Circulation

AHA STATISTICAL UPDATE

2025 Heart Disease and Stroke Statistics: A Report of US and Global Data From the American Heart Association

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BACKGROUND: The American Heart Association (AHA), in conjunction with the National Institutes of Health, annually reports the most up-to-date statistics related to heart disease, stroke, and cardiovascular risk factors, including core health behaviors (smoking, physical activity, nutrition, sleep, and obesity) and health factors (cholesterol, blood pressure, glucose control, and metabolic syndrome) that contribute to cardiovascular health. The AHA Heart Disease and Stroke Statistical Update presents the latest data on a range of major clinical heart and circulatory disease conditions (including stroke, brain health, complications of pregnancy, kidney disease, congenital heart disease, rhythm disorders, sudden cardiac arrest, subclinical atherosclerosis, coronary heart disease, cardiomyopathy, heart failure, valvular disease, venous thromboembolism, and peripheral artery disease) and the associated outcomes (including quality of care, procedures, and economic costs).

METHODS: The AHA, through its Epidemiology and Prevention Statistics Committee, continuously monitors and evaluates sources of data on heart disease and stroke in the United States and globally to provide the most current information available in the annual Statistical Update with review of published literature through the year before writing. The 2025 AHA Statistical Update is the product of a full year's worth of effort in 2024 by dedicated volunteer clinicians and scientists, committed government professionals, and AHA staff members. This year's edition includes a continued focus on health equity across several key domains and enhanced global data that reflect improved methods and incorporation of ≈3000 new data sources since last year's Statistical Update.

The 2025 AHA Statistical Update uses language that conveys respect and specificity when referencing race and ethnicity. Instead of referring to groups very broadly with collective nouns (eg, Blacks, Whites), we use descriptions of race and ethnicity as adjectives (eg, Asian people, Black adults, Hispanic youths, Native American patients, White females).

As the AHA continues its focus on health equity to address structural racism, we are working to reconcile language used in previously published data sources and studies when this information is compiled in the annual Statistical Update. We strive to use terms from the original data sources or published studies (mostly from the past 5 years) that may not be as inclusive as the terms used in 2025. As style guidelines for scientific writing evolve, they will serve as guidance for data sources and publications and how they are cited in future Statistical Updates.

Supplemental Material is available at https://www.ahajournals.org/journal/doi/suppl/10.1161/CIR.0000000000001303

Circulation is available at www.ahajournals.org/journal/circ

RESULTS: Each of the chapters in the Statistical Update focuses on a different topic related to heart disease and stroke statistics.

CONCLUSIONS: The Statistical Update represents a critical resource for the lay public, policymakers, media professionals, clinicians, health care administrators, researchers, health advocates, and others seeking the best available data on these factors and conditions.

Key Words: AHA Scientific Statements ■ cardiovascular diseases ■ epidemiology ■ risk factors ■ statistics ■ stroke

CLINICAL STATEMENTS AND GUIDELINES

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SUMMARY

Each year, the American Heart Association (AHA), in conjunction with the National Institutes of Health and other government agencies, brings together in a single document the most up-to-date statistics related to HD, stroke, and cardiovascular risk factors in the AHA's Life's Essential 8 (Figure),[1] which include core health behaviors (smoking, PA, diet, and weight) and health factors (cholesterol, BP, and glucose control) that contribute to CVH. In 2024, a complementary construct that expands and includes the CVH framework to incorporate kidney health was developed by the AHA and called CKM health.[2,3] The CKM syndrome includes stages 0 to 4, which represent the pathophysiological progression from optimal CKM health to prevalent CVD.

The AHA Heart Disease and Stroke Statistical Update represents a critical resource for the lay public, policymakers, media professionals, clinicians, health care administrators, researchers, health advocates, and others seeking the best available data on these factors and conditions. CVD produces immense health and economic burdens in the United States and globally. The Statistical Update also presents the latest data on a range of major clinical heart and circulatory disease conditions (including stroke, congenital HD, rhythm disorders, subclinical atherosclerosis, CHD, HF, VHD, venous disease, and PAD) and the associated outcomes (including quality of care, procedures, and economic costs).

Each annual version of the Statistical Update undergoes revisions to include the newest nationally and globally representative available data, add additional relevant published scientific findings, remove older information, add new sections or chapters, and increase the number of ways to access and use the assembled information. This year-long process, which begins as soon as the previous Statistical Update is published, is performed by the AHA Statistics Committee faculty volunteers and staff and government agency partners. Following are a few

Figure. AHA's My Life Check–Life's Essential 8.
AHA indicates American Heart Association.
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highlights from this year's Statistical Update. Please see each chapter for references for these highlights, CIs for statistics reported, and additional information.

Cardiovascular Health (Chapter 2)

- The AHA Life's Essential 8 scores among NHANES (National Health and Nutrition Examination Survey) 2007 through 2018 participants were significantly associated with the prevalence of CVD. For every increasing 1-SD increment of the AHA Life's Essential 8 score, there was a lower odds of CVD (OR, 0.64).
- CVH score, as measured by the AHA Life's Essential 8, and components were also shown to predict MACEs (first occurrence of IHD, MI, stroke, and HF) within the UK Biobank. Individuals in the lowest quartile (least healthy) compared with the highest quartile (healthiest) had a greater risk for MACEs (HR, 2.07), which was strongest for HF. The authors estimated that a 10-point improvement in the AHA Life's Essential 8 score could have prevented 9.2% of MACEs.
- CVH measured at multiple times across the life course can be used to assess the cumulative exposure to CVH. In the FHS (Framingham Heart Study), participants who maintained a low AHA Life's Essential 8 score (below the median at each examination; AHA Life's Essential 8 scores at examination 2, 69; median at examination 6, 66) scores over an average of 13 years had the highest CVD and mortality risk (HRs, 2.3 and 1.45) compared with those who had high AHA Life's Essential 8 scores above the examination median at both examinations 2 and 6.

Smoking/Tobacco Use (Chapter 3)

- The prevalence of cigarette use in the past 30 days among middle and high school students in the United States was 1.1% and 1.9%, respectively, in 2023.
- Although there has been a consistent decline in adult and youth cigarette use in the United States in the past 2 decades, significant disparities persist. In 2023, the prevalence of past 30-day cigarette use was comparable between NH White youths (1.6%) and NH multiracial youths (1.6%) compared with Hispanic youths (2.1%). In 2021, 11.7% of NH Black adults, 5.4% of NH Asian adults, 7.7% of Hispanic adults, and 11.7% of NH White adults reported cigarette use every day or some days.
- Electronic cigarettes were the most commonly used tobacco product among adolescents in 2023; the prevalence of use in the past 30 days among middle and high school students in the United States was 4.6% and 10.0%, respectively, with 89.4% of adolescent users reporting use of flavored products and 25.2% reporting daily use.

Physical Activity and Sedentary Behavior (Chapter 4)

- The percentage of high school students who were physically active for ≥60 minutes on all 7 d/wk decreased over the past decade from 28.7% in 2011 to 23.9% in 2021. The percentage of high school students participating in muscle-strengthening activities on ≥3 d/wk decreased over the past decade from 55.6% in 2011 to 44.9% in 2021.
- According to the NHIS (National Health Interview Survey), the percentage of adults meeting the aerobic and muscle-strengthening Physical Activity Guidelines for Americans changed little from 2020 to 2022. The percentage reporting engaging in ≥150 min/wk of moderate-intensity aerobic activity, 75 min/wk of vigorous aerobic activity, or an equivalent combination was 47.9% in 2020 and 48.1% in 2022. The percentage reporting engaging in muscle-strengthening activities of at least moderate intensity and including all major muscle groups ≥2 d/wk was 31.9% in 2020 and 31.5% in 2022. The percentage of adults meeting both aerobic PA and muscle-strengthening guidelines was 25.2% in 2020 and 25.3% in 2022. It is important to note that each of these population prevalence estimates

remains below the goals set by Healthy People 2030.
- In the PROPASS consortium (Prospective Physical Activity, Sitting, and Sleep), among 15 253 adults, a cross-sectional compositional data analysis estimated that replacing less intense activities such as sedentary time, standing, and light-intensity PA, with 4 to 12 min/d of moderate- to vigorous-intensity PA was associated with meaningful cardiometabolic health benefits. For example, the minimum reallocation associated with a statistically significant reduction in body mass index was replacing 7 min/d of sedentary behavior with moderate to vigorous PA. In addition, replacing 4 min/d of light-intensity PA with moderate- to vigorous-intensity PA was associated with a significantly lower hemoglobin A1c.

Nutrition (Chapter 5)

- The evidence for benefits of healthful diet patterns on a range of cardiometabolic and other disease outcomes is strong. The core elements of a healthy dietary pattern are (1) vegetables of all types; (2) fruits, especially whole fruits; (3) grains, of which at least half are whole grains; (4) dairy, including fat-free or low-fat milk, yogurt, and cheese or lactose-free versions and fortified soy beverages and yogurt as alternatives; (5) protein foods, including lean meats, poultry and eggs, seafood, beans, peas, lentils, nuts, seeds, and soy products; and (6) oils, including vegetable oils and oils in food such as seafood and nuts. A healthy dietary pattern is also limited in foods and beverages high in added sugars, saturated fat, sodium, and alcoholic beverages.
- Most of the American population does not consume a healthy dietary pattern, as measured by the Healthy Eating Index, which is a measure of diet quality and compliance with the Dietary Guidelines for Americans. Although average diet quality has slightly improved in the past 10 years, the current average score is 59 (on a scale from 0–100). Differences in overall Healthy Eating Index scores are observed across subgroups characterized by age, sex, race and ethnicity, income, pregnancy status, and lactation status.
- Social and environmental factors observed to be associated with diet quality include education, income, race and ethnicity, neighborhood availability of supermarkets, and cost of food. The US Department of Agriculture reported that food-at-home prices will increase by 2.9% (prediction interval, 0.5%–5.3%) in 2024. This is a deceleration relative to the reported increase of 8.6% (prediction interval, 5.6%–11.8%) in 2023. The retail price of eggs increased 1.8% in January 2024, after an increase of 8.9% in December 2023, albeit 28.6% below prices seen in January 2023. The price for fresh vegetables increased by 2.9% in January 2024 but was almost 1% lower than prices seen in January 2023, at which time the prices remained elevated after a peak in December 2022. Historically, in the first quarter of each year, fresh vegetables experience a seasonal peak in prices. The prices for fresh vegetables are predicted to increase 1.9% in 2024 (prediction interval, −3.0% to 7.0%).

Overweight and Obesity (Chapter 6)

- According to US data from 2017 to 2020, the prevalence of obesity in adults was 41.8% for males and 41.8% for females; among youths 2 to 19 years of age, the prevalence of obesity was 20.9% for males and 18.5% for females.
- In 2022, all US states had an obesity prevalence >20%, 22 states had an obesity prevalence between 30% and 35%, 19 states had a prevalence of 35% to 40%, and 3 states had a prevalence of ≥40%.
- In 2022, it was estimated that among adults ≥18 years of age globally, 16% (890 million) were obese and 43% adults (2.5 billion) were overweight.

High Blood Cholesterol and Other Lipids (Chapter 7)

- Elevated lipoprotein(a), which is defined as ≥125 nmol/L or ≥50 mg/dL and is present in up to 20% of the population, is associated with increased risk of a range of CVD conditions. Recent recommendations call for measuring lipoprotein(a) at least once in every adult, although screening rates remain low in US adults.
- Among US adults with severe dyslipidemia (low-density lipoprotein cholesterol ≥190 mg/dL), 78.0% reported cholesterol evaluation in the preceding 5 years, with no significant change in screening rates between 2011 to 2012 and 2017 to March 2020.
- The landmark CLEAR outcomes trial (Cholesterol Lowering via Bempedoic Acid, an ACL-Inhibiting Regimen) testing bempedoic acid versus placebo among statin-intolerant patients showed an overall 13% relative risk reduction in the primary end point; however, recent analyses among the primary prevention patient subgroup showed a 30% relative risk reduction.

High Blood Pressure (Chapter 8)

- In 2022, the prevalence of high BP in US adults was highest in Mississippi (40.2%) and lowest in Colorado (24.6%). The prevalence of hypertension increases with age and was 28.5% among those 20 to 44 years of age, 58.6% among those 45 to 64

years of age, and 76.5% among those ≥65 years of age.
- A systematic review and meta-analysis of 136 studies with 28 612 children and young adults (between 4 and 25 years of age) showed that the prevalence of masked hypertension was 10.4%.
- A reanalysis from the Spanish Ambulatory Blood Pressure Registry with 10-year follow-up data reported that 24-hour systolic BP was more strongly associated with all-cause mortality (HR, 1.41 per 1-SD increment), which remained robust after adjustment for clinic BP. Elevated all-cause mortality risk was also reported with masked hypertension (HR, 1.24) but not with white-coat hypertension.

Diabetes (Chapter 9)

- CVDs remain the leading causes of death in individuals with diabetes.
- Composite risk factor control in those with diabetes remains suboptimal, with ≤20% at recommended levels of hemoglobin A1c, BP, and lipids.
- In a meta-analysis of 45 cohort studies, a systolic BP of ≥140 mm Hg (but not ≥130 mm Hg) compared with below this number was associated with a greater risk of cardiovascular outcomes (HR, 1.56). The risk was greater for each 10–mm Hg increment in systolic BP (HR, 1.10).

Metabolic Syndrome (Chapter 10)

- A meta-analysis including 28 193 768 participants showed that the global metabolic syndrome prevalence varied from 12.5% to 31.4% according to the definition considered. The prevalence was significantly higher in the Eastern Mediterranean region and Americas compared with other global regions and was directly related to the country's level of income.
- In the United States, according to data from NHANES 2001 to 2020, the prevalence of metabolic syndrome among youths 12 to 18 years of age was 3.73% for Hispanic youths, 1.58% for NH Black youths, and 2.78% for NH White youths. In 2017 to 2018, Mexican American adults generally had the highest prevalence of metabolic syndrome at 52.2%, followed by NH Black adults (47.6%), Asian/other adults and multirace adults (46.7%), NH White adults (46.6%) and other Hispanic adults (45.9%).
- CKM syndrome was defined as a health disorder attributable to connections among obesity, diabetes, chronic kidney disease, and CVD, including HF, AF, CHD, stroke, and PAD. In the NHANES 2011 to 2018 database, among individuals 20 to 44, 45 to 64, and ≥65 years of age, stage 0 CKM was present in 17.35%, 5.45%, and 1.80%, respectively, and risk factors and subclinical CKM (stages 1–3) were present in 80.94%, 85.95%, and 72.03%, respectively.

Adverse Pregnancy Outcomes (Chapter 11)

- The US national prevalence of gestational diabetes was 8.3% in 2021, an increase of 38% from 2016 according to birth data from the National Vital Statistics System.[4] Rates of gestational diabetes rose steadily with maternal age: In 2021, the rate for mothers ≥40 years of age was almost 6 times higher compared with that for mothers <20 years of age (15.6% versus 2.7%).
- Among 51 685 525 live births between 2007 and 2019, age-standardized hypertensive disorders of pregnancy rates doubled (38.4 to 77.8 per 1000 live births). An inflection point was observed in 2014, with an acceleration in the rate of increase of hypertensive disorders of pregnancy (from +4.1%/y before 2014 to +9.1%/y after 2014). Rates of preterm delivery and low birth weight increased significantly when co-occurring in the same pregnancy with hypertensive disorders of pregnancy. Absolute rates of adverse pregnancy outcomes were higher in NH Black individuals and in older age groups. However, similar relative increases were seen across all age and racial and ethnic groups.
- A meta-analysis of 20 studies up to 2019 showed the effectiveness of lifestyle intervention and bariatric surgery on reduced risk of hypertensive disorders of pregnancy (OR, 0.45), gestational hypertension (OR, 0.61), and preeclampsia (OR, 0.67).

Kidney Disease (Chapter 12)

- In 2021, the age-, race-, and sex-adjusted prevalence of ESKD in the United States was 2219 per million people, a decrease of 3.5% from its peak in 2019. The overall prevalence count increased slightly from 807 920 in 2020 to 808 536 in 2021, after having almost doubled from 409 226 in 2001 to 806 939 in 2019.
- The adjusted ESKD incidence decreased in all racial and ethnic groups from 2001 to 2019. After 2019, ESKD incidence increased among Black individuals but not among members of other race and ethnicity groups. In 2021, the incidence of ESKD among Black individuals was 3.8 times the incidence of NH White individuals; the incidence among Native American individuals was 2.3 times as high, and it was twice as high among Hispanic individuals.

- There is a strong and consistent association of reduced estimated glomerular filtration rate and higher urine albuminuria (even within the normal) range with incident and prevalent CVD. The addition of estimated glomerular filtration rate and urine albumin-to-creatinine ratio improves the prediction of CVD beyond traditional risk factors, and they are included in the new American Heart Association Predicting Risk of CVD Events equation. Furthermore, in an analysis of >4 million adults from 35 cohorts, inclusion of estimated glomerular filtration rate and albuminuria significantly improved prediction for CVD mortality beyond the Systematic Coronary Risk Evaluation and atherosclerotic CVD beyond the Pooled Cohort Equations in validation datasets (Δ C statistic, 0.027 and 0.010) and categorical net reclassification improvement (0.080 and 0.056, respectively).

Sleep (Chapter 13)

- Females have ≈1.5 to 2.3 higher odds of reporting insomnia symptoms than males.
- Risks of developing obstructive sleep apnea and reporting a sleep disorder are lower in individuals who consume a healthy diet.
- Obstructive sleep apnea severity is associated with higher odds of white matter hyperintensities (mild: OR, 1.70; moderate to severe: OR, 3.9; severe: OR, 4.3).

Total Cardiovascular Diseases (Chapter 14)

- According to national data, 39.5% of deaths in 2022 attributable to CVD in the United States were caused by CHD.
- In 2022, the age-adjusted mortality rate attributable to CVD in the United States was 224.3 per 100 000. The highest rate was in NH Black males (379.7 per 100 000), and the lowest rate was in NH Asian females (104.9 per 100 000).

Stroke (Cerebrovascular Diseases) (Chapter 15)

- According to BRFSS (Behavioral Risk Factor Surveillance System) 2022 data, stroke prevalence in adults was 3.4% (median) in the United States, with the lowest prevalence in Puerto Rico (1.8%) and South Dakota (2.1%) and the highest prevalence in Arkansas (4.8%).
- A population-based cohort study from Ontario, Canada, followed up 9.2 million adults for a median of 15 years (2003–2018) and observed 280 197 incident stroke or transient ischemic attack events. Women had an overall lower adjusted hazard of stroke or transient ischemic attack than men (HR, 0.82), which held true for all stroke types except subarachnoid hemorrhage (HR, 1.29).
- Among 6214 participants without history of stroke in the ELSA (English Longitudinal Study of Ageing) dataset, over 8 years of follow-up, compared with good sleep quality, poor baseline sleep quality was associated with long-term stroke risk (HR, 2.37). Worsened sleep quality was associated with stroke risk among those with good (HR, 2.08) and intermediate (HR, 2.15) sleep quality; improved sleep quality was associated with decreased stroke risk among those with poor sleep quality (HR, 0.31).

Brain Health (Chapter 16)

- Young-onset dementia, defined as symptoms before 65 years of age, was estimated in a meta-analysis to have a prevalence globally of 1.1 per 100 000 at 30 to 34 years of age, 1.0 per 100 000 at 35 to 39 years of age, 3.8 per 100 000 at 40 to 44 years of age, 6.3 per 100 000 at 45 to 49 years of age, 10.0 per 100 000 at 50 to 54 years of age, 19.2 per 100 000 at 55 to 59 years of age, and 77.4 per 100 000 at 60 to 64 years of age, although data for some age groups were limited in lower- and middle-income countries.
- In a nationally representative cohort study of US veterans (N=1 869 090; individuals ≥55 years of age receiving care in the Veterans Healthcare System between 1999 and 2019), there were significant differences in the incidence of dementia by race and ethnicity. The age-adjusted incidence of dementia was higher among underrepresented racial and ethnic groups than White racial and ethnic groups: 14.2 per 1000 person-years in American Indian or Alaska Native participants, 12.4 in Asian participants, 19.4 in Black participants, and 20.7 in Hispanic participants compared with 11.5 in White participants.
- Among 316 669 participants in the UK Biobank with 4238 incident cases of all-cause dementia (mean, 56 years of age) over a median 12.6 years of follow-up, an optimal AHA Life's Essential 8 score (score, 80–100 on a 100-point scale) was associated with a 14% lower risk of incident all-cause dementia (HR, 0.86) and 30% lower risk of vascular dementia (HR, 0.70) compared with a poor AHA Life's Essential 8 score (score of 0-49).

Congenital Cardiovascular Defects and Kawasaki Disease (Chapter 17)

- Novel technology such as remote cardiac monitoring during the interstage might help to reduce disparities in outcome. Among families from low, mid, and high socioeconomic groups enrolled in a Cardiac High Acuity Monitoring Program, survival

was no different among the highest– and mid–socioeconomic status groups (mortality OR, 0.997; and OR 1.7, respectively).
- Gaps in care are common among youths with congenital cardiovascular defects. According to results from a single-center study, roughly one-third of youths with congenital cardiovascular defects experience a >3-year gap in clinical care. Factors associated with gaps in clinical care include 14 to 29 years of age (OR, 1.20), Black race (OR, 1.50), a distance of >150 miles from the hospital (OR, 1.81), mother's education of high school or less (OR, 1.17), and low neighborhood-level opportunity (eg, high deprivation; OR, 1.22).
- Risk of multisystem inflammatory syndrome in children is higher in children who have not been vaccinated. The pooled OR for multisystem inflammatory syndrome in children in vaccinated children compared with unvaccinated children is 0.4.

Disorders of Heart Rhythm (Chapter 18)

- A nationwide, time-stratified case-crossover study using air pollution data from 322 cities in China observed an increased incidence of symptomatic AF and supraventricular tachycardia in the 24 hours after higher pollutant exposure. The change in the odds of onset of symptomatic AF associated with a 10–μg/m^3 (1 mg/m^3 for carbon monoxide) increase in air pollutant concentrations during lag 0 to 24 hours was 0.6% for fine particulate matter <2.5-μm diameter, 1.6% for NO_2, and 5.7% for carbon monoxide.
- An analysis of the UK Biobank (N=201 856) combined dietary recall of fruit juice and soft drink consumption with the polygenic risk score for AF developed in the cohort. Over a median of 9.9 years of follow-up, consumption of >2 L/wk of sugar-sweetened beverages and artificially sweetened beverages was associated with increased risk of AF in multivariable-adjusted analyses including demographic, social, clinical, and genetic risk factors (HR, 1.10 and 1.20, respectively) compared with nonconsumption of sugar-sweetened beverages or artificially sweetened beverages. Consumption of >2 L/wk of fruit juice was not associated with increased risk of AF (HR, 1.05) compared with nonconsumption. However, consumption of ≤1 L/wk of fruit juice was associated with reduced risk of AF (HR, 0.92) compared with nonconsumption.
- In Canada's single-payer health care environment, screening for AF with a single-lead ECG was determined to improve health outcomes and cost savings. A model indicated that screening 2 929 301 individuals identified 127 670 cases of AF and estimated avoidance of 12 236 strokes with a gain of 59 577 quality-adjusted life-years (0.02 per patient) over the lifetime of screened individuals.

Sudden Cardiac Arrest, Ventricular Arrhythmias, and Inherited Channelopathies (Chapter 19)

- Cardiac arrest secondary to poisoning/overdose continues to rise with a recent study from Sweden highlighting the international concern about polysubstance use as a rising cause of cause of out-of-hospital cardiac arrest. In this recent study, 5.2% of out-of-hospital cardiac arrests were secondary to poisoning primarily secondary to polysubstance use.
- Studies continue to show worse outcomes for individuals with lower socioeconomic status and from people of underrepresented races and ethnicities, highlighting the need for widespread adoption of interventions to improve resuscitative care for at-risk individuals and communities internationally. A recent study showed worse outcomes on all reportable measures for Black individuals with out-of-hospital cardiac arrest, including survival to hospital discharge (OR, 0.81), return of spontaneous circulation (OR, 0.79), and good neurological outcomes (OR, 0.80).
- Data from Get With The Guidelines–Resuscitation have shown a modest improvement in survival in pre–COVID-19 data. Results indicated that both return of spontaneous circulation (unadjusted rate, 55.0%–65.4%; aOR] per year, 1.04) and survival to hospital discharge (unadjusted rate, 16.7%–20.5%; aOR per year, 1.03) improved in an analysis of in-hospital cardiac arrest from 2006 through 2018.

Subclinical Atherosclerosis (Chapter 20)

- In a harmonized data set analysis of 19 725 Black individuals and White individuals 30 to 45 years of age from CARDIA (Coronary Artery Risk Development in Young Adults), the CAC Consortium (Coronary Artery Calcium), and the Walter Reed Cohort, the prevalence of CAC >0 among White males, Black males, White females, and Black females was 26%, 16%, 10%, and 7%, respectively.
- In an analysis of 4511 participants without known coronary artery disease who were compared with 438 individuals with a prior atherosclerotic CVD event in the CONFIRM international registry (Coronary CT Angiography Evaluation for Clinical Outcomes: An International Multicenter Registry), there was a similar MACE rate of ≈53 per 1000 patient-years for individuals with CAC >300 and those with a history of atherosclerotic CVD. Over a median of 4 years of follow-up, there was a similar

cumulative incidence of MACEs for individuals with CAC >300 or a prior atherosclerotic CVD event (*P*=0.329).
- Compared with traditional risk factors, the C statistic for CVD (C=0.756) and CHD (C=0.752) increased the most by the addition of CAC presence (CVD: C=0.776; CHD: C=0.784; *P*<0.001).

Coronary Heart Disease, Acute Coronary Syndrome, and Angina Pectoris (Chapter 21)

- An analysis of data on 17 266 adults with a history of CHD from NHIS 2006 to 2015, the prevalence of premature CHD (<65 years of age for females and <55 years of age for males) was higher among Asian Indian adults (aOR, 1.77) and other Asian adults (aOR, 1.68) than White adults.
- In a study of the 2010 to 2019 National Readmission Database, among 592 015 thirty-day readmissions and 787 008 ninety-day readmissions after index acute MI hospitalization, 30-day and 90-day all-cause readmission rates after acute MI decreased from 12.8% to 11.6% (*P*=0.0001) and 20.6% to 18.8% (*P*=0.0001), respectively.
- In a meta-analysis of 4 retrospective, nonrandomized, observational cohort studies among 184 951 patients ≥18 years of age diagnosed with non–ST-segment–elevation MI, early treatment (administered within 24 hours) with β-blockers was associated with a significant reduction in in-hospital mortality compared with no β-blocker treatment (OR, 0.43).

Cardiomyopathy and Heart Failure (Chapter 22)

- According to recent Global Burden of Disease 2021 estimates, the prevalence of cardiomyopathy or myocarditis was 5.26 million and that of HF was 55.50 million.
- In the United States, 6.7 million people (2.3%) lived with HF in 2017 to 2020, and 87 941 people died of HF in 2022.
- Treatments that improve survival are underused in HF, and age- and population-adjusted mortality in the United States has continued to rise over time, reaching 21.0 per 100 000 people in 2022.

Valvular Diseases (Chapter 23)

- The global prevalence of nonrheumatic VHD in 2021 was 28.4 million.
- Globally, the prevalence of nonrheumatic calcific aortic valve disease was 13.32 million in 2021.
- In 2021, there were 40 000 nonrheumatic degenerative mitral valve deaths globally.

Venous Thromboembolism (Deep Vein Thrombosis and Pulmonary Embolism), Chronic Venous Insufficiency, Pulmonary Hypertension (Chapter 24)

- In 2021, there were an estimated ≈523 994 cases of pulmonary embolism, ≈756 514 cases of deep vein thrombosis, and ≈1 280 508 total venous thromboembolism cases in the United States in inpatient settings. Moreover, an analysis between 2011 and 2018 involving individuals with venous thromboembolism diagnosis observed that 37.6% of all patients were treated as outpatients.
- Data from Pulmonary Embolism Response Team Consortium Registry revealed a mortality rate of 20.6% in high-risk patients and 3.7% in intermediate-risk patients. Among the high-risk individuals, in-hospital mortality rate was 42.1% for those admitted with a catastrophic pulmonary embolism.
- Centers for Disease Control and Prevention Wide-Ranging Online Data for Epidemiologic Research revealed a progressive increase in pulmonary hypertension–related mortality from 2003 to 2020. The age-standardized mortality rate per 1 000 000 patient-years rose from 17.81 in 2003 to 23.89 in 2020, driven by a pronounced increase in deaths in pulmonary hypertension groups 2 through 5.

Peripheral Artery Disease and Aortic Diseases (Chapter 25)

- The prevalence of PAD in the United States is increasing and was estimated at >12.4 million people ≥40 years of age in 2019.
- Social determinants of health, including low socioeconomic status and living in rural communities, are associated with worse outcomes for patients with PAD. For example, lower socioeconomic status (defined as living in a ZIP code with a median household income <$40 000) was associated with greater risk for amputation (HR, 1.12).
- PAD was the underlying cause of death for 11 596 individuals in 2022.

Quality of Care (Chapter 26)

- Among 237 549 survivors of acute MI in the US Nationwide Readmissions Database, sex differences in HF hospitalization risk were explored. In a propensity-matched time-to-event analysis, females had a 13% higher risk of 6-month HF readmission compared with males (6.4% versus 5.8%; HR, 1.13).
- A multicenter, nationwide cross-sectional analysis of Medicare claims data (2012–2018) examined receipt of TAVR among beneficiaries of

fee-for-service Medicare who were ≥66 years of age living in the 25 largest metropolitan core-based statistical areas. When analyzed by ZIP code, every 1-unit increase in the Distressed Communities Index score was associated with 0.4% fewer TAVR procedures performed per 100 000 Medicare beneficiaries.

- Recent work within a large US registry demonstrated that Black individuals and Hispanic individuals were 27% less likely to receive bystander cardiopulmonary resuscitation at home (38.5%) than White individuals (47.4%; aOR, 0.74) and 37% less likely to receive bystander cardiopulmonary resuscitation in public locations than White individuals (45.6% versus 60.0%; aOR, 0.63). Significant disparities in bystander cardiopulmonary resuscitation exist after controlling for income variables, regardless of the racial and ethnic composition of the location of the arrest.

Medical Procedures (Chapter 27)

- Percutaneous coronary intervention was the most common cardiovascular procedure in the United States from 2016 to 2021, followed by angioplasty and related vessel procedures (endovascular, excluding carotid) and saphenous vein harvest and other therapeutic vessel removal.
- In 2019, TAVR volumes (n=72 991) exceeded the volumes for all forms of SAVR (n=57 626). Patients undergoing TAVR in 2019 had a median of 80 years of age (interquartile range, 73–85 years of age) compared with 84 years of age (interquartile range, 78–88 years of age) in the initial years after US Food and Drug Administration approval of TAVR.
- In 2023, 4545 heart transplantations were performed in the United States, the most ever.

