marked variation by race and ethnicity, region, urbanization, and place of death. These findings support continued attention to cardiovascular health among older men with BPH and highlight geographic and care-setting differences that may warrant further investigation.

Trial registration

Not applicable. This study was a retrospective analysis of publicly available, de-identified mortality data from the CDC WONDER database and did not involve a clinical trial.

1. INTRODUCTION

Benign prostatic hyperplasia (BPH) is one of the most common urological diseases affecting men and is characterized by nonmalignant enlargement of the prostate that may cause lower urinary tract symptoms (LUTS) and bladder outlet obstruction (1, 2). Its prevalence increases substantially with advancing age, making BPH an important contributor to morbidity among older men (3). As BPH commonly occurs in the setting of multimorbidity, its clinical relevance extends to other chronic conditions that become increasingly prevalent with aging beyond urinary symptoms (4).

Cardiovascular disease (CVD) remains a major cause of mortality in the United States, with its burden increasing markedly with advancing age (5, 6). BPH and CVD share several important risk factors, including hypertension, obesity, diabetes and dyslipidemia (7). Men with BPH have been reported to have a higher prevalence of cardiovascular risk factors than men without BPH (8). Lower urinary tract symptoms, which are commonly observed with BPH, have also been associated with an increased risk of cardiovascular conditions (9). These findings highlight a potential link between BPH, LUTS, and cardiovascular health. Several mechanisms may contribute to the observed relationship such as shared metabolic abnormalities, vascular dysfunction, autonomic disturbances, chronic inflammation, and sleep disturbance related to nocturia (7, 10, 11). However, the relationship between BPH and CVD remains incompletely understood, and available evidence has not consistently established a direct causal association. Thus, the cardiovascular burden observed among men with BPH may reflect a combination of shared risk factors and advancing age instead of contribution of BPH itself (12).

Despite the clinical relevance of BPH and cardiovascular disease, national mortality patterns involving both these conditions among older men in the United States remain limited. Multiple-cause-of-death

data provide an opportunity to examine conditions reported anywhere on the death certificate, allowing a broader examination of mortality involving co-reported BPH and cardiovascular disease.

Therefore, this study aimed to evaluate temporal trends in mortality involving BPH and diseases of the circulatory system among U.S. males aged 55 years and older from 1999 through 2024 using (CDC WONDER) Multiple Cause of Death database. We further examined mortality patterns across age, race/ethnicity, geographic region, and urbanization to characterize demographic and geographic differences in this co-reported mortality burden. Examining temporal and population-level patterns may help to establish targeted prevention strategies for older men particularly in demographic and geographic groups that have a greater mortality burden. These findings may also be informative to guide future research and improve more integrated approaches to cardiovascular risk assessment and management in men with BPH.

2. METHODS

2.1. Research Design and Data Source

We conducted a retrospective, population-based analysis of U.S. mortality data obtained from the Centers for Disease Control and Prevention (CDC) Wide-Ranging Online Data for Epidemiologic Research (WONDER) Multiple Cause of Death (MCOD) database for 1999–2024 (13). The database is derived from death certificates collected across all 50 U.S. states and the District of Columbia and includes information on both the underlying and contributing causes of death.

The Multiple Cause of Death (MCOD) database was used to identify decedents in whom both BPH and CVD were reported anywhere on the death certificate, regardless of whether either condition was recorded as the underlying or a contributing cause of death. This approach was selected to capture the broader mortality burden of cardiovascular disease co-reported with BPH and to account for the contribution of both conditions to mortality beyond deaths attributed to a single underlying cause. Deaths involving benign prostatic hyperplasia (BPH) were identified using International Classification of Diseases, Tenth Revision (ICD-10) code N40 (14). Cardiovascular disease (CVD) was defined using ICD-10 codes I00–I99, representing diseases of the circulatory system (15). Informed consent and institutional review board approval were not necessary because the CDC WONDER database offers publicly accessible, de-identified mortality data. This observational study was carried out in accordance with Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) standards (16).

2.2. Study Variables

The extracted variables were year of death, age, race/ethnicity, census region, urbanization status, and place of death, with the analysis limited to male descendants. Age-stratified analyses were restricted to descendants aged ≥ 55 years and divided into four groups: 55–64 years, 65–74 years, 75–84 years, and ≥ 85 years. This age restriction was selected because BPH commonly affects older men while BPH related mortality is uncommon among younger age groups.

According to CDC WONDER criteria, race and ethnicity were categorized as non-Hispanic White, non-Hispanic Black or African American, non-Hispanic American Indian or Alaska Native, non-Hispanic Asian or Pacific Islander, and Hispanic or Latino. The Northeast, Midwest, South, and West U.S. Census regions were used for geographic analyzes (17).

Urbanization status was divided into metropolitan and non-metropolitan areas using the National Center for Health Statistics' urban-rural classification scheme. The CDC WONDER database only had county-level urbanization classifications for the years 1999–2020; data for the years after that was unavailable. Data for American Indian or Alaska Native individuals were not available for analysis because the corresponding estimates were suppressed in the CDC WONDER database due to small cell counts.

