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Heart Disease and Stroke Statistics—2012 Update : A Report From the American Heart Association

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Heart Disease and Stroke Statistics—2012 Update

A Report From the American Heart Association

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*The findings and conclusions of this report are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.

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Summary

Each year, the American Heart Association (AHA), in conjunction with the Centers for Disease Control and Prevention, the National Institutes of Health, and other government agencies, brings together the most up-to-date statistics on heart disease, stroke, other vascular diseases, and their risk factors and presents them in its Heart Disease and Stroke Statistical Update. The Statistical Update is a valuable resource for researchers, clinicians, healthcare policy makers, media professionals, the lay public, and many others who seek the best national data available on disease morbidity and mortality and the risks, quality of care, medical procedures and operations, and costs associated with the management of these diseases in a single document. Indeed, since 1999, the Statistical Update has been cited more than 8700 times in the literature (including citations of all annual versions). In 2010 alone, the various Statistical Updates were cited ≈1600 times (data from ISI Web of Science). In recent years, the Statistical Update has undergone some major changes with the addition of new chapters and major updates across multiple areas. For this year's edition, the Statistics Committee, which produces the document for the AHA, updated all of the current chapters with the most recent nationally representative data and inclusion of relevant articles from the literature over the past year and added a new chapter detailing various disorders of heart rhythm. Also, the 2012 Statistical Update is a major source for monitoring both cardiovascular health and disease in the population, with a focus on progress toward achievement of the AHA's 2020 Impact Goals. Below are a few highlights from this year's Update.

Rates of Death Attributable to CVD Have Declined, Yet the Burden of Disease Remains High

- The 2008 overall rate of death attributable to cardiovascular disease (CVD) (*International Classification of Diseases, 10th Revision*, codes I00–I99) was 244.8 per 100 000. The rates were 287.2 per 100 000 for white males, 390.4 per 100 000 for black males, 200.5 per 100 000 for white females, and 277.4 per 100 000 for black females.
- From 1998 to 2008, the rate of death attributable to CVD declined 30.6%. Mortality data for 2008 show that CVD (I00–I99; Q20–Q28) accounted for 32.8% (811 940) of all 2 471 984 deaths in 2008, or 1 of every 3 deaths in the United States.
- On the basis of 2008 mortality rate data, more than 2200 Americans die of CVD each day, an average of 1 death every 39 seconds. About 150 000 Americans killed by CVD (I00–I99) in 2008 were <65 years of age. In 2008, 33% of deaths due to CVD occurred before the age of 75 years, which is well before the average life expectancy of 77.9 years.
- Coronary heart disease caused ≈1 of every 6 deaths in the United States in 2008. Coronary heart disease mortality in 2008 was 405 309. Each year, an estimated 785 000 Americans will have a new coronary attack, and ≈470 000 will have a recurrent attack. It is estimated that an additional 195 000 silent first myocardial infarctions occur each year. Approximately every 25 seconds, an American will have a coronary event, and approximately every minute, someone will die of one.
- Each year, ≈795 000 people experience a new or recurrent stroke. Approximately 610 000 of these are first attacks, and 185 000 are recurrent attacks. Mortality data from 2008 indicate that stroke accounted for ≈1 of every 18 deaths in the United States. On average, every 40 seconds, someone in the United States has a stroke. From 1998 to 2008, the stroke death rate fell 34.8%, and the actual number of stroke deaths declined 19.4%.
- In 2008, 1 in 9 death certificates (281 437 deaths) in the United States mentioned heart failure.

Prevalence and Control of Traditional Risk Factors Remains an Issue for Many Americans

- Data from the National Health and Nutrition Examination Survey (NHANES) 2005–2008 indicate that 33.5% of US adults ≥20 years of age have hypertension (Table 7-1). This amounts to an estimated 76 400 000 US adults with hypertension. The prevalence of hypertension is nearly equal between men and women. African American adults have among the highest rates of hypertension in the world, at 44%.
- Among hypertensive adults, ≈80% are aware of their condition, 71% are using antihypertensive medication, and only 48% of those aware that they have hypertension have their condition controlled.
- Despite 4 decades of progress, in 2010, among Americans ≥18 years of age, 21.2% of men and 17.5% of women continued to be cigarette smokers. In 2009, 19.5% of students in grades 9 through 12 reported current cigarette use.
- The percentage of the nonsmoking population with detectable serum cotinine (indicating exposure to secondhand smoke) declined from 52.5% in 1999 to 2000 to 40.1% in 2007 to 2008, with declines occurring, and was higher for those 3 to 11 years of age (53.6%) and those 12 to 19 years of age (46.5%) than for those 20 years of age and older (36.7%).
- An estimated 33 600 000 adults ≥20 years of age have total serum cholesterol levels ≥240 mg/dL, with a prevalence of 15.0% (Table 14-1).
- In 2008, an estimated 18 300 000 Americans had diagnosed diabetes mellitus, representing 8.0% of the adult population. An additional 7 100 000 had undiagnosed diabetes mellitus, and 36.8% had prediabetes, with abnormal fasting glucose levels. African Americans, Mexican Americans, Hispanic/Latino individuals, and other ethnic minorities bear a strikingly disproportionate burden of diabetes mellitus in the United States (Table 17-1).

The 2012 Update Expands Data Coverage of the Obesity Epidemic and Its Antecedents and Consequences

- The estimated prevalence of overweight and obesity in US adults (≥20 years of age) is 149 300 000, which represents 67.3% of this group in 2008. Fully 33.7% of US adults are obese (body mass index ≥30 kg/m^2). Men and women of all race/ethnic groups in the population are affected by the epidemic of overweight and obesity (Table 16-1).
- Among children 2 to 19 years of age, 31.7% are overweight and obese (which represents 23.6 million children), and 16.9% are obese (12.6 million children). Mexican American boys and girls and African American girls are disproportionately affected. Over the past 3 decades, the prevalence of obesity in children 6 to 11 years of age has increased from ≈4% to >20%.
- Obesity (body mass index ≥30 kg/m^2) is associated with marked excess mortality in the US population. Even more notable is the excess morbidity associated with overweight and obesity in terms of risk factor development and incidence of diabetes mellitus, CVD end points (including coronary heart disease, stroke, and heart failure), and numerous other health conditions, including asthma, cancer, degenerative joint disease, and many others.
- The prevalence of diabetes mellitus is increasing dramatically over time, in parallel with the increases in prevalence of overweight and obesity.
- On the basis of NHANES 2003–2006 data, the age-adjusted prevalence of metabolic syndrome, a cluster of major cardiovascular risk factors related to overweight/obesity and insulin resistance, is ≈34% (35.1% among men and 32.6% among women).
- The proportion of youth (≤18 years of age) who report engaging in no regular physical activity is high, and the proportion increases with age. In 2009, among adolescents in grades 9 through 12, 29.9% of girls and 17.0% of boys reported that they had not engaged in 60 minutes of moderate-to-vigorous physical activity, defined as any activity that increased heart rate or breathing rate, even once in the previous 7 days, despite recommendations that children engage in such activity ≥5 days per week.
- Thirty-three percent of adults reported engaging in no aerobic leisure-time physical activity.
- Data from NHANES indicate that between 1971 and 2004, average total energy consumption among US adults increased by 22% in women (from 1542 to 1886 kcal/d) and by 10% in men (from 2450 to 2693 kcal/d; see Chart 20-1).
- The increases in calories consumed during this time period are attributable primarily to greater average carbohydrate intake, in particular, of starches, refined grains, and sugars. Other specific changes related to increased caloric intake in the United States include larger portion sizes, greater food quantity and calories per meal, and increased consumption of sugar-sweetened beverages, snacks, commercially prepared (especially fast food) meals, and higher energy-density foods.

The 2012 Update Provides Critical Data About Cardiovascular Quality of Care, Procedure Utilization, and Costs

In light of the current national focus on healthcare utilization, costs, and quality, it is critical to monitor and understand the magnitude of healthcare delivery and costs, as well as the quality of healthcare delivery, related to CVDs. The Statistical Update provides these critical data in several sections.

Quality-of-Care Metrics for CVDs

Chapter 21 reviews many metrics related to the quality of care delivered to patients with CVDs, as well as healthcare disparities. In particular, quality data are available from the AHA's "Get With The Guidelines" programs for coronary artery disease and heart failure and from the American Stroke Association/AHA's "Get With The Guidelines" program for acute stroke. Similar data from the Veterans Healthcare Administration, national Medicare and Medicaid data, and Acute Coronary Treatment and Intervention Outcomes Network–"Get With The Guidelines" Registry data are also reviewed. These data show impressive adherence with guideline recommendations for many, but not all, metrics of quality of care for these hospitalized patients. Data are also reviewed on screening for cardiovascular risk factor levels and control.

Cardiovascular Procedure Utilization and Costs

Chapter 22 provides data on trends and current usage of cardiovascular surgical and invasive procedures. For example, the total number of inpatient cardiovascular operations and procedures increased 22%, from 6 133 000 in 1999 to 7 453 000 in 2009 (National Heart, Lung, and Blood Institute computation based on National Center for Health Statistics annual data).

Chapter 23 reviews current estimates of direct and indirect healthcare costs related to CVDs, stroke, and related conditions using Medical Expenditure Panel Survey data. The total direct and indirect cost of CVD and stroke in the United States for 2008 is estimated to be $297.7 billion. This figure includes health expenditures (direct costs, which include the cost of physicians and other professionals, hospital services, prescribed medications, home health care, and other medical durables) and lost productivity resulting from mortality (indirect costs). By comparison, in 2008, the estimated cost of all cancer and benign neoplasms was $228 billion ($93 billion in direct costs, $19 billion in morbidity indirect costs, and $116 billion in mortality indirect costs). CVD costs more than any other diagnostic group.

The AHA, through its Statistics Committee, continuously monitors and evaluates sources of data on heart disease and stroke in the United States to provide the most current data available in the Statistics Update.

Finally, it must be noted that this annual Statistical Update is the product of an entire year's worth of effort by dedicated professionals, volunteer physicians and scientists, and outstand-

ing AHA staff members, without whom publication of this valuable resource would be impossible. Their contributions are gratefully acknowledged.

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On behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee

Note: Population data used in the compilation of NHANES prevalence estimates is for the latest year of the NHANES survey being used. Extrapolations for NHANES prevalence estimates are based on the census resident population for 2008 because this is the most recent year of NHANES data used in the Statistical Update.

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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

1. About These Statistics

The American Heart Association (AHA) works with the Centers for Disease Control and Prevention's (CDC's) National Center for Health Statistics (NCHS); the National Heart, Lung, and Blood Institute (NHLBI); the National Institute of Neurological Disorders and Stroke (NINDS); and other government agencies to derive the annual statistics in this Heart Disease and Stroke Statistical Update. This chapter describes the most important sources and the types of data we use from them. For more details, see Chapter 25 of this document, the Glossary.

The surveys used are:

- Behavioral Risk Factor Surveillance System (BRFSS)—ongoing telephone health survey system
- Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS)—stroke incidence rates and outcomes within a biracial population
- Medical Expenditure Panel Survey (MEPS)—data on specific health services that Americans use, how frequently they use them, the cost of these services, and how the costs are paid
- National Health and Nutrition Examination Survey (NHANES)—disease and risk factor prevalence and nutrition statistics
- National Health Interview Survey (NHIS)—disease and risk factor prevalence
- National Hospital Discharge Survey (NHDS)—hospital inpatient discharges and procedures (discharged alive, dead, or status unknown)
- National Ambulatory Medical Care Survey (NAMCS)—physician office visits
- National Home and Hospice Care Survey (NHHCS)—staff, services, and patients of home health and hospice agencies
- National Hospital Ambulatory Medical Care Survey (NHAMCS)—hospital outpatient and emergency department (ED) visits
- Nationwide Inpatient Sample of the Agency for Healthcare Research and Quality—hospital inpatient discharges, procedures, and charges
- National Nursing Home Survey (NNHS)—nursing home residents
- National Vital Statistics System—national and state mortality data
- World Health Organization—mortality rates by country
- Youth Risk Behavior Surveillance System (YRBSS)—health-risk behaviors in youth and young adults

Abbreviations Used in Chapter 1

AHA	American Heart Association
AP	angina pectoris
ARIC	Atherosclerosis Risk in Communities Study
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHS	Cardiovascular Health Study
CVD	cardiovascular disease
DM	diabetes mellitus
ED	emergency department
FHS	Framingham Heart Study
GCNKSS	Greater Cincinnati/Northern Kentucky Stroke Study
HD	heart disease
HF	heart failure
ICD	International Classification of Diseases
ICD-9-CM	*International Classification of Diseases, Clinical Modification, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHHCS	National Home and Hospice Care Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NINDS	National Institute of Neurological Disorders and Stroke
NNHS	National Nursing Home Survey
PAD	peripheral artery disease
YRBSS	Youth Risk Behavior Surveillance System

See Glossary (Chapter 25) for explanation of terms.

Disease Prevalence

Prevalence is an estimate of how many people have a disease at a given point or period in time. The NCHS conducts health examination and health interview surveys that provide estimates of the prevalence of diseases and risk factors. In this Update, the health interview part of the NHANES is used for the prevalence of cardiovascular diseases (CVDs). NHANES is used more than the NHIS because in NHANES, angina pectoris (AP) is based on the Rose Questionnaire; estimates are made regularly for heart failure (HF); hypertension is based on blood pressure (BP) measurements and interviews; and an estimate can be made for total CVD, including myocardial infarction (MI), AP, HF, stroke, and hypertension.