Economic Cost of Cardiovascular Disease (Chapter 28)

- The average annual direct and indirect costs of CVD in the United States were an estimated $417.9 billion in 2020 to 2021.
- The estimated direct costs of CVD in the United States increased from $189.7 billion in 2012 to 2013 to $233.3 billion in 2020 to 2021.
- Direct costs of hypertension by percentage of event type were largest for prescription medicines (30%) and office-based events (30%).

Conclusions

The AHA, through its Epidemiology and Prevention Statistics Committee, continuously monitors and evaluates sources of data on HD and stroke in the United States and globally to provide the most current information available in the Statistical Update. The 2025 Statistical Update is the product of a full year's worth of effort by dedicated volunteer clinicians and scientists, committed government professionals, and AHA staff members, without whom publication of this valuable resource would be impossible. Their contributions are gratefully acknowledged.

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The American Heart Association requests that this document be cited as follows: Martin SS, Aday AW, Allen NB, Almarzooq ZI, Anderson CAM, Arora P, Avery CL, Baker-Smith CM, Bansal N, Beaton AZ, Commodore-Mensah Y, Currie ME, Elkind MSV, Fan W, Generoso G, Gibbs BB, Heard DG, Hiremath S, Johansen MC, Kazi DS, Ko D, Leppert MH, Magnani JW, Michos ED, Mussolino ME, Parikh NI, Perman SM, Rezk-Hanna M, Roth GA, Shah NS, Springer MV, St-Onge M-P, Thacker EL, Urbut SM, Van Spall HGC, Voeks JH, Whelton SP, Wong ND, Wong S, Yaffe K, Palaniappan LP; on behalf of the American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Committee. 2025 Heart disease and stroke statistics: a report of US and global data from the American Heart Association. *Circulation*. 2025;151:e41–e660. doi: 10.1161/CIR.0000000000001303

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Acknowledgments

The writing group thanks its colleagues Michael Wolz at the National Heart, Lung, and Blood Institute; Kimberly Chapoy Casas, Sandeep Gill, Jason Walchok, Anokhi Dahya, Olivia Larkins, Kathie Thomas, Holly Picotte, Tian Jiang, Chandler Beon, Haoyun Hong, Sophia Zhong, and Shen Li from the AHA Data Science team; Nikki DeCleene, Laura Lara-Castorthe, and team at the Institute for Health Metrics and Evaluation at the University of Washington; and Bryan McNally and Rabab Al-Araji at the CARES program (Cardiac Arrest Registry to Enhance Survival) for their valuable contributions and review.

2. CARDIOVASCULAR HEALTH

See Tables 2-1 through 2-10 and Charts 2-1 through 2-8

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In 2010, the AHA released its 2020 Impact Goals that included 2 objectives that would guide organizational priorities over the next decade: "by 2020, to improve the CVH of all Americans by 20%, while reducing deaths from CVDs and stroke by 20%."[1] The concept of CVH was introduced in this goal and characterized by 7 components (Life's Simple 7) that include health behaviors (diet quality, PA, smoking) and health factors (blood cholesterol, BMI, BP, blood glucose). For an individual to have ideal CVH overall, they must not have clinically manifest CVD and also have optimal levels of all 7 CVH components, including not smoking, a healthy diet pattern, sufficient PA, normal body weight, and normal levels of TC, BP, and FPG in the absence of medication treatment.

To update the construct of CVH metrics on the basis of extensive evidence and insights accumulated over the decade after introduction of Life's Simple 7, the AHA released a presidential advisory in 2022 to introduce an enhanced approach to measuring CVH: AHA's Life's Essential 8.[2] The components of AHA's Life's Essential 8 include updates for the original 7 CVH components to provide metrics that more broadly recognize the scope of current health behaviors and practices with a more refined and continuous scale for better contrasting interindividual differences in CVH at a given point in time and improved tracking of intraindividual changes in CVH over time. Furthermore, sleep health was added into the CVH metrics, given its important role in human biology and sustainment of life, as well as its impact on cardiometabolic health. Table 2-1 summarizes the definitions and scoring algorithms for each of the CVH components under this new approach in both adults and youths. Recent methodological work has demonstrated that statistical models using demographics and those factors often available in routinely collected data such as EHR systems (BMI, smoking, hypertension, hypercholesterolemia and diabetes) may be able to estimate the AHA's Life's Essential 8 score, offering the potential to use the AHA's Life's Essential 8 framework even when data on some of the behavioral metrics are missing.[3] It is important to note that the AHA presidential advisory recognized psychological health and well-being and social determinants of health not merely as individual CVH metrics equivalent to one of the AHA's Life's Essential 8 metrics but as foundational factors underlying all 8 CVH components.

With this updated approach to assessing CVH, this chapter now provides statistical updates focusing on the newer CVH metrics as the health research and clinical practice fields migrate toward the use of AHA's Life's Essential 8, with attention also given to the 2 foundational influences on CVH. Changes in the leading causes and risk factors for YLDs and YLLs between 1990 and 2019, first added to the 2021 Statistical Update, highlight the influence of the components of CVH on premature death and disability in populations.

Relevance of Ideal CVH (See Table 2-2 and Charts 2-1 through 2-8)

- Multiple independent investigations have confirmed the importance of having ideal levels of CVH components, along with the overall concept of CVH, based on the original Life's Simple 7 metrics and updated to include AHA's Life's Essential 8. Findings include strong inverse, stepwise associations in the United States and in recent meta-analyses of the number of CVH components at ideal levels with all-cause mortality, CVD mortality, IHD mortality, CVD, and HF; with subclinical measures of atherosclerosis such as carotid IMT, arterial stiffness, the number of carotid artery plaques, and CAC prevalence and progression; with physical functional impairment and frailty; with cognitive decline and depression; and with longevity.[4-8] CVH has also been shown to be associated with various diseases and conditions, including periodontitis,[9] depression [10], osteoarthritis,[11] MASLD,[12] and cancer.[13] These associations were observed in all populations in the United States, including underrepresented racial and ethnic populations.[14] Similar relationships have also been seen in different populations internationally and in certain patient populations such as cancer survivors.[7,15-25]
- AHA's Life's Essential 8 scores among NHANES 2007 through 2018 participants were significantly associated with the prevalence of CVD. For every increasing 1-SD increment of AHA's Life's Essential 8 score, there was a lower odds of CVD (OR, 0.64 [95% CI, 0.60–0.69]). [26] The strength of the association between AHA's Life's Essential 8 score and CVD was strongest in younger individuals (20–59 years of age) and women. Recent work to elucidate the molecular mechanisms underlying the

The 2025 AHA Statistical Update uses language that conveys respect and specificity when referencing race and ethnicity. Instead of referring to groups very broadly with collective nouns (eg, Blacks, Whites), we use descriptions of race and ethnicity as adjectives (eg, Asian people, Black adults, Hispanic youths, Native American patients, White females).

As the AHA continues its focus on health equity to address structural racism, we are working to reconcile language used in previously published data sources and studies when this information is compiled in the annual Statistical Update. We strive to use terms from the original data sources or published studies (mostly from the past 5 years) that may not be as inclusive as the terms used in 2025. As style guidelines for scientific writing evolve, they will serve as guidance for data sources and publications and how they are cited in future Statistical Updates.

association between CVH and CVD events found that metabolomic profiles were associated with CVH and partially mediated the relationship between CVH and HF in the Framingham cohort.[27] Other biomarkers considered have included early kidney dysfunction; urinary ACR in the normal range moderated the impact of CVH on all-cause mortality.[28]

- Ideal health behaviors and ideal health factors are each independently associated with lower CVD risk in a stepwise fashion: Across any level of health behaviors, having a greater number of ideal health factors is associated with a graded decrease in risk of incident CVD, and conversely, across any level of health factors, having a greater number of ideal health behaviors is associated with a graded lowering of incident CVD risk.[29,30] Use of technology has been shown to be differentially associated with health factors compared with behaviors.[31] Data from the JHS found that among these older adults, 88% of participants used internet and mobile technology. This study demonstrated that although no association of internet and mobile technology was seen with overall CVH score, using technology to track health was associated with having ideal BP, BMI, and cholesterol (all $P<0.05$), and having ideal PA was associated with using smart devices ($P=0.012$).
- Many studies have been published in which investigators have assigned individuals a CVH score ranging from 0 to 14 on the basis of the sum of points assigned to each component of the original Life's Simple 7 CVH metrics (poor=0, intermediate=1, ideal=2 points). With this approach, data from the REGARDS cohort were used to demonstrate an inverse stepwise association between a higher CVH score component and a lower incidence of stroke. On the basis of this score, every 1-unit increase in CVH was associated with an 8% lower risk of incident stroke (HR, 0.92 [95% CI, 0.88–0.95]), with a similar effect size for White participants (HR, 0.91 [95% CI, 0.86–0.96]) and Black participants (HR, 0.93 [95% CI, 0.87–0.98]).[32] A similar association between CVH score and incidence of stroke was also observed in a large Chinese cohort.[33] Arterial stiffness mediates almost 10% of the relationship between CVH and stroke risk.[34]
- CVH score, as measured by AHA's Life's Essential 8, and components were also shown to predict MACEs (first occurrence of IHD, MI, stroke, and HF) within the UK Biobank.[35] Individuals in the lowest quartile (least healthy) compared with the highest quartile (healthiest) had a greater risk for MACEs (HR, 2.07 [95% CI, 1.99–2.16]), which was strongest for HF. The authors estimated that a 10-point improvement in AHA's Life's Essential 8 score could have prevented 9.2% of MACEs. Similar findings were seen in the Heart SCORE study, a biracial community-based population, over a median follow-up of 12 years.[36]
- By combining the 7 CVH component scores and categorizing the total score to define overall CVH (low, 0–8 points; moderate, 9–11 points; high, 12–14 points), a report pooled NHANES 2011 to 2016 data and individual-level data from 7 US community-based cohort studies to estimate the age-, sex-, and race and ethnicity–adjusted PAF of major CVD events (nonfatal MI, stroke, HF, or CVD death) associated with CVH and found that 70.0% (95% CI, 56.5%–79.9%) of major CVD events in the United States were attributable to low and moderate CVH.[37] According to the authors' estimates, 2.0 (95% CI, 1.6–2.3) million major CVD events could potentially be prevented each year if all US adults attain high CVH, and even a partial improvement in CVH scores to the moderate level among all US adults with low overall CVH could lead to a reduction of 1.2 (95% CI, 1.0–1.4) million major CVD events annually.
- A report from the CARDIA study observed a very low rate of CVD (aHR, 0.14 [95% CI, 0.09–0.22]) and CVD mortality (aHR, 0.17 [95% CI, 0.03–0.19]) over 32 years of follow-up being associated with a high (12–14 of 14 points) versus low (<8 points) level of CVH in late adolescence or early adulthood, as classified by Life's Simple 7.[38]
- CVH, as measured at multiple times across the life course, can be used to assess the cumulative exposure to CVH. In the FHS, participants who maintained low AHA's Life's Essential 8 scores (below the median at each examination; AHA's Life's Essential 8 scores at examination 2=69 and median at examination 6=66) over an average of 13 years had the highest CVD and mortality risk (HRs, 2.3 [95% CI, 1.75–3.13] and 1.45 [95% CI, 1.13–1.85]) compared with those who had high AHA's Life's Essential 8 scores above the examination median at both examinations 2 and 6.[39]
- A report from the Framingham Offspring Study showed increased risks of subsequent hypertension, diabetes, CKD, CVD, and mortality associated with having a shorter duration of ideal CVH in adulthood.[40] Another report from the ARIC study estimated CVD risk and all-cause mortality associated with patterns of overall CVH level (classified as poor, intermediate, and ideal to correspond to 0–2, 3–4, and 5–7 of the original CVH metrics at ideal levels) over time. The authors observed that participants attaining ideal CVH at the first follow-up visit had the lowest levels of CVD risks and mortality regardless of subsequent change in CVH level, and improvement from poor CVH over time was consistently associated with lower CVD risk (aHR, 0.67 [95% CI, 0.59–0.75]) and mortality (aHR, 0.80 [95% CI, 0.72–0.89]) subsequently compared with

remaining in poor CVH over time.[41] Reduced CVD risk associated with improvement of CVH over time was also observed in the elderly and very elderly populations without CVD.[42]

- Ideal CVH in parents was associated with greater CVD-free survival in offspring, and maternal CVH (0–4 versus 10–14 CVH scores) was found to be a more robust predictor of an offspring's CVD-free survival (aHR, 2.09 [95% CI, 1.50–2.92]) than paternal CVH (aHR, 1.30 [95% CI, 0.87–1.93]).[43] Furthermore, better maternal CVH at 28 weeks' gestation during pregnancy was significantly associated with better offspring CVH in early adolescence: Having just 1 poor maternal CVH metric (versus all ideal) in pregnancy was associated with a 33% lower chance of offspring attaining ideal CVH (aRR, 0.67 [95% CI, 0.58–0.77]) between 10 and 14 years of age.[44] Long-term data from the FHS show that parental CVH affects offspring DALYs such that offspring of mothers in ideal compared with poor CVH had an additional 3 healthy life-years, although no association was seen with paternal CVH.[45]
- The Cardiovascular Lifetime Risk Pooling Project showed that adults with all optimal risk factor levels (similar to having ideal CVH factor levels of cholesterol, blood sugar, and BP, as well as not smoking) have substantially longer overall and CVD-free survival than those who have poor levels of ≥1 of these CVH factors. For example, at an index of 45 years of age, males with optimal risk factor profiles lived on average 14 years longer free of all CVD events and 12 years longer overall than people with ≥2 risk factors.[46] A large community-based prospective study in China showed that greater CVH was associated with lower lifetime risk of CVD and that improvement in CVH could lower the lifetime risk of CVD and prolong the years of life free of CVD.[47,48] Another report based on a large dataset from the UK Biobank found that having ideal CVH compared with poor CVH attenuated the all-cause and cardiometabolic disease–related mortality for males and females and was associated with life expectancy gains of 5.50 years (95% CI, 3.94–7.05) for males and 4.20 years (95% CI, 2.77–5.62) for females at an index of 45 years of age among participants with cardiometabolic diseases and correspondingly 4.55 years (95% CI, 3.62–5.48) in males and 4.89 years (95% CI, 3.99–5.79) in females for people without cardiometabolic diseases.[49]
- Better CVH as defined by both the Life's Simple 7 and AHA's Life's Essential 8 scores is associated with less subclinical vascular disease,[8,18] better global cognitive and domain-specific performance and cognitive function,[50] higher incidence of MCI,[51] slower cognitive decline,[53] greater total brain volume, lower WMH volume, greater hippocampal volume,[54] and lower hazard of subsequent dementia.[55–57] Among participants of the FHS, having favorable CVH was associated with a marginally lower risk of dementia (HR, 0.45 [95% CI, 0.20–1.01]).[55] A recent systematic review suggests that CVH is associated with incident dementia in a linear manner; however, the shape of the relationship differs, depending on when the risk factors are measured, with a linear relationship with midlife risk factors and a more J-shaped relationship with older-age risk factors.[58] At 5 years of age, children with better CVH have greater neurodevelopment as measured by the intelligence quotient.[59] Data from the longitudinal study ELSA-Brazil found that higher baseline AHA's Life's Essential 8 scores were associated with slower decline in global cognition, memory, verbal fluency, and the Trail-Making Test B.[53] Apolipoprotein E carrier status[54] and acculturation[60] have been demonstrated to modify the effects of CVH on cognition.
- Better CVH is also associated with fewer depressive symptoms,[61–63] lower risks of proteinuria[64] and chronic obstructive pulmonary disease,[65] lower risk of AF,[66] and lower odds of having elevated resting heart rate.[67] Using the CVH scoring approach, the FHS demonstrated significantly lower odds of prevalent hepatic steatosis associated with more favorable CVH scores, and the decrease of liver fat associated with more favorable CVH scores was greater among people with a higher GRS for MASLD.[68] In addition, a study based on NHANES data showed significantly decreased odds of ocular diseases (OR, 0.91 [95% CI, 0.87–0.95]), defined as age-related macular degeneration, any retinopathy, and cataract or glaucoma, and odds of diabetic retinopathy (OR, 0.71 [95% CI, 0.66–0.76]) associated with each 1-unit increase in CVH among US adults.[69]
- CVH has consistently been associated with frailty and multimorbidity in later life.[70] Better CVH in midlife was associated with a lower prevalence of frailty in a large community-based cohort study, ARIC,[71] such that for every 1-unit greater midlife Life's Simple 7 CVH score, there was a 37% higher relative prevalence of being in robust health as opposed to being frail (relative PR, 1.37 [95% CI, 1.30–1.44]). It is important to note that the UK Biobank has also shown that frailty and poor psychosocial health modify the relationship of CVH with CVD such that individuals who are both frail or in poor psychosocial health (social isolation and loneliness) and in poor CVH have the greatest risk of CVD.[70,72] Having ≥5 ideal Life's Simple 7 metrics was associated with a lower odds of having multiple disabilities within the 2017 to 2019 BRFSS data.[73] Among adults ≥65 years of age with ≥5 ideal CVH components, 78.8% (95% CI, 77.6%–79.9%) had

no disabilities compared with only 61.2% (95% CI, 60.1%–61.9%) among those with <5 ideal metrics.

- According to NHANES 1999 to 2006 data, several social risk factors (low family income, low education level, underrepresented racial groups, and single-living status) were related to lower likelihood of attaining better CVH as measured by Life's Simple 7 scores.[74] A recent report from the ARIC study found that people of Black race (versus White race: OR, 0.68 [95% CI, 0.57–0.80]), with low income (OR, 0.71 [95% CI, 0.57–0.87]), or with low education (OR, 0.65 [95% CI, 0.53–0.79]) were at higher odds of having worsening CVH over time,[75] whereas analysis of NHANES data from 2013 to 2016 found that the association between educational attainment and likelihood of ideal CVH differed by race and ethnicity, underscoring the need for elucidating specific barriers preventing achievement of CVH across different racial and ethnic subgroups in the population.[76,77] A recent publication from the MESA study found that greater social disadvantage as measured by an aggregated score across 5 social determinants of health domains was associated with greater odds of unfavorable CVH risk factors, including hypertension, diabetes, smoking, and obesity, and higher risk of CVD, consistent with the notion of social determinants of health as a foundational factor for CVH.[78] The MESA study also found that health literacy, which is highly disparate by race and ethnicity and SES, was associated with Life's Simple 7 scores in older age such that limited personal health literacy had a 31% lower odds of optimal Life's Simple 7 score (OR, 0.69 [95% CI, 0.50–0.95]).[79]
- Other recent reports on CVH disparity include a study focused on people with serious mental illness, which found that individuals of underrepresented races and ethnicities had significantly lower CVH scores based on 5 of the Life's Simple 7 components.[80] Data from BRFSS identifying racial and ethnic and geographic disparities in CVH among females of childbearing age in the United States showed that NH Black females were found to have lower adjusted odds (OR, 0.54 [95% CI, 0.46–0.63]) of attaining ideal CVH compared with NH White females, whereas 5 spatial clusters in the Southwest, South, Midwest, and Mid-Atlantic regions were identified as having significantly lower prevalence of ideal CVH.[81] A systematic review and meta-analysis summarized the finding on demographic differences and socioeconomic disparities in ideal CVH in the literature through June 2020, with females having a significantly higher prevalence of ideal smoking (81% versus 60% in males), BP (41% versus 30% in males), and overall CVH (6% versus 3% in males) and people with higher education and individuals who were economically more affluent being more likely to have ideal CVH.[82] In addition to these broad markers of SES, NHANES data shows disparities in CVH among those who have lower household food security even among those that participate in Supplemental Nutrition Assistance Program with Life's Simple 7 mean CVH scores for those with high, marginal, low and very low food security of 66.9 (SD, 0.4), 65.4 (SD, 0.6), 63.9 (SD, 0.8), and 62.3 (SD, 0.8), respectively.[83]
- Neighborhood factors and contextual relationships have been linked to health disparities in CVH, but more research is needed to better understand these complex relationships.[84] A cross-sectional study from REGARDS found that neighborhood characteristics mediated a portion of the racial disparities in ideal CVH such that neighborhood physical environment, neighborhood safety, neighborhood social cohesion, and discrimination explained 5%, 6%, 1%, and 11%, respectively, of the racial disparities in CVH.[85] This and other recent reports on the association between better neighborhood perceptions and higher CVH score in Black communities[86,87] and the relationship between greater perceived social status and higher CVH score in the Hispanic/Latino population in the United States[88] are some examples of effort toward identifying complex relationships between demographic and socioeconomic factors and attaining ideal CVH. A recently published narrative review[89] described knowledge gaps and outlined potential steps toward equity in CVH, which is the objective of the interim[90] and longer-term[91] Impact Goals set forth by the AHA.
- Reproductive factors, including higher BMI in pregnancy, greater gestational weight gain, and a history of infertility, have been associated with worse CVH in middle-aged females.[92,93] Among Project Viva participants, 34% of female individuals had experienced infertility, and those with a history of infertility had an average CVH score that was 2.94 points lower (95% CI, −5.13 to −0.74) compared with those without a history of infertility after adjustment for demographics, SES, and reproductive factors.[93]
- Having more ideal CVH components in middle age has been associated with lower non-CVD and CVD health care costs in later life.[94] An investigation of 4906 participants in the Cooper Center Longitudinal Study reported that participants with ≥5 ideal CVH components in the original metrics exhibited 24.9% (95% CI, 11.7%–36.0%) lower median annual non-CVD costs and 74.5% (95% CI, 57.5%–84.7%) lower median CVD costs than those with ≤2 ideal CVH components.[94] A report from a large, ethnically diverse insured population found that people with 6 or 7 and those with 3 to 5 of the CVH components in the ideal category had a $2021 and $940 lower annual mean health care expenditure, respectively, than those with 0 to 2 ideal health components.[95]

- The 2022 AHA presidential advisory on AHA's Life's Essential 8 also provided summaries of knowledge gained on CVH since 2010 and evidence supporting psychological health and well-being, as well as social determinants, as foundational factors for CVH.[2] Since the publication of the AHA presidential advisory on AHA's Life's Essential 8, Lloyd-Jones et al[96] reported CVH prevalence estimates in the United States, analyzing NHANES data from 2013 to 2018 using the updated metrics. Independently, another report using 6 cycles of NHANES data from 2007 to 2018 focused on trajectories of overall and component CVH scores under the updated metrics for US adults between 18 and 44 years of age by sex and race and ethnicity subgroups, over 3 periods in time, each with 2 cycles, 4 years of NHANES data combined.[97] Similar statistics produced by the AHA using NHANES data are presented in the next section (Table 2-2 and Charts 2-1 through 2-8).
- Two additional reports used NHANES data from 2005 to 2018 to quantify CVH using the AHA Life's Essential 8 metrics and linked the NHANES participants to the National Death Index mortality file through 2019 to study the association between CVH and life expectancy, as well as all-cause and CVD-specific mortality. From 23 003 US adults 20 to 79 years of age, the life expectancy at 50 years of age, for example, the average number of years of life remaining after age 50, was estimated to be 27.3 years (95% CI, 26.1–28.4) in the low-CVH group, defined as CVH overall score <50, 32.9 (95% CI, 32.3–33.4) in the moderate-CVH group (CVH overall score between 50 and 79), and 36.2 (95% CI, 34.2–38.2) years in the high-CVH group, defined as overall CVH score of ≥80.[98] With 19 951 US adults between 30 and 79 years of age over a median follow-up of 7.6 years, the second report found a 58% reduction (HR, 0.42 [95% CI, 0.32–0.56]) in all-cause mortality rate and a 64% reduction (HR, 0.36 [95% CI, 0.21–0.59]) in CVD-specific mortality rate when the high-CVH (score, 75–100) was compared with the low-CVH (score <50) group and a 40% reduction (HR, 0.60 [95% CI, 0.51–0.71]) in all-cause mortality rate and 38% reduction (HR, 0.62 [95% CI, 0.46–0.83]) in CVD-specific mortality rate when the moderate-CVH group (score, 50–74) was compared with the low-CVH group.[99] In a third report analyzing 23 110 US adults ≥20 years of age from NHANES between 2005 and 2014, also matching with the National Death Index data through 2019, the authors reported a 40% reduction (HR, 0.60 [95% CI, 0.48–0.75]) in all-cause mortality rate and a 54% reduction (HR, 0.46 [95% CI, 0.31–0.68]) in CVD-specific mortality rate over a median follow-up of 9.4 years when they compared the high-CVH group (defined as overall CVH score of 80–100) with the low-CVH group (score <50).[100]
- Several reports using UK Biobank data were also produced with the updated CVH metrics. With 250 825 participants observed over a median follow-up of 10.4 years, people in the lowest quartile of the overall CVH score had 2.1- (95% CI, 2.0–2.2) fold higher risk of MACEs (including IHD, MI, stroke, and HF) compared with participants in the highest quartile of CVH score. HF was the MACE component outcome that experienced the greatest elevated risk (HR, 2.7 [95% CI, 2.4–2.9]).[35] The mean difference in life expectancy at 45 years of age between these 2 groups of people was estimated as 7.2 years (95% CI, 5.5–8.9) in favor of people with ≥4 ideal components in the CVH metrics. According to data from 135 199 participants, the life expectancy free of 4 major chronic diseases, namely CVD, diabetes, cancer, and dementia, at 50 years of age was estimated to be 6.9 years (95% CI, 6.1–7.7) longer for males with high CVH level (overall score, 80–100) compared with males at the low CVH level (overall score <50) and 9.4 years (95% CI, 8.5–10.2) longer for females in the high-CVH category compared with females in the low-CVH category. The corresponding estimates were 4.0 years (95% CI, 3.4–4.5) longer for males and 6.3 years (95% CI, 5.6–7.0) longer for females with moderate CVH level compared with their counterparts in the low CVH category.[101] In a study focusing on 33 236 participants with type 2 diabetes who were 40 to 72 years of age at baseline using the same database, people with ≥4 ideal components in the CVH metrics enjoyed a 65% reduction (HR, 0.35 [95% CI, 0.26–0.47]) in diabetes complications and a 47% reduction (HR, 0.53 [95% CI, 0.43–0.65]) in all-cause mortality rate compared with people with no more than 1 ideal CVH metric over a median of 11.7 years of follow-up.[102] Similar favorable risk reductions for risk of dying before 75 years of age were found for males and females with or without type 2 diabetes at the moderate to high CVH levels compared with low CVH among 309 789 adults from the same database.[103]
- Similar associations between greater CVH in childhood using the revised metrics and more favorable health or mortality outcomes were also reported by a Finnish[104] study and 2 Chinese cohort[105,106] studies. The Healthy Start Study contrasted the original Life's Simple 7 and the revised AHA's Life's Essential 8 CVH metrics in 305 children between 4 and 7 years of age and observed modest concordance between these 2 CVH metrics. The authors noted the important role that sleep health played in classifying childhood CVH levels.[107] Additional information on the relevance of sleep to cardiometabolic health can be found in Chapter 13 (Sleep) of this Statistical Update.

CVH in the United States: Mean CVH Scores (NHANES 2013–March 2020)

(See Table 2-2)

- The national estimates of the 8 CVH components for children (2–19 years of age) and adults (≥20 years of age) are displayed in Table 2-2. Multiple cycles of NHANES data were combined to provide more precise estimates on all CVH components. Dietary, PA, and BMI scores were calculated for all children who were 2 to 19 years of age; blood lipid and BP scores were calculated for children who were 6 to 19 and 8 to 19 years of age, respectively; and blood glucose and nicotine exposure scores were calculated for those who were 12 to 19 years of age in the sample. The sleep health score was available only for youths 16 to 19 years of age, so the mean score of this component and the overall CVH score were derived for this age range only. Dietary estimates were available only through data up to the 2017 to 2018 NHANES cycle at the time of this report.
- For most components of CVH, mean scores were higher in US children (within corresponding age ranges of the components) than in US adults (≥20 years of age), except for the diet score and the sleep health score, for which mean scores in children were lower than in adults. Mean diet scores were the lowest among the 8 CVH components for both US children and adults.
  - Among US children, BP, blood glucose, and nicotine exposure were the CVH components scoring highest compared with the rest of the CVH components, with all mean scores in the 80s and the 90s (of 100 points as the ideal score) across race and ethnicity groups. In contrast, mean PA, lipids, and sleep health scores within the corresponding age ranges were all in the 70s across race and ethnicity categories.
  - Among US adults (Table 2-2), the lowest mean scores for CVH were observed in diet, PA, and BMI components, with mean scores ranging from the 30s to the 50s across all race and ethnicity categories. Sleep health scores were the highest among the CVH components in US adults, with mean scores in the 80s across all race and ethnicity groups except in the NH Black adult population, for whom the mean score was 75.6 (95% CI, 74.5–76.7). Mean scores for blood lipids, blood glucose, and BP among US adults were all in the 60s to the 70s range across race and ethnicity categories.
- From 2013 to March 2020, the overall CVH score combining health scores of all 8 components was, on average, 73.6 (95% CI, 72.4–74.7) for all US children between 16 and 19 years of age (Table 2-2). The corresponding mean overall CVH score was 78.4 (95% CI, 75.7–81.1) for NH Asian children, 74.1 (95% CI, 72.0–76.2) for NH White children, 72.7 (95% CI, 70.6–76.3) for Mexican American children, and 71.3 (95% CI, 68.8–73.8) for NH Black children.
- During the same period, the mean overall CVH score was 65.2 (95% CI, 64.2–66.1) for all US adults, with mean score of 69.6 (95% CI, 68.1–71.1) for NH Asian adults, 66.0 (95% CI, 64.8–67.2) for NH White adults, 63.5 (95% CI, 62.2–64.8) for Mexican American adults, and 59.7 (95% CI, 58.4–60.9) for NH Black adults (Table 2-2).
- An article appeared online ahead of print on the same day as the presidential advisory on AHA's Life's Essential 8 providing CVH score estimates by additional sociodemographic categories under this new CVH metrics using NHANES data from 2013 to 2018.[96]

CVH in the United States: Trend in Mean CVH Scores Over Time (NHANES 2007–March 2020)

(See Charts 2-1 through 2-8)

- The overall trend for national estimates of the 8 CVH components for adults 20 to 79 years of age and trends by race and ethnicity subgroups are displayed in Charts 2-1 through 2-8 (unpublished AHA tabulation using NHANES[108]). Adults who self-reported a history of CHD, MI, angina, or stroke; were pregnant; or were breastfeeding at time of examination were not included in these analyses. Dietary estimates were available only through the 2017 to 2018 NHANES data cycle at the time of this report because of the availability of the Food Patterns Equivalents Database from the US Department of Agriculture, whereas mean scores for the rest of the CVH metrics were derived through the 2017 to March 2020 combined NHANES cycle. As a result, the trends over time for the overall CVH score are not presented here. Furthermore, data for the NH Asian population are available only for CVH evaluation starting from the 2011 to 2012 NHANES data cycle.
  - During this time period, CVH diet scores for US adults remained low and relatively unchanged (Chart 2-1). Adult NH Asian individuals observed slightly higher average diet scores since 2011 to 2012 compared with other race and ethnicity subgroups. The age-adjusted mean score for NH Asian adults in 2017 to 2018 was 47.8 (95% CI, 44.3–55.3). NH Black individuals had the lowest diet score on average during the past decade. In 2017 to 2018, the adjusted mean score for NH Black adults was 22.4 (95% CI, 19.1–27.7).