2.3. Statistical Analysis

The CDC WONDER standard approach was used to compute crude mortality rates and age-adjusted mortality rates (AAMRs) per 100,000 population. The 2000 U.S. standard population was used to standardize AAMRs (18). Temporal trends in mortality were assessed using joinpoint regression analysis. To describe changes in death rates over time, annual percent change (APC) and average annual percent change (AAPC), together with related 95% confidence intervals (CIs), were computed. The ideal number of joinpoints was determined using the Monte Carlo permutation approach; a maximum of four joinpoints was set to combine model flexibility with the risk of overfitting. The standard error estimates for each annual death rate were used to develop joinpoint regression models. The Joinpoint Regression Program's conventional error-based modeling approach was used to account for heteroscedastic errors, and an uncorrelated error structure was given. Permutation testing was used in the final model selection process to choose the model that best reflected the observed temporal trends. Age group, race/ethnicity, census region, and urbanization status were used to stratify trend analyzes. Because BPH is a disorder that only affects men, sex-stratified analyzes were not done. A two-sided p-value of less than 0.05 was considered statistically significant. The Joinpoint Regression Program, version 5.4, was used for all analyzes (19).

3. RESULTS

3.1. Overall

From 1999 to 2024, a total of 88,076 cardiovascular disease deaths were co-reported with benign prostatic hyperplasia (BPH) among U.S. male descendants. The annual number of deaths increased from 2,035 in 1999 to 6,221 in 2024, and the age-adjusted mortality rate (AAMR) rose from 10.05 to 16.41 per 100,000 population. Across the study period, the AAMR ranged from a low of 9.63 (2004) to a high of 16.53 (2021). Because BPH is a sex-specific condition, stratification by gender was not applicable to this analysis. Joinpoint regression identified three distinct trend segments for the overall AAMR: a significant increase of 0.79% per year from 1999 to 2017, followed by a sharp and significant increase of 10.79% per year from 2017 to 2021, and a non-significant decline of 0.79% per year from

2021 to 2024. Across the full study period, the AAMR increased significantly, with an average annual percent change (AAPC) of 2.13% per year (1999–2024) Fig. 1, Table 1, **Supplementary Table 1.**

Table 1
Deaths, age-adjusted mortality rates (AAMR), and temporal trends annual percent change [APC] and average annual percent change [AAPC] of Cardiovascular-Disease Co-reported with Benign prostate Hyperplasia related mortality among U.S. adults, 1999–2024

Year	APC (95% CI)	p-value for APC	AAPC (95% CI)	p-value for AAPC	Deaths	Mean Age-Adjusted Mortality Rate (95% CI)
Male						
1999 to 2017	0.79 (0.46 to 1.12)	< 0.001	2.13 (1.42 to 2.84)	< 0.001	88,076	11.59 (11.19–11.99)
2017 to 2021	10.79 (6.82 to 14.91)	< 0.001				
2021 to 2024	-0.79 (-4.04 to 2.56)	0.620				
Race						
Asian or Pacific Islander						
1999 to 2024	2.32 (1.52 to 3.12)	< 0.001	2.32 (1.52 to 3.12)	< 0.001	2,836	9.28 (7.41–11.44)
Black or African American						
1999 to 2014	0.28 (-1.21 to 1.78)	0.704	2.28 (1.15 to 3.42)	< 0.001	7,066	12.14 (10.60–13.70)
2014 to 2024	5.36 (3.36 to 7.40)	< 0.001				
White						
1999 to 2016	0.52 (0.16 to 0.87)	0.006	2.15 (1.51 to 2.80)	< 0.001	72,068	11.74 (11.29–12.19)
2016 to 2021	8.79 (6.17 to 11.47)	< 0.001				
2021 to 2024	0.82 (-2.49 to 4.23)	0.615				
Hispanic or Latino						
1999 to 2017	2.87 (2.00 to 3.75)	< 0.001	3.42 (1.50 to 5.36)	< 0.001	5,508	9.63 (8.21–11.16)
2017 to 2020	16.67 (0.47 to 35.50)	0.044				
2020 to 2024	-3.26 (-7.03 to 0.66)	0.096				

APC, annual percent change; AAPC, average annual percent change; CI, confidence interval.

Year	APC (95% CI)	p-value for APC	AAPC (95% CI)	p-value for AAPC	Deaths	Mean Age-Adjusted Mortality Rate (95% CI)
Male						
Age group						
55–64 Years						
1999 to 2009	-1.53 (-4.83 to 1.89)	0.358	2.53 (1.02 to 4.06)	< 0.001	3,160	0.68 (0.56–0.81)
2009 to 2024	5.33 (3.95 to 6.73)	< 0.001				
65–74 Years						
1999 to 2016	-0.52 (-1.11 to 0.07)	0.080	1.77 (0.77 to 2.77)	< 0.001	12,302	3.92 (3.55–4.28)
2016 to 2021	12.94 (8.73 to 17.31)	< 0.001				
2021 to 2024	-2.67 (-7.50 to 2.41)	0.279				
75–84 Years						
1999 to 2017	0.03 (-0.31 to 0.37)	0.868	1.62 (0.54 to 2.71)	0.003	29,542	18.42 (17.33–19.50)
2017 to 2020	13.19 (3.67 to 23.58)	0.008				
2020 to 2024	0.63 (-1.65 to 2.97)	0.571				
85 + Years						
1999 to 2017	1.38 (1.00 to 1.75)	< 0.001	2.52 (1.73 to 3.31)	< 0.001	43,072	85.91 (81.67–90.14)
2017 to 2021	9.59 (5.27 to 14.09)	< 0.001				
2021 to 2024	0.29 (-3.37 to 4.09)	0.872				
Census region						
Northeast						
1999 to 2018	1.94 (1.37 to 2.52)	< 0.001	2.76 (0.96 to 4.60)	0.003	17,826	12.06 (11.15–12.99)

APC, annual percent change; AAPC, average annual percent change; CI, confidence interval.