A major emphasis of this Statistical Update is to present the latest estimates of the number of people in the United States who have specific conditions to provide a realistic estimate of burden. Most estimates based on NHANES prevalence rates are based on data collected from 2005 to 2008 (in most cases, these are the latest published figures). These are applied to census population estimates for 2008. Differences in population estimates based on extrapolations of rates beyond the data collection period by use of more recent census population estimates cannot be used to evaluate possible trends in prevalence. Trends can only be evaluated by comparing prevalence rates estimated from surveys conducted in different years.

Risk Factor Prevalence

The NHANES 2005–2008 data are used in this Update to present estimates of the percentage of people with high lipid

values, diabetes mellitus (DM), overweight, and obesity. The NHIS is used for the prevalence of cigarette smoking and physical inactivity. Data for students in grades 9 through 12 are obtained from the YRBSS.

Incidence and Recurrent Attacks

An incidence rate refers to the number of new cases of a disease that develop in a population per unit of time. The unit of time for incidence is not necessarily 1 year, although we often discuss incidence in terms of 1 year. For some statistics, new and recurrent attacks or cases are combined. Our national incidence estimates for the various types of CVD are extrapolations to the US population from the Framingham Heart Study (FHS), the Atherosclerosis Risk in Communities (ARIC) study, and the Cardiovascular Health Study (CHS), all conducted by the NHLBI, as well as the GCNKSS, which is funded by the NINDS. The rates change only when new data are available; they are not computed annually. Do not compare the incidence or the rates with those in past editions of the Heart Disease and Stroke Statistics Update (also known as the Heart and Stroke Statistical Update for editions before 2005). Doing so can lead to serious misinterpretation of time trends.

Mortality

Mortality data are presented according to the underlying cause of death. "Any-mention" mortality means that the condition was nominally selected as the underlying cause or was otherwise mentioned on the death certificate. For many deaths classified as attributable to CVD, selection of the single most likely underlying cause can be difficult when several major comorbidities are present, as is often the case in the elderly population. It is useful, therefore, to know the extent of mortality attributable to a given cause regardless of whether it is the underlying cause or a contributing cause (ie, its "any-mention" status). The number of deaths in 2008 with any mention of specific causes of death was tabulated by the NHLBI from the NCHS public-use electronic files on mortality.

The first set of statistics for each disease in this Update includes the number of deaths for which the disease is the underlying cause. Two exceptions are Chapter 7 (High Blood Pressure) and Chapter 9 (Cardiomyopathy and Heart Failure). High BP, or hypertension, increases the mortality risks of CVD and other diseases, and HF should be selected as an underlying cause only when the true underlying cause is not known. In this Update, hypertension and HF death rates are presented in 2 ways: (1) as nominally classified as the underlying cause and (2) as any-mention mortality.

National and state mortality data presented according to the underlying cause of death were computed from the mortality tables of the NCHS World Wide Web site, the Health Data Interactive data system of the NCHS, or the CDC compressed mortality file. Any-mention numbers of deaths were tabulated from the electronic mortality files of the NCHS World Wide Web site and from Health Data Interactive.

Population Estimates

In this publication, we have used national population estimates from the US Census Bureau for 2008 in the computation of morbidity data. NCHS population estimates for 2008 were used in the computation of death rate data. The Census Bureau World Wide Web site[1] contains these data, as well as information on the file layout.

Hospital Discharges and Ambulatory Care Visits

Estimates of the numbers of hospital discharges and numbers of procedures performed are for inpatients discharged from short-stay hospitals. Discharges include those discharged alive, dead, or with unknown status. Unless otherwise specified, discharges are listed according to the first-listed (primary) diagnosis, and procedures are listed according to all listed procedures (primary plus secondary). These estimates are from the NHDS of the NCHS unless otherwise noted. Ambulatory care visit data include patient visits to physician offices and hospital outpatient departments and EDs. Ambulatory care visit data reflect the first-listed (primary) diagnosis. These estimates are from NAMCS and NHAMCS of the NCHS.

International Classification of Diseases

Morbidity (illness) and mortality (death) data in the United States have a standard classification system: the International Classification of Diseases (ICD). Approximately every 10 to 20 years, the ICD codes are revised to reflect changes over time in medical technology, diagnosis, or terminology. Where necessary for comparability of mortality trends across the 9th and 10th ICD revisions, comparability ratios computed by the NCHS are applied as noted.[2] Effective with mortality data for 1999, we are using the 10th revision (ICD-10). It will be a few more years before the 10th revision is used for hospital discharge data and ambulatory care visit data, which are based on the *International Classification of Diseases, Clinical Modification, 9th Revision* (ICD-9-CM).[3]

Age Adjustment

Prevalence and mortality estimates for the United States or individual states comparing demographic groups or estimates over time either are age specific or are age adjusted to the 2000 standard population by the direct method.[4] International mortality data are age adjusted to the European standard.[5] Unless otherwise stated, all death rates in this publication are age adjusted and are deaths per 100 000 population.

Data Years for National Estimates

In this Update, we estimate the annual number of new (incidence) and recurrent cases of a disease in the United States by extrapolating to the US population in 2008 from rates reported in a community- or hospital-based study or multiple studies. Age-adjusted *incidence* rates by sex and race are also given in this report as observed in the study or studies. For US *mortality*, most numbers and rates are for 2008. For disease and risk factor *prevalence*, most rates in this report are calculated from the 2005–2008 NHANES. Because NHANES is conducted only in the noninstitutionalized population, we extrapolated the rates to the total US population in 2008, recognizing that this probably underestimates the total prevalence, given the relatively high prevalence in the institutionalized population. The numbers and rates of

hospital inpatient discharges for the United States are for 2009. Numbers of visits to *physician offices*, *hospital EDs*, and *hospital outpatient departments* are for 2009. Except as noted, *economic cost* estimates are for 2008.

Cardiovascular Disease

For data on hospitalizations, physician office visits, and mortality, CVD is defined according to ICD codes given in Chapter 25 of the present document. This definition includes all diseases of the circulatory system, as well as congenital CVD. Unless so specified, an estimate for total CVD does not include congenital CVD. Prevalence of CVD includes people with hypertension, heart disease (HD), stroke, peripheral artery disease (PAD), and diseases of the veins.

Race

Data published by governmental agencies for some racial groups are considered unreliable because of the small sample size in the studies. Because we try to provide data for as many racial groups as possible, we show these data for informational and comparative purposes.

Contacts

If you have questions about statistics or any points made in this Update, please contact the AHA National Center, Office of Science & Medicine at statistics@heart.org. Direct all media inquiries to News Media Relations at inquiries@heart.org or 214-706-1173.

We do our utmost to ensure that this Update is error free. If we discover errors after publication, we will provide corrections at our World Wide Web site, http://www.heart.org/statistics, and in the journal *Circulation*.

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2. American Heart Association's 2020 Impact Goals

See Tables 2-1 through 2-4 and Charts 2-1 through 2-9.

After achieving its major Impact Goals for 2010, the AHA recently created a new set of Impact Goals for the current decade.[1] Specifically, the AHA committed to the following organizational goals:

> *By 2020, to improve the cardiovascular health of all Americans by 20%, while reducing deaths from cardiovascular diseases and stroke by 20%.*[1]

These goals include a novel concept, "cardiovascular health," which encompasses 7 health behaviors and health factors (Table 2-1). "Ideal cardiovascular health" is defined by the absence of clinically manifest CVD and the simultaneous presence of optimal levels of all 7 health behaviors (lean body mass, avoidance of smoking, participation in physical activity [PA], and healthy dietary intake consistent with a Dietary Approaches to Stop Hypertension [DASH]–like eating pattern) and health factors (untreated total cholesterol <200 mg/dL, untreated BP <120/<80 mm Hg, and fasting blood glucose <100 mg/dL). Because the ideal cardiovascular health profile is known to be rare in the population, the entire spectrum of cardiovascular health can also be represented as being "ideal," "intermediate," or "poor" for each of the health behaviors and health factors, as shown in Table 2-1.[1]

Beginning in 2011, and recognizing the substantial time lag in the nationally representative data sets, the annual Statistical Update began to evaluate and publish metrics and information that gives the AHA directional insights into progress and/or areas critical for greater concentration, to meet their 2020 goals.

Cardiovascular Health

- Table 2-1 provides the specific definitions for ideal, intermediate, and poor cardiovascular health for each of the 7 health behaviors and health factors, for adults ≥20 years of age and children of selected ages (depending on data availability).
- The prevalences of ideal, intermediate, and poor levels of each of the 7 cardiovascular health metrics are shown in Chart 2-1 (for children ages 12–19 years) and Chart 2-2 (for adults ≥20 years of age).
  - — Among children (Chart 2-1), the prevalence (unadjusted) of ideal levels of cardiovascular health behaviors and factors currently varies from 0% for the healthy diet score (ie, essentially no children meet 4 or 5 of the 5 dietary components) to >80% for the smoking and BP metrics. More than 90% of US children meet 0 or only 1 of the 5 healthy dietary components.
  - — Among US adults (Chart 2-2), the age-standardized prevalence of ideal levels of cardiovascular health behaviors and factors currently varies from 0.1% for having 4 to 5 components of the healthy diet score up to 75% for the smoking metric (ie, 75% of US adults have never smoked or are current nonsmokers who have quit for >12 months).
  - — In general, the prevalence of ideal levels of health behaviors and health factors is higher in US children than in US adults.
- Age-standardized and age-specific prevalence estimates for ideal cardiovascular health and for ideal levels of each of its components are shown in Table 2-2.
  - — The prevalence of ideal levels of all of the 7 health factors and health behaviors decreases dramatically from younger to older ages.
- Chart 2-3 displays the prevalence estimates for the population of US children meeting different numbers of criteria for ideal cardiovascular health (out of 7 possible).
  - — Half of US children ages 12 to 19 years meet 4 or fewer criteria for ideal cardiovascular health.
  - — The distributions are similar overall in boys and girls.
- Charts 2-4 and 2-5 display the age-standardized prevalence estimates for the population of US adults meeting different numbers of criteria for ideal cardiovascular health (out of 7 possible), overall and stratified by age groups, sex, and race.
  - — Approximately 2.5% of US adults have 0 of the 7 criteria at ideal levels, with 26% having 3 at ideal levels and 4% having 6 metrics at ideal levels (Chart 2-4).
  - — Compared with younger adults, older adults tend to have fewer of the 7 metrics at ideal levels; more than 60% of those >60 years of age have only 2 or fewer metrics at ideal levels (Chart 2-4).

Abbreviations Used in Chapter 2

AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities Study
BMI	body mass index
BP	blood pressure
CVD	cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	diastolic blood pressure
DM	diabetes mellitus
HD	heart disease
HF	heart failure
HR	hazard ratio
MI	myocardial infarction
NHANES	National Health and Nutrition Examination Survey
PA	physical activity
SBP	systolic blood pressure
SE	standard error

— Women tend to have more metrics at ideal levels than do men (Chart 2-4).
— Approximately 63% of white adults and 71% of black and Mexican American adults have 3 or fewer metrics (out of 7) at ideal levels (Chart 2-5).

- Chart 2-6 displays the age-standardized percentages of US adults and percentages of children who have 5 or more of the metrics (out of 7 possible) at ideal levels.

— Only ≈41% of US children aged 12 to 19 years have 5 or more metrics at ideal levels, including somewhat more girls than boys.
— However, only 16% of US adults have 5 or more metrics with ideal levels, including 12% of men and 21% of women.
— Whites have approximately twice the percentage of adults with 5 or more metrics with ideal levels as Mexican Americans.

- Chart 2-7 displays the age-standardized percentages of US adults meeting different numbers of criteria for poor and ideal cardiovascular health. Meeting the AHA 2020 Strategic Impact Goals is predicated on reducing the relative percentage of those with poor levels while increasing the relative percentage of those with ideal levels for each of the 7 metrics.

— Approximately 94% of US adults have at least 1 metric at poor levels.
— Approximately 38% of US adults have at least 3 metrics at poor levels.

- The prevalence of risk factors and their awareness, treatment, and control are displayed in Table 2-3 separately for those with and without self-reported CVD. Among those without CVD, NHANES 2007–2008 data indicate the following:

— Approximately 26% of US adults are current smokers or have recently quit for <12 months.
— Prevalence of hypertension is estimated to be 27%; 71% are aware of their hypertension, and 57% are treated. Among those with hypertension who are treated, control to goal BP levels of <140/<90 mm Hg is 77%.
— Prevalence of dyslipidemia (defined by total cholesterol ≥240 mg/dL or receiving medication) is 25%; 63% are aware of their dyslipidemia, and 38% are treated. Among those with dyslipidemia who are treated, 81% have total cholesterol <200 mg/dL.
— Prevalence of obesity is 33%, and prevalence of overweight or obesity is 68%.
— Prevalence of DM is 9%; 64% are aware of their DM, and 63% are treated. Among those with DM who are treated, 23% have controlled blood glucose levels.
— As measured by objective accelerometer data, 60% of adults have intermediate or poor levels of PA, with 47% having no moderate or vigorous activity at all.
— 79% of US adults without CVD meet 0 or only 1 of the 5 healthy diet metrics.