- Although still low overall, a gradual upward trend in mean CVH PA scores was observed for adults in every race and ethnicity subgroup presented, except for NH Asian adults, for whom the trend is less obvious (Chart 2-2). In the period of 2017 to March 2020, the age-adjusted mean PA scores ranged from 47.9 (95% CI, 45.6–50.3) for Hispanic adults to 57.7 (95% CI, 54.0–61.4) for NH White adults.
- Upward trends in mean nicotine exposure CVH scores were observed for adults in all race and ethnicity subgroups presented (Chart 2-3). The mean scores for the updated nicotine exposure CVH score, which now takes into account secondhand smoking exposure as well, were significantly higher in NH Asian and Hispanic individuals compared with NH White and NH Black individuals. The age-adjusted mean scores ranged between 66.8 (95% CI, 62.7–70.8) for NH Black adults and 87.0 (95% CI, 84.4–89.5) for NH Asian adults during 2017 to March 2020.
- Upward trends were also observed across all race and ethnicity subgroups for the newest addition to the updated CVH metrics, the sleep health score, although the age-adjusted mean scores were significantly lower for NH Black individuals, ranging from 71.5 (95% CI, 69.3–73.6) in 2007 to 2008 to 78.5 (95% CI, 76.4–80.6) in 2015 to 2016 and then to 76.6 (95% CI, 74.9–78.3) in 2017 to March 2020 compared with other race and ethnicity subgroups (Chart 2-4).
- Although mean CVH BMI scores were higher in NH White individuals and NH Asian individuals compared with NH Black individuals and Hispanic individuals, all race and ethnicity subgroups presented here observed a steep downward trend in this CVH metric over the past decade (Chart 2-5). In the period of 2017 to March 2020, the age-adjusted mean BMI scores ranged between 57.5 (95% CI, 54.8–60.2) for NH White adults and 50.3 (95% CI, 48.5–52.2) for NH Black adults.
- Trends in age-adjusted mean scores of the non-HDL lipids metric over the past decade improved for all race and ethnicity subgroups, except for the NH Asian population, for which the mean scores were relatively unchanged (Chart 2-6). NH Black individuals had significantly higher mean scores in this metric, ranging from 69.0 (95% CI, 67.0–71.1) in 2007 to 2008 to 74.9 (95% CI, 72.8–77.0) in 2017 to March 2020, compared with the other race and ethnicity subgroups.
- Although they remained relatively stable through 2014, the mean CVH blood glucose scores had a steady worsening for all race and ethnicity subgroups over the past 6 years (Chart 2-7). The mean scores for all US individuals were 79.4 (95% CI, 78.2–80.6) in 2007 to 2008 and 80.5 (95% CI, 79.4–81.5) in 2013 to 2014 but declined to 76.0 (95% CI, 75.2–76.9) in 2017 to March 2020.
- During this time period, age-adjusted mean BP scores for US adults remained relatively unchanged (Chart 2-8). NH Black individuals had the lowest mean BP score and had a seemingly more pronounced downward trend over time in this CVH metric, from 65.8 (95% CI, 62.2–69.3) in 2007 to 2008 to 57.9 (95% CI, 55.8–59.9) in 2017 to March 2020, compared with the rest of US adult populations.

Trends in Risk Factors and Causes for YLL and YLD in the United States: 1990 to 2021

(See Tables 2-3 through 2-6)

- The leading risk factors for YLLs from 1990 to 2021 in the United States and the corresponding percent change in age-standardized YLL rates attributable to these risk factors are presented in Table 2-3.
  - High SBP and smoking remained the first and second leading YLL risk factors in both 1990 and 2021. Age-standardized rates of YLL attributable to smoking declined by 53.9%, whereas age-standardized rates attributable to high SBP declined 47.4%.
  - In 2021, CVH components accounted for 12 (among which 7 were related to poor diet) of the 20 leading YLL risk factors, with 6 of the 7 diet-related risk factors rising in the risk factor rankings since 1990.
- The leading causes of YLLs from 1990 to 2021 in the United States and the corresponding percent change in age-standardized YLL rates attributable to these risk factors are presented in Table 2-4.
  - IHD was the leading YLL cause in1990 and second leading YLL cause 2021, with COVID-19 ranking first. Age-standardized YLL rates attributable to IHD declined 55.5%, whereas age-standardized YLL rates resulting from tracheal, bronchus, and lung cancer declined 50.0%.
  - Type 2 diabetes also rose from the 13th to 9th leading YLL cause, whereas AD and other dementias also rose from the 10th to 6th leading YLL cause.
- The leading risk factors for YLDs from 1990 to 2021 in the United States and the corresponding percent change in age-standardized YLD rates attributable to these risk factors are presented in Table 2-5.
  - High BMI, high FPG, and smoking are among the top 4 leading YLD risk factors in both 1990 and 2021, with high FPG rising in ranking and smoking dropping from the second to the fourth leading YLD risk factor during this time period. Age-standardized YLD rates attributable to smoking declined by 22.8%, and age-standardized rates

attributable to high BMI and high FPG increased by 69.3% and 123.7%, respectively, between 1990 and 2021.

- The leading causes of YLDs from 1990 to 2021 in the United States and the corresponding percent change in age-standardized YLD rates attributable to these risk factors are presented in Table 2-6.
  - From 1990 to 2021, type 2 diabetes rose from the 10th to 3rd leading YLD cause with a 157.9% increase in the age-standardized YLD rates.

Trends in Global Risk Factors and Causes for YLL and YLD: 1990 to 2021

(See Tables 2-7 through 2-10)

- The leading global YLL risk factors from 1990 to 2021 and the corresponding percent change in age-standardized YLL rates attributable to these risk factors are presented in Table 2-7.
  - High SBP and smoking were the first and second leading YLL risk factors globally in 2021. Age-standardized YLL rates attributable to high SBP and smoking declined 32.3% and 42.5%, respectively, between 1990 and 2021.
  - From 1990 to 2021, high FPG rose from the 14th to 5th leading risk factor of global YLLs with a 1.6% decrease in the age-standardized YLL rates over this period.
- The leading global YLL causes from 1990 to 2021 and the corresponding percent change in age-standardized YLL rates attributable to these risk factors are presented in Table 2-8.
  - IHD rose from the third to second leading global YLL cause between 1990 and 2021, whereas age-standardized YLL rates declined by 31.6% during this period.
  - ICH and ischemic stroke rose from the seventh to fifth and from the 12th to 8th leading cause of global YLL, respectively, between 1990 and 2021.
  - Type 2 diabetes also rose from the 26th to 14th leading global YLL cause, showing a 9.8% increase in age-standardized YLL rate.
- The leading global risk factors for YLDs from 1990 to 2021 and the corresponding percent change in age-standardized YLD rates attributable to these risk factors are presented in Table 2-9.
  - High FPG and high BMI were the first and second leading YLD risk factors globally in 2021, replacing iron deficiency and smoking, which were first and second in 1990. Age-standardized YLD rates attributable to high FPG and high BMI increased 74.6% and 69.0%.
- The leading global causes of YLDs from 1990 to 2021 and the corresponding percent change in age-standardized YLD rates attributable to these risk factors are presented in Table 2-10.
  - From 1990 to 2021, type 2 diabetes rose from the 10th to 7th leading global cause of YLD during this time period with a 94.7% increase in the age-standardized global YLD rate.

Furthering the AHA's Impact Through Continued Efforts to Improve CVH

(See Tables 2-3 through 2-6)

- Renewed efforts to maintain and improve CVH will be foundational to successful reductions in mortality and disability in the United States and globally. Individuals with more favorable levels of CVH have significantly lower risk for several of the leading causes of death and YLD, including IHD,[29] AD,[109] stroke,[110,111] CKD,[112] diabetes,[113] and breast cancer[114] (Tables 2-4 and 2-6). In addition, 6 of the 10 leading US risk factors for YLL and 4 of the 10 leading risk factors for YLD in 2019 were components of CVH (Tables 2-3 and 2-5). Taken together, these data demonstrate the tremendous importance of continued efforts to improve CVH.
- There is increasing recognition that optimizing prepregnancy and maternal CVH will result in long-term benefits to the health of the birthing individual and the offspring. See Chapter 11 (Adverse Pregnancy Outcomes) for additional information.
- As the benefits of ideal CVH become increasingly evident, attention is turning to developing interventions to promote CVH with a focus on implementation science.[115] An increasing number of studies are using community-based, school-based, or technology-enhanced interventions to directly improve CVH score.[116-120]

Global Efforts to Improve CVH

- A recent scoping review examining our knowledge of CVH across low- and middle-income countries revealed that limited data exist on CVH in these countries, 85% are cross-sectional, and 71% came from only 10 countries.[121]
- Many challenges exist related to implementation of prevention and treatment programs in international settings; some challenges are unique to individual countries/cultures, whereas others are universal. Partnerships and collaborations with local, national, regional, and global partners are foundational to effectively address relevant national health priorities in ways that facilitate contextualization within individual countries and cultures.

21. CORONARY HEART DISEASE, ACUTE CORONARY SYNDROME, AND ANGINA PECTORIS

See Tables 21-1 through 21-3 and Charts 21-1 through 21-8

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Coronary Heart Disease

ICD-9 410 to 414, 429.2; *ICD-10* I20 to I25 (includes MI *ICD-10* I21 to I22).

Prevalence

(See Tables 21-1 and 21-2 and Charts 21-1 through 21-4)

- In an analysis of data on 17 266 adults with a history of CHD from NHIS 2006 to 2015, the prevalence of premature CHD (<65 years of age for females and <55 years of age for males) was higher among Asian Indian adults (aOR, 1.77 [95% CI, 1.05–2.97]) and other Asian adults (aOR, 1.68 [95% CI, 1.17–2.42]) than White adults.[1]
- On the basis of data from NHANES 2017 to 2020,[2] an estimated 20.5 million Americans ≥20 years of age have CHD (Table 21-1). The prevalence of CHD was higher for males than females in all age groups (Chart 21-1).
- According to NHANES 2017 to 2020, total CHD prevalence is 7.1% in US adults ≥20 years of age. CHD prevalence is 8.7% for males and 5.8% for females. CHD prevalence by sex and ethnicity is shown in Table 21-1.
- Based on data from the NHIS 2018, the CHD prevalence estimates are 5.7% among White people, 5.4% among Black people, 8.6% among American Indian/Alaska Native people, and 4.4% among Asian people ≥18 years of age.[3]
- According to data from NHANES 2017 to 2020 (unpublished NHLBI tabulation),[2] the overall prevalence of MI is 3.2% in US adults ≥20 years of age. Males have a higher prevalence of MI than females for all age groups (Chart 21-2). Overall MI prevalence is 4.5% for males and 2.1% for females. MI prevalence by sex and ethnicity is shown in Table 21-1.
- According to data from NHANES 2017 to 2020,[2] the overall prevalence of angina is 3.9% in US adults ≥20 years of age (Table 21-2).
- Data from the BRFSS 2022 survey indicate that 4.5% of respondents had been told that they had had an MI. The highest age-adjusted prevalence was in West Virginia (6.1%), and the lowest was in Colorado (2.8%; Chart 21-3; unpublished NHLBI tabulation using BRFSS[4]).
- In the same survey in 2022, 4.4% of respondents had been told that they had angina or CHD. The highest age-adjusted prevalence was in Mississippi (5.8%), and the lowest was in Colorado (2.5%; Chart 21-4).[4]

Incidence

- Approximately every 40 seconds, an American will have an MI (AHA computation based on incidence data from the ARIC study of the NHLBI[5]).
- On the basis of data tabulated by the NHLBI from the 2005 to 2014 ARIC study[5]:
  - Approximately 720 000 Americans will have a new coronary event (defined as first hospitalized MI or CHD death), and ≈335 000 will have a recurrent event.
  - The estimated annual incidence of MI is 605 000 new attacks and 200 000 recurrent attacks. Of these 805 000 first and recurrent events, it is estimated that 170 000 are silent (without significant symptoms).
  - Average age at first MI is 65.6 years for males and 72.0 years for females.
- After adjustment for social determinants of health and cardiovascular risk factors, Black males and females have similar risk for fatal CHD (ARIC, 0.67 [95% CI, 0.36–1.24]; REGARDS, 1.00 [95% CI, 0.54–1.85]) but lower risk for nonfatal CHD (ARIC, 0.70 [95% CI, 0.51–0.97]; REGARDS, 0.70 [95% CI, 0.46–1.06]) compared with White males and females.[6]

Secular Trends

- In a study of the 2010 to 2019 National Readmission Database, among 592 015 30-day readmissions and 787 008 ninety-day readmissions after index AMI hospitalization, 30-day and 90-day all-cause readmission rates after AMI decreased from 12.8% to 11.6% (P=0.0001) and 20.6% to 18.8% (P=0.0001), respectively.[7] Downward trends were observed in both pre-HRRP (2010–2012) and post-HRRP periods (2013–2015, 2016–2019).

The 2025 AHA Statistical Update uses language that conveys respect and specificity when referencing race and ethnicity. Instead of referring to groups very broadly with collective nouns (eg, Blacks, Whites), we use descriptions of race and ethnicity as adjectives (eg, Asian people, Black adults, Hispanic youths, Native American patients, White females).

As the AHA continues its focus on health equity to address structural racism, we are working to reconcile language used in previously published data sources and studies when this information is compiled in the annual Statistical Update. We strive to use terms from the original data sources or published studies (mostly from the past 5 years) that may not be as inclusive as the terms used in 2025. As style guidelines for scientific writing evolve, they will serve as guidance for data sources and publications and how they are cited in future Statistical Updates.

However, the mean length of stay (4.54–4.96 days; P=0.0001) and adjusted total cost ($13 449–$16 938; P=0.0001) for 30-day all-cause readmission increased over the decade.

- In a retrospective study of 2 881 746 PCIs for MI from the NIS 2008 to 2019, the use of intravascular ultrasound–guided PCI increased by 309.9% (6180 versus 25 330; P_{trend}<0.001), whereas optical coherence tomography–guided PCI increased by 548.4% (246 versus 1595; P_{trend}<0.001).[8] The percentage of intravascular ultrasound and optical coherence tomography use in PCIs increased from 3.4% to 8.7% and 0.0% to 0.6%, respectively (P_{trend}<0.001). Intravascular imaging–guided PCI was associated with lower odds of in-hospital mortality (aOR, 0.66 [95% CI, 0.60–0.72]; P<0.001), suggesting a significant increase in use and improved outcomes.
- Among Medicare beneficiaries between 2002 and 2011, the rates of MI hospitalization declined from 1485 to 1122 per 100 000 PY.[9]
  - The rates of MI as the primary reason for hospitalization decreased over time (from 1063 to 677 per 100 000 PY between 2002 and 2011). The percentage of MIs that were attributable to a primary reason for hospitalization decreased from 72% to 60% between 2002 and 2011.
  - However, the rates of MI as a secondary reason for hospitalization increased (from 190 to 245 per 100 000 PY). The percentage of MIs that were attributable to a secondary diagnosis increased from 28% to 40%.
- In Olmsted County, Minnesota, between 2003 and 2012, the annual incidence declined for both type 1 MI (from 202 to 84 per 100 000; P<0.001) and type 2 MI (from 130 to 78 per 100 000; P=0.02).[10]
- According to data from inpatient and ambulatory databases from 4 states (Michigan, Maryland, New York, and Florida), population trends in PCI use were examined between January 2010 and December 2017. Among a cohort of 333 819 patients (32% female; mean±SD age, 65.7±12.2 years), 1 044 698 PCIs were performed: 57.1% were elective, and 42.9% were urgent. PCI rates declined from 260.2 to 232.8 per 100 000 (–10.5%; P_{trend}<0.001) between 2010 and 2017. In the same period, outpatient PCI rates increased from 33.8 to 66.7 per 100 000 (+97.1%; P_{trend}<0.001), whereas inpatient PCI rates declined from 226.4 to 166.2 per 100 000 (–26.6%; P_{trend}<0.001).[11]

Admissions and Mortality Trends

- In England, AMI hospitalizations during the COVID-19 period (February 1–May 14, 2020; n=9325) declined >50% compared with the pre–COVID-19 period (February 1–May 14, 2019; n=20 310), with a corresponding increase in the incidence of OHCA (see Chapter 19 [Sudden Cardiac Arrest, Ventricular Arrhythmias, and Inherited Channelopathies]).[12] A similar multisite study in France observed a reduction in STEMIs (IRR, 0.72 [95% CI, 0.62–0.85]) and NSTEMIs (IRR, 0.64 [95% CI, 0.55–0.76]) when the 4 weeks before and after lockdown were compared.[13]
- In a cohort of 1533 patients admitted with AMI (STEMI and NSTEMI) in a large health system in Washington, DC, and Maryland between March 1, 2020, and June 30, 2020, 86 had confirmed COVID-19. Furthermore, 20.0% of patients (n=17) with COVID-19 underwent coronary angiography. Those with concomitant COVID-19 and AMI had higher in-hospital mortality (27.9%) than patients without COVID-19 in the same period (3.7%; P<0.001).[14]
- Among 21 738 patients with type 2 MI in the National Readmission Database, in-hospital mortality and 30-day readmission for patients with type 2 MI were 9.0% and 19.1%, respectively. AF, PAD, male sex, coagulopathy, and fluid/electrolyte imbalances were associated with higher in-hospital mortality. In addition, AF/flutter, carotid artery stenosis, diabetes, anemia, COPD, CKD, and history of MI were associated with higher odds of 30-day readmission.[15]
- An observational analysis of ED and inpatient data among patients with HF (n=21 262) or AMI (n=6165) from 12 hospital health systems across the St. Louis metropolitan area found that patient volume decreased for AMI during COVID-19 (6.1–6.6 events/d before COVID-19, 4.9–5.5 events/d during COVID-19; P<0.001).[16] However, the proportion of patients with STEMI increased during COVID-19 (32.5%–37.6%) compared with before COVID-19 (29.0%–29.3%; P=0.005). Furthermore, in-hospital mortality increased for AMI (OR, 1.46 [95% CI, 1.21–1.76]) and STEMI (OR, 2.57 [95% CI, 2.24–2.96]) during the pandemic.
- A meta-analysis comparing AMI admissions during the COVID-19 pandemic with pre–COVID-19 levels found 35% fewer AMI hospitalizations during COVID-19 compared with the pre–COVID-19 period (OR, 0.65 [95% CI, 0.56–0.74]; I^2=99%; P<0.001; 28 studies).[17] Hospitalizations also declined for STEMI (OR, 0.71 [95% CI, 0.65–0.78]; I^2=93%; P<0.001; 22 studies) and NSTEMI (OR, 0.66 [95% CI, 0.58–0.73]; I^2=95%; P<0.001; 14 studies) during COVID-19 compared with before COVID-19.[17] Another meta-analysis of 79 articles across 57 countries found that during the height of the COVID-19 pandemic, the IRR of STEMI hospitalizations decreased (0.80 [95% CI, 0.76–0.84]; P<0.05) over the reference period. However,

there was significant heterogeneity across studies (I^2=89%; P<0.0001).[18] There was an inverse association between IRRs for STEMI admissions and hospital bed availability in each country (P<0.05).

Social Determinants of Health/Health Equity

- Data from the NIS October 2017 to December 2018 including 294 540 hospitalizations among adults with type 2 MI were analyzed for racial differences in clinical outcomes.[19] Compared with White individuals, individuals of other races and ethnicities (Native American, Asian or Pacific Islander, and other underrepresented racial and ethnic groups) had higher in-hospital mortality (aOR, 1.17 [95% CI, 1.03–1.33]; P=0.016) and longer length of stay (adjusted parameter estimate, 0.59 [95% CI, 0.22–0.97]; P=0.002).
- In a 2013 to 2018 cross-sectional cohort study of 289 376 patients with STEMI and 843 046 patients with NSTEMI ≥66 years of age from the United States, Canada, England, the Netherlands, Taiwan, and Israel, Patients with high income (those living in an area [eg, postal code] in the top 20% of the income distribution) had lower 30-day mortality (1–3 percentage points) and 1-year mortality (up to 9.1 percentage points in Israel for STEMI [95% CI, −16.7 to −1.6]) compared with patients with low income (those living in areas in the bottom 20% of the distribution).[20] Patients with high income also had higher rates of cardiac catheterization and PCI (1−-6 percentage points), lower 30-day readmission rates (1–3 percentage points), and shorter hospital stays (0.2–0.5 days) across all 6 countries.
- A longitudinal cohort study examined racial and cohort differences in the relationship between debt and MI risk in the HRS 1992 to 2018.[21] The study compared the prewar cohort (born 1931–1941, N=8698 in 1992) and Baby Boomers (born 1948–1959, N=6792 in 2010). Higher unsecured debt was associated with increased MI risk for Black adults, especially Baby Boomers (aHR, 1.11; P<0.001) and during recessions (aHR, 1.05; P<0.001). Higher mortgage debt was associated with lower risk for White Baby Boomers (aHR, 0.97; P<0.001) but not Black Baby Boomers (aHR, 1.13; P<0.001).
- In an observational cohort study of 135 358 patients with STEMI in California EDs from 2010 to 2019, uninsured patients had lower odds of interfacility transfer compared with insured patients (aOR, 0.93[95% CI, 0.88–0.98]; P=0.01) after adjustment for time trends, patient factors, and hospital characteristics, including PCI capabilities.[22]
- In a retrospective study of 9 million adults with NSTEMI (72%) and STEMI (28%) from 2005 through 2019, no significant improvement was found in the use of diagnostic angiograms, PCI, and CABG for Asian and Pacific Islander individuals, Black individuals, Hispanic individuals, and Native American individuals, compared with White individuals over 15 years (P>0.05), except for CABG in STEMI for Black individuals versus White individuals (difference, 2.6% to 1.4%; P=0.03).[23]
- In an analysis of data from 72 173 census tracts (N=308 243 060) in the 2018 BRFSS, higher social vulnerability index, a measure of a neighborhood's risk for deleterious outcomes in the event of natural disasters or disease outbreaks, was associated with higher CHD prevalence (β=0.0520; SE, 0.0008; R^2=0.57; P<0.0001) when accounting for census-tract median age.[24]
- An NIS analysis of sex differences spanning 2004 to 2015 identified 7 026 432 hospitalizations for AMI. Compared with males, females were less likely to undergo coronary angiography (aOR, 0.92 [95% CI, 0.91–0.93]) and PCI (aOR, 0.82 [95% CI, 0.81–0.83]). Females had a higher risk of mortality (aOR, 1.03 [95% CI, 1.02–1.04]) compared with males.[25]
- An observational cohort analysis of Medicare beneficiaries hospitalized with MI (N=155 397) in a national MI registry between April 2018 and September 2019 showed that Black adults (compared with non-Black adults) had lower 30-day mortality rates in low-performing hospitals (OR: before the HRRP, 0.79 [95% CI, 0.63–0.97]; P=0.03; after the HRRP, 0.80 [95% CI, 0.68–0.95]; P=0.01) but not in high-performing hospitals.[26]
- In 3635 patients who underwent left-sided heart catheterization for CAD at Emory University between 2004 and 2014, low neighborhood SES (a composite measure using 6 census measures capturing income, housing, education, and occupation) was associated with increased risk of cardiovascular death or MI in patients without a prior MI (HR, 2.72 [95% CI, 1.73–4.28] for the lowest versus highest quartile of neighborhood SES), but no association was observed for those with a prior MI (HR, 1.02 [95% CI, 0.58–1.81]; $P_{interaction}$=0.02).[27]
- According to the CMS Hospital Inpatient Quality Reporting Program data on 2363 hospitals in 2018, the average 30-day mortality after AMI was 13.6% (interquartile range, 12.8%–14.3%), with higher mortality observed in rural hospitals (from 13.4%–13.8% for the most urban to most rural hospitals).[28]
- Among 3006 older adults in the SILVER-AMI study who were recruited across 94 hospitals in the United States, low emotional support, measured with the Medical Outcomes Study Social Support Survey, was associated with higher odds of mortality (OR, 1.43 [95% CI, 1.04–1.97]), whereas low informational support was associated with higher odds of readmission (OR, 1.22 [95% CI, 1.01–1.47]).[29]

- In a retrospective cohort study of Medicare fee-for-service patients (N=453 783) diagnosed with CAD, there was no significant difference in adherence to guideline-recommended care in practices that served the highest proportion of patients who were socioeconomically disadvantaged compared with practices serving the lowest proportion.[30] Yet, at the most socioeconomically disadvantaged–serving practices, patients had higher odds of being admitted for unstable angina (aOR, 1.46 [95% CI, 1.04–2.05]) and higher 30-day mortality rates after AMI (aOR, 1.31 [95% CI, 1.02–1.68]). After additional adjustment for patient-level Area Deprivation Index, these associations were attenuated (unstable angina: aOR, 1.20 [95% CI, 1.02–1.68]; 30-day mortality after MI: aOR, 1.31 [95% CI, 1.02–1.68]).
- An NHANES analysis spanning the 2007 to 2016 cycles examined differences in self-reported history of CAD by limited English proficiency status in individuals reporting angina. Participants with limited English proficiency were 2.8 times more likely not to report a history of CVD compared with those without limited English proficiency (aOR, 2.77 [95% CI, 1.38–5.55]).[31]
- Disparities in cardiac rehabilitation are well recognized: Individuals who are female, of Black race, of Hispanic ethnicity, of lower educational attainment, and eligible for dual Medicare/Medicaid coverage have significantly reduced attendance compared with referents.[32–34] Among Medicare beneficiaries, participation in cardiac rehabilitation is lower among females (18.9%) compared with males (28.6%; adjusted PR, 0.91 [95% CI, 0.90–0.93]) and among Hispanic adults (13.2%) and NH Black adults (13.6%) compared with NH White adults (25.8%; adjusted PR, 0.63 [95% CI, 0.61–0.66] and 0.70 [95% CI, 0.67–0.72], respectively).[32] Likewise, in the BRFSS 2011 to 2015, participants in cardiac rehabilitation were less likely to be female (OR, 0.76 [95% CI, 0.65–0.90]), Black (OR, 0.70 [95% CI, 0.53–0.93]), uninsured (OR, 0.53 [95% CI, 0.37–0.75]), and less educated (OR, 0.47 [95% CI, 0.37–0.61]) compared with the referents.[33] In Optum's Clinformatics database (N=107 199), cardiac rehabilitation attendance was 31% lower for Asian adults (95% CI, 27%–36%), 43% lower for Hispanic adults (95% CI, 40%–45%), and 19% lower for Black adults (95% CI, 16%–22%) after adjustment.[34]
- An administrative claims analysis of Medicaid, commercial insurance, and Medicare claims from 2015 to 2018 identified that patients with Medicaid were less likely to receive guideline-concordant testing for MI (aOR, 0.84 [95% CI, 0.73–0.98]) and HF (aOR, 0.59 [95% CI, 0.51–0.70]) than those with commercial insurance.[35]
- A study of 2 182 903 Medicare beneficiaries hospitalized with MI, HF, or stroke from 2016 to 2018 compared outcomes in rural hospitals with outcomes in urban hospitals. Patients at rural hospitals were less likely to undergo cardiac catheterization (49.7% versus 63.6%; $P<0.001$), PCI (42.1% versus 45.7%; $P<0.001$), or CABG (9.0% versus 10.2%; $P<0.001$). Mortality at 30 days was higher for patients at rural hospitals presenting with MI (aHR, 1.10 [95% CI, 1.08–1.12]), HF (aHR, 1.15 [95% CI, 1.13–1.16]), and ischemic stroke (aHR, 1.20 [95% CI, 1.18–1.22]) compared with their counterparts presenting at metropolitan hospitals.[36]
- In a subset of SILVER-AMI, a community-based longitudinal study of older adults (N=1345, ≥75 years of age), there was no association between neighborhood walkability scores and hospital-free survival time or physical or mental health.[37]
- REGARDS investigators tabulated the number of social determinants of health to determine a progressive increase in fatal CHD (0 social determinants of health, 1.30; 1 social determinant of health, 1.44; 2 social determinants of health, 2.05; ≥3 social determinants of health, 2.86) and nonfatal MI (0 social determinants of health, 3.91; 1 social determinant of health, 4.33; ≥2 social determinants of health, 5.44). Compared with those with no social determinants of health, those with ≥3 social determinants of health had an aHR of 1.67 (95% CI, 1.18–2.37) for risk of fatal CHD.[38]
- Among 22 152 participants free of CHD at baseline in the REGARDS cohort study, there were 463 fatal incident CHD events and 932 nonfatal MIs over a median of 10.7 years (interquartile range, 6.6–12.7 years). Compared with those without social determinants of health, those with ≥3 social determinants of health had a higher risk (aHR, 1.67 [95% CI, 1.18–2.37]) of fatal incident CHD, and those with ≥2 social determinants of health had a nonsignificant higher risk (aHR, 1.14 [95% CI, 0.93–1.41]) of nonfatal MI.[38]
- In an analysis of NIS data from, January 1, 2012, through December 31, 2017, Black adults and individuals from other racial and ethnic groups with AMI compared with White individuals were less likely to undergo coronary angiography (61.9% versus 70.2% versus 73.1%) and PCI (44.6% versus 53.0% versus 58.1%; $P<0.001$).[39]
- A systematic review of 181 studies conducted primarily in high-income countries found that lower socioeconomic position (education, income, insurance, occupation, or composite) was associated with higher incidence of ACS (IRR, 1.1–4.7), higher prevalence of ACS (OR, 1.8–3.9), higher odds of receiving suboptimal medical care (OR, 1.1–10.0), and higher mortality after ACS (HR,1.1–4.13).[40]