Year	APC (95% CI)	p-value for APC	AAPC (95% CI)	p-value for AAPC	Deaths	Mean Age-Adjusted Mortality Rate (95% CI)
Male						
2018 to 2021	15.69 (0.59 to 33.06)	0.042				
2021 to 2024	-3.99 (-9.97 to 2.38)	0.200				
Midwest						
1999 to 2007	0.55 (-1.19 to 2.31)	0.515	0.00 (-1.40 to 1.43)	0.995	18,903	11.62 (10.76–12.48)
2007 to 2013	-4.43 (-7.65 to -1.09)	0.013				
2013 to 2022	3.70 (2.13 to 5.29)	< 0.001				
2022 to 2024	-4.76 (-16.36 to 8.47)	0.437				
South						
1999 to 2004	-1.03 (-3.34 to 1.33)	0.357	2.35 (1.16 to 3.54)	< 0.001	26,651	9.57 (8.96–10.18)
2004 to 2013	1.76 (0.74 to 2.79)	0.003				
2013 to 2016	-2.72 (-10.22 to 5.41)	0.469				
2016 to 2020	11.61 (7.79 to 15.56)	< 0.001				
2020 to 2024	2.99 (1.14 to 4.87)	0.004				
West						
1999 to 2013	0.76 (-0.18 to 1.71)	0.106	2.88 (1.76 to 4.00)	< 0.001	24,696	14.24 (13.31–15.19)
2013 to 2022	7.24 (5.65 to 8.86)	< 0.001				
2022 to 2024	-1.30 (-11.70 to 10.31)	0.807				
Urbanization						

APC, annual percent change; AAPC, average annual percent change; CI, confidence interval.

Year	APC (95% CI)	p-value for APC	AAPC (95% CI)	p-value for AAPC	Deaths	Mean Age-Adjusted Mortality Rate (95% CI)
Male						
Metropolitan						
1999 to 2017	1.11 (0.75 to 1.47)	< 0.001	2.43 (1.82 to 3.04)	< 0.001	50,760	10.24 (9.81–10.67)
2017 to 2020	10.70 (6.39 to 15.18)	< 0.001				
Non-metropolitan						
1999 to 2016	-0.49 (-1.04 to 0.06)	0.078	1.38 (0.55 to 2.21)	0.001	13,873	12.85 (11.83–13.88)
2016 to 2020	9.73 (5.44 to 14.20)	< 0.001				

APC, annual percent change; AAPC, average annual percent change; CI, confidence interval.

3.2. Stratified by Age

Crude mortality rates increased consistently with advancing age throughout the study period. In 2024, the highest crude rate was observed among descendants aged ≥ 85 years (123.38 per 100,000), followed by those aged 75–84 years (25.33), 65–74 years (5.57), and 55–64 years (1.03). This same pattern was present in 1999, when crude rates were 68.80 (≥ 85 years), 17.45 (75–84 years), 3.77 (65–74 years), and 0.54 (55–64 years) (**Supplementary Table 2).**

Joinpoint analysis showed that trends varied by age group. Among decedents aged 55–64 years, rates declined non-significantly by 1.53% per year from 1999 to 2009, then increased significantly by 5.33% per year from 2009 to 2024 (AAPC: 2.53% per year). Among those aged 65–74 years, rates declined non-significantly by 0.52% per year from 1999 to 2016, rose sharply and significantly by 12.94% per year from 2016 to 2021, and then declined non-significantly by 2.67% per year from 2021 to 2024 (AAPC: 1.77% per year). Among decedents aged 75–84 years, rates were stable from 1999 to 2017 (APC: 0.03%, not significant), increased significantly by 13.19% per year from 2017 to 2020, and then remained stable from 2020 to 2024 (APC: 0.63%, not significant) (AAPC: 1.62% per year). Among decedents aged ≥ 85 years, rates increased significantly by 1.38% per year from 1999 to 2017 and by 9.59% per year from 2017 to 2021, before stabilizing from 2021 to 2024 (APC: 0.29%, not significant) (AAPC: 2.52% per year). AAPC values were statistically significant for all four age groups ($p < 0.05$) Fig. 2, Table 1,

3.3. Stratified by Race/Ethnicity

Across racial and ethnic groups, the highest mean AAMR over the study period was observed among Black or African American individuals (12.14), followed by White (11.74), Hispanic or Latino (9.63), and

Asian or Pacific Islander (9.28) populations. By 2024, the highest AAMR was observed among White individuals (16.88), followed by Black or African American (16.48), Hispanic or Latino (14.68), and Asian or Pacific Islander (11.26) individuals (**Supplementary Table 3).**

Joinpoint analysis showed that AAMRs increased significantly across all four racial and ethnic groups over the full study period. Among Asian or Pacific Islander individuals, rates increased significantly throughout the period at 2.32% per year (1999–2024; a single segment with no joinpoints). Among Black or African American individuals, rates were stable from 1999 to 2014 (APC: 0.28%, not significant), then increased significantly by 5.36% per year from 2014 to 2024 (AAPC: 2.28% per year). Among White individuals, rates increased significantly by 0.52% per year from 1999 to 2016, rose sharply by 8.79% per year from 2016 to 2021, and then stabilized from 2021 to 2024 (APC: 0.82%, not significant) (AAPC: 2.15% per year). Among Hispanic or Latino individuals, rates increased significantly by 2.87% per year from 1999 to 2017 and by 16.67% per year from 2017 to 2020, before declining non-significantly by 3.26% per year from 2020 to 2024 (AAPC: 3.42% per year) Fig. 3, Table 1.