Cardiovascular Disease

- In 2007, the age-standardized death rate attributable to all CVDs was 251.2 per 100 000 (Chart 2-8), down 4.3% from 262.5 in 2006 (baseline data for the 2020 Impact Goals on CVD and stroke mortality).

— Death rates attributable to stroke, heart diseases (HDs), and other cardiovascular causes were 42.2, 126.0, and 82.9 per 100 000, respectively.

- Data from NHANES 2007–2008 reveal that overall, 6.6% of Americans self-reported having some type of CVD (Table 2-3).

— 2.8% reported having coronary heart disease
— 2.6% reported having a stroke
— 2.0% reported having congestive heart failure
— 2.7% reported having a heart attack

- Among those with CVD, risk factor prevalence, awareness, treatment, and control in NHANES 2007 to 2008 were variable (Table 2-3).

— Nearly 48% were current smokers or had quit for <12 months.
— Prevalence of hypertension was estimated to be 45%; 96% were aware of their hypertension, and 89% were treated. Among those with hypertension who were treated, control to goal BP levels of <140/<90 mm Hg was 62%.
— Prevalence of dyslipidemia (defined by total cholesterol ≥240 mg/dL or receiving medication) was 35%; 83% were aware of their dyslipidemia, and 76% were treated. Among those with dyslipidemia who were treated, 85% had total cholesterol <200 mg/dL.
— Prevalence of obesity was 44%, and prevalence of overweight or obesity was 71%.
— Prevalence of DM was 17%; 85% were aware of their DM, and 82% were treated.
— As measured by objective accelerometer data, 74% of adults had intermediate or poor levels of PA; 66% had no moderate or vigorous activity at all.
— 70% of US adults without CVD met 0 or only 1 of the 5 healthy dietary metrics.

Prognosis of Ideal Cardiovascular Health

- Folsom et al[2] recently published the first examination of the community prevalence of ideal cardiovascular health and its association with incident CVD events in 12 744 white and African American participants of the ARIC study aged 45 to 64 years at baseline who were followed up for up to 20 years.

— Overall, only 0.1% of participants, and fewer African Americans than whites, had all 7 metrics at ideal levels, consistent with national data.
— There was a stepwise decrease in the 20-year incidence of CVD events (defined as stroke, HF, MI, or fatal coronary disease) with greater numbers of health metrics at ideal levels. Age-, sex-, and race-adjusted CVD incidence rates per 1000 person-years were 32.1, 21.9, 16.0, 12.0, 8.6, 6.4, 3.9, and 0, respectively, for participants with 0, 1, 2, 3, 4, 5, 6, and 7 metrics at ideal levels.
— The corresponding age-, sex-, and race-adjusted hazard ratios (HRs) for incident CVD were 1.0 (reference), 0.65, 0.46, 0.34, 0.24, 0.18, 0.11, and 0 with increasing numbers of ideal health metrics. Thus, 20-year CVD incidence rates for those with 6 ideal health metrics were one-tenth those of participants with 0 ideal health metrics.
— The pattern of outcomes across number of ideal health metrics was similar for African-Americans and whites.
— Importantly, both ideal health behaviors and ideal health factors were associated in a stepwise fashion with lower CVD risk (Chart 2-9).

Implications

- Taken together, these data continue to indicate the substantial progress that will need to occur for the AHA to achieve its 2020 Impact Goals over the next decade. If the goals can be met, there is evidence suggesting that CVD event rates could decrease significantly.
  — To achieve improvements in cardiovascular health, all segments of the population will need to focus on improved cardiovascular health behaviors, in particular with regard to diet and weight, as well as on an increase in PA and further reduction of the prevalence of smoking.
  — More children, adolescents, and young adults will need to learn how to preserve their ideal levels of cardiovascular health factors and health behaviors into older ages.
  — With regard to reducing the burden of CVD and stroke morbidity and mortality, renewed emphasis will be needed on treatment of acute events and secondary and primary prevention through treatment and control of risk factors.
- As shown in Table 2-4, relatively modest changes in population levels of health factors could result in important changes in the prevalence of overall and ideal cardiovascular health. For example, NHANES 2007–2008 data indicate that the current prevalence of ideal levels of BP among US adults is 43.8%. A 20% relative improvement by 2020 would mean the prevalence of ideal BP would need to increase to 52.6%. NHANES data indicate that a reduction in the population mean BP by just 2 mm Hg would result in 55.5% of US adults having ideal levels of BP. Further reductions in BP would mean even more people would achieve ideal levels. Such modest reductions could result from decreased salt intake at the population level of as little as 1 to 2 g per day, with significant projected decreases in CVD rates in US adults.[3]
- Future issues of the Statistical Update will track progress toward the 2020 Strategic Impact Goals.

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Table 2-1. Definitions of Poor, Intermediate, and Ideal Cardiovascular Health for Each Metric, in the AHA 2020 Goals

	Level of Cardiovascular Health for Each Metric		
	Poor	Intermediate	Ideal
Current smoking			
Adults ≥20 y of age	Yes	Former ≤12 mo	Never or quit >12 mo
Children 12–19 y of age	Tried in prior 30 d	. . .	Never tried; never smoked whole cigarette
BMI			
Adults ≥20 y of age	≥30 kg/m^2	25–29.9 kg/m^2	<25 kg/m^2
Children 2–19 y of age	>95th percentile	85th–95th percentile	<85th percentile
Physical activity			
Adults ≥20 y of age	None	1–149 min/wk moderate or 1–74 min/wk vigorous or 1–149 min/wk moderate+2×vigorous	≥150 min/wk moderate or ≥75 min/wk vigorous or ≥150 min/wk moderate+2×vigorous
Children 12–19 y of age	None	>0 and <60 min of moderate or vigorous every day	≥60 min of moderate or vigorous every day
Healthy Diet Score, no. of components			
Adults ≥20 y of age	0–1	2–3	4–5
Children 5–19 y of age	0–1	2–3	4–5
Total cholesterol, mg/dL			
Adults ≥20 y of age	≥240	200–239 or treated to goal	<200
Children 6–19 y of age	≥200	170–199	<170
Blood pressure			
Adults ≥20 y of age	SBP ≥140 or DBP ≥90 mm Hg	SBP 120–139 or DBP 80–89 mm Hg or treated to goal	<120/<80 mm Hg
Children 8–19 y of age	>95th percentile	90th–95th percentile or SBP ≥120 or DBP ≥80 mm Hg	<90th percentile
Fasting plasma glucose, mg/dL			
Adults ≥20 y of age	≥126	100–125 or treated to goal	<100
Children 12–19 y of age	≥126	100–125	<100

AHA indicates American Heart Association; . . ., no definition for this stratum; BMI, body mass index; SBP, systolic blood pressure; and DBP, diastolic blood pressure.

Table 2-2. Prevalence (%) of US Population With Ideal Cardiovascular Health and With Components of Ideal Cardiovascular Health, Overall and in Selected Age Strata From NHANES 2007–2008 (Available Data as of June 1, 2011)

	Ages 12–19 y	Ages ≥20 y*	Ages 20–39 y	Ages 40–59 y	Ages ≥60 y
Ideal CV health profile (composite–all 7)	0.0	0.0	0.0	0.0	0.0
≥6 Ideal CV health components	9.1	3.8	7.2	2.1	0.1
≥5 Ideal CV health components	41.2	16.2	29.4	9.7	2.5
Ideal CV health factors (composite–all 4)	37.9	14.4	27.5	7.3	1.0
Individual components					
Total cholesterol <200 mg/dL (untreated)	75.1	46.8	64.0	37.1	28.4
SBP <120 mm Hg and DBP <80 mm Hg (untreated)	82.3	43.8	63.8	36.9	14.6
Not current smoker (never or quit ≥12 mo)	83.7	72.9	66.4	72.9	86.1
Fasting blood glucose <100 mg/dL	76.2	52.0	67.4	45.6	31.9
Ideal health behaviors (composite–all 4)	0.0	0.1	0.1	0.0	0.0
Individual components					
Physical activity at goal	39.0	39.5	45.6	36.4	33.7
Not current smoker (never or quit ≥12 mo)	83.7	72.9	66.4	72.9	86.1
BMI <25 kg/m^2	62.5	31.9	39.1	28.0	25.3
4–5 Diet goals met†	0.0	0.3	0.3	0.1	0.5
Fruits and vegetables ≥4.5 cups/d	7.9	12.3	11.7	11.4	15.8
Fish ≥2 3.5-oz servings/wk (preferably oily fish)	9.2	18.3	16.8	19.7	19.4
Sodium <1500 mg/d	0.0	0.6	0.6	0.8	0.3
Sugar-sweetened beverages ≤450 kcal/wk	32.0	51.9	41.0	54.6	71.2
Whole grains (1.1 g fiber/10 g carbohydrates) ≥3 1-oz equivalents/d	3.2	7.3	7.0	7.1	8.4
Other dietary measures					
Nuts, legumes, seeds ≥4 servings/wk	8.7	21.7	19.6	22.5	24.7
Processed meats ≤2 servings/wk	56.3	57.6	54.0	59.7	61.1
Saturated fat <7% of total energy intake (kcal)	4.5	8.7	9.3	8.0	9.0

NHANES indicates National Health and Nutrition Examination Survey; CV, cardiovascular; SBP, systolic blood pressure; DBP, diastolic blood pressure; and BMI, body mass index.

*Standardized to the age distribution of the 2000 US standard population.

†Scaled for 2000 kcal/d and in the context of intake with appropriate energy balance and a DASH (Dietary Approaches to Stop Hypertension)–like eating plan.

Table 2–3. Selected Secondary Metrics for Monitoring Cardiovascular Disease, NHANES 2007–2008

	In the Presence of CVD			In the Absence of CVD		
	N*	%†	(SE)	N*	%†	(SE)
Risk factor control						
Smoking	13 775 054			187 189 147		
Current smoker or smokers who quit <12 mo ago	3 482 092	40.64	(5.20)	44 333 396	23.35	(1.42)
BP	13 042 362			178 481 116		
Prevalence of BP ≥140/90 mm Hg or taking medications	8 790 237	44.58	(3.71)	47 737 172	27.18	(0.63)
Awareness among those with hypertension	8 277 582	95.80	(1.50)	36 832 906	70.63	(3.84)
Treatment among those with hypertension	7 739 839	88.72	(2.48)	32 685 394	57.25	(2.14)
BP control to <140/<90 mm Hg among treated	4 731 044	62.03	(7.97)	23 440 265	76.97	(2.66)
Cholesterol	12 935 387			177 322 590		
Prevalence of total cholesterol ≥240 mg/dL or taking medications	6 847 388	34.83	(3.57)	45 453 440	25.44	(1.08)
Awareness among those with hypercholesterolemia	6 218 269	83.43	(6.43)	33 326 995	62.57	(2.36)
Treatment among those with hypercholesterolemia	5 722 826	76.39	(6.01)	22 922 768	38.46	(2.54)
Cholesterol control to <200 mg/dL among treated	5 110 272	84.61	(7.08)	19 890 862	80.73	(5.43)
Weight	13 232 271			185 443 123		
Overweight or obese BMI ≥25.0 kg/m^2	10 401 572	70.65	(4.93)	125 175 950	67.69	(0.97)
Obese BMI ≥30.0 kg/m^2	6 221 362	43.73	(6.12)	61 956 664	33.42	(1.11)
Diabetes mellitus	14 292 850			188 058 669		
Prevalence of fasting glucose ≥125 mg/dL or taking medications	5 174 893	17.44	(3.71)	16 987 130	9.26	(0.60)
Awareness among diabetics	3 909 379	84.51	(5.78)	12 446 506	64.28	(4.56)
Treatment among diabetics	3 798 559	82.03	(5.69)	12 028 826	62.54	(4.46)
Blood glucose control among treated	1 460 295	‡		4 026 301	23.44	(3.74)
Physical activity	13 775 054			187 296 417		
Physical activity: intermediate or poor§	9 914 277	74.10	(4.77)	111 901 937	59.93	(2.40)
Physical activity: none	9 045 113	65.70	(5.86)	87 091 042	46.70	(2.70)
Diet	12 665 860			161 854 617		
Total diet score 0–3 of 5	12 665 860	100.00	(0.00)	161 370 154	99.71	(0.11)
Total diet score 0–1 of 5	9 540 532	70.06	(4.69)	127 156 293	78.84	(1.42)

NHANES indicates National Health and Nutrition Examination Survey; CVD, cardiovascular disease; SE, standard error; BP, blood pressure; and BMI, body mass index.

*Weighted sample size.

†Standardized to the age distribution of the 2000 US Standard population.

‡Estimate suppressed because of instability by National Center for Health Statistics standards (relative SE >30%).

§Moderate <150 min/wk AND Vigorous <75 min/wk AND Combined <150 min/wk.

Table 2-4. Reduction in BP Required to Increase Prevalence of Ideal BP Among Adults ≥20 Years of Age, NHANES 2007–2008

Percent BP ideal among adults, 2007–2008	43.82
20% Relative increase	52.58
Percent who would have ideal BP if population mean BP were lowered by*	
2 mm Hg	55.47
3 mm Hg	59.79
4 mm Hg	61.48
5 mm Hg	65.49

NHANES indicates National Health and Nutrition Examination Survey; BP, blood pressure.