Risk Prediction

- In a prospective study of 128 322 males and 135 103 females from the UK Biobank study, higher serum levels of sex hormone–binding globulin were associated with a decreased risk of CHD in both males (aHR per 1–log nmol/L increase, 0.88 [95% CI, 0.83–0.94]) and females (aHR, 0.89 [95% CI, 0.83–0.96]).[41] A meta-analysis of 216 417 males and 138 282 females from 11 studies showed that higher sex hormone–binding globulin levels were associated with decreased CHD risk in males (aRR, 0.81 [95% CI, 0.74–0.89]) and females (aRR, 0.86 [95% CI, 0.78–0.94]) when the highest quartile was compared with the lowest quartile.[41]
- Two large cohort studies (MESA, N=1991; Rotterdam Study, N=1217) examining adults 45 to 79 years of age (median: MESA, 61 years of age; Rotterdam Study, 67 years of age) demonstrated that incorporating a CAC score significantly improved risk discrimination compared with a traditional risk factor model alone (MESA: change in C statistic, 0.09; Rotterdam Study: change in C statistic, 0.06). Notably, the addition of a PRS did not yield a similar improvement.[42]
- In 9066 participants 45 to 79 years of age from the REGARDS study, the observed and predicted ASCVD risks using the Pooled Cohort Risk Equations were similar in people with high social deprivation, although ASCVD risk was overestimated in those with low social deprivation (observed incident rate, 6.23 [95% CI, 5.31–7.31] versus predicted incident rate, 8.02; Hosmer-Lemeshow χ^2=12.43; *P*=0.01).[43]
- In the WHI, although the risk of ASCVD was overestimated with the Pooled Cohort Risk Equations, adding ASCVD events identified through linkage with CMS claims that were not self-reported resulted in alignment of the observed and predicted risks (observed [predicted] risks for baseline 10-year risk categories of <5%, 5%–7.5%, 7.5%–10%, and ≥10% were 3.8 [4.3], 7.1 [6.4], 8.3 [8.7], and 18.9 [18.7], respectively).[44]
- In 14 169 patients with ASCVD risk <5% and self-reported family history of CHD from the multicenter CAC Consortium followed up for ≈12 years, those with CAC scores >100 had a >10-fold higher risk of CHD mortality than patients with CAC=0 (HR, 10.4 [95% CI, 3.2–33.7]).[45] Furthermore, addition of CAC to a model with traditional risk factors (age, sex, race, hypertension, hyperlipidemia, diabetes, and smoking status) improved the prediction for CHD mortality (AUC, 0.72 for the model with traditional risk factors and 0.82 for the model adding CAC; *P*=0.03).
- In a large competing-risk analysis among 66 363 adults from the CAC Consortium, participants with CAC >10 had higher risk of CHD death (aHR, 2.83 [95% CI, 2.07–3.86]) than those with CAC=0.[46] This risk was not significantly higher among adults <40 years of age but was significantly higher among adults >40 to 50 years of age (aHR, 2.97 [95% CI, 1.32–6.69]), 50 to 60 years of age (aHR, 5.08 [95% CI, 2.68–9.63]), 60 to 70 years of age (aHR, 1.89 [95% CI, 1.08–3.31]), and ≥70 years of age (aHR, 2.43 [95% CI, 1.33–4.46]) compared with their age counterparts with CAC=0.
- Among 66 636 asymptomatic adults in the CAC Consortium, those with extremely high CAC scores (≥1000) had higher adjusted risk of CVD (HR, 5.04 [95% CI, 3.92–6.48]), CHD (HR, 6.79 [95% CI, 4.74–9.73]), all-cause mortality (HR, 2.89 [95% CI, 2.53–3.31]), and cancer (HR, 1.55 [95% CI, 1.23–1.95]) than those with CAC=0.[47] Moreover, those with CAC ≥1000 had higher adjusted risk of CVD (HR, 1.71 [95% CI, 1.41–2.08]), CHD (HR, 1.84 [95% CI, 1.43–2.36]), all-cause mortality (HR, 1.51 [95% CI, 1.33–1.70]), and cancer (HR, 1.36 [95% CI, 1.07–1.73]) than those with CAC scores of 400 to 999.
- Among 16 289 adults (6526 males, 9763 females) in the HCHS/SOL, WC cut points of >102 cm in males (current joint interim statement criterion) and >97 cm (9 points above the joint interim statement criterion) in females provide optimal discrimination for CHD (evidence of prior MI from ECG or self-report of MI, angina, or coronary procedures).[48]
- A precatheterization model and bedside risk score were developed and validated with data from 706 263 PCIs at 1608 sites between July 2018 and June 2019 to predict in-hospital mortality. Variables that predicted in-hospital mortality included cardiovascular instability, level of consciousness after cardiac arrest, and procedural urgency. The C indexes of the precatheterization model and bedside risk score were 0.940 and 0.923, respectively. The simplified bedside score includes age, CKD, cardiovascular instability, and the presence or absence of cardiac arrest before PCI. The total score ranges from 2 to 31 points, with an overall score ≤5 corresponding to a predicted mortality rate of <0.1% and a score of ≥27 associated with mortality rate of >85%.[49]
- A coronary age calculator was derived with traditional risk factors and CAC score in a MESA cohort of 6727 adults and compared with chronological age, the MESA CHD Risk Score, and CAC alone. The derived coronary age with CAC was identical to the MESA CHD Risk Score in predicting 10-year risk of CHD and had the highest discrimination (AUC, 0.76) compared with chronological age (AUC, 0.63) and coronary age without CAC (AUC, 0.70).[50]

- In a cohort of 272 307 White adults in the UK Biobank study, the integrated PRS, PCE, and PRS-enhanced PCE were compared to predict incident CAD cases.[51] The C statistics for the integrated PRS, PCE, and PRS-enhanced PCE were 0.640 (95% CI, 0.634–0.646), 0.718 (95% CI, 0.713–0.723), and 0.753 (95% CI, 0.748–0.758), respectively. The addition of the integrated PRS to the PCE at a 7.5% risk threshold yielded an NRI of 0.117 (95% CI, 0.102–0.129) for cases and − 0.023 (95% CI, − 0.025 to − 0.022) for controls (overall, 0.093 [95% CI, 0.08–0.104]). Among the incident CAD cases, 14.2% were correctly reclassified to the higher-risk category, and 2.6% were incorrectly reclassified to the lower-risk category.
- The T2-risk score, a risk stratification tool for predicting the primary outcome of death or future MI among patients with type 2 MI, was derived from the High-STEACS trial (2013–2016), the APACE study (2006–2018), and single-center consecutive patients at a hospital in Stockholm (2011–2014).[52] The T2-risk score, which includes age, IHD, diabetes, HF, myocardial ischemia on ECG, anemia, heart rate, eGFR, and maximal cardiac troponin concentration, had good discrimination (AUC, 0.76 [95% CI, 0.73–0.79]) for the primary outcome and was well calibrated. The T2-risk score improved discrimination over the Global Registry of Acute Coronary Events 2.0 risk score in all cohorts.

Genetics and Family History

Family History as a Risk Factor

- Among adults ≥20 years of age, 13.8% (SE, 0.6%) reported having a parent or sibling with a heart attack or angina before 50 years of age. The racial and ethnic breakdown from NHANES 2017 to 2020 is as follows (unpublished NHLBI tabulation)[2]:
  - For NH White people, 14.0% (SE, 1.5%) for males and 15.7% (SE, 0.9%) for females.
  - For NH Black people, 9.7% (SE, 1.5%) for males and 14.4% (SE, 1.2%) for females.
  - For Hispanic people, 8.1% (SE, 1.1%) for males and 12.9% (SE, 1.4%) for females.
  - For NH Asian people, 6.3% (SE, 1.3%) for males and 8.4% (SE, 1.5%) for females.
- Because the incidence of HD increases with age, the prevalence of family history will vary depending on the age at which family history is assessed. The distribution of reported family history of heart attack by age of survey respondent in the US population as measured by NHANES 2017 to 2020 is as follows (unpublished NHLBI tabulation)[2]:
  - 20 to 39 years of age, 7.8% (SE, 1.3%) for males and 10.1% (SE, 0.8%) for females.
  - 40 to 59 years of age, 16.1% (SE, 1.7%) for males and 16.9% (SE, 1.4%) for females.
  - 60 to 79 years of age, 15.8% (SE, 2.1%) for males and 21.2% (SE, 2.6%) for females.
  - ≥80 years of age, 11.1% (SE, 2.9%) for males and 13.3% (SE, 2.1%) for females.
- Data from a longitudinal observational study (N=49 255) demonstrated an association between family history of premature angina, MI, angioplasty, or bypass surgery and increased lifetime risk by ≈50% for both HD (from 8.9% to 13.7%) and CVD mortality (from 14.1% to 21%).[53]

Genetic Predictors of CHD

- CHD is heritable. From 36 years of follow-up data in 20 966 Swedish twins, the heritability of CHD mortality was 57% for males and 38% for females.[54] Of note, estimated heritability was operative throughout the life span but more prominently at younger ages of death, particularly for males.
- The application of GWASs to large cohorts of subjects with CHD has identified consistent genetic variants associated with CHD. Although several CHD loci indicate roles for atherosclerosis and traditional CVD risk factors, other loci highlight the importance of biological processes (ie, cellular adhesion, leukocyte migration and atherosclerosis, coagulation and inflammation, and vascular smooth muscle cell differentiation) in the arterial wall.[55]
- The first GWAS identified a locus on chromosome 9p21.3, which is the most consistently replicated genetic marker for CHD and MI in populations of European ancestry.[56] The primary SNP at 9p21.3 is common; 50% of the population of European ancestry is estimated to harbor 1 risk allele, and 23% harbor 2 risk alleles.[57]
  - A meta-analysis of 22 studies (N=35 872 cases; N=95 837 controls) identified the 10-year HD risk for a male 65 years of age with two 9p21.3 risk alleles and no other traditional risk factors as ≈13.2%, whereas a similar male with 0 alleles would have a 10-year risk of ≈9.2%. The 10-year HD risk for a female 40 years of age with 2 alleles and no other traditional risk factors is ≈2.4%, whereas a similar female with 0 alleles would have a 10-year risk of ≈1.7%.[57]
- GWASs have identified multiple loci associated with CAD implicating pathways in blood vessel morphogenesis, lipid metabolism, nitric oxide signaling, inflammation, and basic cellular processes governing the cell cycle,[58] division/replication, and growth. One large, ancestrally diverse GWAS included n=243 392 CAD case and n=849 686 control MVP participants.[59] After meta-analysis with predominantly European ancestry GWASs from CARDIoGRAMplusC4D and the UK Biobank, this GWAS identified 33 novel loci. Further meta-analysis with Biobank Japan and inclusion of MVP

Black participants and Hispanic participants identified 66 novel loci. These loci did not demonstrate heterogeneity across ancestral populations. Most of these novel loci (58%) were associated with CAD risk factors (eg, blood lipids, BP, diabetes, obesity, or smoking). Large-scale collaborative genetic studies of CAD (n=72 868 cases and n=120 770 controls) focused on the coding regions of the genome (exons) have identified additional loci, including loss-of-function variants in *ANGPTL4* (angiopoietin-like 4), which is an inhibitor of lipoprotein lipase.[60] These variants are associated with low plasma triglycerides and high HDL-C.

- A study of X chromosome genetic variation in >500 000 multiancestry individuals from the TOPMed Consortium found common alleles on chromosome Xq23 to be strongly associated with lower TC, LDL-C, and triglycerides in both females and males and associated with reduced odds for CHD and type 2 diabetes.[61] Every additional rs5942634-T allele, the lead cholesterol-lowering variant in chromosome Xq23, was associated with estimated ORs of 0.98 (95% CI, 0.96–0.99) for CHD and 0.97 (95% CI, 0.96–0.99) for type 2 diabetes.
- Hematopoietic somatic variants (clonal hematopoiesis of indeterminate potential) that accumulate with age are also independent predictors of CHD events. Carriers of clonal hematopoiesis of indeterminate potential had a risk of CHD 1.9 times greater than that of noncarriers (95% CI, 1.4–2.7) and a risk of MI 4.0 times greater than that of noncarriers (95% CI, 2.4–6.7).[62] Clonal hematopoiesis of indeterminate potential itself has germline genetic determinants.[63]

Clinical Utility of Genetic Markers

- Studies have shown that patients with early-onset MI have a higher proportion of high PRS than those with FH variants; for example, ≈2% carry a rare FH genetic variant, whereas ≈17% have a high PRS.[64]
- Even in individuals with high genetic risk, prevention strategies may have benefit. For example, in 4 studies across 55 685 individuals, genetic and lifestyle factors were independently associated with CHD, but even in individuals at high genetic risk, a favorable lifestyle was associated with a nearly 50% lower RR of CHD than an unfavorable lifestyle (HR, 0.54 [95% CI, 0.47–0.63]).[65]
- A summary of the 5 most highly cited studies of PRS concluded that the change in C statistic with the addition of PRS to the standard risk model improves the C statistic by −0.001 to 0.021 and that PRS has a limited contribution to primary prevention of CAD.[66]
- In the FOURIER study (N=14 298), patients without multiple clinical risk factors or high genetic risk as defined by a 27-CHD-variant PRS did not derive benefit from evolocumab, whereas patients with high genetic risk, regardless of clinical risk, had reduced risk of major coronary events (HR, 0.69 [95% CI, 0.55–0.86]; *P*=0.0012).[67]
- Studies suggest that the addition of a PRS contributes modestly to clinical risk prediction. In the UK Biobank with >350 000 participants, the change in C statistic for incident CAD prediction between a PCE and GRS model was 0.02 (95% CI, 0.01–0.03) with an overall NRI of 4.0% (95% CI, 3.1%–4.9%).[68] In the ARIC and MESA studies, adding a GRS to the PCE did not significantly increase the C statistic in either cohort for prediction of incident CHD events (change in C statistic: ARIC, −0.001 [95% CI, −0.009 to 0.006]; MESA, 0.021 [95% CI, −0.0004 to 0.043]).[69] In an East Asian cohort (N=41 271), addition of a PRS including 540 genetic variants to clinical risk factors had an NRI for CAD of 3.2% (95% CI, 0.9%–5.8%).[70]
- GRSs derived from 1 ancestry may have limited generalizability to individuals of different ancestries, necessitating the development of GRSs that are ancestry specific.[71] An example is a GRS for CAD derived and validated in South Asian individuals (OR per 1 SD, 1.58 [95% CI, 1.42–1.76]) that outperformed previous scores based on European ancestral populations.[72]

Awareness, Treatment, and Control

Awareness of Warning Signs and Risk for HD

- Data from the NHIS 2017 indicate that being unaware of all 5 MI symptoms was more common in males (OR, 1.23 [95% CI, 1.05–1.44]), Hispanic individuals (OR, 1.89 [95% CI, 1.47–2.43]), those not born in the United States (OR, 1.85 [95% CI, 1.47–2.33]), and those with a high school or lower education (OR, 1.31 [95% CI, 1.09–1.58]).[73] Compared with adults born in the United States, adults born in Europe, Russia, Africa, the Middle East, the Indian subcontinent, Asia, and Southeast Asia were likely to be aware of all 5 MI symptoms in the NHIS 2017 cycle.[74]
- Data from an online survey of US females (≥25 years of age) showed that awareness related to CHD as a leading cause of death among females declined from 65% in 2009 to 44% in 2019. The decline in awareness was observed in all racial and ethnic groups and ages except females ≥65 years of age. Moreover, NH Black females (OR, 0.31 [95% CI, 0.19–0.49]) and Hispanic females (OR, 0.14 [95% CI, 0.07–0.28]) and 25- to 34-year-old females (OR, 0.19 [95% CI, 0.10–0.34]) experienced the

greatest 10-year decline in awareness from 2019 to 2009.[75]

Time of Symptom Onset and Arrival at Hospital

- The weekend effect, that is, presentation with ACS on a weekend rather than weekday, has been examined with regard to timing and use of invasive management strategies. An analysis of NIS data spanning 2000 to 2016 identified statistically different rates of coronary angiography (59.9% versus 58.8%; $P<0.001$) and PCI (38.4% versus 37.6%; $P<0.001$) between weekend and weekday ACS presentations, more pronounced when early coronary angiography was examined (26% versus 21%; $P<0.001$).[76] Weekend presentation was not associated with increased risk of mortality compared with weekday presentation with ACS (OR, 1.01 [95% CI, 1.00–1.01]). A meta-analysis of 56 studies (N=384 452) concluded that individuals with STEMI presenting during off-hours had similar short-term (RR, 1.07 [95% CI, 1.00–1.14]), midterm (RR, 1.00 [95% CI, 0.95–1.05]), and long-term (RR, 0.95 [95% CI, 0.86–1.04]) mortality compared with those presenting during regular working hours.[77]
- A European registry of 6609 patients treated at 77 high-volume PCI centers determined that the COVID-19 pandemic was associated with a significant increase in door-to-balloon and total ischemia times.[78] Door-to-balloon time >30 minutes was 57.0% in the period of March to April 2020 compared with 52.9% in March to April 2019 ($P=0.003$), and total ischemia time >12 hours was 11.7% in the 2020 period compared with 9.1% in 2019 ($P=0.001$).
- In a meta-analysis including 57 136 patients from 10 studies, door-to-balloon time of >90 minutes versus ≤90 minutes was associated with higher in-hospital or 30-day mortality (OR, 1.52 [95% CI, 1.40–1.65]). An increased risk of 6-month to 12-month mortality was also observed for >90-minute door-to-balloon delay in 14 261 patients from 8 studies (OR, 1.53 [95% CI, 1.13–2.06]).[79]
- Rural EMS response has been longer than activation from suburban or metropolitan locations. National data from 2015 indicated that the mean response time for EMS was 14.5 minutes (9.5 minutes) in rural zip codes, 7.0 minutes (4.4 minutes) in urban zip codes, and 7.7 minutes (5.4 minutes) in suburban zip codes.[80]
- Analysis of a multinational registry of PCI for STEMI that included 109 high-volume centers determined that in 2020 the incidence of PCI was significantly less than in 2019 (IRR, 0.84 [95% CI, 0.83–0.86]), accompanied by increased likelihood of door-to-balloon time >30 minutes (OR, 1.1 [95% CI, 1.03–1.17]).[81]

Operations and Procedures

- In 2021, an estimated 444 730 PCIs, 184 000 CABGs, 110 245 carotid endarterectomy and stenting procedures, and 84 020 pacemaker and defibrillator procedures were performed for inpatients in the United States (unpublished NHLBI tabulation using HCUP[82]).

Comparison of Outcomes: Surgery Versus Percutaneous Intervention

- An analysis of 30 studies determined that compared with males, females undergoing CABG and combined CABG and valve surgery had higher short-term (ie, in hospital or within 30 days) mortality (OR, 1.40 [95% CI, 1.32–1.49]; $I^2=79\%$) and postoperative stroke (OR, 1.2 [95% CI, 1.07–1.34]; $I^2=90\%$) risks.[83]
- In an analysis of the BEST, PRECOMBAT, and SYNTAX trials comparing individuals with a previous MI and left main or multivessel CAD, CABG (versus PCI) was associated with a lower risk of MI (HR, 0.29 [95% CI, 0.16–0.55]) over a median follow-up of 59.8 months (interquartile range, 50.7–60.3 months).[84]
- At 10 years of follow-up in the SYNTAX trial, among 1800 trial participants, no difference in all-cause death was observed between PCI and CABG overall and among the subgroup of patients with left main CAD; however, for patients with 3-vessel disease, a greater risk of death was observed for those treated with PCI (HR, 1.42 [95% CI, 1.11–1.81]).[85]
- The ISCHEMIA trial randomized 5179 individuals with stable CAD and moderate or severe ischemia on stress testing to invasive or initial conservative treatment. Over the 4-year follow-up, there was no difference in primary end-point events (defined as cardiovascular death, MI, hospitalization for unstable angina, HF, or cardiac arrest) between those randomized to the invasive (18.2 per 100 patients [95% CI, 15.8–20.9]) and conservative (19.7 per 100 patients [95% CI, 17.5–22.2]) management arms.[86]
- In patients (N=1905) with left main CAD with low or intermediate complexity (SYNTAX scores ≤32), no difference in the composite outcome of MI, stroke, or death was observed between PCI (n=948) and CABG (n=957) at 5 years of follow-up, although ischemia-driven revascularization (OR, 1.84 [95% CI, 1.39–2.44]) and all-cause death (OR, 1.39 [95% CI, 1.03–1.85]) were more common after PCI.[87]
- In the NCDR CathPCI Registry, 1% of PCI procedures were for unprotected left main coronary lesions. A composite end point of in-hospital MI, stroke, emergency CABG, or death was more frequent in unprotected left main PCI (OR, 1.46 [95% CI, 1.39–1.53]) compared with all other PCIs.[88]

- In 4041 patients with STEMI with multivessel CAD randomized to complete revascularization versus culprit lesion–only PCI, those with complete revascularization experienced lower rates of a composite end point of cardiovascular death or MI (HR, 0.74 [95% CI, 0.60–0.91]; *P*=0.004) and a composite end point of cardiovascular death, MI, or ischemia-driven revascularization (HR, 0.51 [95% CI, 0.43–0.61]; *P*<0.001) at a median follow-up of 3 years.[89]
- In 27 840 patients with STEMI transported by EMS to 744 hospitals in the ACTION registry, preactivation of the catheterization laboratory >10 minutes before hospital arrival compared with no preactivation was associated with shorter times to the catheterization laboratory (median, 17 minutes [interquartile range, 7–25 minutes] versus 28 minutes [interquartile range, 18–39 minutes]), shorter door-to-device time (median, 40 minutes [interquartile range, 30–51 minutes] versus 52 minutes [interquartile range, 41–65 minutes]), and lower in-hospital mortality (2.8% versus 3.4%; *P*=0.01).[90]
- In the ISCHEMIA randomized trial including 5179 patients with stable coronary disease and moderate or severe ischemia, an initial invasive strategy did not reduce ischemic cardiovascular events or death compared with an initial conservative strategy (risk difference, −1.8% [95% CI, −4.7% to 1%] at 5 years).[91]

Secular Trends in Procedures

- In an analysis of the NIS, among patients ≥70 years of age with non–ST-segment–elevation ACS or STEMI, the proportion of patients undergoing PCI increased from 7.3% in 1998 to 24.9% in 2013 in those with non–ST-segment–elevation ACS and from 11% in 1998 to 35.7% in 2013 in those with STEMI.[92]
- An analysis of HCUP Inpatient and State Ambulatory and Surgery and Services Databases quantified the number of patients who underwent PCI from 2010 to 2017 in Florida, Maryland, Michigan, and New York.[11] In these 4 states, PCI rates declined from 260.2 per 100 000 individuals in 2010 to 232.8 per 100 000 individuals in 2017 (−10.5%; P_{trend}<0.001). This decline was attributed to a decrease in elective PCI across these years of −34.4%. Rates of urgent PCI increased from 95.0 per 100 000 individuals in 2010 to 109.2 in 2017 (+15.0%; P_{trend}<0.001).
- Among 216 657 adults with type 1 MI, 37 675 adults with type 2 MI, and 1521 with both type 1 and type 2 MI in the Nationwide Readmissions Database, use of coronary angiography (10.9% versus 57.3%; *P*<0.001), PCI (1.7% versus 38.5%; *P*<0.001), and CABG (0.4% versus 7.8%; *P*<0.001) was lower among patients with type 2 MI than those with type 1 MI. Furthermore, the risks of in-hospital mortality (aOR, 0.57 [95% CI, 0.54–0.60]) and 30-day MI readmission (aOR, 0.46 [95% CI, 0.35–0.59]) were lower among those with type 2 MI than those with type 1 MI.[93]
- In a Swedish population–based registry (N=4085), PCI for unprotected left main CAD increased from 121 procedures in 2005 to 589 in 2017.[94] The risk of major adverse cardiovascular and cerebrovascular events was 44% less in 2017 compared with 2005 (HR, 0.56 [95% CI, 0.41–0.78]).

Cardiac Rehabilitation

- In the BRFSS from 2005 to 2015, <40% of patients self-reported participation in cardiac rehabilitation after AMI. Between 2011 and 2015, patients who declared participation in cardiac rehabilitation were less likely to be female (OR, 0.76 [95% CI, 0.65–0.90]; *P*=0.002) or Black (OR, 0.70 [95% CI, 0.53–0.93]; *P*=0.014), were less well educated (high school versus college graduate: OR, 0.69 [95% CI, 0.59–0.81]; *P*<0.001; less than high school versus college graduate: OR, 0.47 [95% CI, 0.37–0.61]; *P*<0.001), and were more likely to be retired or self-employed (OR, 1.39 [95% CI, 1.24–1.73]; *P*=0.003) than patients who did not participate in cardiac rehabilitation.[33]
- Among 366 103 Medicare fee-for-service beneficiaries eligible for cardiac rehabilitation in 2016, only 24.4% participated in cardiac rehabilitation; among those who participated, the mean time to initiation was 47.0 days (SD, 38.6 days), and 26.9% completed cardiac rehabilitation with ≥36 sessions. Participation decreased with increasing age and was lower in females, Hispanic people, Asian people, those eligible for dual Medicare/Medicaid coverage, and those with ≥5 comorbidities.[32]
- A systematic review of 9 studies concluded that home-based cardiac rehabilitation is cost-effective, albeit recognizing heterogeneity across studies, limited duration of follow-up, and absence of consideration of diversity of cardiac rehabilitation participants.[95]
- In an administrative analysis of individuals eligible for cardiac rehabilitation (N=107 199), 28 433 (26.5%) attended cardiac rehabilitation.[34] After adjustment, compared with White individuals, the probability of attending cardiac rehabilitation was 31% lower for Asian individuals (95% CI, 27%–36%), 19% lower for Black individuals (95% CI, 16%–22%), and 43% lower for Hispanic individuals (95% CI, 40%–45%).
- In a randomized trial in patients undergoing cardiac rehabilitation after ACS with PCI, patients receiving digital health lifestyle interventions had more weight loss at 90 days than the control group (−5.1±6.5 kg versus −0.8±3.8 kg [mean±SD]; *P*=0.02) and

a nonsignificant decrease in cardiovascular-related rehospitalizations and ED visits at 180 days (8.1% versus 26.6%; RR, 0.30 [95% CI, 0.08–1.10]; *P*=0.054).[96]

- In an observational study (N=1120) of individuals with IHD, the 1-year mortality risk did not differ between those who accepted home-based cardiac rehabilitation (n=490) compared with those who did not (HR, 0.67 [95% CI, 0.31–1.45]).[97] In contrast, during a median follow-up of 4.2 years, those who participated in home-based cardiac rehabilitation had an HR of 0.64 (95% CI, 0.45–0.90) compared with those who declined.

Mortality (See Table 21-1)

- In an observational study using the NIS and National Readmission Database between 2017 and 2019, which compared Takotsubo syndrome (n=43 335), type 1 MI (n=2 035 055), and type 2 MI (n=639 075), mortality risk was lower in Takotsubo syndrome compared with type 1 MI (aOR, 0.3; *P*<0.001) and type 2 MI (aOR, 0.3; *P*<0.001). Type 1 MI had higher mortality (aOR, 1.2; *P*<0.001) than type 2 MI.[98]
- In a meta-analysis of 81 studies involving 157 439 patients with COVID-19, those with preexisting CHD had a higher risk of mortality (OR, 2.45 [95% CI, 2.04–2.94]; *P*<0.001), severe/critical COVID-19 (OR, 2.57 [95% CI, 1.98–3.33]; *P*<0.001), ICU/coronary care unit admission (OR, 2.75 [95% CI, 1.61–4.72]; *P*=0.002), and lower odds of discharge/recovery (OR, 0.43 [95% CI, 0.28–0.66]; *P*<0.001) compared with those without preexisting CHD.[99]
- In a propensity score–matched analysis of 159 890 STEMI hospitalizations from the 2020 NIS database, 1.38% had concurrent COVID-19.[100] These patients had higher in-hospital mortality (17.8% versus 9.1%) and lower rates of same-day PCI (63.6% versus 70.6%) and CABG (3.0% versus 6.8%) compared with those without COVID-19. However, COVID-19–positive patients with STEMI receiving same-day PCI had lower odds of in-hospital mortality (aOR, 0.42 [95% CI, 0.20–0.85]).
- In a meta-analysis of 4 retrospective, nonrandomized, observational cohort studies among 184 951 patients ≥18 years of age diagnosed with NSTEMI, early treatment (administered within 24 hours) with β-blockers was associated with a significant reduction in in-hospital mortality compared with no β-blocker treatment (OR, 0.43 [95% CI, 0.36–0.51]; *P*=0.0022).[101]
- On the basis of 2022 mortality data (unpublished NHLBI tabulation using NVSS[102]):
  - CHD mortality was 371 506 (Table 21-1), and CHD any-mention mortality was 595 121.
  - MI mortality was 103 905 (Table 21-1). MI any-mention mortality was 151 312.
- From 2012 to 2022, the annual death rate attributable to CHD declined 16.9%, whereas the actual number of deaths stayed relatively the same (unpublished NHLBI tabulation using CDC WONDER[103]).
- The age-adjusted death rates for CHD and MI by sex, race, and ethnicity can be found in Table 21-1.
- In 2022, 79% of CHD deaths occurred out of hospital. According to US mortality data, 294 458 CHD deaths occurred out of hospital or in hospital EDs in 2022 (unpublished NHLBI tabulation using CDC WONDER[103]).
- The estimated average number of YLL because of an MI death was 14.6 in 2022 (unpublished NHLBI tabulation using CDC WONDER[103]).
- Approximately 35% of the people who experience a coronary event in a given year will die as a result of it, and ≈14% who experience an MI will die of it (unpublished NHLBI tabulation using ARIC Community Surveillance [2005–2014]).[5]
- An analysis of the multicenter NCDR Chest Pain–MI Registry (N=155 397 patients and 763 hospitals) reported that 30-day mortality among hospitalized patients with MI decreased from 6.6% to 5.0% in Black individuals and from 5.2% to 4.0% in non-Black individuals in the period of 2008 to 2016. Furthermore, racial differences in readmission were not significant after covariate adjustment.[26]
- According to data on >4 million Medicare fee-for-service beneficiaries with AMI, 30-day mortality declined from 1995 through 2014 (20.0% to 12.4%). Mortality was higher in females, but over time, the difference in 30-day mortality between males and females reduced.[104]
- Other data indicate that the rapid increase in the population ≥65 years of age has contributed to the reduction of HD mortality. From CDC WONDER data from 2011 through 2017, a deceleration in the decline in HD mortality was observed with a <1% annualized decrease. Taking into account the increase in the growth of the population ≥65 years of age combined with the slowing of the decrease in HD mortality resulted in an increase in the absolute number of HD deaths since 2011 (50 880 deaths; 8.5% total increase). However, the age-adjusted mortality for CHD continued to decline (2.7% annualized decrease) and the absolute number of CHD deaths declined (2.5% total decrease over the time period) between 2011 and 2017.[105]
- An analysis of those enrolled in Medicare Advantage or traditional Medicare from 2009 to 2018 presenting with STEMI (n=557 309) and NSTEMI (n=1 670 193) identified significant 30-day

mortality rate differences in 2009 that were no longer present in 2018.[106] In 2018, the 30-day mortality for STEMI was 17.7% in those with Medicare Advantage and 17.8% in those with traditional Medicare (difference, 0.0 percentage points [95% CI, −0.7 to 0.6]); for NSTEMI, the 30-day mortality rate was 10.9% in those with Medicare Advantage and 11.1% in those with traditional Medicare (difference, −0.2 percentage points [95% CI, −0.4 to 0.1]).