3.4. Stratified by Census Region

Across census regions, the highest mean AAMR (1999–2024) was observed in the West (14.24), followed by the Northeast (12.06), Midwest (11.62), and South (9.57) **(Supplementary Table 4).**

Joinpoint analysis revealed distinct patterns by region. In the Northeast, rates increased significantly by 1.94% per year from 1999 to 2018 and by 15.69% per year from 2018 to 2021, before declining non-significantly by 3.99% per year from 2021 to 2024 (AAPC: 2.76% per year). In the Midwest, rates were stable from 1999 to 2007 (APC: 0.55%, not significant), declined significantly by 4.43% per year from 2007 to 2013, increased significantly by 3.70% per year from 2013 to 2022, and then declined non-significantly by 4.76% per year from 2022 to 2024; over the full period the AAPC for the Midwest was not statistically significant (0.005% per year, p = 0.995), indicating no net change from 1999 to 2024. In the South, rates declined non-significantly from 1999 to 2004 (APC: −1.03%, not significant), increased significantly by 1.76% per year from 2004 to 2013, declined non-significantly from 2013 to 2016 (APC: −2.72%), rose sharply and significantly by 11.61% per year from 2016 to 2020, and increased significantly by 2.99% per year from 2020 to 2024 (AAPC: 2.35% per year, 1999–2024). In the West, rates were stable from 1999 to 2013 (APC: 0.76%, not significant), increased significantly by 7.24% per year from 2013 to 2022, and then declined non-significantly by 1.30% per year from 2022 to 2024 (AAPC: 2.88% per year) Fig. 4, Table 1.

3.5. Stratified by Urbanization

Across urbanization categories, the mean AAMR between 1999 and 2020 was higher in non-metropolitan areas (12.85) than in metropolitan areas (10.24) **(Supplementary Table 5).**

Joinpoint analysis showed that in metropolitan areas, rates increased significantly by 1.11% per year from 1999 to 2017 and by 10.70% per year from 2017 to 2020 (AAPC: 2.43% per year, 1999–2020). In

non-metropolitan areas, rates declined non-significantly by 0.49% per year from 1999 to 2016, followed by a significant increase of 9.73% per year from 2016 to 2020 (AAPC: 1.38% per year, 1999–2020) Fig. 5, Table 1.

3.6. Stratified by State

In 2020, the lowest AAMRs were observed in Nevada (4.26), followed by Louisiana (4.77), Utah (5.20), Georgia (5.64), and Florida (5.78). In contrast, the highest AAMRs were reported in Vermont (21.08), West Virginia (20.53), Ohio (18.80), California (16.64), and Alaska (16.23). In 2024, the lowest AAMR was observed in Massachusetts (5.62), followed by Illinois (6.50), Alabama (6.67), Alaska (7.10), and Delaware (7.29). The highest AAMRs were observed in Wyoming (53.30), Colorado (44.62), Oregon (27.19), Minnesota (26.90), and Maryland (26.30) **(Supplementary Table 6).**

3.7. Stratified by Place of Death

Place-of-death analysis (1999–2024) revealed that the majority of deaths occurred in nursing home or long-term care facilities (33.1%), followed by decedents' homes (28.6%) and medical facility inpatient settings (24.3%). Medical facility outpatient or emergency department settings accounted for 4.8% of deaths, while other locations accounted for 4.7%. Hospice facilities accounted for 3.9% of deaths, whereas medical facility dead-on-arrival (0.4%) and medical facility status unknown (< 0.1%) comprised only a small proportion of deaths. Place of death was unknown for at least 127 decedents in 1999–2020; the corresponding count for 2021–2024 was suppressed, accounting for the 6-death difference between the overall total (88,076) and the place-of-death total (88,070) (**Supplementary Table 7).**

4. DISCUSSION

This study provides an extensive 25-year analysis of mortality trends among individuals with Cardiovascular Diseases (CVD) and concomitant benign prostatic hyperplasia (BPH). Based on data from the CDC WONDER database, we report several important findings. A total of 88,076 deaths were identified between 1999 and 2024, with an overall increase in BPH and CVD-related mortality over the study period. Mortality rates were highest among Black or African American and White individuals, whereas Hispanic or Latino and non-Hispanic Asian or Pacific Islander populations consistently had lower mortality rates. Crude mortality rates increased consistently with advancing age throughout the study period. Geographically, highest AAMR was observed in the Western region followed by Northeast, Midwest and Southern regions. Mortality was higher in non-metropolitan areas than in metropolitan areas. Additionally, the majority of deaths occurred in nursing home or long-term care facilities, followed by decedents' homes and medical facility inpatient settings. These patterns highlight persistent demographic and geographic disparities contributing to the rising burden of CVD- related mortality among adults with BPH.