*Reduction in BP=observed average systolic BP−X mm Hg AND observed average diastolic−X mm Hg.

Standardized to the age distribution of the 2000 US standard population.

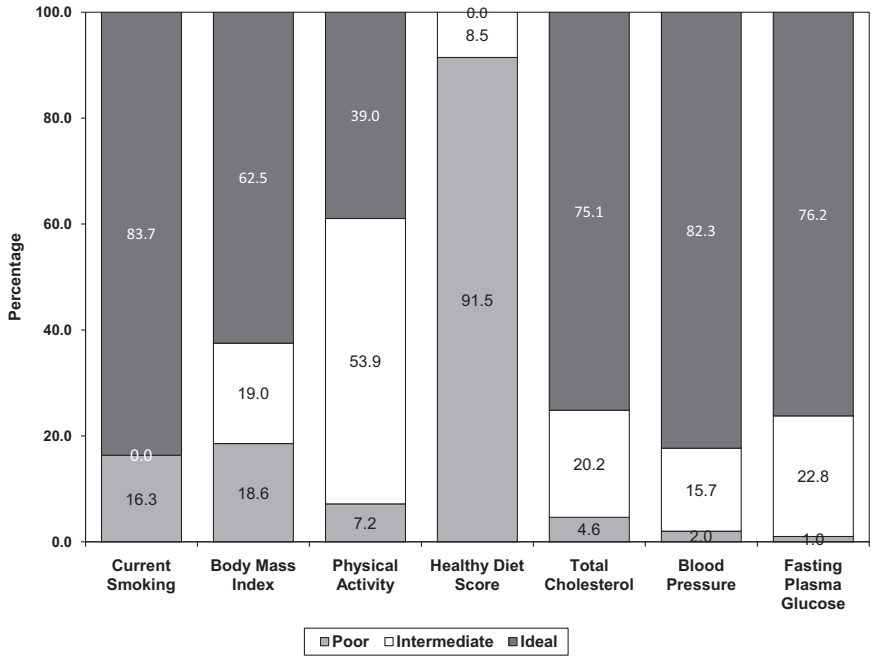

Chart 2-1. Prevalence (unadjusted) estimates for poor, intermediate, and ideal cardiovascular health for each of the 7 metrics of cardiovascular health in the American Heart Association 2020 goals, US children aged 12 to 19 years, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2011).

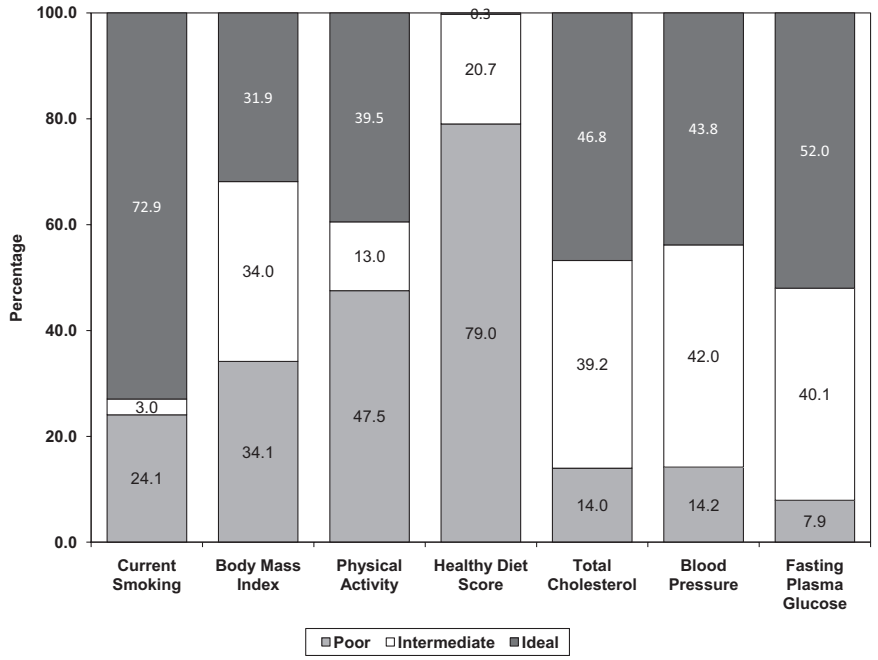

Chart 2-2. Age-standardized prevalence estimates for poor, intermediate, and ideal cardiovascular health for each of the 7 metrics of cardiovascular health in the American Heart Association 2020 goals, among US adults aged ≥20 years, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2011).

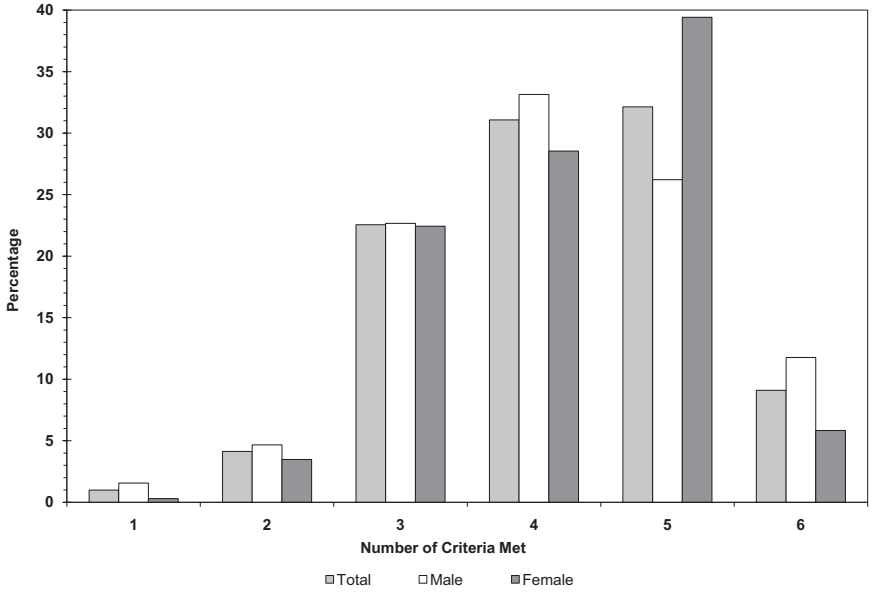

Chart 2-3. Proportion (unadjusted) of US children meeting different numbers of criteria for ideal cardiovascular health, overall and by sex, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2011). No children meet all 7 criteria.

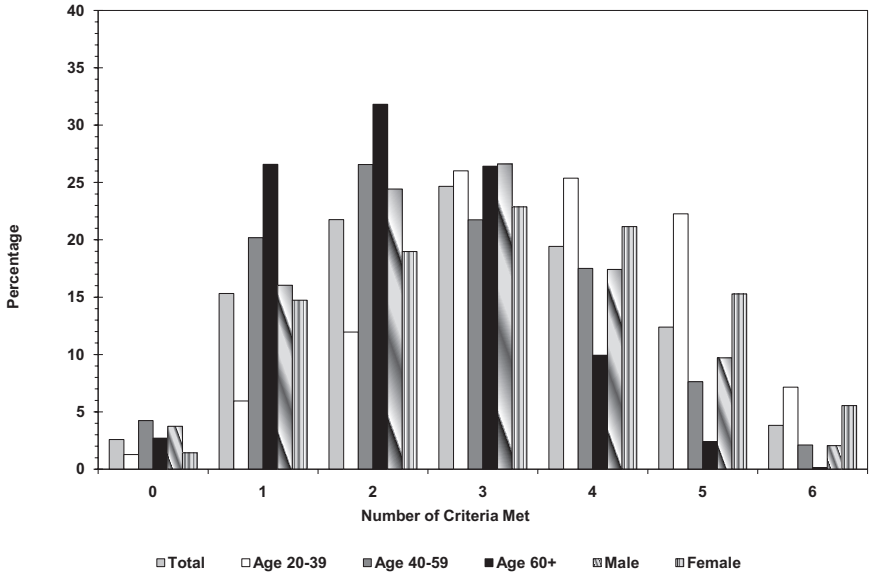

Chart 2-4. Age-standardized prevalence estimates of US adults meeting different numbers of criteria for ideal cardiovascular health, overall and by age and sex subgroups, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2011). No adults meet all 7 criteria.

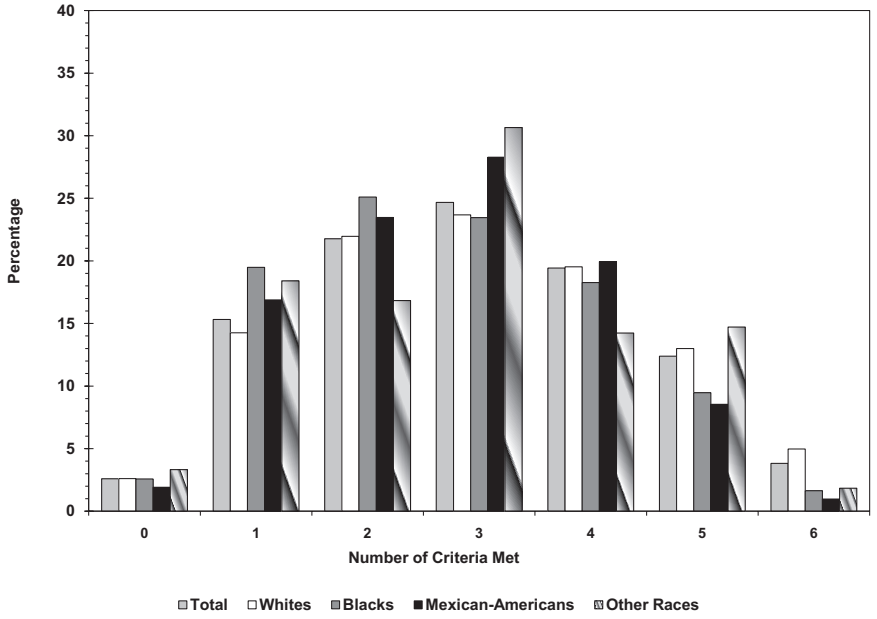

Chart 2-5. Age-standardized prevalence estimates of US adults meeting different numbers of criteria for ideal cardiovascular health, overall and in selected race subgroups from National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2011). No adults meet all 7 criteria.

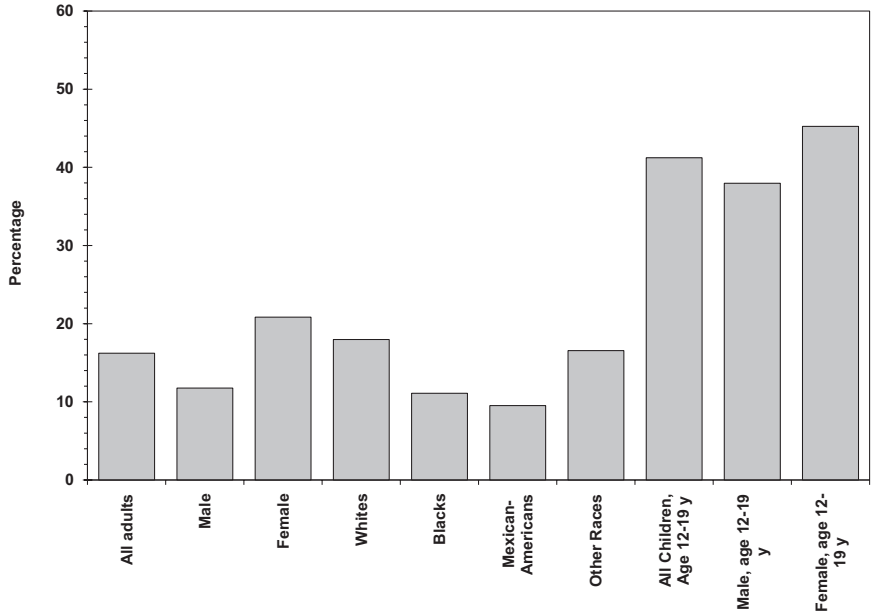

Chart 2-6. Prevalence estimates of meeting at least 5 criteria for ideal cardiovascular health, US adults (age-standardized), overall and by sex and race, and US children (unadjusted), by sex, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2011). No adults meet all 7 criteria.

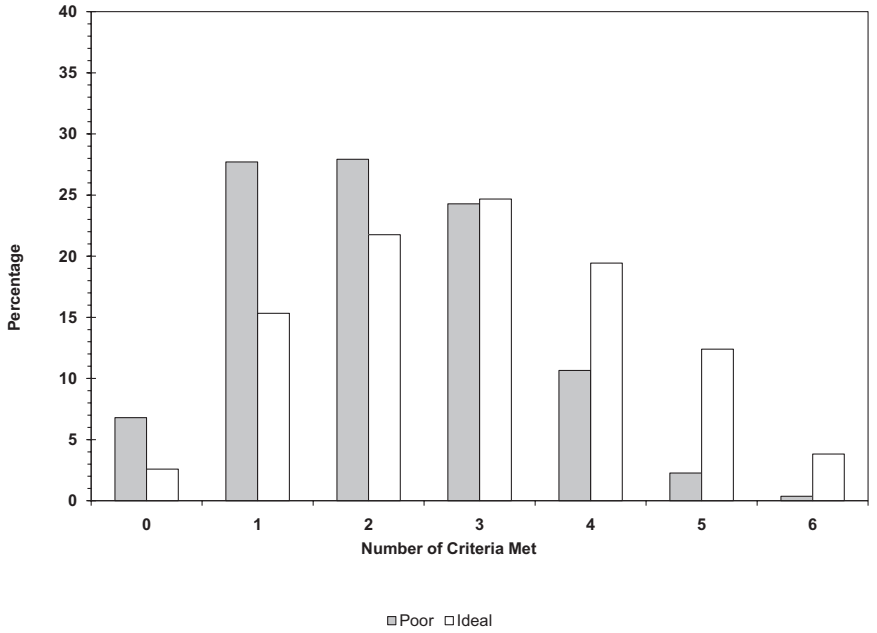

Chart 2-7. Age-standardized prevalence estimates of US adults meeting different numbers of cardiovascular health criteria for ideal and poor cardiovascular health, among US adults aged ≥20 years, National Health and Nutrition Examination Survey (NHANES) 2007-2008 (available data as of June 1, 2011).