- An analysis of the ISCHEMIA trial (N=5179) compared 4-year mortality in trial participants classified as having mild/no ischemia, moderate ischemia, or severe ischemia. Compared with those with mild/no ischemia, 4-year mortality rates were similar in those with moderate (HR, 0.89 [95% CI, 0.61–1.30]) and severe (HR, 0.83 [95% CI, 0.57–1.21]) ischemia.[107]
- In extended follow-up (median, 5.7 years), ISCHEMIA participants randomized to an initial invasive strategy did not have increased mortality (HR, 1.00 [95% CI, 0.85–1.18]) compared with those randomized to an initial invasive strategy.[108]
- A meta-analysis of 56 studies determined that females with STEMI have higher mortality risk (OR, 1.91 [95% CI, 1.84–1.99]) than males.[109]
- An NIS analysis spanning 2004 to 2018 determined that females had a higher incidence of mortality after PCI than males (1.12% mortality compared with 0.78%).[110]
- A prospective analysis of data on 5064 Black adults in the JHS between 2019 and 2021 found that participants with CHD (HR, 1.59 [95% CI, 1.22–2.08]), diabetes (HR, 1.50 [95% CI, 1.22–1.85]), or stroke (HR, 1.74 [95% CI, 1.24–2.42]) had higher risk for all-cause mortality compared with those with no cardiometabolic morbidities.[111] Those with ≥2 cardiometabolic morbidities had higher risk of all-cause mortality with the highest risk among those with diabetes, stroke, and CHD (HR, 3.68 [95% CI, 1.96–6.93].

Social Determinants and Health Equity of Mortality

- In-hospital mortality is higher in females than in males with STEMI (7.4% versus 4.6%) and NSTEMI (4.8% versus 3.9%). An analysis of NCDR data from 2010 to 2015 reported that females admitted with STEMI had decreased survival to discharge compared with males (OR, 0.63 [95% CI, 0.52–0.76]).[112,113] Females experience longer door-to-balloon times and lower rates of GDMT than males; however, a 4-step systems-based approach to minimize STEMI care variability at the Cleveland Clinic decreased the difference in 30-day mortality between males and females.[114]
- An analysis of the STS database including 1 042 056 patients who underwent isolated CABG between 2011 and 2018 found that Black individuals had higher overall mortality than White individuals (OR, 1.11 [95% CI, 1.05–1.18]).[115] Likewise, odds of death were higher in females compared with males (OR, 1.26 [95% CI, 1.21–1.30]).
- A pooled analysis of 21 randomized PCI trials including 32 877 patients (27.8% females) found that in multivariable-adjusted analyses, female sex was associated with 5-year risks of MACEs (HR, 1.14 [95% CI, 1.01–1.30]) and ischemia-driven target lesion vascularization (HR, 1.23 [95% CI, 1.05–1.44]) but not all-cause or cardiovascular mortality (HR, 0.91 [95% CI, 0.75–1.09] and 0.97 [95% CI, 0.73–1.29], respectively).[116]
- On the basis of pooled data from the FHS, ARIC, CHS, MESA, CARDIA, and JHS studies of the NHLBI (1995–2012), within 1 year after a first MI (unpublished NHLBI tabulation):
  - At ≥45 years of age, 18% of males and 23% of females will die.
  - At 45 to 64 years of age, 3% of White males, 5% of White females, 9% of Black males, and 10% of Black females will die.
  - At 65 to 74 years of age, 14% of White males, 18% of White females, 22% of Black males, and 21% of Black females will die.
  - At ≥75 years of age, 27% of White males, 29% of White females, 19% of Black males, and 31% of Black females will die.
  - In part because females have MIs at older ages than males, they are more likely to die of MI within a few weeks.
- On the basis of pooled data from the FHS, ARIC, CHS, MESA, CARDIA, and JHS studies of the NHLBI (1995–2012), within 5 years after a first MI (unpublished NHLBI tabulation):
  - At ≥45 years of age, 36% of males and 47% of females will die.
  - At 45 to 64 years of age, 11% of White males, 17% of White females, 16% of Black males, and 28% of Black females will die.
  - At 65 to 74 years of age, 25% of White males, 30% of White females, 33% of Black males, and 44% of Black females will die.
  - At ≥75 years of age, 55% of White males, 60% of White females, 61% of Black males, and 64% of Black females will die.
- An analysis conducted in the CARDIA study (N=5112) with a median follow-up >33 years identified that premature CVD risk in Black participants was attenuated after adjustment for lifestyle, neighborhood, and socioeconomic factors.[117] For example, the 2.4-fold increased CVD risk in Black females (95% CI, 1.71–3.49) relative to White females was no longer significant after adjustment for clinical, lifestyle, socioeconomic, and neighborhood factors.

The largest decreases in the race-specific estimate for CVD risk occurred with adjustment for clinical (87%), neighborhood (32%), and socioeconomic (23%) factors.

- In MESA, an analysis (N=6814) similarly reported that compared with White participants, Black participants had increased risk of mortality (HR, 1.34 [95% CI, 1.19–1.51]), which decreased after adjustment for socioeconomic factors (HR, 1.16 [95% CI, 1.01–1.34]).[118]
- A large regional health care system in Northern California conducted an analysis of 1-year mean residential-level estimates of PM2.5 in individuals with ASCVD. A 10–μg/m^3 increase in PM2.5 exposure was associated with an HR of 1.20 (95%, 1.11–1.30) increased risk of cardiovascular mortality but not stroke or MI.[119]
- A meta-analysis of 30 cardiac surgery studies identified that females have an increased risk of short-term mortality after CABG (aOR, 1.40 [95% CI, 1.32–1.49]; I^2=79%) compared with males.[83]
- Sex differences in outcomes after MI are well established. In Olmsted County, Minnesota, mortality risk after premature MI (defined as 18–55 years of age in males and 18–65 years of age in females) declined by 66% in females (HR, 0.34 [95% CI, 0.17–0.68]) from 1987 through 2012. In contrast, no significant decline in mortality was observed in males.[120] A multicenter study in London, UK (N=26 799), determined that multivariable-adjusted sex differences in survival after STEMI over a median of 4.1 years (interquartile range, 2.2–5.8 years) of follow-up were significant in those >55 years of age (HR, 1.20 [95% CI, 1.09–1.41] for females compared with males).[121]

Complications

- A comparison of 2 NIS cohorts of young adults (18–44 years of age) hospitalized with AMI in 2007 and 2017 revealed an overall increased admission rate, with a decline in males (77.1% to 66.1%) and a rise in females (28.9% to 33.9%).[122] Post-AMI complications, including cardiogenic shock (aOR, 1.16 [95% CI, 1.06–1.27]) and SVT (aOR, 3.76 [95% CI, 3.18–4.44]), increased, whereas all-cause mortality was comparable in these 2 time periods. (aOR, 1.01 [95% CI, 0.93–1.10]; *P*=0.749).
- STEMI confers greater in-hospital risks than NSTEMI, including death (6.4% for STEMI, 3.4% for NSTEMI), cardiogenic shock (4.4% versus 1.6%, respectively), and bleeding (8.5% versus 5.5%, respectively).[123] In the NCDR ACTION Registry–GWTG, a measure of neighborhood SES based on census data was associated with in-hospital deaths and major bleeding in patients with AMI. Compared with those in the highest quintile of neighborhood SES, those residing in the lowest SES quintile experienced higher rates of in-hospital death (OR, 1.10 [95% CI, 1.02–1.18]) and major bleeding (OR, 1.10 [95% CI, 1.05–1.15]).[124]
- In an analysis of the NIS, females with AMI presenting with spontaneous coronary artery dissection had higher odds of in-hospital mortality (6.8%) than females without spontaneous coronary artery dissection (3.8%; OR, 1.87 [95% CI, 1.65–2.11]; *P*<0.001) in a propensity-matched analysis.[125]
- In the NCDR ACTION Registry–GWTG, patients with STEMI or NSTEMI with nonobstructive coronary arteries (<50% stenosis) had lower in-hospital mortality than patients with obstructive CAD (1.1% versus 2.9%; *P*<0.001). Nonobstructive coronary arteries were more common in females than males (10.5% versus 3.4%; *P*<0.001), but no difference in in-hospital mortality was observed between females and males with nonobstructive coronary arteries (*P*=0.84).[126]
- In a propensity score–matched analysis from the NIS HCUP that included discharges with MI as the principal diagnosis from 2012 to 2014, patients with concomitant delirium had higher rates of in-hospital mortality than those without delirium (10.5% versus 7.6%; RR, 1.39 [95% CI, 1.2–1.6]; *P*<0.001).[127]
- In a trial of patients presenting with STEMI (N=402), those with HF symptoms (New York Heart Association functional class ≥2; n=76) within 30 days after PCI for STEMI experienced increased risk of death or hospitalization for HF within 1 year compared with those without HF symptoms (HR, 3.78 [95% CI, 1.16–12.22]; *P*=0.03).[128]
- The burden of rehospitalizations for AMI is substantial. Among Medicare fee-for-service patients ≥65 years of age who were discharged alive after AMI in 2009 to 2014, the rate of 1-year recurrent AMI was 5.3% (95% CI, 5.27%–5.41%) with a median of 115 days (interquartile range, 34–230 days) of time from discharge to recurrent AMI.[129]
- Sudden death after MI is common. A secondary analysis of IMPROVE-IT (N=18 144) determined the cumulative incidence rate of sudden death after MI as 2.47% (95% CI, 2.23%–2.73%) at the 7-year follow-up.[130]

Age, Sex, Race, and Complications

- On the basis of pooled data from the FHS, ARIC, CHS, MESA, CARDIA, and JHS studies of the NHLBI (1995–2012; unpublished NHLBI tabulation), of those who have a first MI, the percentage with a recurrent MI or fatal CHD within 5 years is as follows:
  - At ≥45 years of age, 17% of males and 21% of females.

  - At 45 to 64 years of age, 11% of White males, 15% of White females, 22% of Black males, and 32% of Black females.
  - At 65 to 74 years of age, 12% of White males, 17% of White females, 30% of Black males, and 30% of Black females.
  - At ≥75 years of age, 21% of White males, 20% of White females, 45% of Black males, and 20% of Black females.
- The percentage of people with a first MI who will have HF in 5 years is as follows:
  - At ≥45 years of age, 16% of males and 22% of females.
  - At 45 to 64 years of age, 6% of White males, 10% of White females, 13% of Black males, and 25% of Black females.
  - At 65 to 74 years of age, 12% of White males, 16% of White females, 20% of Black males, and 32% of Black females.
  - At ≥75 years of age, 25% of White males, 27% of White females, 23% of Black males, and 19% of NH Black females.
- The percentage of people with a first MI who will have an incident stroke within 5 years is as follows:
  - At ≥45 years of age, 4% of males and 7% of females.
  - At ≥45 years of age, 5% of White males, 6% of White females, 4% of Black males, and 10% of Black females.
- The median survival time (in years) after a first MI is as follows:
  - At ≥45 years of age, 8.2 for males and 5.5 for females.
  - At ≥45 years of age, 8.4 for White males, 5.6 for White females, 7.0 for Black males, and 5.5 for Black females.
- A systematic review and pooled analysis of 4 CABG trials compared sex differences in outcomes between females (n=2714) and males (n=10 479). Over the 5-year follow-up, females had a significantly increased risk of major adverse cardiac and cerebrovascular events (aHR, 1.12 [95% CI, 1.04–1.21]), MI (aHR, 1.30 [95% CI, 1.11–1.52]), and repeat revascularization (aHR, 1.22 [95% CI, 1.04–1.43]) but not stroke (aHR, 1.17 [95% CI, 0.90–1.43]).[131]
- A meta-analysis of 56 studies of STEMI identified that compared with males, females hospitalized with STEMI are more likely to experience repeat MI (OR, 1.25 [95% CI, 1.00–1.56]), stroke (OR, 1.67 [95% CI, 1.27–2.20]), and major bleeding (OR, 1.82 [95% CI, 1.56–2.12]).[109]
- An analysis of the US Nationwide Readmissions Database determined that after hospitalization for AMI, females had 13% increased risk of 6-month HF hospitalization compared with males (6.4% in females versus 5.8% in males; HR, 1.13 [95% CI, 1.05–1.21]).[132]
- An Australian registry of individuals who had undergone PCI (N=13 996) from 2008 to 2020 determined that female sex was associated with increased 2-year readmission (HR, 1.29 [95% CI, 1.11–1.48]) compared with male sex.[133]

Hospital Discharges and Ambulatory Care (See Chart 21-5)

- From 2011 to 2021, the number of inpatient discharges from short-stay hospitals with CHD as the first-listed diagnosis decreased from 1 193 438 to 886 904.
- From 1997 through 2021, the number of hospital discharges for CHD generally declined (Chart 21-5).
- In 2019, there were 14 167 000 physician office visits for CHD (unpublished NHLBI tabulation using NAMCS[134]). In 2021, there were 909 467 ED visits with a primary diagnosis of CHD (unpublished NHLBI tabulation using HCUP[82]).
- In the NIS, the mean length of hospital stay for patients with STEMI with primary PCI declined from 3.3 days in 2005 to 2.7 days in 2014; the proportion of hospitalizations with length of stay >3 days declined from 31.9% in 2005 to 16.9% in 2014.[135]
- In the CathPCI registry, a composite of use of evidence-based medical therapies, including aspirin, P2Y12 inhibitors, and statins, was high (89.1% in 2011 and 93.5% in 2014). However, in the ACTION-GWTG registry, metrics shown to need improvement were defect-free care (median hospital performance rate, 78.4% in 2014), P2Y12 inhibitor use in eligible medically treated patients with AMI (56.7%), and use of aldosterone antagonists in patients with LV systolic dysfunction and either diabetes or HF (12.8%).[123]
- Among 147 600 individuals with premature ASCVD (≤55 years of age) receiving care in the Veterans Affairs health care system from October 1, 2014, through September 30, 2015, there were 10 413 females and 137 187 males. In adjusted analyses, females were less likely to receive antiplatelet therapy (OR, 0.47 [95% CI, 0.45–0.50]), any statin (OR, 0.62 [95% CI, 0.59–0.66]), or high-intensity statin (OR, 0.63 [95% CI, 0.59–0.66]) than males.[136]
- Among individuals presenting with an MI or undergoing coronary revascularization in the Veterans Affairs health care system from July 24, 2015, through December 9, 2019 (N=81 372), the proportions receiving lipid-lowering intensification were 33.3% at 14 days, 41.9% at 3 months, and 47.3% at 12 months after hospitalization.[137] Lipid-lowering

intensification was defined as increasing or initiating therapies to achieve LDL target goals of 70 or 100 mg/dL.
- An analysis of the ISCHEMIA trial (N=5179) compared days alive out of the hospital or extended care facilities among trial participants classified as having mild/no ischemia, moderate ischemia, or severe ischemia and randomized to invasive or initially conservative management strategies. At 4 years, there was no significant difference between the 2 groups (1415.0 days with conservative management and 1412.2 days with invasive management; *P*=0.65).[138]

Cost

- The estimated direct cost of CHD in 2020 to 2021 (average annual) was $52.8 billion (MEPS,[139] unpublished NHLBI tabulation).
- The estimated direct and indirect cost of CHD in 2020 to 2021 (average annual) was $129.3 billion (MEPS,[139] unpublished NHLBI tabulation).
- MI ($14.3 billion) and CHD ($8.7 billion) were 2 of the 10 most expensive conditions treated in US hospitals in 2017.[140]
- In 642 105 Medicare beneficiaries hospitalized for AMI between 2011 and 2014, 30-day episode payments averaged $22 128 but varied 2-fold across hospitals. Median costs were $20 207 in the lowest quartile versus $24 174 in the highest quartile of hospitals.[141]
- In Medicare beneficiaries hospitalized with AMI, the 180-day expenditures increased from an average of $32 182 per person in 1999 to 2000 to $36 836 in 2008 and remained relatively stable thereafter, with expenditures of $36 668 in 2013 to 2014.[142]
- In 11 969 patients with AMI from 233 US hospitals who underwent PCI from 2010 to 2013, average hospital costs were higher for patients with STEMI ($19 327) compared with patients with NSTEMI ($18 465; *P*=0.002) and higher among elderly patients ($19 575 for those ≥65 years of age versus $18 652 for those <65 years of age; *P*=0.004). Forty-five percent of costs were related to the catheterization laboratory, 22% to room and board, 14% to supplies, and 9% to pharmacy costs. At 1 year after discharge, hospital and ED costs averaged $8037, with three-quarters attributable to hospitalizations ($6116 for hospitalizations, $1334 for outpatient hospital stays, and $587 for ED visits).[143]
- Among 26 255 patients with isolated CABG in a regional STS database between 2012 and 2019, the median hospital cost was higher among those with open CABG ($35 011) than those with minimally invasive CABG surgery ($27 906; *P*<0.001) after propensity score matching. There were no significant differences in mortality or morbidity, although patients with open CABG had longer hospital stays (7 days versus 6 days; *P*=0.005) than those with minimally invasive CABG surgery.[144]
- An observational analysis of data on young adults (18–45 years of age) who underwent PCI in the 2004 to 2018 NIS found that the inflation-adjusted care cost significantly increased from $21 567 in 2004 to $24 173 in 2018 ($P_{trend}$<0.01).[110]

Global Burden

(See Table 21-3 and Charts 21-6 and 21-7)

- Based on 204 countries and territories in 2021[145]:
  - An estimated 8.99 (95% UI, 8.26–9.53) million total deaths attributable to IHD occurred (Table 21-3). Among regions, IHD mortality rates were highest for Central Asia, Eastern Europe, and North Africa and the Middle East. Mortality was lowest for high-income Asia Pacific (Chart 21-6).
  - Globally, it was estimated that 254.28 (95% UI, 221.45–295.49) million people lived with IHD, and it was more prevalent in males than in females (145.31 [95% UI, 125.89–167.45] and 108.97 [95% UI, 95.25–127.39] million people, respectively). North Africa and the Middle East had the highest prevalence rates of IHD among regions, followed by Eastern Europe and South and Central Asia (Chart 21-7).
- Among 31 443 respondents ≥50 years of age from 6 low- and middle-income countries participating in the WHO SAGE Wave 1, prevalence of angina ranged between 8% in China and 39% in Russia and was higher in females than males.[146]

Acute Coronary Syndrome

ICD-9 410, 411; *ICD-10* I20.0, I21, I22.

- In a study of 9450 patients with ACS, a modified GRACE risk score incorporating continuous high-sensitivity cardiac troponin at presentation showed improved discrimination and reclassification for in-hospital (AUC, 0.878 versus 0.780; NRI, 0.097), 30-day (AUC, 0.858 versus 0.771; NRI, 0.08), and 1-year (AUC, 0.813 versus 0.797; NRI, 0.056) all-cause mortality compared with the original GRACE score.[147]
- In a 2019 to 2020 NIS study (N=32 355 827), patients with STEMI with COVID-19 had higher mortality (aOR, 3.10 [95% CI, 2.40–4.02]; *P*<0.01) and longer length of stay (aOR, 1.66 [95% CI, 1.07–2.25]; *P* <0.01), and unstable patients with NSTEMI had longer admission-to-PCI times (0.45 days [95% CI, 0.16–0.76 days]; *P*<0.01).[148]
- In 2021, there were 591 129 ACS principal diagnosis discharges. This estimate was derived by adding the principal diagnoses for MI (584 499) to those

for unstable angina (6630; unpublished NHLBI tabulation using HCUP[82]).

- When all listed discharge diagnoses in 2021 were included, the corresponding number of inpatient hospital discharges was 1 391 719 unique hospitalizations for ACS. Of the total, 1 375 674 were for MI alone, and 16 045 were for unstable angina alone (unpublished NHLBI tabulation[82]).
- In a population-level study in Italy, the incidence rate of PCI for ACS decreased from 178 (before the COVID-19 outbreak) to 120 (after the COVID-19 outbreak) cases per 100 000 residents per year (IRR, 0.68 [95% CI, 0.65–0.70]).[149] Females (IRR, 0.60 [95% CI, 0.57–0.65]) had fewer PCIs for ACS than males (IRR, 0.70 [95% CI: 0.68–0.73]; $P_{interaction}$<0.011).
- Among 17 562 patients with ACS between 2005 and 2017 who lived beyond 30 days in a large PCI registry in Australia, 83.3% were on a β-blocker. Risk of overall mortality was lower among those who were on a β-blocker (aHR, 0.87 [95% CI, 0.78–0.97]; *P*=0.014) compared with those who were not. This mortality benefit was observed among patients with LVEF <35% (aHR, 0.63 [95% CI, 0.44–0.91]; *P*=0.013) and 35% to 50% (aHR, 0.80 [95% CI, 0.68–0.95]; *P*=0.01]) but not among those with LVEF >50%.[150]
- In a retrospective analysis of 43 446 patients who were referred for cardiac catheterization at a medical center in Massachusetts between January 2006 and June 2017, 26 545 patients had ACS. Younger patients with ACS (<35 years of age) were more likely to be White, obese, and a smoker and to report a family history of CAD, but they were less likely to have diabetes, hypertension, and hyperlipidemia than older patients. Younger patients with ACS also had a higher prevalence of elevated troponin, late-presentation STEMI, and cardiogenic shock than older patients. Compared with patients with ACS who were 36 to 54 years of age, those who were ≤35 years of age had higher odds of 30-day mortality (aOR, 5.65 [95% CI, 2.49–12.82]; *P*<0.001).[151]
- A retrospective analysis of 801 195 patients with ACS in the NIS identified disparities in outcomes of patients admitted based on insurance (Medicaid, Medicare, private, and no insurance). Patients who had no insurance (aOR,1.46 [95% CI, 1.26–1.69]; *P*≤0.01) or were on Medicaid (aOR, 1.16 [95% CI, 1.03–1.30]; *P*=0.01) had higher mortality than those who had private insurance.[152]
- A retrospective analysis of data on 10 019 patients from the Epi-Cardio Registry in Argentina was conducted to examine sex differences in the presentation of ACS.[153] Females were more likely than males to present with non–ST-segment–elevation ACS (60.3% versus 46.7%; *P*<0.001). This sex difference was driven mainly by a higher prevalence of ACS with nonobstructive coronary arteries (20.9% versus 6.6%) in young females because ACS without coronary lesions was mostly non–ST-segment–elevation ACS (77.7% versus 22.3%). There was no significant sex difference in the clinical presentation among patients with obstructive CHD.
- Among adults with ACS from the PLATO trial, the ABC-ACS ischemia model for predicting 1-year risk of CVD and MI that included growth differentiation factor 15 and NT-proBNP had greater prognostic value than all candidate variables (C indices, 0.71 and 0.72 in the development and validation cohorts, respectively).[154]
- A retrospective cohort study of 257 948 adults in the NIH Research Health Informatics Collaborative with suspected ACS in the United Kingdom between 2010 and 2017 found a positive and graded association between high-sensitivity CRP level and mortality at baseline.[155] This association persisted after 3 years for those with high-sensitivity CRP of 2.0 to 4.9 mg/L (aHR, 1.32 [95% CI, 1.18–1.48]), 5 to 9.9 mg/L (aHR, 1.40 [95% CI, 1.26–1.57]), and 10 to 15 mg/L (aHR, 2.00 [95% CI, 1.75–2.28]).

Stable AP

ICD-9 413; *ICD-10* I20.1 to I20.9.

Prevalence

(See Table 21-2 and Chart 21-8)

- According to data from NHANES 2017 to 2020, the prevalence of AP among adults (≥20 years of age) was 3.9% (10.8 million adults; Table 21-2).
- On the basis of NHANES 2017 to 2020, the prevalence of AP increased with age from <1% among males and females 20 to 39 years of age to >9% among males and females ≥80 years of age (Chart 21-8).
- On the basis of data from NHANES in 2009 to 2012, an average of 3.4 million people ≥40 years of age in the United States had angina each year compared with 4 million in 1988 to 1994. Declines in angina symptoms have occurred for NH White people but not for NH Black people.[156]
- Among 1612 of 4139 eligible patients diagnosed with CAD in a network consisting of 15 primary care clinics in Massachusetts, the prevalence of angina was measured with the Seattle Angina Questionnaire-7; 21.2% reported angina symptoms at least once monthly, and among those, 12.5% reported daily or weekly angina symptoms, and 8.7% reported monthly angina symptoms.[157]

Table 21-1. CHD in the United States

Population group	Prevalence, CHD, 2017–2020, ≥20 y of age	Prevalence, MI, 2017–2020, ≥20 y of age	Mortality,* CHD, 2022, all ages	Age-adjusted mortality rates per 100 000 (95% CI),* CHD, 2022	Mortality,* MI, 2022, all ages	Age-adjusted mortality rates per 100 000 (95% CI),* MI, 2022
Both sexes	20 500 000 (7.1%) [95% CI, 6.1%–8.3%]	9 300 000 (3.2%) [95% CI, 2.5%–4.0%]	371 506	87.6 (87.3–87.9)	103 905	24.5 (24.4–24.7)
Males	11 700 000 (8.7%)	6 100 000 (4.5%)	223 952 (60.3%)†	121.9 (121.4–122.4)	62 571 (60.2%)†	33.3 (33.0–33.6)
Females	8 800 000 (5.8%)	3 200 000 (2.1%)	147 554 (39.7%)†	60.3 (60.0–60.6)	41 334 (39.8%)†	17.2 (17.0–17.3)
NH White males	9.4%	4.8%	172 181	126.8 (126.2–127.4)	48 545	35.3 (34.9–35.6)
NH White females	5.9%	2.2%	112 164	61.9 (61.5–62.3)	31 205	17.7 (17.5–18.0)
NH Black males	6.2%	4.0%	24 839	144.1 (142.2–146.0)	6695	38.2 (37.2–39.1)
NH Black females	6.3%	2.3%	18 264	75.8 (74.7–76.9)	5193	21.5 (20.9–22.1)
Hispanic males	6.8%	3.1%	16 840	90.7 (89.3–92.2)	4664	24.3 (23.5–25.0)
Hispanic females	6.1%	1.9%	10 754	46.7 (45.8–47.6)	3141	13.5 (13.0–14.0)
NH Asian males	5.2%	2.8%	6538‡	70.8 (69.1–72.6)‡	1855‡	19.8 (18.9–20.8)‡
NH Asian females	3.9%	0.5%	4418‡	35.2 (34.1–36.2)‡	1278‡	10.2 (9.6–10.8)‡
NH American Indian or Alaska Native	…	…	1973	76.5 (73.1–80.0)	572	22.3 (20.4–24.1)
NH Native Hawaiian or Pacific Islander			551	94.1 (86.0–102.1)	164	27.1 (22.8–31.3)

In March 2020, the COVID-19 pandemic halted NHANES field operations. Because data collected in the partial 2019 to 2020 cycle are not nationally representative, they were combined with previously released 2017 to 2018 data to produce nationally representative estimates.[158] CHD includes people who responded "yes" to at least 1 of the questions in "Has a doctor or other health professional ever told you that you had CHD, angina or AP, heart attack, or MI?" Those who answered "no" but were diagnosed with Rose angina are also included (the Rose questionnaire is administered only to survey participants >40 years of age). CIs have been added for overall prevalence estimates in key chapters. CIs have not been included in this table for all subcategories of prevalence for ease of reading.

AP indicates angina pectoris; CHD, coronary heart disease; COVID-19, coronavirus disease 2019; ellipses (…), data not available; MI, myocardial infarction; NH, non-Hispanic; and NHANES, National Health and Nutrition Examination Survey.

*Mortality for Hispanic people, NH American Indian or Alaska Native people, and NH Asian and Pacific Islander people should be interpreted with caution because of inconsistencies in reporting Hispanic origin or race on the death certificate compared with censuses, surveys, and birth certificates. Studies have shown underreporting on death certificates of American Indian or Alaska Native decedents, Asian and Pacific Islander decedents, and Hispanic decedents, as well as undercounts of these groups in censuses.

†These percentages represent the portion of total CHD and MI mortality that is for males vs females.

‡Includes Chinese people, Filipino people, Japanese people, and other Asian people.

Sources: Prevalence: unpublished National Heart, Lung, and Blood Institute (NHLBI) tabulation using National Health and Nutrition Examination Survey.[2] Percentages for racial and ethnic groups are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2020 US population estimates. These data are based on self-reports. Incidence: Atherosclerosis Risk in Communities study (2005–2014),[5] unpublished tabulation by NHLBI, extrapolated to the 2014 US population. Mortality (for underlying cause of CHD): unpublished NHLBI tabulation using National Vital Statistics System[102] and Centers for Disease Control and Prevention Wide-Ranging Online Data for Epidemiologic Research.[103]

Table 21-2. AP* in the United States

Population group	Prevalence, 2017–2020, ≥20 y of age
Both sexes	10 800 000 (3.9%) [95% CI, 3.3%–4.5%]
Males	5 600 000 (4.3%)
Females	5 200 000 (3.6%)
NH White males	4.7%
NH White females	3.5%
NH Black males	2.7%
NH Black females	4.1%
Hispanic males	3.6%
Hispanic females	4.3%
NH Asian or Pacific Islander males	2.7%
NH Asian or Pacific Islander females	2.7%

In March 2020, the COVID-19 pandemic halted NHANES field operations. Because data collected in the partial 2019 to 2020 cycle are not nationally representative, they were combined with previously released 2017 to 2018 data to produce nationally representative estimates.[158] AP includes people who either answered "yes" to the question of ever having angina or AP or being diagnosed with Rose angina (the Rose questionnaire is administered only to survey participants >40 years of age).

AP indicates angina pectoris; COVID-19, coronavirus disease 2019; ellipses (…), data not available; NH, non-Hispanic; and NHANES, National Health and Nutrition Examination Survey.

*AP is chest pain or discomfort that results from insufficient blood flow to the heart muscle. Stable AP is predictable chest pain on exertion or under mental or emotional stress. The incidence estimate is for AP without myocardial infarction.

Sources: Prevalence: unpublished National Heart, Lung, and Blood Institute tabulation using NHANES.[2] Percentages for racial and ethnic groups are age adjusted for US adults ≥20 years of age. Estimates from NHANES 2017 to 2020 were applied to 2020 population estimates (≥20 years of age).