Central illustration

Our findings are consistent with existing literature linking BPH to an elevated risk of developing CVD. In a retrospective cohort study with 2,370,986 patients, Suzuki et al (8) reported that BPH was associated with a significantly increased risk of developing angina, stroke, heart failure, and atrial fibrillation. Similarly, in a Chinese study by Wang et al, men with BPH had an increased risk of developing CVD, heart disease and stroke (20). A meta-analysis by Gacci et al reported that patients with moderate-to-severe Lower Urinary Tract Symptoms (LUTS) from BPH were associated with 68% higher odds of developing MACE (9). Several biological pathways can explain the link between BPH and CVD. Notably, nocturia from BPH leads to sleep dysregulation which results in circadian dysregulation and ANS imbalance(21, 22). Research has shown that circadian disruption has been associated with reduced heart rate variability (HRV) leading to diminished parasympathetic regulation and/or increased sympathetic activity (22). Additionally, reduced nocturnal blood pressure dipping (nondipping), indicative of increased sympathetic activity during sleep, has been significantly associated with nocturia among men with LUTS and is an early indicator of cardiovascular disease (23–25). Furthermore, chronic low-grade inflammation may represent another potential link between BPH and CVD. Inflammatory processes are commonly observed in BPH and have been associated with greater prostate volume and more severe LUTS. Pro-inflammatory mediators, including interleukin-6, tumor necrosis factor-α, and C-reactive protein, may promote prostatic stromal proliferation and tissue remodeling, while also contributing to endothelial dysfunction and atherosclerotic processes (26, 27). BPH and CVD share several cardiometabolic risk factors, including obesity, hypertension, diabetes, and metabolic syndrome. These metabolic abnormalities have been associated with the development and progression of BPH/LUTS, while also representing established risk factors for CVD (28, 29). Advancing age may further contribute to this overlap, as age is the principal unmodifiable risk factor for BPH and is associated with an increasing burden of cardiometabolic risk factors and multimorbidity(30, 31). This is consistent with our finding that mortality rates among individuals with BPH and CVD increased consistently with advancing age throughout the study period.

Our analysis revealed a significant increase in BPH and CVD-related mortality from 1999 to 2017, followed by a sharp and significant rise from 2017 to 2021. This increase coincided with the COVID-19 pandemic, which may have disrupted access to routine cardiovascular care, including screening and medication management, potentially affecting the management of chronic cardiovascular conditions (32) Indirect effects of the pandemic, such as increased psychological stress, reduced physical activity, and unhealthy dietary behaviors, may have contributed to worsening modifiable cardiovascular risk factors (33, 34). Additionally, the pandemic substantially disrupted urological healthcare in the United States. Data from the American Urological Association Quality (AQUA) Registry demonstrated a greater than 50% decline in outpatient urology visits during the initial months of the pandemic, with nonurgent conditions and procedures experiencing particularly pronounced reductions (35). Such disruptions may have delayed routine BPH evaluation, follow-up, medication management, and treatment escalation. This is particularly relevant because discontinuation of pharmacological therapy for BPH has been associated with increased risks of BPH-related hospitalization and surgery, while delays in initiating 5-

alpha reductase inhibitor therapy have been associated with greater risks of clinical progression, acute urinary retention, and prostate-related surgery (36, 37).

Furthermore, fear of SARS-CoV-2 exposure, adherence to stay-at-home measures, appointment cancellations, and changes in healthcare-seeking behavior may have discouraged patients from seeking timely medical attention for progressive urinary and cardiovascular symptoms (38).

Significant disparities in mortality rates were observed across racial and ethnic groups, with the highest mortality among NH Black or African American adults, followed by NH White and other groups. The higher mortality observed among NH Black adults may partly reflect their greater burden of cardiometabolic risk factors. Previous studies have reported higher prevalences of obesity, diabetes, and hypertension among NH Black populations (39, 40), conditions that contribute to cardiovascular disease and may also promote BPH progression. Moreover, insulin resistance and hyperinsulinemia have been identified as independent risk factors for BPH, suggesting that metabolic dysregulation may contribute to the greater BPH burden observed in this population (29, 41).Differences in BPH management may also contribute to the observed disparity. Studies have reported that NH Black individuals are less likely to receive treatment for BPH and may have a higher risk of postoperative morbidity and mortality following surgical intervention (42–44). A retrospective analysis of approximately 7 million men in the California Medi-Cal database found that Black men were 28.6% less likely to receive medical treatment within the first year following diagnosis than men from other racial groups (45). These disparities may reflect socioeconomic determinants of health, including differences in healthcare access, financial barriers, and potential bias within healthcare settings. Financial difficulties and limited access to quality healthcare have also been associated with greater LUTS severity (46).

Circadian rhythm disruption may represent another potential contributor to cardiovascular risk and progression of BPH among NH Black individuals. Factors such as night-shift work, socioeconomic conditions, and environmental exposures to nighttime noise and artificial light may increase susceptibility to circadian disruption. Black individuals are disproportionately represented in night-shift occupations and are approximately twice as likely as White individuals to work night shifts (47, 48). Historical residential segregation has also resulted in greater exposure to environmental stressors in some marginalized communities, including nighttime noise and artificial light (49, 50). Persistent circadian disruption may adversely affect cardiometabolic health and has been associated with cardiovascular disease, potentially contributing to the observed mortality burden.

From a geographical perspective, substantial disparities were evident in our analysis.Residents of nonmetropolitan areas experienced higher mortality than those residing in metropolitan areas. These disparities may be related to limited access to specialty services, greater travel distances to healthcare facilities, and a higher burden of chronic diseases (51–53). Urologist availability has declined disproportionately in rural counties. An 18-year analysis found a 29.5% decline in urologist availability in rural counties, compared with 9.4% in metropolitan counties (54). Furthermore, rural communities may face limited access to telemedicine, as areas without local urologists are also more likely to have

inadequate broadband coverage (55). In terms of Census region, the highest AAMRs were observed in the West, followed by the Northeast, Midwest, and South. Although regional patterns of overall CVD mortality have been heterogeneous, with higher rates reported in the South (56), the relatively high burden of cardiometabolic conditions, including diabetes, metabolic disorders, and hypertension, in parts of the Western United States may contribute to the observed pattern. These conditions contribute to the progression of both CVD and BPH and can lead to the higher mortality observed in our study (57–60). However, the specific factors underlying the higher mortality observed in the West remain uncertain and warrant further investigation.