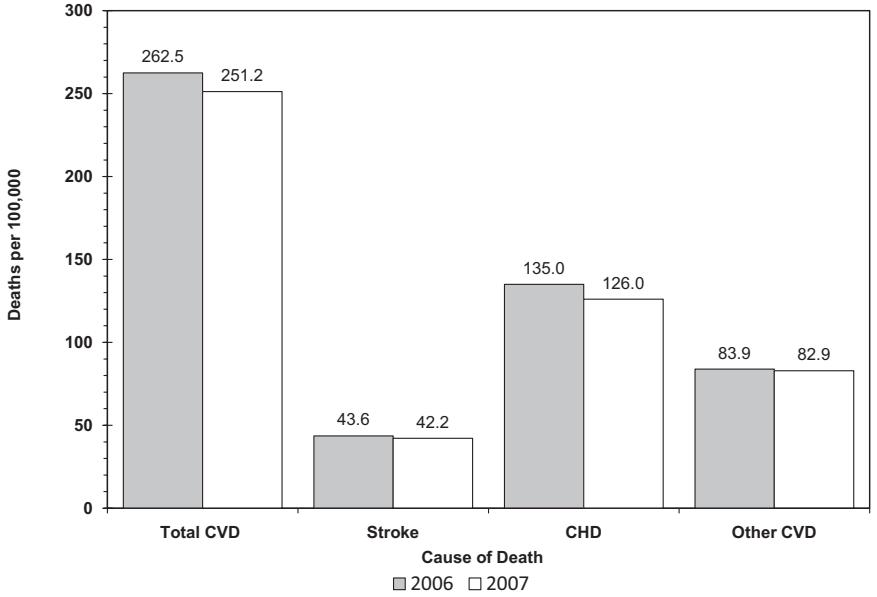

Chart 2-8. US age-standardized death rates attributable to cardiovascular diseases, 2006 and 2007. CVD indicates cardiovascular disease; CHD, coronary heart disease. Total CVD, *International Classification of Diseases, 10th Revision* (ICD-10) I00–I99; stroke, ICD-10 I60–I69; CHD, ICD-10 I20–I25; other CVD, ICD-10 I00–I15, I26–I51, I70–I78, I80–I89, I95–I99. Data derived from Heron et al[4] and Xu et al.[5]

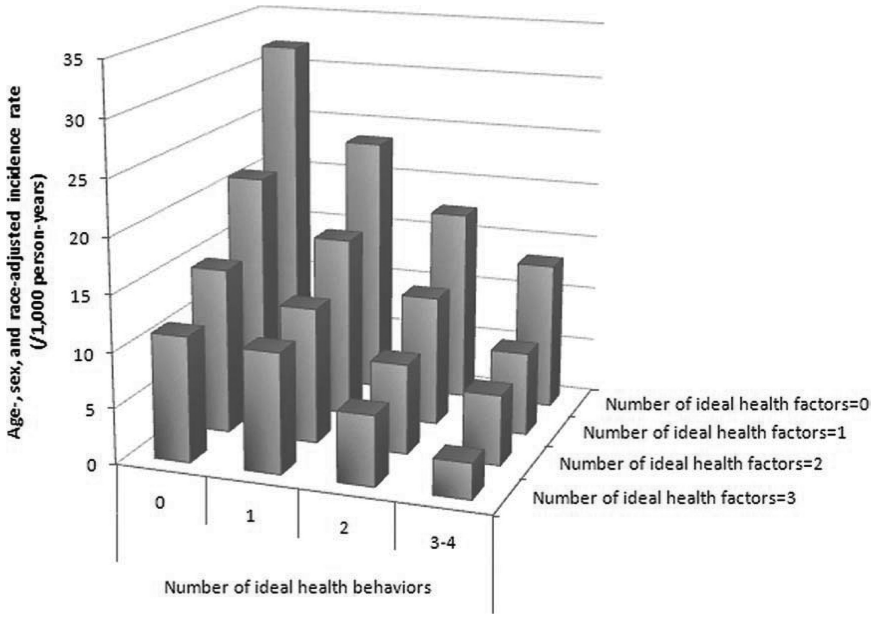

Chart 2-9. Incidence of cardiovascular disease according to the number of ideal health behaviors and health factors. Reprinted from Folsom et al[2] with permission of the publisher. Copyright © 2011, American College of Cardiology Foundation.

3. Cardiovascular Diseases

ICD-9 390 to 459, 745 to 747, ICD-10 I00 to I99, Q20 to Q28; see Glossary (Chapter 25) for details and definitions. See Tables 3-1 through 3-4 and Charts 3-1 through 3-21.

Abbreviations Used in Chapter 3

AHA	American Heart Association
AIDS	acquired immune deficiency syndrome
AMI	acute myocardial infarction
AP	angina pectoris
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CABG	cardiac revascularization (coronary artery bypass graft)
CAD	coronary artery disease
CARDIA	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHF	congestive heart failure
CHS	Cardiovascular Health Study
CLRD	chronic lower respiratory disease
CVD	cardiovascular disease
DM	diabetes mellitus
ED	emergency department
FHS	Framingham Heart Study
HBP	high blood pressure
HD	heart disease
HF	heart failure
HIV	human immunodeficiency virus
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
LDL	low-density lipoprotein
MEPS	Medical Expenditure Panel Survey
MESA	Multi-Ethnic Study of Atherosclerosis
MI	myocardial infarction
MRFIT	Multiple Risk Factor Intervention Trial
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHES	National Health Examination Survey
NHIS	National Health Interview Survey
NHHCS	National Home and Hospice Care Survey
NHLBI	National Heart, Lung, and Blood Institute
NIS	National Inpatient Sample
NNHS	National Nursing Home Survey
PA	physical activity
PCI	percutaneous coronary intervention
RR	relative risk
SBP	systolic blood pressure
UA	unstable angina

Prevalence

An estimated 82 600 000 American adults (>1 in 3) have 1 or more types of CVD. Of these, 40 400 000 are estimated to be ≥60 years of age. Total CVD includes diseases listed in the bullet points below, with the exception of congenital CVD. Because of overlap across conditions, it is not possible to add these conditions to arrive at a total.

- High BP (HBP)—6 400 000 (defined as systolic pressure ≥140 mm Hg and/or diastolic pressure ≥90 mm Hg, use of antihypertensive medication, or being told at least twice by a physician or other health professional that one has HBP).
- Coronary heart disease (CHD)—16 300 000
  - MI (heart attack)—7 900 000
  - AP (chest pain)—9 000 000
  - HF—5 700 000
  - Stroke (all types)—7 000 000
  - Congenital cardiovascular defects—650 000 to 1 300 000
- The following age-adjusted prevalence estimates from the NHIS, NCHS are for diagnosed conditions for people ≥18 years of age in 2010[1]:
  - Among whites only, 11.7% have HD, 6.4% have CHD, 23.6% have hypertension, and 2.5% have had a stroke.
  - Among blacks or African Americans, 10.9% have HD, 6.3% have CHD, 33.8% have hypertension, and 3.9% have had a stroke.
  - Among Hispanics or Latinos, 8.1% have HD, 5.2% have CHD, 22.5% have hypertension, and 2.6% have had a stroke.
  - Among Asians, 7.2% have HD, 4.9% have CHD, 20.5% have hypertension, and 2.0% have had a stroke.
  - Among American Indians or Alaska Natives, 12.5% have HD, 5.9% have CHD, 30.0% have hypertension, and 5.9% have had a stroke (estimate considered unreliable). Among Native Hawaiians or other Pacific Islanders, 20.2% have HD, 19.7% have CHD, 40.8% have hypertension, and 10.6% have had a stroke.
- Asian Indian adults (9%) are ≈2-fold more likely than Korean adults (4%) to have ever been told they have HD, based on data for 2004 to 2006.[2]
- By 2030, 40.5% of the US population is projected to have some form of CVD.[3]

Incidence

- On the basis of the NHLBI's FHS original and offspring cohort data from 1980 to 2003[4]:
  - The average annual rates of first cardiovascular events rise from 3 per 1000 men at 35 to 44 years of age to 74 per 1000 men at 85 to 94 years of age. For women, comparable rates occur 10 years later in life. The gap narrows with advancing age.
  - Before 75 years of age, a higher proportion of CVD events attributable to CHD occur in men than in women, and a higher proportion of events attributable to stroke occur in women than in men.

- Among American Indian men 45 to 74 years of age, the incidence of CVD ranges from 15 to 28 per 1000 population. Among women, it ranges from 9 to 15 per 1000.[5]
- Data from the FHS indicate that the subsequent lifetime risk for all CVD in recipients starting free of known disease is 2 in 3 for men and >1 in 2 for women at 40 years of age (personal communication, Donald Lloyd-Jones, MD, Northwestern University, Chicago, IL; Table 3-4).
- Analysis of FHS data among participants free of CVD at 50 years of age showed the lifetime risk for developing CVD was 51.7% for men and 39.2% for women. Median overall survival was 30 years for men and 36 years for women.[6]

Mortality

ICD-10 I00 to I99, Q20 to Q28 for CVD (CVD mortality includes congenital cardiovascular defects); C00 to C97 for cancer; C33 to C34 for lung cancer; C50 for breast cancer; J40 to J47 for chronic lower respiratory disease (CLRD); G30 for Alzheimer disease; E10 to E14 for DM; and V01 to X59, Y85 to Y86 for accidents.

- Mortality data show that CVD (I00–I99, Q20–Q28) as the listed underlying cause of death (including congenital cardiovascular defects) accounted for 32.8% (811 940) of all 2 471 984 deaths in 2008, or 1 of every 3 deaths in the United States. CVD any-mentions (1 354 527 deaths in 2008) constituted 55.0% of all deaths that year (NHLBI; NCHS public-use data files).[7]
- In every year since 1900 except 1918, CVD accounted for more deaths than any other major cause of death in the United States.[8,9]
- On average, >2200 Americans die of CVD each day, an average of 1 death every 39 seconds. CVD currently claims more lives each year than cancer, CLRD, and accidents combined.[7]
- The 2008 death rate attributable to CVD (I00–I99) was 244.8 (excluding congenital cardiovascular defects) (NCHS).[7] The rates were 287.2 for white males, 390.4 for black males, 200.5 for white females, and 277.4 for black females. From 1998 to 2008, death rates attributable to CVD (ICD-10 I00–I99) declined 30.6%. In the same 10-year period, the actual number of CVD deaths per year declined 14.1% (NHLBI tabulation).[7] (Appropriate comparability ratios were applied.)
- Among other causes of death in 2008, cancer caused 565 469 deaths; CLRD, 141 090; accidents, 121 902; and Alzheimer disease, 82 435.[7]
- The 2008 CVD (I00–I99) death rates were 292.6 for males and 206.1 for females. There were 40 589 deaths due to breast cancer in females in 2008; lung cancer claimed 70 070 in females. Death rates for females were 22.5 for breast cancer and 39.0 for lung cancer. One in 31 deaths in females was attributable to breast cancer, whereas 1 in 6.6 was attributable to CHD. For comparison, 1 in 4.6 females died of cancer, whereas 1 in 3.0 died of CVD (I00–I99, Q20–Q28). On the basis of 2008 mortality data, CVD caused ≈1 death per minute among females, or 419 730 deaths in females in 2008. That represents more female lives than were claimed by cancer, CLRD, and Alzheimer disease combined (unpublished NHLBI tabulation).[7]
- About 150 000 Americans died of CVD (I00–I99) in 2008 who were <65 years of age, and 33% of deaths attributed to CVD occurred before the age of 75 years, which is well below the average life expectancy of 77.9 years.[7]
- According to the NCHS, if all forms of major CVD were eliminated, life expectancy could rise by almost 7 years. If all forms of cancer were eliminated, the estimated gain could be 3 years. According to the same study, the probability at birth of eventually dying of major CVD (I00–I78) is 47%, and the chance of dying of cancer is 22%. Additional probabilities are 3% for accidents, 2% for DM (unrelated to CVD), and 0.7% for HIV.[10]
- In 2008, the leading causes of death in women ≥65 years of age were diseases of the heart (No. 1), cancer (No. 2), stroke (No. 3), and CLRD (No. 4). In older men, they were diseases of the heart (No. 1), cancer (No. 2), CLRD (No. 3), and stroke (No. 4).[7]
- A study of the decrease in US deaths attributable to CHD from 1980 to 2000 suggests that ≈47% of the decrease was attributable to increased use of evidence-based medical therapies and 44% to changes in risk factors in the population attributable to lifestyle and environmental changes.[11]
- Analysis of data from NCHS was used to determine the number of disease-specific deaths attributable to all nonoptimal levels of each risk factor exposure, by age and sex. In 2005, tobacco smoking and HBP were estimated to be responsible for 467 000 deaths, accounting for ≈1 in 5 or 6 deaths among US adults. Overweight/obesity and physical inactivity were each estimated to be responsible for nearly 1 in 10 deaths. High dietary salt, low dietary omega-3 fatty acids, and high dietary trans fatty acids were the dietary risks with the largest estimated excess mortality effects.[12]