Table 21-3. Global Mortality and Prevalence of IHD, by Sex, 2021

	Both sexes		Male		Female	
	Deaths (95% UI)	Prevalence (95% UI)	Deaths (95% UI)	Prevalence (95% UI)	Deaths (95% UI)	Prevalence (95% UI)
Total number (millions), 2021	8.99 (8.26 to 9.53)	254.28 (221.45 to 295.49)	5.00 (4.68 to 5.34)	145.31 (125.89 to 167.45)	3.99 (3.54 to 4.32)	108.97 (95.25 to 127.39)
Percent change (%) in total number, 1990–2021	67.53 (58.76 to 75.87)	126.69 (117.83 to 136.62)	78.35 (66.61 to 91.90)	123.62 (115.17 to 132.70)	55.69 (45.92 to 66.83)	130.92 (121.31 to 141.66)
Percent change (%) in total number, 2010–2021	21.03 (15.88 to 26.46)	39.56 (32.12 to 48.00)	22.96 (15.84 to 30.99)	36.63 (29.29 to 44.87)	18.69 (11.82 to 25.33)	43.66 (35.95 to 52.64)
Rate per 100 000, age standardized, 2021	108.73 (99.60 to 115.38)	2,946.38 (2,572.69 to 3,424.32)	136.84 (127.37 to 145.90)	3,610.24 (3,153.05 to 4,164.95)	85.32 (75.90 to 92.31)	2,357.61 (2,063.31 to 2,751.95)
Percent change (%) in rate, age standardized, 1990–2021	−31.57 (−34.86 to −28.33)	1.43 (−2.55 to 5.94)	−27.08 (−31.50 to −21.72)	−2.12 (−5.88 to 2.23)	−36.57 (−40.44 to −32.49)	4.76 (0.56 to 9.85)
Percent change (%) in rate, age standardized, 2010–2021	−13.02 (−16.59 to −9.23)	2.90 (−2.36 to 8.95)	−11.37 (−16.30 to −5.67)	0.47 (−4.68 to 6.24)	−14.96 (−19.81 to −10.01)	5.99 (0.31 to 12.51)

These estimates reflect improvements in demography and population estimation, statistical and geospatial modeling methods, and the addition of nearly 3000 new data sources since the 2024 AHA Statistical Update. During each annual GBD Study cycle, population health estimates are produced for the full time series. Improvements in statistical and geospatial modeling methods and the addition of new data sources may lead to changes in past results across GBD Study cycles.

GBD indicates Global Burden of Diseases, Injuries, and Risk Factors; IHD, ischemic heart disease, and UI, uncertainty interval.

Source: Data courtesy of the GBD Study. Institute for Health Metrics and Evaluation. Used with permission. All rights reserved.[145]

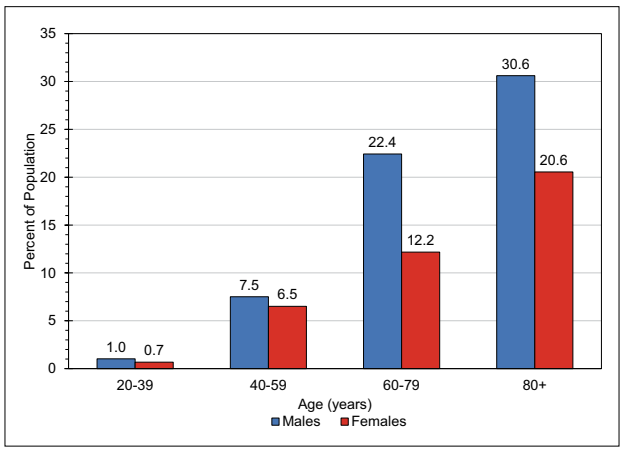

Chart 21-1. Prevalence of CHD by age and sex, United States (NHANES, 2017–2020).

In March 2020, the COVID-19 pandemic halted NHANES field operations. Because data collected in the partial 2019 to 2020 cycle are not nationally representative, they were combined with previously released 2017 to 2018 data to produce nationally representative estimates.[158]

CHD indicates coronary heart disease; COVID-19, coronavirus disease 2019; and NHANES, National Health and Nutrition Examination Survey.

Source: Unpublished National Heart, Lung, and Blood Institute tabulation using NHANES.[2]

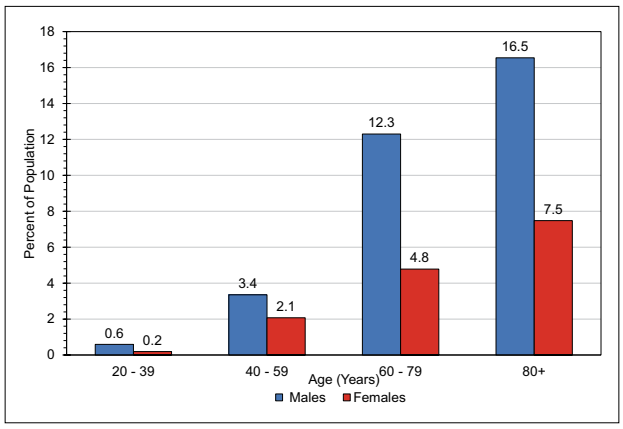

Chart 21-2. Prevalence of MI by age and sex, United States (NHANES, 2017–2020).

In March 2020, the COVID-19 pandemic halted NHANES field operations. Because data collected in the partial 2019 to 2020 cycle are not nationally representative, they were combined with previously released 2017 to 2018 data to produce nationally representative estimates.[158]

MI includes people who answered "yes" to the question of ever having had a heart attack or MI.

COVID-19 indicates coronavirus disease 2019; MI, myocardial infarction; and NHANES, National Health and Nutrition Examination Survey.

Source: Unpublished National Heart, Lung, and Blood Institute tabulation using NHANES.[2]

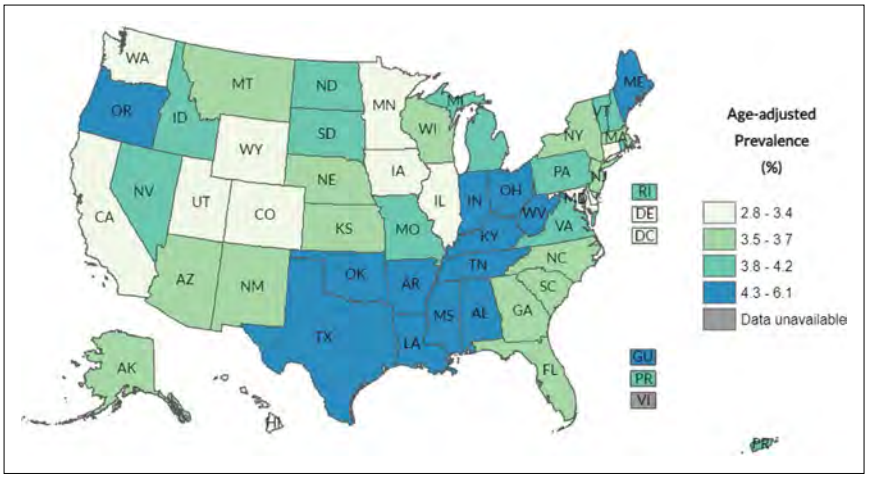

Chart 21-3. "Ever told you had a heart attack (MI)?" Age-adjusted US prevalence by state (BRFSS prevalence and trends data, 2022).
Original chart has been modified to remove white space between map and legend.
BRFSS indicates Behavioral Risk Factor Surveillance System; and MI, myocardial infarction.
Source: BRFSS prevalence and trends data.[4]

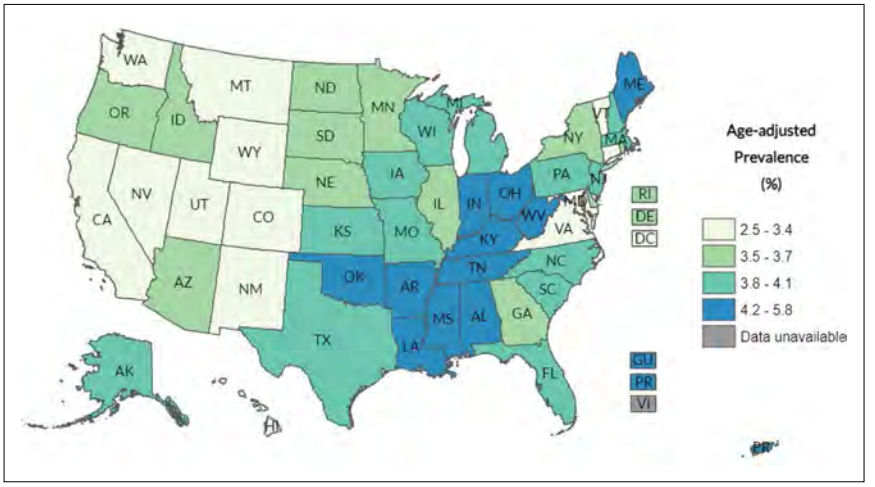

Chart 21-4. "Ever told you had angina or CHD?" Age-adjusted US prevalence by state (BRFSS prevalence and trends data, 2022).
Original chart has been modified to remove white space between map and legend.
BRFSS indicates Behavioral Risk Factor Surveillance System; and CHD, coronary heart disease.
Source: BRFSS prevalence and trends data.[4]

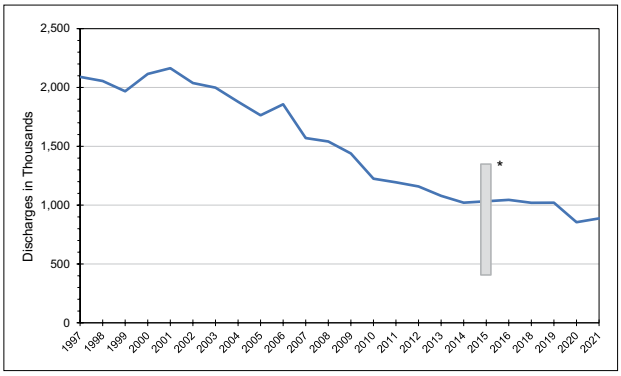

Chart 21-5. Hospital discharges for CHD, United States (HCUP, 1997–2021).
Hospital discharges include people discharged alive, dead, and status unknown.
CHD indicates coronary heart disease; and HCUP, Healthcare Cost and Utilization Project.
*Data not available for 2015. Readers comparing data across years should note that beginning October 1, 2015, a transition was made from the 9th revision to the 10th revision of the *International Classification of Diseases*. This should be kept in consideration because coding changes could affect some statistics, especially when comparisons are made across these years.
Source: Unpublished National Heart, Lung, and Blood Institute tabulation using HCUP.[82]

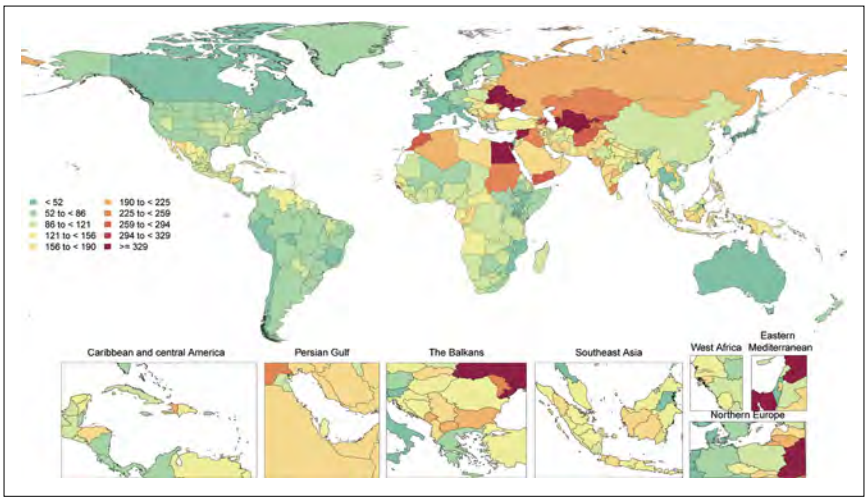

Chart 21-6. Age-standardized global mortality rates of IHD per 100 000, both sexes, 2021.
These estimates reflect improvements in demography and population estimation, statistical and geospatial modeling methods, and the addition of nearly 3000 new data sources since the 2024 AHA Statistical Update. During each annual GBD Study cycle, population health estimates are produced for the full time series. Improvements in statistical and geospatial modeling methods and the addition of new data sources may lead to changes in past results across GBD Study cycles.
GBD indicates Global Burden of Diseases, Injuries, and Risk Factors; and IHD, ischemic heart disease.
Source: Data courtesy of the GBD Study. Institute for Health Metrics and Evaluation. Used with permission. All rights reserved.[145]

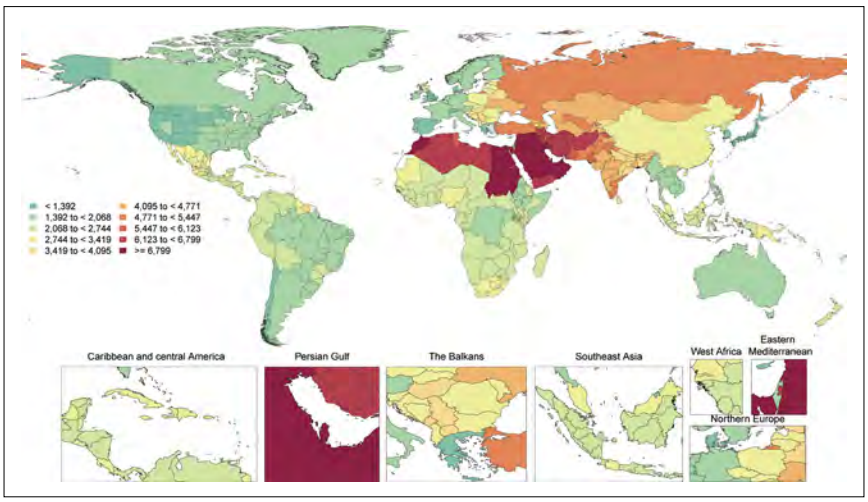

Chart 21-7. Age-standardized global prevalence rates of IHD per 100 000, both sexes, 2021.
These estimates reflect improvements in demography and population estimation, statistical and geospatial modeling methods, and the addition of nearly 3000 new data sources since the 2024 AHA Statistical Update. During each annual GBD Study cycle, population health estimates are produced for the full time series. Improvements in statistical and geospatial modeling methods and the addition of new data sources may lead to changes in past results across GBD Study cycles.
GBD indicates Global Burden of Diseases, Injuries, and Risk Factors; and IHD, ischemic heart disease.
Source: Data courtesy of the GBD Study. Institute for Health Metrics and Evaluation. Used with permission. All rights reserved.[145]

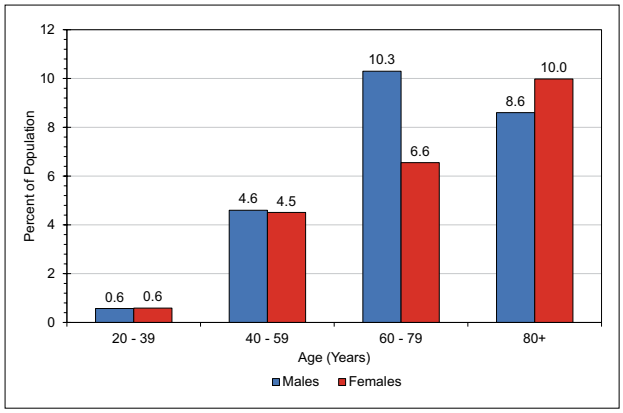

Chart 21-8. Prevalence of AP by age and sex, United States (NHANES, 2017–2020).
In March 2020, the COVID-19 pandemic halted NHANES field operations. Because data collected in the partial 2019 to 2020 cycle are not nationally representative, they were combined with previously released 2017 to 2018 data to produce nationally representative estimates.[158] AP includes people who either answered "yes" to the question of ever having angina or AP or being diagnosed with Rose angina.
AP indicates anginal pectoris; COVID-19, coronavirus disease 2019; and NHANES, National Health and Nutrition Examination Survey.
Source: Unpublished National Heart, Lung, and Blood Institute tabulation using NHANES.[2]

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22. CARDIOMYOPATHY AND HEART FAILURE

See Tables 22-1 through 22-3 and Charts 22-1 through 22-4

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Cardiomyopathy

ICD-9 425; *ICD-10* I42

2022, United States: Underlying cause mortality—19 739. Any-mention mortality—45 314.

Cardiomyopathy diagnoses account for a substantial number of inpatient and outpatient encounters annually.[1] According to HCUP 2021 data[2] for inpatient hospitalizations, cardiomyopathy was the principal diagnosis for 14 770, and it was included among all-listed diagnoses for 1 154 769.

Hypertrophic Cardiomyopathy

- HCM is a monogenic disorder with primarily autosomal dominant inheritance that is caused by 1 of hundreds of variants in >30 genes that primarily encode components of the sarcomere, with variants in *MYH7* and *MYBPC3* (cardiac myosin-binding protein C) being the most common.[3,4] A variant is identifiable in 30% to 60% of cases of familial HCM.
- A meta-analysis of prior GWASs found a strong correlation between common genetic variants associated with several LV traits, including increased LV mass, mean LV wall thickness, and radial strain, and HCM.[5] Two-sample mendelian randomization suggests a causal link between increased LV contractility and risk of developing HCM.
- The Sarcomeric Human Cardiomyopathy Registry studied 4591 patients with HCM contributing >24 000 PY of follow-up and observed a higher mortality rate in patients with HCM compared with unaffected individuals of a similar age in the US general population: 20 to 29 years of age, 0.39% versus 0.09% ($P<0.05$); 40 to 49 years of age, 0.66% versus 0.28% ($P=0.09$); and 60 to 69 years of age, 3.99% versus 1.33% ($P<0.01$). Risk for adverse events (ie, any ventricular arrhythmia, HF, AF, stroke, or death) was highest in patients diagnosed before 40 years of age versus after 60 years of age (cumulative incidence, 77% [95% CI, 72%–80%] by 60 years of age versus 32% [95% CI, 29%–36%] by 70 years of age). Adverse events were also higher in patients with versus without pathogenic sarcomere variants (HR, 1.98 [95% CI, 1.72–2.28]). AF (HR, 2.41 [95% CI, 1.98–2.94]) and HF (HR, 2.03 [95% CI, 1.68–2.45]) accounted for a substantial proportion of the adverse events despite typically not manifesting until years to decades after the initial diagnosis. Compared with males, females with HCM were at lower risk for ventricular arrhythmia (HR, 0.69 [95% CI, 0.51–0.94]; $P<0.05$) and AF (HR, 0.72 [95% CI, 0.60–0.87]; $P<0.001$) but higher risk for HF (HR, 1.28 [95% CI, 1.07–1.52]; $P<0.01$). There was no statistically significant difference in risk of each outcome for patients from underrepresented racial groups (all $P>0.05$).[6]
- A meta-analysis of 98 studies encompassing 70 510 patients with HCM from 1985 to 2020 demonstrated an overall incidence rate of SCD of 0.43%/y (95% CI, 0.37%/y–0.50%/y).[7] This rate decreased over time from 0.73%/y (95% CI, 0.53%/y–1.02%/y) in 1985 to 2000 to 0.32%/y (95% CI, 0.20%/y –0.52%/y) in 2015 to 2020.
- Sex disparities exist in the treatment of HCM. Among 9306 patients with obstructive HCM in the MarketScan database, females were less likely to be prescribed β-blockers (42.7% versus 45.2%; $P=0.600$) or to receive an ICD (1.7% versus 2.6%; $P=0.005$).[8]

Genetic Testing

- The NIH-funded Clinical Genome Resource framework identified that of the 33 speculated HCM genes, 8 genes (*MYBPC3*, *MYH7*, *TNNT2*, *TNNI3*, *TPM1*, *ACTC1*, *MYL2*, and *MYL3*) have definitive evidence, 3 genes (*CSRP3*, *TNNC1*, and *JPH2*) have moderate evidence, and the remaining genes have limited to no evidence supporting an association with HCM.[9]
- Given the heterogeneous nature of the underlying genetics, manifestation of the disease is highly variable, even in cases for which the causal variant has been identified.[10] Among clinically unaffected individuals with pathogenic sarcomere variants discovered as part of cascade testing, 46% developed HCM over 15 years of follow-up.[11]

Dilated Cardiomyopathy

- DCM has a prevalence of 1 in 2500, but it is likely underestimated.[12]

The 2025 AHA Statistical Update uses language that conveys respect and specificity when referencing race and ethnicity. Instead of referring to groups very broadly with collective nouns (eg, Blacks, Whites), we use descriptions of race and ethnicity as adjectives (eg, Asian people, Black adults, Hispanic youths, Native American patients, White females).

As the AHA continues its focus on health equity to address structural racism, we are working to reconcile language used in previously published data sources and studies when this information is compiled in the annual Statistical Update. We strive to use terms from the original data sources or published studies (mostly from the past 5 years) that may not be as inclusive as the terms used in 2025. As style guidelines for scientific writing evolve, they will serve as guidance for data sources and publications and how they are cited in future Statistical Updates.

- Potential causes of DCM of variable chronicity and reversibility include cardiomyopathies developing after an identifiable exposure such as tachyarrhythmia, stress, neurohormonal disorder, alcoholism, chemotherapy, infection, autoimmunity, or pregnancy (see the Peripartum Cardiomyopathy section).[13]
- Among 23 341 participants with HF from 40 countries in the G-CHF registry, 15.4% had idiopathic DCM.[14]
- Many cases of DCM have a genetic cause, called familial DCM.[15] Familial DCM has an estimated prevalence of 30% to 50% among all cases of DCM, and a causal genetic variant has been identified in 10% to 14% of cases.[3,15]
- In a cross-sectional survey of 1220 probands with confirmed DCM and 1693 first-degree relatives who underwent clinical screening, including transthoracic echocardiography, the prevalence of familial DCM was 11.6%.[16]
  - If all living first-degree relatives had been screened, the estimated prevalence of familial DCM was 29.7% (95% CI, 23.5%–36.0%).
  - The estimated prevalence of familial DCM was higher in Black probands compared with White probands (difference, 11.3% [95% CI, 1.9%–20.8%]).
  - With the use of an expanded definition of familial DCM, which included the presence of DCM, LV enlargement, or LV systolic dysfunction without a known cause in at least 1 first-degree relative, the estimated prevalence was 56.9% (95% CI, 50.8%–63.0%).
- Missense and truncating variants in the titin gene have been linked to autosomal dominant cardiomyopathy and to DCM with incomplete penetrance in the general population.[17] Analysis of sequence data in 7855 cases with cardiomyopathy and >60 000 controls revealed a range in penetrance of putative disease variants, which further highlights the challenges in clinical interpretation of genetic variation in cardiomyopathy-associated genes.[18]

Genetic Testing

- Among patients with DCM, a recent multisite nationwide cross-sectional study indicates an estimated familial prevalence of ≈30% in first-degree relatives and an estimated 19% risk of developing DCM by 80 years of age.[16] This study also indicates that first-degree relatives of NH Black probands (index patients with DCM) or probands diagnosed at a young age have a higher risk of DCM. These findings suggest a potential yield of phenotypic screening of first-degree relatives of index DCM cases, especially those identified at a young age. The clinical outcomes in familial DCM have been described recently.[19]
- In 186 families who underwent genetic screening because of having a relative with DCM, 37% (95% CI, 30%–45%) were discovered to have a likely pathogenic or pathogenic genetic variant for DCM.[20]
- In an appraisal of the 51 genes hypothesized to be associated with DCM, the recent Clinical Genome Resource framework panel noted that only 12 genes from 8 gene ontologies have definitive (*BAG3*, *DES*, *FLNC*, *LMNA*, *MYH7*, *PLN*, *RBM20*, *SCN5A*, *TNNC1*, *TNNT2*, and *TTN*) or strong (*DSP*) evidence and only 7 genes from the additional 2 ontologies (*ACTC1*, *ACTN2*, *JPH2*, *NEXN*, *TNNI3*, *TPM1*, and *VCL*) have moderate evidence supporting a robust association with DCM.[21] Because DCM is often the final disease manifestation of several cardiomyopathies, it shares genetic architecture with other inherited cardiomyopathies. Among the previously mentioned 19 genes linked to DCM, the Clinical Genome Resource panel noted that 6 had a similar classification for HCM and 3 had a similar classification for arrhythmogenic right ventricular cardiomyopathy.[21]
- Missense and truncating variants in the titin gene have been linked to autosomal dominant cardiomyopathy, as well as to DCM, with incomplete penetrance in the general population.[17] Analysis of sequence data in 7855 cases with cardiomyopathy and >60 000 controls revealed the variance in penetrance of putative disease variants, which further highlights the challenges in clinical interpretation of variation in mendelian disease genes.[18]
- A recent GWAS has identified common genetic variants associated with HCM (16 loci identified) and DCM (13 loci identified), indicating a potential oligogenic pattern (instead of a conventionally understood monogenic pattern) for the genetic risk of HCM and DCM.[5,22] It is notable that 2 HCM loci (chromosome 1 near *HSPB7* and chromosome 10 near *BAG3*) have opposite directions of effect for DCM and require further evaluation in subsequent investigations.

Peripartum Cardiomyopathy

- PPCM is a global problem with significant geographic variation in its incidence.[23] The highest incidence (1 in 102 births) is seen in Nigeria, and the lowest incidence (1 in 15 533 births) is seen in Japan.[24] Accordingly, worldwide and in the United States, females with Black ancestry appear to have highest risk, especially females with Nigerian (1 per 100 live births) and Haitian (1 per 300 live births) background.[23,25]
- In the United States, according to NIS data, the incidence of PPCM increased between 2004 and 2011 from 8.5 to 11.8 per 10 000 live births

(P_{trend}<0.001), likely related to rising average maternal age and prevalence of PPCM risk factors such as obesity, hypertension, pregnancy-related hypertension, and diabetes.[26] Stratified by race and ethnicity, incidence of PPCM was lowest in Hispanic females (3.6 per 10 000 live births) and highest in Black females (22.8 per 10 000 live births). Stratified by region, incidence was lowest in the West (6.5 [95% CI, 6.3–6.7] per 10 000 live births) and highest in the South (13.1 [95% CI, 12.9–13.1] per 10 000 live births).[26]

- In a cohort of 55 804 hospitalized patients with PPCM in the United States, Black individuals (OR, 1.17 [95% CI, 1.15–1.57]; *P*<0.001) and Hispanic individuals (OR, 1.37 [95% CI, 1.17–1.59]; *P*<0.001) were more likely to develop cardiogenic shock than White individuals.[27] Similarly, Black individuals (OR, 1.67 [95% CI, 1.21–2.23]; *P*=0.002) and Hispanic individuals (OR, 2.20 [95% CI, 1.45–3.33]; *P*<0.001) were more likely to have in-hospital mortality than White individuals.
- In a UK case-control study of women with PPCM from 1998 to 2017, the incidence was 2.02 (95% CI, 1.76–2.29) per 10 000 deliveries in Scotland, which was similar to the incidence rate of 2.12 (95% CI, 2.03–2.21) per 10 000 deliveries in England from 2003 to 2017.[28] From 1998 to 2017 in Scotland, 7.7% of women with PPCM died over a median follow-up of 8.3 years, and 30-day mortality was 2.3% (95% CI, 0.9%–5.3%).[28]
- In a global prospective PPCM registry, which included 739 women enrolled between 2012 and 2018 from 49 countries, the 6-month maternal mortality rate was 5.9% overall but negatively correlated with the health expenditure of the countries, defined as a proportion of GDP allocated to health care expenses.[29] Maternal mortality rates at 6 months ranged from 2.0% in high-health-expenditure (>8% GDP) countries, 5.5% in medium-health-expenditure (5%-8% GDP) countries, and 10.0% in low-health-expenditure (<5% GDP) countries (*P*=0.002).
- In a registry of 535 women with PPCM from 51 countries from 2012 to 2018, overall 1-year mortality was 8.4% with rates varying by region: Europe, 4.9%; Africa, 6.5%; Asia Pacific, 9.2%; and the Middle East, 18.9% (*P*<0.001).[30] Within 1 year of diagnosis, 14.0% of women had at least 1 rehospitalization, and 3.5% had ≥2 rehospitalizations.
- In many cases of PPCM (47%–66%), LVEF recovers to at least near-normal (≥50%) function and often within 6 months.[30–33] However, an initial LVEF <30%, LV end-diastolic dimension ≥6.0 cm, Black race, and initial presentation >6 weeks after delivery are associated with lower LVEF at 1 year.[34]

Genetics of PPCM

- Genetic analyses suggest that ≈15% of individuals with PPCM have rare truncating variants in genes also linked to idiopathic DCM. The majority of these are truncating variants in *TTN*, which encodes the sarcomeric protein titin, and truncating variants in *TTN* in females with PPCM are associated with lower EF after 1 year of follow-up.[34]
- Global mortality from PPCM is 9%[31,35] and is lower in developed (4%) than developing (14%) countries; in addition, a high prevalence of females of African descent was positively correlated with mortality (weight correlation coefficient, 0.29 [95% CI, 0.13–0.52]).[35]

Youths

- Since 1996, the Pediatric Cardiomyopathy Registry has collected prospective data on children with cardiomyopathy in New England and central southwestern states.[36,37]
  - Overall incidence of cardiomyopathy is 1.13 cases per 100 000 in children <18 years of age.
  - Incidence is 8.34 (95% CI, 7.21–9.61) per 100 000 in children <1 year of age.
  - Annual incidence (cases per 100 000) is higher in Black children (1.47) than in White children (1.06; *P*=0.02), in male (1.32) than in female (0.92) children (*P*<0.001), and in New England (1.44) than in the central Southwest (0.98; *P*<0.001).
- The annual incidence of HCM in children is ≈4.7 per 1 million (95% CI, 4.1–5.3) with higher incidence in New England (5.9 per 1 million [95% CI, 4.8–7.2]) than in the central Southwest region (4.2 per 1 million [95% CI, 3.5–4.9]) and in males (5.9 per 1 million [95% CI, 5.0–6.9]) than in females (3.4 per 1 million [95% CI, 2.8–4.2]).[38] Approximately 9% progress to HF and 12% to SCD over a median follow-up of 6.5 years.[39] Chapter 18 (Disorders of Heart Rhythm) provides statistics on SCD. Data from the NIS indicate that hospitalization is more likely with increasing age (OR, 5.59 [95% CI, 2.03–15.37] for ≥10 years of age versus 1–9 years of age) and in Black individuals compared with White individuals (OR, 2.78 [95% CI, 1.19–6.47]).[40]
- The annual incidence of DCM in children is ≈0.57 per 100 000 (95% CI, 0.52–0.63) with a higher incidence in males than females (0.66 versus 0.47; *P*<0.001) and in Black children than White children (0.98 versus 0.46; *P*<0.001). Commonly recognized causes include myocarditis (46%) and neuromuscular disease (26%).[41] The 5-year incidence rate of SCD is 3% at the time of DCM diagnosis.[42]
- For all cardiomyopathies seen in children, 5-year transplantation-free survival rate of DCM, HCM, restrictive cardiomyopathy, and LV noncompaction is 50%, 90%, 30%, and 60%, respectively.[43]

- Data from the Childhood Cancer Survivor Study cohort of 14 358 survivors of childhood or adolescent cancers showed a 5.9-fold (95% CI, 3.4–9.6) increased risk for HF compared with siblings,[44] usually preceded by asymptomatic cardiomyopathy persisting up to 30 years after the cancer diagnosis, especially in patients treated with chest radiation or anthracycline chemotherapy.