Limitations

Our study has several limitations that should be acknowledged. First, the analysis relied on death certificate data and International Classification of Diseases codes, which may be subject to underreporting and misclassification of causes of death. Errors in death certificate completion may further affect the accuracy of mortality estimates (61). Second, CDC WONDER lacks detailed information on individual-level comorbidities, medication use, and vital parameters, which may influence mortality outcomes. Third, race and ethnicity categorization in CDC WONDER was revised in 2018 to include multiracial groups, potentially limiting the comparability of race-specific mortality estimates across the study period and introducing misclassification bias. Finally, urbanization data were unavailable beyond 2020, restricting assessment of metropolitan versus nonmetropolitan mortality trends for subsequent years.

Conclusion

Our study provides a comprehensive analysis of CVD-related mortality among adults with BPH in the U.S. from 1999 to 2024, revealing a rising trend over time. Mortality rates were higher in NH Black or African American individuals, residents of the Western region, and those living in rural areas. These findings underscore the urgent need for targeted interventions to reduce the burden of BPH and CVD-related mortality and to address the persistent demographic and geographic disparities.

Declarations

Conflicts of interest:

The authors report no relationships that could be construed as a conflict of interest

Ethics approval and consent to participate:

Not applicable

Consent for publication:

Not applicable

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Author Contribution

AUTHOR CONTRIBUTIONS: HA, OR: conceptualization, supervision, formal analysis, validation, visualization, writing–original draft, writing–review and editing; SM: project administration, visualization, writing–original draft, writing–review and editing; RA,MJ: methodology, investigation, validation, writing–original draft, writing–review and editing; MV, BL: data curation, software analysis, methodology, visualization; AL, RK , MK: data curation, resources, writing–original draft and editing.

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Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Figures

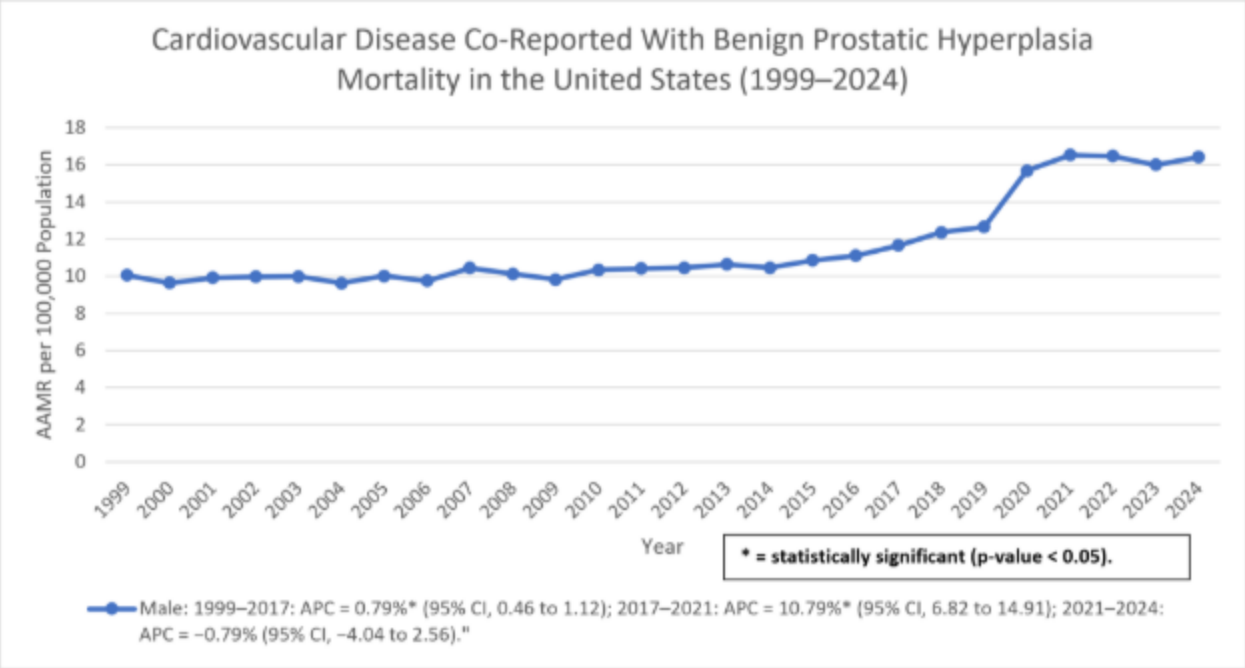


Figure 1

Overall and age-adjusted mortality rates (per 100,000 population) for cardiovascular disease co-reported with benign prostatic hyperplasia among U.S. male decedents, 1999 to 2024.