Aftermath

- Among an estimated 45 million people with functional disabilities in the United States, HD, stroke, and hypertension are among the 15 leading conditions that caused those disabilities. Disabilities were defined as difficulty with activities of daily living or instrumental activities of daily living, specific functional limitations (except vision, hearing, or speech), and limitation in ability to do housework or work at a job or business.[13]

Awareness of Warning Signs and Risk Factors for CVD

- Surveys conducted by the AHA in 1997, 2000, 2003, and 2006 to evaluate trends in women's awareness, knowledge, and perceptions related to CVD found that in 2006, awareness of HD as the leading cause of death among women was 57%, significantly higher than in prior surveys. Awareness was lower among black and Hispanic women than among white women, and the racial/ethnic difference has not changed appreciably over time. In 2006, more than

twice as many women felt uninformed about stroke compared with HD. Hispanic women were more likely than white women to report that there is nothing they can do to keep themselves from getting CVD. The majority of respondents reported confusion related to basic CVD prevention strategies.[14]

- A nationally representative sample of women responded to a questionnaire about history of CVD risk factors, self-reported actions taken to reduce risk, and barriers to heart health. According to the study, published in 2006, the rate of awareness of CVD as the leading cause of death had nearly doubled since 1997, was significantly greater for whites than for blacks and Hispanics, and was independently correlated with increased PA and weight loss in the previous year. Fewer than half of the respondents were aware of healthy levels of risk factors. Awareness that their personal level was not healthy was positively associated with preventive action. Most women took steps to lower risk in family members and themselves.[15]
- A total of 875 students in 4 Michigan high schools were given a survey to obtain data on the perception of risk factors and other knowledge-based assessment questions about CVD. Accidents were rated as the greatest perceived lifetime health risk (39%). Nearly 17% selected CVD as the greatest lifetime risk, which made it the third most popular choice after accidents and cancer. When asked to identify the greatest cause of death for each sex, 42% correctly recognized CVD for men, and 14% correctly recognized CVD for women; 40% incorrectly chose abuse/use behavior with a substance other than cigarettes as the most important CVD risk behavior.[16]

Awareness of Cardiopulmonary Resuscitation

- Seventy-nine percent of the lay public are confident that they know what actions to take in a medical emergency; 98% recognize an automated external defibrillator as something that administers an electric shock to restore a normal heart beat among victims of sudden cardiac arrest; and 60% are familiar with cardiopulmonary resuscitation (Harris Interactive survey conducted on behalf of the AHA among 1132 US residents ≥18 years of age, January 8, 2008, through January 21, 2008).

Risk Factors

- Data from the 2003 CDC BRFSS survey of adults ≥18 years of age showed the prevalence of respondents who reported having ≥2 risk factors for HD and stroke was successively higher at higher age groups. The prevalence of having ≥2 risk factors was highest among blacks (48.7%) and American Indian/Alaska Natives (46.7%) and lowest among Asians (25.9%); prevalence was similar in women (36.4%) and men (37.8%). The prevalence of multiple risk factors ranged from 25.9% among college graduates to 52.5% among those with less than a high school diploma (or its equivalent). People reporting household income of ≥$50 000 had the lowest prevalence (28.8%), and those reporting household income of <$10 000 had the highest prevalence (52.5%). Adults who reported being unable to work had the highest prevalence (69.3%) of ≥2 risk factors, followed by retired people (45.1%), unemployed adults (43.4%), homemakers (34.3%), and employed people (34.0%). Prevalence of ≥2 risk factors varied by state/territory and ranged from 27.0% (Hawaii) to 46.2% (Kentucky). Twelve states and 2 territories had a multiple risk factor prevalence of ≥40%: Alabama, Arkansas, Georgia, Indiana, Kentucky, Louisiana, Mississippi, North Carolina, Ohio, Oklahoma, Tennessee, West Virginia, Guam, and Puerto Rico.[17]
- Data from the Chicago Heart Association Detection Project (1967–1973, with an average follow-up of 31 years) showed that in younger women (18–39 years of age) with favorable levels for all 5 major risk factors (BP, serum cholesterol, body mass index [BMI], DM, and smoking), future incidence of CHD and CVD is rare, and long-term and all-cause mortality are much lower than for those who have unfavorable or elevated risk factor levels at young ages. Similar findings applied to men in this study.[18,19]
- Analysis of several data sets by the CDC showed that in adults ≥18 years of age, disparities were common in all risk factors examined. In men, the highest prevalence of obesity (29.7%) was found in Mexican Americans who had completed a high school education. Black women with or without a high school education had a high prevalence of obesity (48.4%). Hypertension prevalence was high among blacks (41.2%) regardless of sex or educational status. Hypercholesterolemia was high among white and Mexican American men and white women regardless of educational status. CHD and stroke were inversely related to education, income, and poverty status. Hospitalization for total HD and acute MI (AMI) was greater among men, but hospitalization for congestive heart failure (CHF) and stroke was greater among women. Among Medicare enrollees, CHF hospitalization was higher among blacks, Hispanics, and American Indian/Alaska Natives than among whites, and stroke hospitalization was highest among blacks. Hospitalizations for CHF and stroke were highest in the southeastern United States. Life expectancy remains higher in women than in men and in whites than in blacks by ≈5 years. CVD mortality at all ages tended to be highest in blacks.[20]
- Analysis of 5 cross-sectional, nationally representative surveys from the National Health Examination Survey (NHES) 1960 to 1962 to the NHANES 1999 to 2000 showed that the prevalence of key risk factors (ie, high cholesterol, HBP, current smoking, and total DM) decreased over time across all BMI groups, with the greatest reductions observed among overweight and obese groups. Total DM prevalence

was stable within BMI groups over time; however, the trend has leveled off or been reversed for some of the risk factors in more recent years.[21]

- Data from BRFSS 2006 to 2008 demonstrated that during this 3-year period, 25.6% of non-Hispanic blacks, non-Hispanic whites, and Hispanics were obese, but prevalent obesity varied across groups: 35.7% for non-Hispanic blacks, 28.7% for Hispanics, and 23.7% for non-Hispanic whites.
- Data from NHANES 2005 to 2006 showed that 90.4% of US adults exceeded their recommended target limit of daily dietary sodium intake.[22]
- Analysis of >14 000 middle-aged subjects in the ARIC study sponsored by the NHLBI showed that >90% of CVD events in black subjects, compared with ≈70% in white subjects, appeared to be explained by elevated or borderline risk factors. Furthermore, the prevalence of participants with elevated risk factors was higher in black subjects; after accounting for education and known CVD risk factors, the incidence of CVD was identical in black and white subjects. Thus, the observed higher CVD incidence rate in black subjects appears to be largely attributable to a greater prevalence of elevated risk factors. These results suggest that the primary prevention of elevated risk factors might substantially impact the future incidence of CVD, and these beneficial effects would likely be applicable not only for white but also for black subjects.[23]
- Data from the MEPS 2004 Full-Year Data File showed that nearly 26 million US adults ≥18 years of age were told by a doctor that they had HD, stroke, or any other heart-related disease[24]:
  - 38.6% maintained a healthy weight. Among those told that they had HD, 33.9% had a healthy weight compared with 39.3% who had never been told they had HD.
  - 78.8% did not currently smoke. Among those ever told that they had indicators of HD, 18.3% continued to smoke.
  - More than 93% engaged in at least 1 recommended behavior for prevention of HD: 75.5% engaged in 1 or 2; 18% engaged in all 3; and 6.5% did not engage in any of the recommended behaviors.
  - Age-based variations:
    - Moderate to vigorous PA ≥3 times per week varied according to age. Younger people (18–44 years of age) were more likely (59.9%) than those who were older (45–64 and ≥65 years of age, 55.3% and 48.5%, respectively) to engage in regular PA.
    - A greater percentage of those 18 to 44 years of age had a healthy weight (43.7%) than did those 45 to 64 years of age and ≥65 years of age (31.4% and 37.3%, respectively).
    - People ≥65 years of age were more likely to be current nonsmokers (89.7%) than were people 18 to 44 years of age and 45 to 64 years of age (76.1% and 77.7%, respectively).
  - Race/ethnicity-based variations:
    - Non-Hispanic whites were more likely than Hispanics or non-Hispanic blacks to engage in moderate-to-vigorous PA (58.5% versus 51.4% and 52.5%, respectively).
    - Non-Hispanic whites were more likely to have maintained a healthy weight than were Hispanics or non-Hispanic blacks (39.8% versus 32.1% and 29.7%, respectively).
    - Hispanics were more likely to be nonsmokers (84.2%) than were non-Hispanic whites and non-Hispanic blacks (77.8% and 76.3%, respectively).
  - Sex-based variations:
    - Men were more likely to have engaged in moderate-to-vigorous PA ≥3 times per week than women (60.3% versus 53.1%, respectively).
    - Women were more likely than men to have maintained a healthy weight (45.1% versus 31.7%, respectively).
    - 81.7% of women did not currently smoke, compared with 75.7% of men.
  - Variations based on education level:
    - A greater percentage of adults with at least some college education engaged in moderate-to-vigorous PA ≥3 times per week (60.8%) than did those with a high school education or less than a high school education (55.3% and 48.3%, respectively).
    - A greater percentage of adults with at least some college education had a healthy weight (41.2%) than did those with a high school or less than high school education (36.2% and 36.1%, respectively).
    - There was a greater percentage of nonsmokers among those with a college education (85.5%) than among those with a high school or less than high school education (73.8% and 69.9%, respectively).
- Participants (18–64 years of age at baseline) in the Chicago Heart Association Detection Project in Industry without a history of MI were investigated to determine whether traditional CVD risk factors were similarly associated with CVD mortality in black and white men and women. In general, the magnitude and direction of associations were similar by race. Most traditional risk factors demonstrated similar associations with mortality in black and white adults of the

same sex. Small differences were primarily in the strength and not the direction of the association.[25]

- A study of nearly 1500 participants in the Multi-Ethnic Study of Atherosclerosis (MESA) study found that Hispanics with hypertension, hypercholesterolemia, and/or DM who speak Spanish at home and/or have spent less than half a year in the United States have higher systolic BP (SBP), low-density lipoprotein (LDL) cholesterol, and fasting blood glucose, respectively, than Hispanics who speak English and who have lived a longer period of time in the United States.[26]

Family History of CVD

- A family history of CVD increases risk of CVD, with the largest increase in risk if the family member's CVD was premature.[27]
- There is consistent evidence from multiple large-scale prospective epidemiology studies for a strong and significant association of a reported family history of premature parental CHD with incident MI or CHD in offspring. In the FHS, the occurrence of a validated premature atherosclerotic CVD event in either a parent[28] or a sibling[29] was associated with an ≈2-fold elevated risk for CVD, independent of other traditional risk factors.
- Addition of family history of premature CVD to a model that contained traditional risk factors provided modestly improved prognostic value in the FHS.[28] Family history of premature MI is also an independent risk factor in other multivariable risk models that contain traditional risk factors in large cohorts of women[30] and men.[31]
- Parental history of premature CHD is associated with increased burden of subclinical atherosclerosis in the coronary arteries and the abdominal aorta.[32,33]
- In the FHS, a parental history of validated HF is associated with a 1.7-fold higher risk of HF in offspring, after multivariable adjustment.[34]
- A family history of early-onset sudden cardiac death in a first-degree relative is associated with a >2-fold higher risk for sudden cardiac death in offspring on the basis of available case-control studies.[35]
- The 2004 HealthStyles survey of 4345 people in the United States indicated that most respondents believe that knowing their family history is important for their own health, but few are aware of the specific health information from relatives necessary to develop a family history.[36]
- An accurate and complete family history may identify rare mendelian conditions such as hypertrophic cardiomyopathy (HCM), long-QT syndrome, or familial hypercholesterolemia. However, in the majority of people with a family history of a CVD event, a known rare mendelian condition is not identified.
- Studies are under way to determine genetic variants that may help identify individuals at increased risk of CVD.

Impact of Healthy Lifestyle and Low Risk Factor Levels

Much of the literature on CVD has focused on factors associated with increasing risk for CVD and on factors associated with poorer outcomes in the presence of CVD; however, in recent years, a number of studies have defined the potential beneficial effects of healthy lifestyle factors and lower CVD risk factor burden on CVD outcomes and longevity. These studies suggest that prevention of risk factor development at younger ages may be the key to "successful aging," and they highlight the need for evaluating the potential benefits of intensive prevention efforts at younger and middle ages once risk factors develop to increase the likelihood of healthy longevity.