Global Burden of Cardiomyopathy (See Table 22-1 and Charts 22-1 and 22-2)

- Based on 204 countries and territories in 2021[45]:
  - There were 0.40 (95% UI, 0.37–0.43) million total deaths estimated for cardiomyopathy and myocarditis and an age-standardized mortality rate of 4.89 (95% UI, 4.47–5.26) per 100 000 (Table 22-1).
  - The highest age-standardized death rates among regions estimated for cardiomyopathy and myocarditis were for Eastern Europe followed by central sub-Saharan Africa and Central Asia. Rates were lowest for Andean Latin America (Chart 22-1).
  - Globally, there were 5.26 (95% UI, 4.36–6.09) million prevalent cases of cardiomyopathy and myocarditis and an age-standardized prevalence rate of 65.90 (95% UI, 54.98–76.83) per 100 000 (Table 22-1).
  - Age-standardized prevalence of cardiomyopathy and myocarditis among regions was highest for high-income North America followed by Australasia and Eastern Europe. The lowest prevalence rates were for East Asia and Oceania (Chart 22-2).
- Rates of SCD in patients with HCM vary by geographic region. In a meta-analysis of data from 2015 to 2020, the reported incidence rate per 100 PY was highest in Asia (0.67% [95% CI, 0.54%–0.84%]) followed by Europe (0.37% [95% CI, 0.31%–0.46%]) and North America (0.28% [95% CI, 0.18%–0.43%]).[7]

Heart Failure

ICD-9 428; *ICD-10* I50. For hospital discharges, *ICD-10* I50, I11.0, I13.0, I13.2, I09.81

2022, United States: Underlying cause mortality—87 941. Any-mention mortality—425 147.

2021, United States: Hospital discharges, principal diagnosis—1 200 188

Prevalence (See Table 22-2 and Chart 22-3)

- According to data from NHANES 2017 to 2020, 6.7 million Americans ≥20 years of age had HF (Table 22-2), up from the estimate of 6.0 million in 2015 to 2018 (NHLBI unpublished tabulation using NHANES). The breakdown of HF prevalence by age and sex is shown in Chart 22-3.
- Based on temporal trends, the prevalence of HF is projected to increase further, affecting >8 million people ≥18 years of age by 2030. The total percentage of the population with HF is projected to rise from 2.4% in 2012 to 3.0% in 2030.
- Overall, 1.9% to 2.6% of adults in the United States have HF.[46]

Incidence

- Of 1 799 027 unique Medicare beneficiaries at risk for HF (median, 73 years of age [interquartile range, 68–79 years]; 56% female), 249 832 had a new diagnosis of HF.[47] HF incidence was 26.5 cases per 1000 beneficiaries in 2016, consistent across subgroups based on sex and race or ethnicity.

Risk Factors

- Hypertension, smoking, diabetes, and obesity account for 52% of incident HF with PARs as follows[48]: CHD, 20% (23% in males versus 16% in females); hypertension, 20% (28% in females versus 13% in males); cigarette smoking, 14%; obesity, 12%; and diabetes, 12%.
- Data from NHANES show that one-third of US adults have at least 1 HF risk factor.[49]
- Risk factors differ by HF subtype: among 4 community-based studies (CHS, FHS, PREVEND, MESA)[50]:
  - Older age was more strongly associated with incident HFpEF versus HFrEF (subdistribution HR, 1.91 [95% CI, 1.78–2.06] versus 1.69 [95% CI, 1.59–1.81], respectively, per 10-year age increase; *P* for equality=0.02).
  - In contrast, the following risk factors were more strongly associated with incident HFrEF than HFpEF: male sex (subdistribution HR, 1.87 [95% CI, 1.63–2.16] in HFrEF versus 0.91 [95% CI, 0.79–1.05] in HFpEF; *P* for equality <0.0001), previous MI (subdistribution HR, 2.70 [95% CI, 2.25–3.24] in HFrEF versus 1.30 [95% CI, 1.02–1.67] in HFpEF; *P* for equality <0.0001), electrocardiographic LVH (subdistribution HR, 2.08 [95% CI, 1.60–2.69] in HFrEF versus 1.16 [95% CI, 0.84–1.60] in HFpEF; *P* for equality=0.009), and left bundle-branch block (subdistribution HR, 3.65 [95% CI, 2.62–5.09] in HFrEF versus 1.30 [95% CI, 0.81–2.09] in HFpEF; *P* for equality=0.0008).
- Age dependency of risk factors: Although the absolute risk of HF is lower among younger individuals, the PAR of modifiable risk factors is greater among young (<55 years of age) compared with older (≥75 years of age) individuals:

obesity, 21% versus 13%; hypertension, 35% versus 23%; diabetes, 14% versus 7%; and smoking, 32% versus 1%.[51]
- Lifestyle factors also affect HF risk. Among WHI, MESA, and CHS participants, individuals with more than twice the minimum guideline-recommended leisure-time PA had lower risk of HFpEF compared with those with no leisure-time PA (HR, 0.81 [95% CI, 0.68–0.97]), whereas no such association was observed for risk of HFrEF.[52]
- In the ARIC study, greater alignment with the AHA's Life's Simple 7 guidelines (better profiles in smoking, BMI, PA, diet, cholesterol, BP, and glucose) was associated with lower lifetime risk of HF.[53] Specifically, the lifetime risk of HF among those with 5 to 7 ideal components in middle age was 12% (95% CI, 9%–15%), whereas those with 0 ideal components had a lifetime risk of 45% (95% CI, 35%–52%).

Race and Sex Differences

- In 6 US longitudinal population-based cohorts, hypertension had the highest PAR among Black males and females (28% [95% CI, 19%–37%] and 26% [95% CI, 16%–34%], respectively), whereas obesity had the highest PAR among White males and females (21% [95% CI, 15%–27%] and 18% [95% CI, 13%–23%]).[54]
- Sex-specific risk factors for incident HF include disorders of pregnancy (eclampsia/preeclampsia, gestational diabetes), PPCM, polycystic ovarian syndrome, and premature menopause, although the exact contribution of these conditions to the incidence of HF among women is unknown.[55] The penetrance of genetic cardiomyopathies may be influenced by sex with males often more severely affected.

Family History and Genetics

- In the multigenerational FHS, HF in at least 1 parent was associated with a higher prevalence of asymptomatic LV systolic dysfunction (5.7% versus 3.1%; *P* adjusted for age, sex, and height=0.046) and greater risk of incident HF (age- and sex-adjusted 10-year incidence rate, 2.72% [95% CI, 1.80%–4.11%] versus 1.62% [95% CI, 1.10%–2.39%]; age- and sex-adjusted HR, 1.72 [95% CI, 1.13–2.61]; *P*=0.01).[56]
- Several GWASs have been conducted to identify common variations associated with cardiomyopathy and HF in the general population, albeit with modest results, highlighting a small number of putative loci, including *HSPB7*[57–59] and *CACNB4*.[60] In a GWAS of >47 000 cases and >930 000 controls, 11 HF loci were identified, all of which have known relationships with other CVD traits.[61]
- Multiple GWASs of cardiac structure and function have highlighted the association of genetic architecture of LV phenotypes with the risk of future HF.[62,63] A transancestry meta-analysis of GWASs including >1 500 000 individuals identified 47 risk loci for all-cause HF. Integrating cardiac MRI intermediary phenotypes into this GWAS led to the identification of 61 loci.[64]
- A large GWAS meta-analysis for HF, including 90 000 cases and 1 000 000 controls, identified 18 novel loci.[65] Mendelian randomization and colocalization analyses identified an additional 10 putatively causal loci associated with HF.[65] According to the GWAS and mendelian randomization proteomics analysis, 7 potential proteins (*CAMK2D*, *PRKD1*, *PRKD3*, *MAPK3*, *TNFSF12*, *APOC3*, and *NAE1*) were identified as drug targets for HF.[65]
- Human induced pluripotent stem cell cardiomyocyte–based functional studies investigating the molecular mechanism of decreased incidence of HF linked to the *BAG3* gene demonstrated that a coding variant ($BAG3^{C151R}$) is responsible for the maintenance of myofibrillary integrity and regulation response to proteotoxic stress.[66]
- A single-cell profiling study showed that HCM and DCM share a common final transcriptional pathway at the cellular level. Furthermore, cardiomyopathy was associated with a shift in the macrophage population and the presence of a unique population of activated fibroblasts.[67]
- The genetic basis of specific cardiomyopathies is summarized in the previous Cardiomyopathy section.

Treatment

- Improvement in survival has been attributed primarily to evidence-based approaches to treat HFrEF, including pharmacotherapies, ICDs, and cardiac resynchronization therapy.[68]
- Based on modeling from clinical trial data, initiation of contemporary GDMT for HFrEF (quadruple therapy with angiotensin receptor/neprilysin inhibitors, ACE inhibitors, or ARBs; β-blockers; mineralocorticoid receptor antagonists; and SGLT-2 inhibitors) may reduce the hazard of cardiovascular death or HF hospitalization in HFrEF by up to 62% (HR, 0.38 [95% CI, 0.30–0.47]) compared with limited conventional therapy, resulting in an estimated 1.4 to 6.3 additional years alive.[69] Treatment efficacy with these classes for the outcome of death is attenuated as LVEF increases, and there is no clear evidence to support β-blockers in HFpEF.[70,71]
- Across jurisdictions, there are significant gaps in the use and dose of GDMT, particularly in females.[72] In an analysis of a US administrative health claims database of 63 759 patients (mean age, 71.3 years;

56.6% male), only 6.2% achieved optimal GDMT within 12 months of HFrEF diagnosis; optimal GDMT was defined as ≥50% of the target dose of evidence-based β-blocker plus ≥50% of the target dose of ACE inhibitors or ARBs or any dose of angiotensin receptor/neprilysin inhibitor plus any dose of mineralocorticoid receptor antagonist.[72] Treatment gaps were wider in female than male patients; relative to males, females with HFrEF had lower use of each GDMT class and of optimal GDMT at every time point at follow-up. In adjusted analyses, female sex was associated with a 23% lower probability of achieving optimal GDMT after diagnosis (HR, 0.77 [95% CI, 0.71–0.83]; $P<0.001$). Females were also less likely to receive cardiac resynchronization and intracardiac device therapy than males.

- ICD and cardiac resynchronization therapy reduce all-cause mortality in eligible patients with HFrEF[71,72] but remain underused. Females receive this intervention less frequently than males. In a pooled analysis of 98 cohorts who had received CRT with or without ICD, men received the devices at a median ratio of 3.16 (25th–75th interquartile range, 2.48–3.62) relative to women.[73]

Mortality

Secular Trends

- Among adults ≥75 years of age with HF in the CDC WONDER dataset[74]:
  - AAMR per 100 000 declined from 141.0 in 1999 to 108.3 in 2012 (annual percent change, −2.1 [95% CI, −2.4 to −1.9]), after which it increased to 121.3 in 2019 (annual percent change, 1.7 [95% CI, 1.2–2.2]).
- Across jurisdictions, the COVID-19 pandemic was associated with increased mortality among those with decompensated HF and with a shift in deaths from hospital to community.[75,76] There was an increase in both in-hospital and postdischarge mortality among patients hospitalized with HF despite similar care quality. In the GWTG–Heart Failure registry, in-hospital mortality increased from 2.5% in 2019 to 2020 to 3.0% during 2020 to 2021, with in-hospital mortality as high as 8.2% among those with concurrent COVID-19 infection.[76]

Mortality by HF Subtype

- Among 4 community-based cohorts, including CHS, FHS, PREVEND, and MESA, all-cause mortality rates after HF diagnosis were 459 per 10 000 PY among those with HFrEF and 394 per 10 000 PY in individuals with HFpEF.[77]
- Phenotypes based on clinical comorbidities may stratify all-cause death or readmissions with greater discrimination than LVEF categories after hospitalization for HF.[78] In an unsupervised machine-learning cluster analysis of 1693 patients hospitalized for HF and discharged alive, 6 discrete phenogroups characterized by a predominant comorbidity were identified: CHD, valvular HD, AF, sleep apnea, chronic obstructive pulmonary disease, or minimal comorbidities. Phenogroups were LVEF independent, with each phenogroup encompassing a wide range of LVEFs. For the composite outcome of all-cause death or rehospitalization at 6 months, the HRs for phenogroups ranged from 1.25 (95% CI, 1.00–1.58) for AF to 2.04 (95% CI, 1.62–2.57) for chronic obstructive pulmonary disease (log-rank $P<0.001$) relative to the phenogroup with minimal comorbidities. In comparison, LVEF-based classification did not separate patients into different risk categories for composite of all-cause death or rehospitalization at 6 months; ($P=0.69$); relative to patients with LVEF ≤40%, those with LVEF ≥50% had no difference in risk of all-cause death or rehospitalization at 6 months (HR, 1.01 [95% CI, 0.87–1.17]; $P=0.94$).[78]

CVD Mortality (See Table 22-2)

- Among optimally treated clinical trial patients with HF across the LVEF continuum, 53.5% of deaths were ascribed to CVD causes (of which 33.1% were from HF and 50.6% from SCD), 29.9% to non-CVD causes, and 16.5% to undetermined causes.[79] The proportion of non-CVD death was higher in those with higher EF. In the same analysis, the rate of death per 100 000 patient-years resulting from sudden death, HF, and cardiovascular causes decreased as LVEF increased.
- Data from the CDC WONDER database show that age-adjusted rates of HF-related CVD death declined from 1999 (78.7 per 100 000 [95% CI, 78.2–79.2]) to 2012 (53.7 per 100 000 [95% CI, 53.3–54.1]) and subsequently increased through 2017 (59.3 per 100 000 [95% CI, 58.9–59.6]).[80] There is geographic variation in HF-related CVD mortality, with the highest increases in annual AAMR after 2011 occurring in the Midwest (1.14 per 100 000 per year [95% CI, 0.75–1.53]) and South (0.96 per 100 000 per year [95% CI, 0.66–1.26]) compared with the Northeast (0.35 per 100 000 per year [95% CI, 0.03–0.68]).[81]
- Given improvements in HF survival overall, the number of individuals carrying a diagnosis of HF at death has increased. Mortality associated with HF is substantial such that ≈1 in 8 deaths in 2021 has HF mentioned on the death certificate (unpublished NHLBI tabulation).[82]
- In 2022, HF was the underlying cause in 87 941 deaths (41 657 males and 46 284 females; Table 22-2).

- The number of deaths attributable to HF was 45.7% higher in 2022 than in 2012 (60 341; unpublished NHLBI tabulation using CDC WONDER).[83]

Age, Sex, and Race and Ethnicity Differences in Mortality (See Table 22-2)

- Among older adults in the CDC WONDER dataset between 1999 and 2019[74]:
  - Males had consistently a higher AAMR than females throughout the period, with an AAMR of 141.1 in males and 107.8 in females in 2019.
  - NH White adults had the highest overall AAMR (127.2) followed by NH Black adults (108.7), NH American Indian/Alaska Native adults (102.0), Hispanic or Latino adults (78.0), and NH Asian or Pacific Islander adults (57.1).
- In the Southern Community Cohort Study, all-cause mortality after a diagnosis code for HF varied by sex, with HRs of 1.63 (95% CI, 1.27–2.08), 1.38 (95% CI, 1.11–1.72), and 0.90 (95% CI, 0.73–1.12) for White males, Black males, and Black females, respectively, compared with White females.[84] In the ARIC study, the 30-day, 1-year, and 5-year case fatality rates after hospitalization for HF were 10.4%, 22%, and 42.3%, respectively, with Black individuals having a greater 5-year case fatality rate than White individuals ($P<0.05$).[85]
- Underlying cause and any-mention age-adjusted HF mortality rates by gender, race, and ethnicity are listed in Table 22-2.

Rural-Urban Disparities

- Among Medicare fee-for-service beneficiaries, 30-day mortality was higher among patients with HF presenting to rural versus urban hospitals (HR, 1.15 [95% CI, 1.13–1.16]).[86]

Health Care Use: Hospital Use

- In 2019, there were 8 054 000 physician office visits with a primary diagnosis of HF (NAMCS,[87] unpublished NHLBI tabulation). In 2021, there were 1 390 365 ED visits for HF (HCUP,[2] unpublished NHLBI tabulation). In 2021, there were 1 200 188 principal diagnosis hospital discharges for HF (HCUP,[2] unpublished NHLBI tabulation).
- In the NCDR PINNACLE, 1 in 6 patients with HFrEF developed worsening HF within 18 months of diagnosis and was more likely to be Black, to be >80 years of age, and to have greater comorbidity burden; overall, the 2-year mortality rate was 22.5%.[88]
- Outcomes remain poor after hospitalization for HF. In a pragmatic trial of 2494 patients discharged alive after hospitalization for HF in Canada in 2015 to 2016, 49.1% of patients were rehospitalized (47.4% of these for HF), an additional 34.1% visited the ED without being rehospitalized, and 15.5% died within 6 months of discharge.[89]

Secular Trends

- In the US NIS, hospitalizations for HF increased from 1 060 540 in 2008 to 1 270 360 in 2018 with a greater proportion among individuals from underrepresented racial and ethnic groups (Black individuals: 18.4% in 2008, 21.2% in 2018; Hispanic individuals: 7.1% in 2008, 9.0% in 2018; $P<0.001$ for all).[90]
- Hospitalizations by HF subtype increased from 2008 to 2018 in the United States for both HFrEF (n=283 193 to n=679 815) and HFpEF (n=189 260 to n=495 095).[90] A greater proportion of HFrEF hospitalizations occurred in males (60.5%), and a greater proportion of HFpEF hospitalizations occurred in females (62.5%; $P<0.001$ for sex difference).
- Among 11 806 679 cases of HF hospitalization in the US NIS 2002 to 2016, there was a decrease in adjusted mortality from 6.8% in 2002 to 4.9% in 2016 ($P_{trend}<0.001$), with consistent findings across age, sex, and race.[91] The adjusted mean length of stay decreased from 8.6 to 6.5 days ($P<0.001$), and discharge to a long-term care facility increased from 20.8% to 25.6% ($P<0.001$).

Age, Sex, Race, and Socioeconomic Differences

- Among 4 287 478 weighted hospitalizations in NIS dataset in the United States, the median age was 73.4 years (interquartile range, 62.4–82.9 years), 51.3% of hospitalizations occurred in male patients, and race and ethnicity composition included White individuals (70.0%), Black individuals (17.5%), Hispanic individuals (7.6%), Asian or Pacific Islander individuals (2.2%), and Native American individuals (0.5%). Among the hospitalizations, 33.1% were patients from zip codes in the lowest quartile of national household income (including 0.6% experiencing homelessness).[92] In models adjusted for baseline characteristics, male sex (RR, 1.09 [95% CI, 1.07–1.11]) and low SES (RR, 1.02 [95% CI, 1.00–1.05]) were associated with a higher risk of in-hospital mortality relative to female sex and high SES, whereas Black race (RR, 0.79 [95% CI, 0.76–0.81]) and Hispanic ethnicity (RR, 0.90 [95% CI, 0.86–0.93]) were associated with a lower risk of in-hospital mortality than White race.
- Among 767 180 weighted hospitalizations for HF among young adults <50 years of age in the NIS dataset in the US, Black adults (50.1%) accounted for disproportionately higher HF

hospitalizations compared with White adults (31.9%) and Hispanic adults (12.2%). Nearly half of hospitalizations (45.8%) represented patients from the lowest quartile of national household income.[93]

- Data from the 2005 to 2014 ARIC Community Surveillance study have shown that HF hospitalization rates are increasing over time with the average annual percentage change ranging from 1.9% (95% CI, 0.7%–3.1%) in White females to 4.3% (95% CI, 2.7%–5.9%) in Black females from 2005 to 2014. This increase in HF hospitalizations is driven largely by HFpEF events. Age-adjusted 28-day and 1-year case fatality rates after first-time hospitalized HF were higher among White individuals versus Black individuals. Specifically, 28-day age-adjusted case fatality was 12.1% (White males), 11.7% (White females), 10.2% (Black females), and 9.2% (Black males).[94]
- In a pragmatic clinical trial of 2494 patients hospitalized for HF in Canada, females were on average ≈5 years older (mean±SD age, 80.0±10.9 years versus 75.4±12.8 years), more commonly resided in a nursing home (16.2% versus 8.2%), had a 6.89 (5.51–8.28) higher associated mean LVEF,[78] and experienced worse quality of life as measured by the European Quality of Life 5 Dimensions 5 Level Version scores (range, 0–1; 0.37 [95% CI, 0.30–0.44] females versus 0.62 [95% CI, 0.57–0.67] males).[89,95] Event rates were high after index hospitalization; at 3 years of follow-up, 51.4% of patients receiving usual care had died, 80.7% were readmitted for any cause, and 42.3% were readmitted for HF.[96]
- Among those who die or are rehospitalized for any cause after hospitalization for HF, the proportion of cardiovascular deaths or HF hospitalizations decreases as LVEF increases. In a cohort of 1693 patients hospitalized for HF in 2015 to 2016 and discharged alive in Canadian hospitals, patients with LVEF ≥50% had a similar risk of all-cause death or rehospitalization at 6 months (HR, 1.01 [95% CI, 0.87–1.17]; *P*=0.94), but a lower risk of cardiovascular death or HF rehospitalization (HR, 0.75 [95% CI, 0.62–0.92]; *P*=0.005) than those with LVEF ≤40%.[78] Among those who died or were rehospitalized for any cause at 6 months, the proportion of cardiovascular deaths or HF hospitalizations was higher in patients with LVEF <40% (61.1%) than in those with LVEF >50% (48.4%).
- In the CHARM program, rates of cardiovascular hospitalization were higher among those with LVEF ≤40% (23.6 [95% CI, 22.6–24.7] per 100 patient-years) versus LVEF >40% (19.3 [95% CI, 18.2–20.5] per 100 patient-years; *P*<0.001 for difference), whereas rates of noncardiovascular hospitalization were similar (14.3 [95% CI, 13.5–15.2] versus 14.3 [95% CI, 13.3–15.3] per 100 patient-years, respectively).[97]

Orthotopic Heart Transplantation and Mechanical Circulatory Support Device Placement in the United States

Heart Transplantation

(See Chapter 27 [Medical Procedures] for additional heart transplantation data)

Transplant Recipients

- The annual number of transplantations in the United States has increased over time from 1676 in 1988 to 4111 in 2022.[98]
- In 2022, the annual transplantation rate was 122.5 per 100 patient-years.[99]
  - Among 3668 adult transplant recipients in 2022, the most common age group (46.6%) was 50 to 64 years of age, 26.9% were female, and 56.4% were White.
  - The demographic characteristics of the typical heart transplant recipient—50 to 64 years of age, male, White—remained unchanged between 2012 and 2022; however, there has been an increase in the proportion of recipients who are 18 to 34 years of age (10.9% to 12.5%), ≥65 years of age (17.8% to 20.3%), and of Black race (19.7%–27.0%) or Hispanic ethnicity (7.9% to 11.2%); there has been a decrease in private insurance payers (50.9% to 44.8%). The proportion of recipients who are female remained unchanged between 2012 and 2022 (27.7% to 26.9%).
  - Among 3668 transplant recipients in 2022, the primary diagnosis was cardiomyopathy (64.1%) followed by CAD (25.9%), congenital HD (5.1%), and valvular disease (1.0%), with unknown underlying diagnoses in 3.9%. Coronary disease has become a less common primary diagnosis for heart transplantation over time (38.8% in 2012), and cardiomyopathy remains the most common diagnosis for heart transplantation (54.1% in 2012).
  - Among 3668 transplant recipients in 2022, a majority lived <50 miles from the transplantation center (59.2%) and in metropolitan areas (83.6%).
  - A ventricular assist device was present in 35.2% of transplant recipients in 2022, down from 41.4% in 2012.[99]
  - The proportion of transplant recipients who waited <90 days for a transplantation increased from 47.6% in 2012 to 65.9% in 2022.
  - Multiorgan transplantation has increased more rapidly than heart transplantation alone. In 2012, 6.0% of heart transplantations were combined

with transplantation of other organs, and this increased to 14.0% in 2022. Between 2012 and 2022, heart-kidney transplantations increased from 3.7% to 10.5% of total heart transplantations, heart-liver transplantations increased from 0.9% to 2.0%, and heart-lung transplantations changed from 1.3% to 1.2%; the absolute numbers of each type of multiorgan transplantations increased substantially during this period in the order in which they are presented here.

Transplantation Listings

- In 2022, a total of 7519 adults were awaiting heart transplantation in the United States, a 28.1% increase from 2011.[99]
- A majority (61.6%) of adult heart transplantation candidates lived <50 miles from the transplantation center in 2022.[99]
- The largest adult age group on the waiting list in 2022 continued to be 50 to 64 years of age (46.7%, followed by 35 to 49 years of age (21.7%), ≥65 years of age (20.3%), and 18 to 34 years of age (11.3%).[99]
- In 2022, more than half of adult heart transplantation candidates were White (56.3%); 28.2% were Black, 10.8% were Hispanic, 3.8% were Asian, 0.4% were Native American, and 0.5% multiracial.[99]
- Among 8747 US adults listed for heart transplantation in the Scientific Registry of Transplant Recipients from 2017 to 2019, 84.7% were from metropolitan, 8.6% were from micropolitan, and 6.6% were from rural settings; >70% were male candidates.[100]
- Time on the waiting list, which is determined by the earliest of transplantation, death, removal, or December 31 of the year, has decreased, and the proportion of candidates waiting <90 days increased from 2011 to 45.8% in 2022.[99]
- The proportion of candidates on the waiting list with a ventricular assist device increased from 22.4% in 2012 to 35.0% in 2022.[99]
- Overall pretransplantation mortality declined from 15 deaths per 100 patient-years in 2011 to 8.7 deaths per 100 patient-years in 2019 and remained steady through 2022. The decrease in pretransplantation mortality has been consistent in all age and race or ethnicity groups other than Asian Americans, who experienced a slight increase with 9.7 deaths per 100 patient-years in 2022 compared with 8.5 deaths per 100 patient-years in 2012.[99]

Outcomes After Transplantation

- In 2022, 6-month, 1-year, 3-year, and 5-year mortality after transplantation were 7.3%, 9.2%, 15.3%, and 19.9%, respectively. Mortality after transplantation has decreased since 2009.[99]
- Five-year survival was modestly lower in those 18 to 34 years of age (78.9%) and ≥65 years of age (77.8%) compared with the other age groups (35–49 and 50–64 years of age, 82.1% and 81.0%, respectively).[99]
- Five-year survival was highest in White transplant recipients (81.9%) followed by recipients identifying as Asian, multiracial, Black, Hispanic, and Native American, ranging from 76.0% to 80.4%.[99]
- Among 32 353 adult heart transplant recipients in the United Network for Organ Sharing database, the proportion of Black individuals and Hispanic individuals listed increased from 2011 to 2020 (21.7% to 28.2% [P=0.003] and 7.7% to 9.0% [P=0.002], respectively).[101] Black individuals had a higher risk of death after transplantation (aHR, 1.14 [95% CI, 1.04–1.24]; P=0.004) compared with White individuals.
- Among 34 198 heart transplant recipients in the International Society for Heart and Lung Transplantation registry between 2004 and 2014, when matched for recipient and donor characteristics, there was no significant difference in survival between male and female recipients.[102] Data from the Scientific Registry of Heart Transplant Recipients 2015 to 2017 also demonstrate no sex differences in survival through 5 years after transplantation.[99]
- Among 15 036 adult candidates for heart transplantation between 2011 and 2016 in the United States, there was significant state-level variation in outcomes, ranging from 1.0 to 7.8 deaths per 1000 wait-list person-days for wait-list mortality.[103] One-year risk-adjusted graft survival ranged from 87% to 92%.

Mechanical Circulatory Support

- The 14th Annual Report from the STS INTERMACS described outcomes of 27 493 patients with a continuous-flow LVAD from 2013 to 2022.[104] During this period, there was a shift to nearly exclusive use of fully magnetically levitated devices.
- In 2022, of 2517 primary LVADs implanted, 99.8% were fully magnetically levitated devices.[104]
- The outcomes of magnetically levitated devices are superior to those of nonmagnetically levitated devices in contemporary (2018–2022) and historical (2013–2017) cohorts.[104]
- Patients supported by a magnetically levitated device (n=10 920) had a higher 1-year survival of 86% versus 79% and 81% in contemporary and historical nonmagnetically levitated groups, respectively (P<0.0001). They also had a higher 5-year survival of 64% versus 44% and 44% in contemporary and historical nonmagnetically levitated groups, respectively (P<0.0001).[104]
- Over 5 years, a higher proportion of patients with magnetically levitated devices had freedom from gastrointestinal bleeding (72% versus 60%;

P<0.0001), stroke (87% versus 67%; *P*<0.0001), and device malfunction/pump thrombus (83% versus 54%; *P*<0.0001) but not device-related infection (61% versus 64%; *P*=0.93) than patients with nonmagnetically levitated support during the contemporary era.[104]

- INTERMACS reported outcomes on 29 143 adults receiving an FDA-approved durable mechanical circulatory support device from 2012 to 2021.[105] During the reported 10-year period, there was not a significant change in the acuity of illness of patients receiving an implant. INTERMACS profiles 1 and 2 still represented approximately half of the population (51% in 2021 versus 54.9% in 2012), with a minority of patients belonging to INTERMACS profile 4 (12.7% in 2021 versus 14.9% in 2012).
- Among LVAD recipients, destination therapy is now the predominant indication (increased from 56.5% in 2018 to 81.1% in 2021), whereas bridge to transplantation is uncommon (decreased from 18.9% in 2018 to 5.3% in 2021).[105]
  - 1-year and 5-year survival was 86.5% and 52.9% for bridge to transplantation, 84.7% and 49.4% % for bridge to candidacy, and 70.9% and 42.4% for destination therapy in 2012 to 2021. During this period, there was a consistent improvement in survival for all device indications, with the most striking improvement in destination therapy as the predominant implantation strategy.[105]
  - Improvements in LVAD technology and the use of LVAD as destination therapy have led to more prolonged time on support. Currently, withdrawal from support is the leading cause of death (19.4%), replacing neurological dysfunction (17.9%) in the prior era.[105]
- In a study that used the United Network for Organ Sharing registry between 2006 and 2015 and addressed insurance status, among those with bridge to transplantation LVADs, Medicaid insurance was associated with worse survival compared with private insurance (subdistribution HR, 1.57 [95% CI, 1.15–2.16]), although access to transplantation was not different.[106]

Sex Differences

- According to INTERMACS data from 2017 to 2019, for patients receiving contemporary centrifugal LVADs, the risk of death appeared to be higher in males (HR, 1.63; *P*=0.01) relative to females.[107]

Cost

Overall Costs

The overall cost of HF continues to rise. See Chapter 28 (Economic Cost of Cardiovascular Disease) for further statistics.