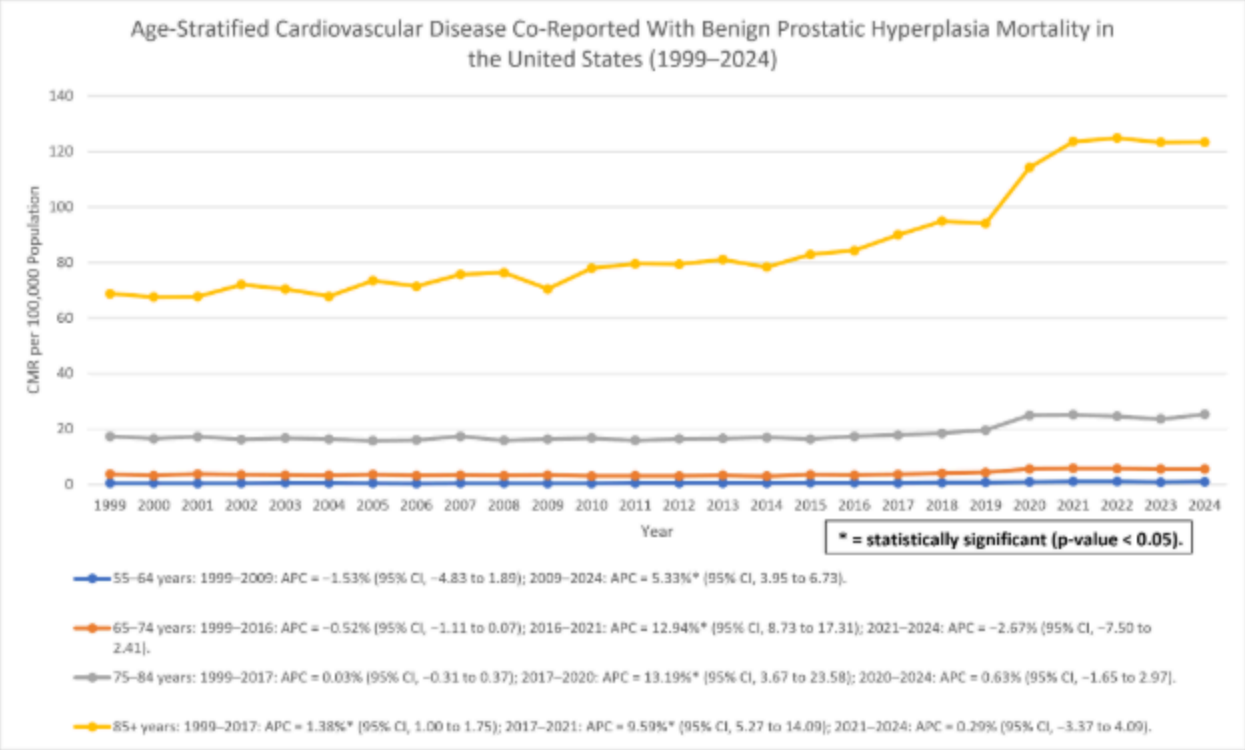


Figure 2

Age-stratified crude mortality rates (per 100,000 population) for cardiovascular disease co-reported with benign prostatic hyperplasia among U.S. male decedents, 1999–2024.

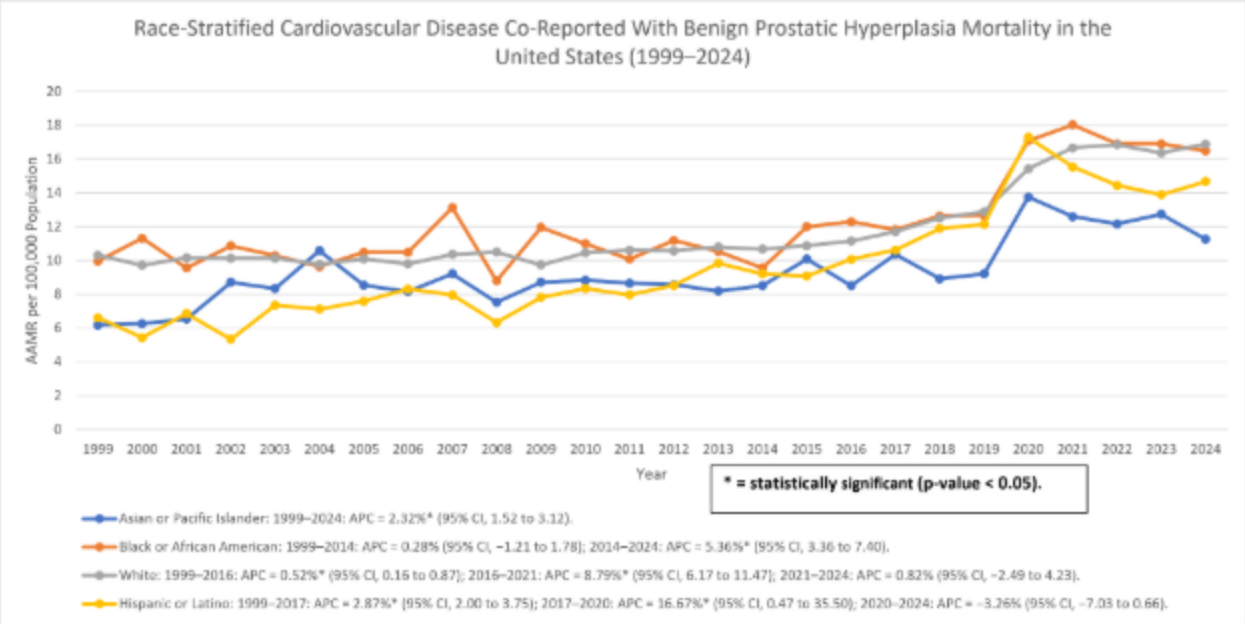


Figure 3

Race-stratified age-adjusted mortality rates (per 100,000 population) for cardiovascular disease co-reported with benign prostatic hyperplasia among U.S. male decedents, 1999 to 2024.