- The lifetime risk for CVD and median survival were highly associated with risk factor presence and burden at 50 years of age among >7900 men and women from the FHS followed up for 111 000 person-years. In this study, optimal risk factor burden at 50 years of age was defined as BP <120/80 mm Hg, total cholesterol <180 mg/dL, absence of DM, and absence of smoking. Elevated risk factors were defined as stage 1 hypertension or borderline high cholesterol (200–239 mg/dL). Major risk factors were defined as stage 2 hypertension, elevated cholesterol (≥240 mg/dL), current smoking, and DM. Remaining lifetime risks for atherosclerotic CVD events were only 5.2% in men and 8.2% in women with optimal risk factors at 50 years of age compared with 68.9% in men and 50.2% in women with ≥2 major risk factors at age 50. In addition, men and women with optimal risk factors had a median life expectancy ≥10 years longer than those with ≥2 major risk factors at age 50 years.[6]
- A recent study examined the association between low lifetime predicted risk for CVD (ie, having all optimal or near-optimal risk factor levels) and burden of subclinical atherosclerosis in younger adults in the Coronary Artery Risk Development in Young Adults (CARDIA) and MESA studies of the NHLBI. Among participants <50 years of age, nearly half had low and half had high predicted lifetime risks for CVD. Those with low predicted lifetime risk had lower prevalence and less severe amounts of coronary calcification and less carotid intima-media thickening, even at these younger ages, than those with high predicted lifetime risk. During follow-up, those with low predicted lifetime risk also had less progression of coronary calcium.[37]
- In another study, FHS investigators followed up 2531 men and women who were examined between the ages of 40 and 50 years and observed their overall rates of survival and survival free of CVD to 85 years of age and beyond. Low levels of the major risk factors in middle age were associated with overall survival and morbidity-free survival to ≥85 years of age.[38]
  - — Overall, 35.7% survived to the age of 85 years, and 22% survived to that age free of major morbidities.
  - — Factors associated with survival to the age of 85 years included female sex, lower SBP, lower total cholester-

ol, better glucose tolerance, absence of current smoking, and higher level of education attained. Factors associated with survival to the age of 85 years free of MI, unstable angina (UA), HF, stroke, dementia, and cancer were nearly identical.
  - When adverse levels of 4 of these factors were present in middle age, <5% of men and ≈15% of women survived to 85 years of age.

- A study of 366 000 men and women from the Multiple Risk Factor Intervention Trial (MRFIT) and Chicago Heart Association Detection Project in Industry defined low-risk status as follows: Serum cholesterol level <200 mg/dL, untreated BP 120/80 mm Hg, absence of current smoking, absence of DM, and absence of major electrocardiographic abnormalities. Compared with those who did not have low risk factor burden, those with low risk factor burden had between 73% and 85% lower relative risk (RR) for CVD mortality, 40% to 60% lower relative total mortality rates, and 6 to 10 years' longer life expectancy.[19]
- A study of 84 129 women enrolled in the Nurses' Health Study identified 5 healthy lifestyle factors, including absence of current smoking, drinking half a glass or more of wine per day (or equivalent alcohol consumption), half an hour or more per day of moderate or vigorous PA, BMI <25 kg/m^2, and dietary score in the top 40% (which included diets with lower amounts of trans fats, lower glycemic load, higher cereal fiber, higher marine omega-3 fatty acids, higher folate, and higher polyunsaturated-to-saturated fat ratio). When 3 of the 5 healthy lifestyle factors were present, the RR for CHD over a 14-year period was 57% lower; when 4 were present, RR was 66% lower; and when all 5 factors were present, RR was 83% lower.[39] However, data from NHANES 1999 to 2002 showed that only about one third of adults complied with ≥6 of the recommended heart-healthy behaviors. Dietary recommendations, in general, and daily fruit intake recommendations, in particular, were least likely to be followed.[40]
- In the Chicago Heart Association Detection Project in Industry, remaining lifetime risks for CVD death were noted to increase substantially and in a graded fashion according to the number of risk factors present in middle age (40–59 years of age). However, remaining lifetime risks for non-CVD death also increased dramatically with increasing CVD risk factor burden. These data help to explain the markedly greater longevity experienced by those who reach middle age free of major CVD risk factors.[41]
- Among individuals 70 to 90 years of age, adherence to a Mediterranean-style diet and greater PA are associated with 65% to 73% relatively lower rates of all-cause mortality, as well as lower mortality rates attributable to CHD, CVD, and cancer.[42]
- Seventeen-year mortality data from the NHANES II Mortality Follow-Up Study indicated that the RR for fatal CHD was 51% lower for men and 71% lower for women with none of 3 major risk factors (hypertension, current smoking, and elevated total cholesterol [≥ 240 mg/dL]) than for those with ≥1 risk factors. Had all 3 major risk factors not occurred, it is hypothesized that 64% of all CHD deaths among women and 45% of CHD deaths in men could theoretically have been avoided.[43]
- Investigators from the Chicago Heart Association Detection Project in Industry have also observed that risk factor burden in middle age is associated with better quality of life at follow-up in older age (≈25 years later) and lower average annual Medicare costs at older ages.
- The presence of a greater number of risk factors in middle age is associated with lower scores at older ages on assessment of social functioning, mental health, walking, and health perception in women, with similar findings in men.[44]
- Similarly, the existence of a greater number of risk factors in middle age is associated with higher average annual CVD-related and total Medicare costs (once Medicare eligibility is attained).[45]

Hospital Discharges, Ambulatory Care Visits, and Nursing Home Residents

- From 1999 to 2009, the number of inpatient discharges from short-stay hospitals with CVD as the first-listed diagnosis decreased from 6 344 000 to 6 165 000 (NHDS, NCHS, and NHLBI). In 2009, CVD ranked highest among all disease categories in hospital discharges (NHDS, NCHS, and NHLBI).
- In 2009, there were 94 871 000 physician office visits with a primary diagnosis of CVD (NCHS, NAMCS, NHLBI tabulation). In 2009, there were 4 761 000 ED visits and 7 261 000 hospital outpatient department visits with a primary diagnosis of CVD (NCHS, NHAMCS, NHLBI tabulation).
- In 2005, ≈1 of every 6 hospital stays, or almost 6 million, resulted from CVD (Agency for Healthcare Research and Quality, NIS). The total inpatient hospital cost for CVD was $71.2 billion, approximately one fourth of the total cost of inpatient hospital care in the United States. The average cost per hospitalization was ≈41% higher than the average cost for all stays. Hospital admissions that originated in the ED accounted for 60.7% of all hospital stays for CVD. This was 41% higher than the rate of 43.1% for all types of hospital stays; 3.3% of patients admitted to the hospital for CVD died in the hospital, which was significantly higher than the average in-hospital death rate of 2.1% for all hospitalized patients.[46]
- In 2004, coronary artery disease (CAD) was estimated to be responsible for 1.2 million hospital stays and was the most expensive condition treated. This condition resulted in >$44 billion in expenses. More than half of the hospital stays for CAD were among patients who also received percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) during their stay. AMI resulted in $31 billion of inpatient hospital charges for 695 000 hospital stays. The 1.1 million hospitalizations for CHF amounted to nearly $29 billion in hospital charges.[47]

- In 2003, ≈48.3% of inpatient hospital stays for CVD were for women, who accounted for 42.8% of the national cost ($187 billion) associated with these conditions. Although only 40% of hospital stays for AMI and CAD were for women, more than half of all stays for nonspecific chest pain, CHF, and stroke were for women. There was no difference between men and women in hospitalizations for cardiac dysrhythmias.[48]
- Circulatory disorders were the most frequent reason for admission to the hospital through the ED, accounting for 26.3% of all admissions through the ED. After pneumonia, the most common heart-related conditions (in descending order) were CHF, chest pain, hardening of the arteries, and heart attack, which together accounted for >15% of all admissions through the ED. Stroke and irregular heart beat ranked seventh and eighth, respectively.[49]
- In 2004, 23.7% of nursing home residents had a primary diagnosis of CVD at admission, and 25% had CVD as the primary diagnosis at the time of interview. This was the leading primary diagnosis for these residents (NCHS, NNHS).[49]
- Among current home healthcare patients in 2007, 18.3% had a primary diagnosis of CVD at admission and 62.9% had any diagnosis of CVD at the time of interview (NCHS, NHHCS unpublished data).
- Among patients discharged from hospice in 2007, 15.8% had a primary diagnosis of CVD at admission (NCHS, NHHCS unpublished data).

Operations and Procedures

- In 2009, an estimated 7 453 000 inpatient cardiovascular operations and procedures were performed in the United States; 4.2 million were performed on males, and 3.3 million were performed on females (NHLBI tabulation of NHDS, NCHS).

Cost

- The estimated direct and indirect cost of CVD for 2008 is $297.7 billion (MEPS, Agency for Healthcare Research and Quality, and NHLBI).
- In 2006, $32.7 billion in program payments were made to Medicare beneficiaries discharged from short-stay hospitals with a principal diagnosis of CVD. That was an average of $10 201 per discharge.[50]
- Between 2010 and 2030, real (2008$) total direct medical costs of CVD are projected to triple, from $273 billion to $818 billion.[3]

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Table 3-1. Cardiovascular Diseases

Population Group	Prevalence, 2008—Age ≥20 y	Mortality, 2008—All Ages*	Hospital Discharges, 2009—All Ages	Cost, 2008
Both sexes	82 600 000 (36.2%)	811 940	6 165 000	$297.7 Billion
Males	39 900 000 (37.4%)	392 210 (48.3%)†	3 230 000	. . .
Females	42 700 000 (35.0%)	419 730 (51.7%)†	2 935 000	. . .
NH white males	37.4%	335 247	. . .	. . .
NH white females	33.8%	360 441	. . .	. . .
NH black males	44.8%	46 819	. . .	. . .
NH black females	47.3%	49 819	. . .	. . .
Mexican American males	30.7%	. . .	. . .	. . .
Mexican American females	30.9%	. . .	. . .	. . .

Ellipses (. . .) indicate data not available; NH, non-Hispanic.

*Mortality data are for whites and blacks and include Hispanics.

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

Sources: Prevalence: National Health and Nutrition Examination Survey (NHANES) 2005–2008, National Center for Health Statistics (NCHS) and National Heart, Lung, and Blood Institute (NHLBI). Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 y of age. Age-specific percentages are extrapolated to the 2008 US population estimates. Mortality: NCHS. These data represent underlying cause of death only. Data include congenital cardiovascular disease mortality. Hospital discharges: National Hospital Discharge Survey, NCHS. Data include those inpatients discharged alive, dead, or of unknown status. Cost: NHLBI. Data include estimated direct and indirect costs for 2008.

Table 3-2. Age-Adjusted Death Rates per 100 000 Population for CVD, CHD, and Stroke by State, 2005–2007

	CVD*			CHD†			Stroke‡		
State	Rank§	Death Rate	% Change 1999–2001 to 2005–2007	Rank§	Death Rate	% Change 1999–2001 to 2005–2007	Rank§	Death Rate	% Change 1999–2001 to 2005–2007
Alabama	51	326.9	−16.0	20	116.6	−28.9	50	56.3	−19.3
Alaska	6	221.6	−22.3	4	87.9	−31.7	35	48.1	−26.1
Arizona	5	217.0	−24.3	25	121.5	−25.7	4	35.3	−32.8
Arkansas	45	307.4	−19.5	46	159.5	−14.8	51	58.0	−24.8
California	27	252.9	−23.0	33	136.3	−28.7	29	44.9	−29.4
Colorado	4	216.1	−21.8	7	98.2	−23.9	11	40.1	−29.6
Connecticut	14	229.6	−23.4	14	110.1	−31.4	5	35.7	−29.4
Delaware	28	262.2	−21.8	39	143.1	−28.1	14	41.3	−21.3
District of Columbia	50	325.6	−13.4	52	187.6	−7.2	18	42.5	−11.5
Florida	12	227.7	−26.4	27	128.5	−32.1	6	35.7	−27.7
Georgia	41	287.6	−23.8	10	107.9	−33.6	44	51.5	−28.5
Hawaii	2	206.0	−24.3	3	82.0	−28.4	20	43.0	−31.1
Idaho	19	234.4	−21.2	11	108.9	−26.3	38	48.9	−24.8
Illinois	31	264.8	−23.9	29	132.8	−30.8	30	45.2	−26.6
Indiana	39	284.7	−21.6	32	136.2	−27.4	37	48.6	−28.9
Iowa	24	247.6	−21.8	37	140.8	−24.5	25	44.5	−25.7
Kansas	25	252.7	−20.9	15	112.4	−27.4	34	46.8	−23.4
Kentucky	44	304.5	−22.3	42	149.6	−25.5	42	49.6	−26.7
Louisiana	47	311.0	−17.9	36	139.6	−26.0	45	52.6	−19.8
Maine	18	234.2	−24.3	16	113.5	−30.9	16	41.8	−27.7
Maryland	33	269.2	−21.2	40	144.9	−23.9	22	44.0	−29.6
Massachusetts	9	224.1	−22.6	9	106.9	−27.2	10	37.9	−24.7
Michigan	42	293.2	−20.9	45	158.0	−25.4	32	45.5	−26.3
Minnesota	1	193.1	−25.9	2	80.5	−33.2	12	40.1	−28.8
Mississippi	52	349.7	−19.3	41	147.2	−29.4	46	53.3	−25.4
Missouri	43	293.9	−21.0	44	154.2	−25.1	43	50.4	−21.6
Montana	11	226.6	−20.9	6	97.6	−21.5	19	42.7	−29.2
Nebraska	17	232.8	−23.0	5	94.4	−29.2	28	44.8	−22.8
Nevada	40	287.4	−16.8	21	117.4	−29.1	17	42.3	−26.8
New Hampshire	13	229.1	−26.9	24	120.9	−33.9	2	35.2	−36.8
New Jersey	25	252.2	−23.7	38	141.1	−29.0	7	35.9	−23.8
New Mexico	7	222.1	−19.8	17	114.7	−25.0	9	37.7	−26.2
New York	37	278.6	−21.1	51	182.1	−23.6	1	29.6	−27.1
North Carolina	34	270.4	−24.4	28	128.7	−29.9	47	53.4	−30.0
North Dakota	22	241.3	−20.3	30	133.5	−19.6	24	44.2	−26.1
Ohio	38	283.2	−22.0	43	151.4	−25.3	33	46.5	−23.3
Oklahoma	49	322.4	−20.6	50	176.2	−23.5	49	54.4	−20.5
Oregon	15	230.6	−22.1	8	98.7	−26.8	40	49.3	−33.1
Pennsylvania	35	271.4	−22.1	34	137.9	−28.0	26	44.6	−22.6
Puerto Rico¶	8	223.5	−15.9	13	109.4	−15.8	23	44.1	−15.5
Rhode Island	29	260.4	−16.8	48	170.6	−19.0	3	35.2	−26.7
South Carolina	36	274.1	−25.2	23	119.8	−32.3	48	53.7	−33.1
South Dakota	20	238.1	−21.2	35	137.9	−17.5	31	45.4	−21.9
Tennessee	48	315.3	−19.4	49	171.1	−22.3	52	58.1	−23.7
Texas	32	266.9	−24.0	31	134.9	−30.5	41	49.3	−25.1