- In 2012, total cost for HF was estimated to be $30.7 billion (2010 dollars), of which more than two-thirds was attributed to direct medical costs.[108] Projections suggest that by 2030 the total cost of HF will increase by 127% to $69.8 billion, amounting to ≈$244 for every US adult.
- In a systematic review of HF-associated medical costs in the United States from 2014 to 2020, the annual median total cost was estimated at $24 383 per patient, with HF hospitalizations accounting for the majority ($15 879 per patient).[109]
- Data from the US NIS for 4 287 478 primary HF hospitalizations 2014 to 2017 highlight differences in cost of care across demographic groups.[92] The median direct cost of admission was higher in high- than in low-SES groups ($10 940.40 versus $9324.60), male versus female patients ($10 217.10 versus $9866.60), and White versus Black individuals ($10 019.80 versus $9077.20). The median costs increased with SES in all demographic groups, related to greater procedural use.
- Among 11 806 679 HF hospitalizations in the US NIS database between 2002 and 2016, inflation-adjusted mean cost of stay increased from $14 301 to $17 925 (*P*<0.001; average annual increase, 1.52%).[91] This trend may have been related to the temporal increase in procedures (echocardiogram, right-sided heart catheterization, use of ventricular assist devices, CABG) and the higher incidence of HF complications (cardiogenic shock, respiratory failure, ventilator, and renal failure requiring dialysis).
- The costs associated with treating HF comorbidities and HF exacerbations in youths are significant, totaling nearly $1 billion in inpatient costs, and may be rising. The associated cost burden of HF is anticipated to constitute a large portion of total pediatric health care costs.[111]

Global Burden of HF

(See Table 22-3 and Chart 22-4)

- Based on 204 countries and territories in 2021[45]:
  - There were 55.50 (95% UI, 49.00–63.84) million prevalent cases of HF and an age-standardized prevalence rate of 676.68 (95% UI, 598.68–776.84) per 100 000 globally (Table 22-3).
  - Among regions, age-standardized prevalence of HF was highest for high-income North America followed by Central Europe, North Africa and the Middle East, and eastern sub-Saharan Africa. The lowest prevalence rates were for high-income Asia Pacific (Chart 22-4).
- In 2019:
  - Adults >70 years of age accounted for 62.2% of the world's HF cases, with female predominance

in this age group and male predominance in younger adults; 50.3% of those living with HF were females, but age-standardized prevalence was greater in males.[112]
  - 69.2% lived in low- and middle-income countries, although the highest age-standardized prevalence was highest in North America and lowest in South Asia.[112] Age-standardized HF prevalence in 2019 was highest in high-income North America (993.84 [95% CI, 866.22–1140.37] per 100 000 in females; 1344.62 [95% CI, 1159.53–1556.54] per 100 000 in males) and East Asia (1001.01 [95% CI, 819.06–1245.62] per 100 000 in females; 991.23 [95% CI, 808.02–1228.71] per 100 000 in males) followed by Oceania and eastern sub-Saharan Africa.[113]
  - There were 5.1 (95% UI, 3.3–7.3) million years lived with disability from HF, distributed equally between the sexes.[112]
  - In sequence, ischemic, hypertensive, and rheumatic HDs were the most common causes of HF in the world. IHD and hypertensive HD were the top causes of HF in males and females, respectively.[112]
- Between 1990 and 2019[112]:
  - There was a doubling in the global number of HF cases from 27.2 (95% UI, 22.2–33.4) million to 56.2 (95% UI, 46.4–67.8) million, with a doubling in both males and females.
  - Accounting for population growth, the age-standardized rate of HF per 100 000 people decreased by 7.1% worldwide, from 766.0 (95% UI, 626.3–936.0) in 1990 to 711.9 (95% UI, 591.1–858.3) in 2019. There were 9.1% (from 864.2 to 785.7) and 5.8% (from 686.0 to 646.1) decreases in age-standardized rates per 100 000 in males and females, respectively.
  - High-income regions experienced a 16.0% decrease in age-standardized rates (from 877.5 to 736.8), whereas low-income regions experienced a 3.9% increase (from 612.1 to 636.0), largely consistent across sexes.
  - There was a temporal increase in age-standardized HF from hypertensive, rheumatic, and calcific aortic valvular HD, as well as a temporal decrease from IHD, with some regional and sex differences. Age-standardized HF rates from hypertensive HD were largely stable but increased by as much as 22.3% in females in high-middle–SDI regions. Age-standardized prevalence of HF from rheumatic HD increased over time; this was driven by increasing rates in males in low- (5% increase) and low-middle– (9.2% increase) SDI regions and most notably in Andean Latin America (16.7% increase). Despite an overall decrease in the age-standardized HF attributable to IHD, low- and low-middle–SDI regions (including South and Southeast Asia and eastern and western sub-Saharan Africa) experienced increases ranging from 5% to 25% over time; this trend was consistent in both sexes.

Table 22-1. Global Prevalence and Mortality of Cardiomyopathy and Myocarditis, by Sex, 2021

	Both sexes		Male		Female	
	Deaths (95% UI)	Prevalence (95% UI)	Deaths (95% UI)	Prevalence (95% UI)	Deaths (95% UI)	Prevalence (95% UI)
Total number (millions), 2021	0.40 (0.37 to 0.43)	5.26 (4.36 to 6.09)	0.25 (0.22 to 0.27)	3.03 (2.51 to 3.53)	0.16 (0.14 to 0.17)	2.22 (1.84 to 2.62)
Percent change (%) in total number, 1990–2021	45.67 (33.04 to 59.51)	77.47 (67.74 to 87.17)	63.31 (46.26 to 80.27)	82.04 (72.71 to 91.54)	24.63 (11.99 to 39.24)	71.60 (61.49 to 81.79)
Percent change (%) in total number, 2010–2021	−0.53 (−6.38 to 5.73)	20.11 (13.65 to 26.46)	0.85 (−7.51 to 7.93)	20.27 (13.82 to 25.90)	−2.61 (−9.32 to 4.93)	19.90 (13.12 to 27.15)
Rate per 100 000, age standardized, 2021	4.89 (4.47 to 5.26)	65.90 (54.98 to 76.83)	6.47 (5.86 to 7.07)	78.26 (65.37 to 90.93)	3.46 (3.05 to 3.76)	54.20 (45.01 to 63.96)
Percent change (%) in rate, age standardized, 1990–2021	−37.54 (−42.40 to −32.03)	3.22 (−2.63 to 9.13)	−30.30 (−36.37 to −23.64)	5.97 (0.28 to 11.49)	−46.71 (−51.37 to −41.47)	−0.01 (−6.07 to 6.48)
Percent change (%) in rate, age standardized, 2010–2021	−25.16 (−29.44 to −20.50)	−0.58 (−5.89 to 4.35)	−23.01 (−28.99 to −17.91)	−0.47 (−5.61 to 4.10)	−27.96 (−32.80 to −22.55)	−0.75 (−6.42 to 4.55)

These estimates reflect improvements in demography and population estimation, statistical and geospatial modeling methods, and the addition of nearly 3000 new data sources since the 2024 AHA Statistical Update. During each annual GBD Study cycle, population health estimates are produced for the full time series. Improvements in statistical and geospatial modeling methods and the addition of new data sources may lead to changes in past results across GBD Study cycles.

GBD indicates Global Burden of Diseases, Injuries, and Risk Factors; and UI, uncertainty interval.

Source: Data courtesy of the GBD Study. Institute for Health Metrics and Evaluation. Used with permission. All rights reserved.[45]

Table 22-2. HF in the United States

Population group	Prevalence, 2017–2020, ≥20 y of age	Underlying cause mortality, 2022, all ages*	Underlying cause age-adjusted mortality rates per 100 000 (95% CI),* 2022	Any-mention mortality, 2022, all ages*	Any-mention age-adjusted mortality rates per 100 000 (95% CI),* 2022
Both sexes	6 700 000 (2.3%) (95% CI, 1.9%–2.8%)	87 941	21.0 (20.8–21.1)	425 147	101.1 (100.8–101.4)
Males	3 700 000 (2.7%)	41 657 (47.4%)†	24.2 (24.0–24.5)	213 137 (50.1%)†	121.9 (121.3–122.4)
Females	3 000 000 (1.9%)	46 284 (52.6%)†	18.5 (18.3–18.6)	212 010 (49.9%)†	85.6 (85.2–86.0)
NH White males	2.9%	32 820	25.3 (25.0–25.6)	169 070	128.2 (127.6–128.8)
NH White females	1.6%	37 152	19.6 (19.4–19.8)	168 537	90.4 (89.9–90.8)
NH Black males	3.8%	5101	32.1 (31.2–33.1)	23 112	143.8 (141.8–145.8)
NH Black females	3.3%	5375	22.7 (22.1–23.3)	23 818	99.8 (98.5–101.1)
Hispanic males	1.8%	2434	15.0 (14.3–15.6)	13 304	79.3 (77.9–80.8)
Hispanic females	1.6%	2478	11.0 (10.5–11.4)	12 611	55.2 (54.3–56.2)
NH Asian males	1.4%	835‡	9.7 (9.0–10.4)‡	4715‡	53.7 (52.1–55.2)‡
NH Asian females	0.5%	883‡	7.0 (6.5–7.5)‡	4611‡	36.7 (35.7–37.8)‡
NH American Indian or Alaska Native	…	354	14.4 (12.9–16.0)	2308	92.8 (88.9–96.6)
NH Native Hawaiian or Pacific Islander		94	17.1 (13.7–21.0)	609	108.8 (99.9–117.6)

HF includes people who answered "yes" to the question of ever having congestive HF. CIs have been added for overall prevalence estimates in key chapters. CIs have not been included in this table for all subcategories of prevalence for ease of reading. In March 2020, the COVID-19 pandemic halted NHANES field operations. Because data collected in the partial 2019 to 2020 cycle are not nationally representative, they were combined with previously released 2017 to 2018 data to produce nationally representative estimates.[114]

COVID-19 indicates coronavirus disease 2019; ellipses (…), data not available; HF, heart failure; NH, non-Hispanic; and NHANES, National Health and Nutrition Examination Survey.

*Mortality data for cause of death listed as HF on death certificates for Hispanic people, NH American Indian or Alaska Native people, and NH Asian and Pacific Islander people should be interpreted with caution because of inconsistencies in reporting Hispanic origin or race on the death certificate compared with censuses, surveys, and birth certificates. Studies have shown underreporting on death certificates of American Indian or Alaska Native decedents, Asian and Pacific Islander decedents, and Hispanic decedents, as well as undercounts of these groups in censuses. For reference to all-cause mortality in setting of prevalent HF, please see the Mortality section.

†These percentages represent the portion of total mortality attributable to HF that is for males vs females.

‡ Includes Chinese people, Filipino people, Japanese people, and other Asian people.

Sources: Prevalence: Unpublished National Heart, Lung, and Blood Institute (NHLBI) tabulation using NHANES.[115] Percentages are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2020 US population estimates. These data are based on self-reports. Mortality: Unpublished NHLBI tabulation using National Vital Statistics System[82] and Centers for Disease Control and Prevention Wide-Ranging Online Data for Epidemiologic Research.[83]

Table 22-3. Global Prevalence of Heart Failure, by Sex, 2021

	Prevalence		
	Both sexes (95% UI)	Male (95% UI)	Female (95% UI)
Total number (millions), 2021	55.50 (49.00 to 63.84)	28.59 (25.35 to 32.83)	26.91 (23.72 to 30.98)
Percent change (%) in total number, 1990–2021	118.21 (112.40 to 124.14)	119.11 (112.83 to 125.49)	117.25 (111.26 to 123.99)
Percent change (%) in total number, 2010–2021	33.28 (30.67 to 36.34)	32.28 (29.62 to 35.21)	34.36 (31.31 to 37.50)
Rate per 100 000, age standardized, 2021	676.68 (598.68 to 776.84)	760.78 (673.19 to 874.71)	604.00 (534.95 to 692.29)
Percent change (%) in rate, age standardized, 1990–2021	5.54 (2.70 to 8.49)	4.78 (1.74 to 7.81)	5.57 (2.35 to 8.69)
Percent change (%) in rate, age standardized, 2010–2021	1.02 (−0.54 to 2.73)	0.16 (−1.43 to 1.85)	1.81 (0.00 to 3.59)

These estimates reflect improvements in demography and population estimation, statistical and geospatial modeling methods, and the addition of nearly 3000 new data sources since the 2024 AHA Statistical Update. During each annual GBD Study cycle, population health estimates are produced for the full time series. Improvements in statistical and geospatial modeling methods and the addition of new data sources may lead to changes in past results across GBD Study cycles.

GBD indicates Global Burden of Diseases, Injuries, and Risk Factors; and UI, uncertainty interval.

Source: Data courtesy of the GBD Study. Institute for Health Metrics and Evaluation. Used with permission. All rights reserved.[45]

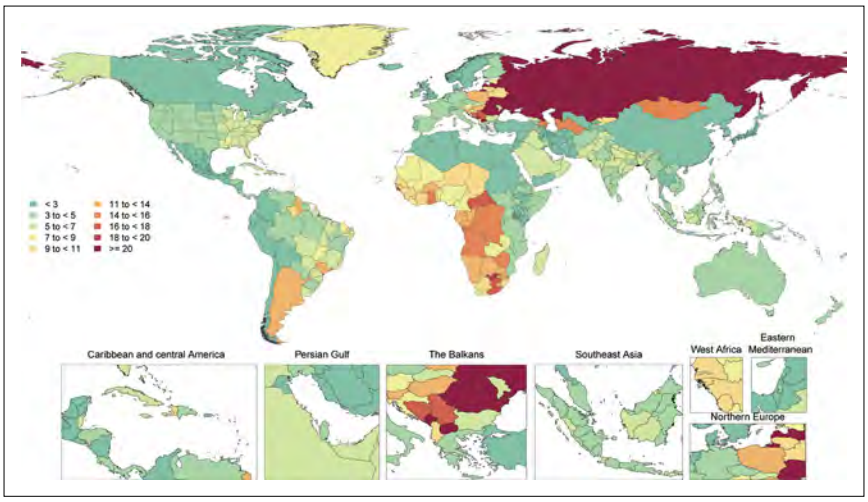

Chart 22-1. Age-standardized global mortality rates of cardiomyopathy and myocarditis per 100 000, both sexes, 2021.
These estimates reflect improvements in demography and population estimation, statistical and geospatial modeling methods, and the addition of nearly 3000 new data sources since the 2024 AHA Statistical Update. During each annual GBD Study cycle, population health estimates are produced for the full time series. Improvements in statistical and geospatial modeling methods and the addition of new data sources may lead to changes in past results across GBD Study cycles.
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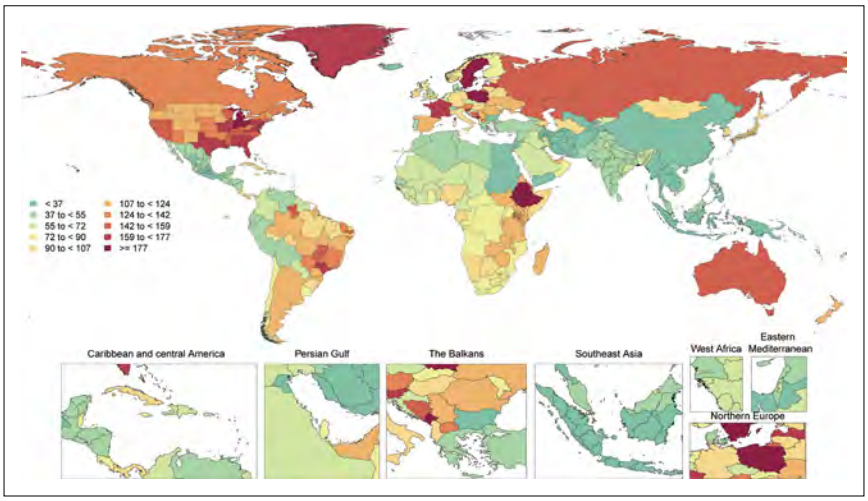

Chart 22-2. Age-standardized global prevalence rates of cardiomyopathy and myocarditis per 100 000, both sexes, 2021.
These estimates reflect improvements in demography and population estimation, statistical and geospatial modeling methods, and the addition of nearly 3000 new data sources since the 2024 AHA Statistical Update. During each annual GBD Study cycle, population health estimates are produced for the full time series. Improvements in statistical and geospatial modeling methods and the addition of new data sources may lead to changes in past results across GBD Study cycles.
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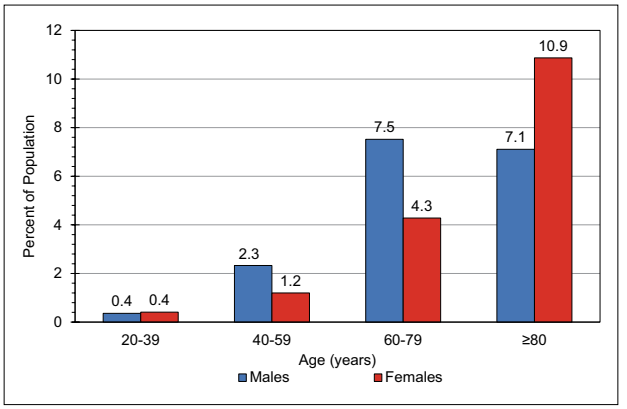

Chart 22-3. Prevalence of HF among US adults ≥20 years of age, by sex and age (NHANES, 2017–2020).
HF indicates heart failure; and NHANES, National Health and Nutrition Examination Survey.
Source: Unpublished National Heart, Lung, and Blood Institute tabulation using NHANES.[115]

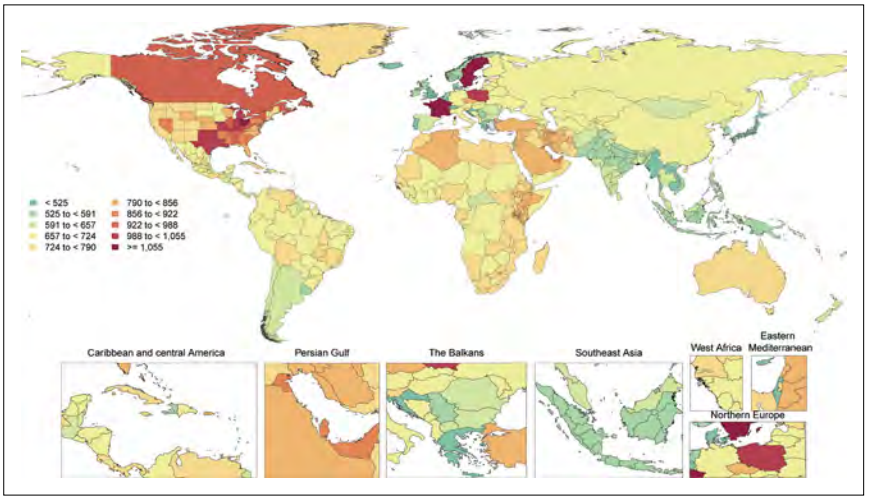

Chart 22-4. Age-standardized global prevalence rates of HF per 100 000, both sexes, 2021.
These estimates reflect improvements in demography and population estimation, statistical and geospatial modeling methods, and the addition of nearly 3000 new data sources since the 2024 AHA Statistical Update.
GBD indicates Global Burden of Disease, Injuries, and Risk Factors; and HF, heart failure.
Source: Data courtesy of the GBD Study. Institute for Health Metrics and Evaluation. Used with permission. All rights reserved.[45]

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30. GLOSSARY

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- *Age-adjusted rates*—Used mainly to compare the rates of ≥2 communities or population groups or the nation as a whole over time. The American Heart Association (AHA) uses a standard population (2000), so these rates are not affected by changes or differences in the age composition of the population. Unless otherwise noted, all death rates in this publication are age adjusted per 100 000 population and are based on underlying cause of death.
- *Agency for Healthcare Research and Quality (AHRQ)*—A part of the US Department of Health and Human Services, this is the lead agency charged with supporting research designed to improve the quality of health care, to reduce the cost of health care, to improve patient safety, to decrease the number of medical errors, and to broaden access to essential services. The AHRQ sponsors and conducts research that provides evidence-based information on health care outcomes, quality, cost, use, and access. The information helps health care decision makers (patients, clinicians, health system leaders, and policymakers) make more informed decisions and improve the quality of health care services. The AHRQ conducts the Medical Expenditure Panel Survey (MEPS; ongoing) and sponsors the Healthcare Cost and Utilization Project (HCUP; ongoing).
- *Body mass index (BMI)*—A mathematical formula to assess body weight relative to height. The measure correlates highly with body fat. It is calculated as weight in kilograms divided by the square of height in meters (kg/m^2).
- *Centers for Disease Control and Prevention/National Center for Health Statistics (CDC/NCHS)*—The CDC is an agency within the US Department of Health and Human Services. The CDC conducts the Behavioral Risk Factor Surveillance System (BRFSS), an ongoing survey. The CDC/NCHS conducts or has conducted these surveys (among others):
  - National Health Examination Survey (NHES I, 1960–1962; NHES II, 1963–1965; NHES III, 1966–1970)
  - National Health and Nutrition Examination Survey I (NHANES I; 1971–1975)
  - National Health and Nutrition Examination Survey II (NHANES II; 1976–1980)
  - National Health and Nutrition Examination Survey III (NHANES III; 1988–1994)
  - National Health and Nutrition Examination Survey (NHANES; 1999–…) (ongoing)
  - National Health Interview Survey (NHIS; ongoing)
  - National Hospital Discharge Survey (NHDS; 1965–2010)
  - National Ambulatory Medical Care Survey (NAMCS; ongoing)
  - National Hospital Ambulatory Medical Care Survey (NHAMCS;1992–2022)
  - National Nursing Home Survey (periodic)
  - National Home and Hospice Care Survey (periodic)
  - National Vital Statistics System (ongoing)
- *Centers for Medicare & Medicaid Services*—The federal agency that administers the Medicare, Medicaid, and Child Health Insurance programs.
- *Comparability ratio*—Provided by the NCHS to allow time-trend analysis from one *International Classification of Diseases* (*ICD*) revision to another. It compensates for the "shifting" of deaths from one causal code number to another. Its application to mortality based on one *ICD* revision means that mortality is "comparability modified" to be more comparable to mortality coded to the other *ICD* revision.
- *Coronary heart disease (CHD) (ICD-10 codes I20–I25)*—This category includes acute myocardial infarction (AMI; I21–I22); certain current complications after AMI (I23); other acute ischemic (coronary) heart disease (I24); angina pectoris (I20); atherosclerotic cardiovascular disease (I25.0); and all other forms of chronic ischemic (coronary) heart disease (I25.1–I25.9).
- *Death rate*—The relative frequency with which death occurs within some specified interval of time in a population. National death rates are computed per 100 000 population. Dividing the total number of deaths by the total population gives a crude death rate for the total population. Rates calculated within specific subgroups such as age-specific or sex-specific rates are often more meaningful and informative. They allow well-defined subgroups of the total population to be examined. Unless otherwise stated, all death rates in this publication are age adjusted and are per 100 000 population.
- *Diseases of the circulatory system (ICD-10 codes I00–I99)*—Included as part of what the AHA calls

The 2025 AHA Statistical Update uses language that conveys respect and specificity when referencing race and ethnicity. Instead of referring to groups very broadly with collective nouns (eg, Blacks, Whites), we use descriptions of race and ethnicity as adjectives (eg, Asian people, Black adults, Hispanic youths, Native American patients, White females).

As the AHA continues its focus on health equity to address structural racism, we are working to reconcile language used in previously published data sources and studies when this information is compiled in the annual Statistical Update. We strive to use terms from the original data sources or published studies (mostly from the past 5 years) that may not be as inclusive as the terms used in 2025. As style guidelines for scientific writing evolve, they will serve as guidance for data sources and publications and how they are cited in future Statistical Updates.

"cardiovascular disease" ("total cardiovascular disease" in this Glossary).

- *Diseases of the heart (ICD-10 codes I00–I09, 111, 113, 120–151)*—Classification that the NCHS uses in compiling the leading causes of death. Includes acute rheumatic fever/chronic rheumatic heart diseases (I00–I09); hypertensive heart disease (I11); hypertensive heart and renal disease (I13); CHD (I20–I25); pulmonary heart disease and diseases of pulmonary circulation (I26–I28); heart failure (I50); and other forms of heart disease (I30–I49, I51). "Diseases of the heart" are not equivalent to "total cardiovascular disease," which the AHA prefers to use to describe the leading causes of death.
- *Hispanic origin*—In US government statistics, "Hispanic" includes people who trace their ancestry to Mexico, Puerto Rico, Cuba, Spain, the Spanish-speaking countries of Central or South America, the Dominican Republic, or other Spanish cultures, regardless of race. It does not include people from Brazil, Guyana, Suriname, Trinidad, Belize, or Portugal because Spanish is not the first language in those countries. Most of the data in this update are for all Hispanic people, as reported by government agencies or specific studies. In certain time-trend charts and tables, data for Mexican American people are shown because data are not available for all Hispanic people.
- *Hospital discharges*—The number of inpatients (including newborn infants) discharged from short-stay hospitals for whom some type of disease was the principal diagnosis. Discharges include those discharged alive, dead, or "status unknown."
- *Incidence*—An estimate of the number of new cases of a disease that develop in a population, usually in a 1-year period. For some statistics, new and recurrent attacks, or cases, are combined. The incidence of a specific disease is estimated by multiplying the incidence rates reported in community- or hospital-based studies by the US population. The rates in this report change only when new data are available; they are not computed annually.
- *Infective endocarditis*—An infection of the inner lining (endocardium) of the heart or of the heart valves. The bacteria that most often cause endocarditis are streptococci, staphylococci, and enterococci.
- *International Classification of Diseases (ICD) codes*—A classification system in standard use in the United States. The *ICD* is published by the World Health Organization. This system is reviewed and revised approximately every 10 to 20 years to ensure its continued flexibility and feasibility. The 10th revision (*ICD-10*) began with the release of 1999 final mortality data. The *ICD* revisions can cause considerable change in the number of deaths reported for a given disease. The NCHS provides "comparability ratios" to compensate for the "shifting" of deaths from one *ICD* code to another. To compare the number or rate of deaths with that of an earlier year, the "comparability-modified" number or rate is used.
- *Major cardiovascular diseases*—Disease classification commonly reported by the NCHS; represents *ICD-10* codes I00 to I78. The AHA does not use "major cardiovascular disease" for any calculations. See "total cardiovascular disease" in this Glossary.
- *Metabolic syndrome*—Metabolic syndrome is defined as the presence of any 3 of the following 5 diagnostic measures: elevated waist circumference (>102 cm in males or >88 cm in females), elevated triglycerides (≥150 mg/dL [1.7 mmol/L] or drug treatment for elevated triglycerides), reduced high-density lipoprotein cholesterol (<40 mg/dL [0.9 mmol/L] in males, <50 mg/dL [1.1 mmol/L] in females, or drug treatment for reduced high-density lipoprotein cholesterol), elevated blood pressure (≥130 mm Hg systolic blood pressure, ≥85 mm Hg diastolic blood pressure, or drug treatment for hypertension), and elevated fasting glucose (≥100 mg/dL or drug treatment for elevated glucose).
- *Morbidity*—Both incidence and prevalence rates are measures of morbidity (ie, measures of various effects of disease on a population).
- *Mortality*—Mortality data for states can be obtained from the NCHS website (http://cdc.gov/nchs/), by direct communication with the CDC/NCHS, or from the AHA on request. The total number of deaths attributable to a given disease in a population during a specific interval of time, usually 1 year, is reported. These data are compiled from death certificates and sent by state health agencies to the NCHS. The process of verifying and tabulating the data takes ≈2 years.
- *National Heart, Lung, and Blood Institute (NHLBI)*—An institute in the National Institutes of Health in the US Department of Health and Human Services. The NHLBI conducts such studies as the following:
  - Framingham Heart Study (FHS; 1948–…) (ongoing)
  - Honolulu Heart Program (HHP; 1965–2002)
  - Cardiovascular Health Study (CHS; 1989–…) (ongoing)
  - Atherosclerosis Risk in Communities (ARIC) study (1987–…) (ongoing)
  - Strong Heart Study (SHS; 1989–…) (ongoing)
  - Multi-Ethnic Study of Atherosclerosis (MESA; 2000–…) (ongoing)
- *National Institute of Neurological Disorders and Stroke (NINDS)*—An institute in the National Institutes of Health of the US Department of Health and Human Services. The NINDS sponsors and conducts research studies such as these:

  - Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS)
  - Rochester (Minnesota) Stroke Epidemiology Project
  - Northern Manhattan Study (NOMAS)
  - Brain Attack Surveillance in Corpus Christi (BASIC) Project
- *Physical activity*—Any bodily movement produced by the contraction of skeletal muscle that increases energy expenditure above a basal level.
- *Physical fitness*—The ability to perform daily tasks with vigor and alertness, without undue fatigue, and with ample energy to enjoy leisure-time pursuits and to respond to emergencies. Physical fitness includes a number of components consisting of cardiorespiratory endurance (aerobic power), skeletal muscle endurance, skeletal muscle strength, skeletal muscle power, flexibility, balance, speed of movement, reaction time, and body composition.
- *Prevalence*—An estimate of the total number of cases of a disease existing in a population during a specified period. Prevalence is sometimes expressed as a percentage of population. Rates for specific diseases are calculated from periodic health examination surveys that government agencies conduct. Annual changes in prevalence as reported in this Statistical Update reflect changes in the population size. Changes in rates can be evaluated only by comparing prevalence rates estimated from surveys conducted in different years. Note: In the data tables, which are located in the different disease and risk factor chapters, if the percentages shown are age adjusted, they will not add to the total.
- *Race and Hispanic origin*—Race and Hispanic origin are reported separately on death certificates. In this publication, unless otherwise specified, deaths of people of Hispanic origin are included in the totals for White people, Black people, American Indian or Alaska Native people, and Asian or Pacific Islander people according to the race listed on the decedent's death certificate. Data for Hispanic people include all people of Hispanic origin of any race. See "Hispanic origin" in this Glossary.
- *Stroke (ICD-10 codes I60–I69)*—This category includes subarachnoid hemorrhage (I60); intracerebral hemorrhage (I61); other nontraumatic intracranial hemorrhage (I62); cerebral infarction (I63); stroke, not specified as hemorrhage or infarction (I64); occlusion and stenosis of precerebral arteries not resulting in cerebral infarction (I65); occlusion and stenosis of cerebral arteries not resulting in cerebral infarction (I66); other cerebrovascular diseases (I67); cerebrovascular disorders in diseases classified elsewhere (I68); and sequelae of cerebrovascular disease (I69).
- *Total cardiovascular disease (ICD-10 codes I00–I99)*—This category includes rheumatic fever/rheumatic heart disease (I00–I09); hypertensive diseases (I10–I15); ischemic (coronary) heart disease (I20–I25); pulmonary heart disease and diseases of pulmonary circulation (I26–I28); other forms of heart disease (I30–I52); cerebrovascular disease (stroke) (I60–I69); atherosclerosis (I70); other diseases of arteries, arterioles, and capillaries (I71–I79); diseases of veins, lymphatics, and lymph nodes not classified elsewhere (I80–I89); and other and unspecified disorders of the circulatory system (I95–I99).
- *Underlying cause of death or any-mention cause of death*—These terms are used by the NCHS when defining mortality. Underlying cause of death is defined by the World Health Organization as "the disease or injury which initiated the chain of events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury." Any-mention cause of death includes the underlying cause of death and up to 20 additional multiple causes listed on the death certificate.