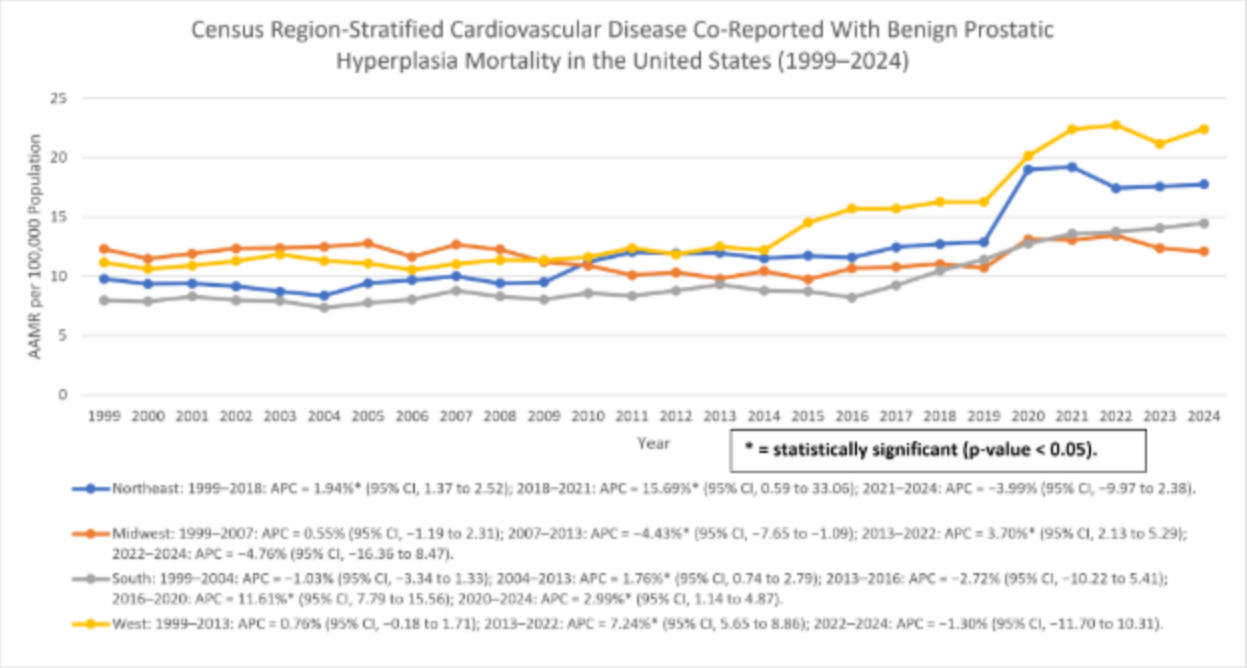


Figure 4

Census region-stratified age-adjusted mortality rates (per 100,000 population) for cardiovascular disease co-reported with benign prostatic hyperplasia among U.S. male decedents, 1999 to 2024.

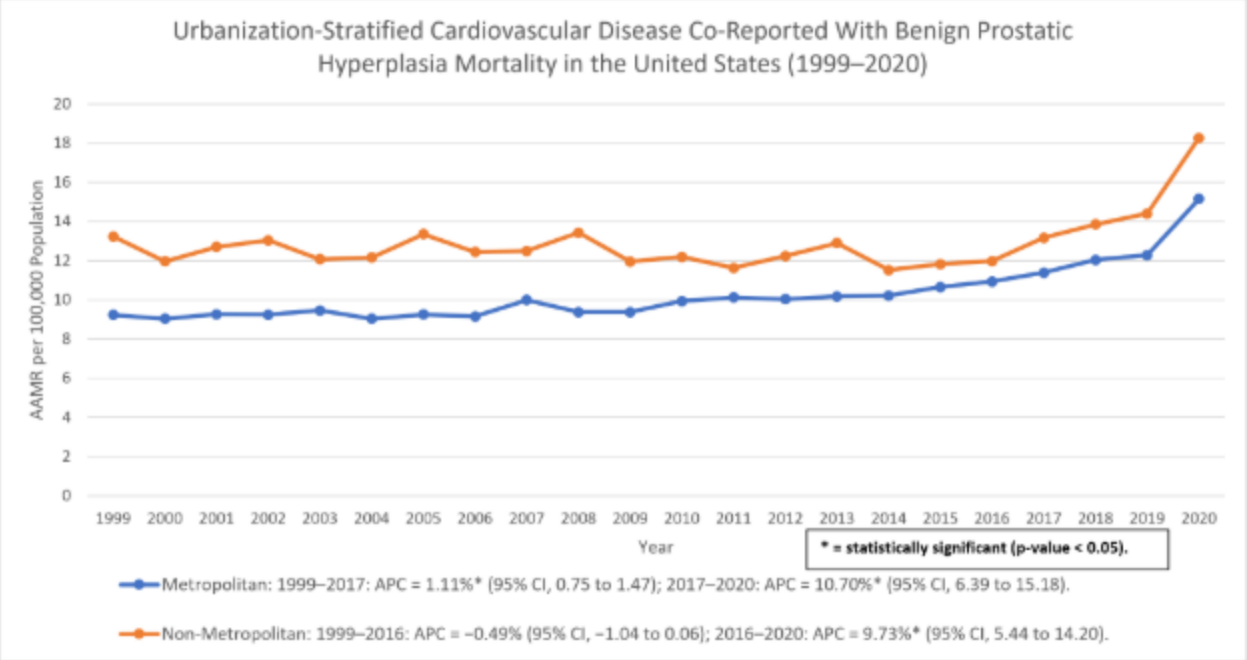


Figure 5

Urbanization-stratified age-adjusted mortality rates (per 100,000 population) for cardiovascular disease co-reported with benign prostatic hyperplasia among U.S. male decedents, 1999 to 2020.

Supplementary Files

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- SupplementarydocxBPHCVD.docx
- floatimage6.png