(*Continued*)

Table 3-2. Continued

	CVD*			CHD†			Stroke‡		
State	Rank§	Death Rate	% Change 1999–2001 to 2005–2007	Rank§	Death Rate	% Change 1999–2001 to 2005–2007	Rank§	Death Rate	% Change 1999–2001 to 2005–2007
Utah	3	213.2	−21.3	1	78.6	−30.8	13	40.4	−34.0
Vermont	10	226.3	−24.0	26	121.6	−26.0	8	37.1	−31.2
Virginia	28	254.7	−23.1	19	114.8	−27.4	36	48.3	−28.4
Washington	16	232.4	−22.6	22	117.5	−26.0	21	43.7	−36.8
West Virginia	46	309.1	−22.0	47	160.7	−27.1	39	49.2	−19.9
Wisconsin	23	242.6	−24.0	18	114.7	−29.9	27	44.7	−30.3
Wyoming	21	238.7	−19.4	13	109.3	−25.1	15	41.4	−29.2
Total United States		262.7	−22.6		135.1	−27.7		44.1	−26.9

CVD indicates cardiovascular disease; CHD, coronary heart disease.

*CVD is defined here as International Classification of Diseases, 10th Revision (ICD-10) codes I00–I78.

†CHD is defined here as ICD-10 I20–I25.

‡Stroke is defined here as ICD-10 I60–I69.

§Rank is lowest to highest.

¶Percent changes for Puerto Rico are for 2000 to 2005–2007.

Source: Health Data Interactive, 2005–2007. Data provided by personal communication with the National Heart, Lung, and Blood Institute.

The Agency for Healthcare Research and Quality has released state-level data for heart disease for all 50 states and the District of Columbia. The data are taken from the congressionally mandated National Healthcare Quality Report (NHQR), available at http://statesnapshots.ahrq.gov/snaps07/index.jsp. In addition, the *Women's Health and Mortality Chartbook* of the National Center for Health Statistics has state-related data for women available at http://www.cdc.gov/nchs/data/healthywomen/womenschartbook_aug2004.pdf. Also, at http://apps.nccd.cdc.gov/brfss-smart/index.asp, Metropolitan/Micropolitan Area Risk (MMSA) data are available for 500 such areas nationwide. Behavioral Risk Factor Surveillance System data are also collected within each state (www.cdc.gov/brfss). The Centers for Disease Control and Prevention (CDC) has the Geographic Information Systems (GIS), which provides mortality rates down to the county level, by sex and ethnicity, available at http://www.cdc.gov/gis/. The 2008 Atlas of Stroke Hospitalizations Among Medicare Beneficiaries (CDC, 2008) is a new resource that provides data down to the county level, by sex and race (available at http://www.cdc.gov/dhdsp/atlas/2008_stroke_atlas/index.htm).

Table 3-3. International Death Rates (Revised May 2011): Death Rates (Per 100 000 Population) for Total CVD, CHD, Stroke, and Total Deaths in Selected Countries (Most Recent Year Available)

	CVD Deaths	CHD Deaths	Stroke Deaths	Total Deaths
Men ages 35–74 y				
Russian Federation (2006)	1299.2	706.0	351.4	2683.4
Bulgaria (2008)	803.7	219.4	218.2	1554.3
Lithuania (2009)	734.7	444.6	138.3	1842.3
Romania (2009)	677.9	276.4	200.2	1572.4
Slovakia (2005)	634.2	320.1	91.8	1528.3
Hungary (2009)	605.6	319.1	121.1	1652.3
Poland (2008)	495.2	180.0	100.8	1412.7
Croatia (2009)	419.3	202.2	113.6	1184.7
Czech Republic (2009)	386.6	198.6	64.4	1080.8
Kuwait (2009)	319.6	187.0	62.1	563.9
Finland (2009)	284.4	170.0	43.8	833.2
United States (2008)	256.0	149.2	30.0	862.7
Greece (2009)	251.6	136.7	50.8	721.6
Germany (2006)	242.1	125.3	34.5	788.5
Ireland (2009)	210.0	140.6	29.2	701.3
Belgium (2005)	209.6	99.5	35.9	821.7
Denmark (2006)	206.6	84.8	45.6	865.6
New Zealand (2007)	204.2	135.6	29.1	635.7
United Kingdom (2009)	202.0	125.8	29.9	687.6
Canada (2004)	198.3	130.8	24.2	705.3
Austria (2009)	189.3	110.2	26.3	736.3
Sweden (2008)	187.8	109.4	31.0	591.8
Portugal (2009)	168.7	61.3	62.1	825.3
Spain (2008)	168.2	77.6	33.7	714.0
Italy (2007)	160.6	75.6	29.9	625.8
Netherlands (2009)	157.9	64.6	24.6	649.4
Israel (2007)	156.3	86.3	32.5	655.9
Norway (2009)	154.4	84.6	29.0	607.0
Switzerland (2007)	150.4	78.2	16.6	587.5
Japan (2009)	145.2	46.5	52.2	605.0
France (2007)	145.0	57.1	26.5	774.6
Australia (2006)	141.3	88.9	22.0	553.4
Korea, South (2009)	138.4	41.0	65.9	783.6
Women ages 35–74 y				
Russian Federation (2006)	521.4	237.1	189.2	1001.8
Bulgaria (2008)	368.6	70.9	120.6	699.3
Romania (2009)	325.5	109.5	116.2	706.0
Slovakia (2005)	269.5	129.5	41.9	643.7
Lithuania (2009)	253.9	127.5	73.8	648.6
Kuwait (2009)	246.1	94.8	56.1	568.1
Hungary (2009)	239.2	113.7	56.0	719.4
Croatia (2009)	190.8	71.9	68.7	520.1
Poland (2008)	181.5	51.6	50.1	570.0
Czech Republic (2009)	164.3	69.9	34.8	506.6
United States (2008)	129.2	59.5	23.5	544.7
Denmark (2006)	100.0	32.4	32.1	557.8
Germany (2006)	97.8	38.2	20.1	402.4
Greece (2009)	97.1	33.3	29.3	319.0
Belgium (2005)	94.4	30.8	24.8	436.3
New Zealand (2007)	89.8	43.9	21.7	418.2
United Kingdom (2009)	88.1	38.5	22.5	438.5
Ireland (2009)	86.8	40.9	21.9	419.8
Finland (2009)	83.4	36.1	23.0	377.8
Canada (2004)	83.1	42.8	17.3	432.7
Portugal (2009)	76.5	20.0	33.5	377.6
Austria (2009)	75.5	33.7	16.4	368.2
Sweden (2008)	74.6	35.5	18.5	374.1
Netherlands (2009)	74.0	20.6	20.1	416.8
Italy (2007)	67.3	22.2	18.2	326.0
Israel (2007)	65.4	22.2	17.3	388.7
Korea, South (2009)	63.5	41.0	33.2	312.3
Spain (2008)	62.4	18.7	17.8	304.4
Norway (2009)	60.5	26.3	15.2	377.0
Australia (2006)	60.4	26.8	16.3	327.5
Japan (2009)	54.4	12.8	22.7	266.9
Switzerland (2007)	54.1	19.4	12.4	327.6
France (2007)	51.3	12.1	13.9	346.0

CVD indicates cardiovascular disease; CHD, coronary heart disease.

Rates are adjusted to the European Standard population. International Classification of Diseases, 10th Revision (ICD-10) codes are used for all countries except Greece, for which International Classification of Diseases, 9th Revision (ICD-9) codes are used. For countries using ICD-9, the ICD-9 codes are 390–459 for CVD, 410–414 for CHD, and 430–438 for stroke. ICD-10 codes are I00–I99 for CVD, I20–I25 for CHD, and I60–I69 for stroke.

The following countries have been dropped from the table because data on number of deaths or population are no longer furnished to the World Health Organization: Argentina, China, Colombia, and Mexico.

Sources: The World Health Organization, National Center for Health Statistics, and National Heart, Lung, and Blood Institute.

Table 3-4. Remaining Lifetime Risks for CVD and Other Diseases Among Men and Women Free of Disease at 40 and 70 Years of Age

	Remaining Lifetime Risk at Age 40 y		Remaining Lifetime Risk at Age 70 y	
Diseases	Men	Women	Men	Women
Any CVD*	2 in 3	1 in 2	1 in 2	1 in 2
CHD[6]	1 in 2	1 in 3	1 in 3	1 in 4
AF[23]	1 in 4	1 in 4	1 in 4	1 in 4
CHF[24]	1 in 5	1 in 5	1 in 5	1 in 5
Stroke[25]	1 in 6†	1 in 5†	1 in 6	1 in 5
Dementia[25]	. . .	. . .	1 in 7	1 in 5
Hip fracture[38]	1 in 20	1 in 6	. . .	. . .
Breast cancer[39,42]	1 in 1000	1 in 8	. . .	1 in 14
Prostate cancer[39]	1 in 6	. . .	. . .	. . .
Lung cancer[39]	1 in 12	1 in 17	. . .	. . .
Colon cancer[39]	1 in 16	1 in 17	. . .	. . .
Diabetes[43]	1 in 3	1 in 3	1 in 9	1 in 7
Hypertension[44]	9 in 10†	9 in 10†	9 in 10‡	9 in 10‡
Obesity[45]	1 in 3	1 in 3	. . .	. . .

CVD indicates cardiovascular disease; ellipses (. . .), not estimated; CHD, coronary heart disease; AF, atrial fibrillation; and CHF, congestive heart failure.

*Personal communication from Donald Lloyd-Jones, based on Framingham Heart Study data.

†Age 55 y.

‡Age 65 y.

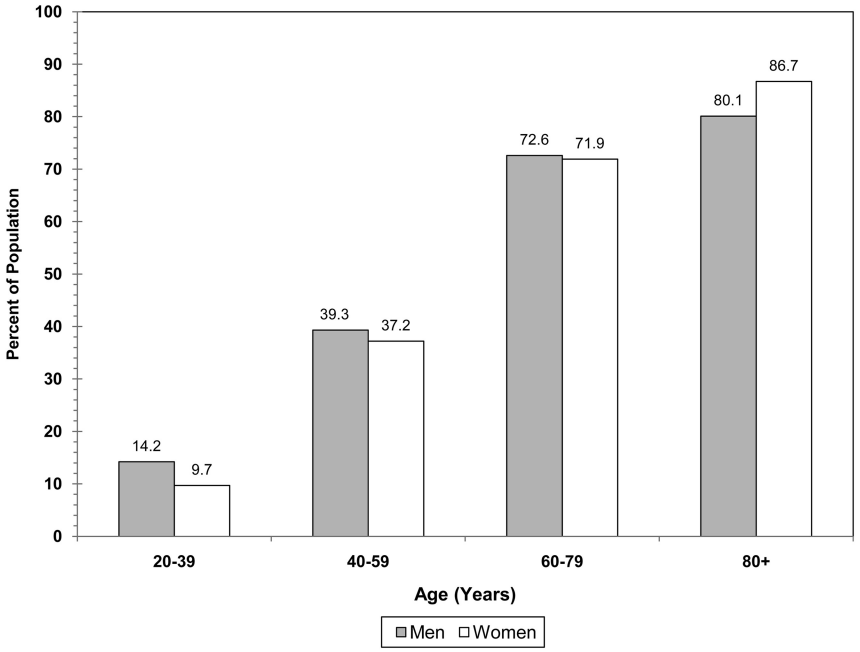


Chart 3-1. Prevalence of cardiovascular disease in adults ≥20 years of age by age and sex (National Health and Nutrition Examination Survey: 2005–2008). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute. These data include coronary heart disease, heart failure, stroke, and hypertension.

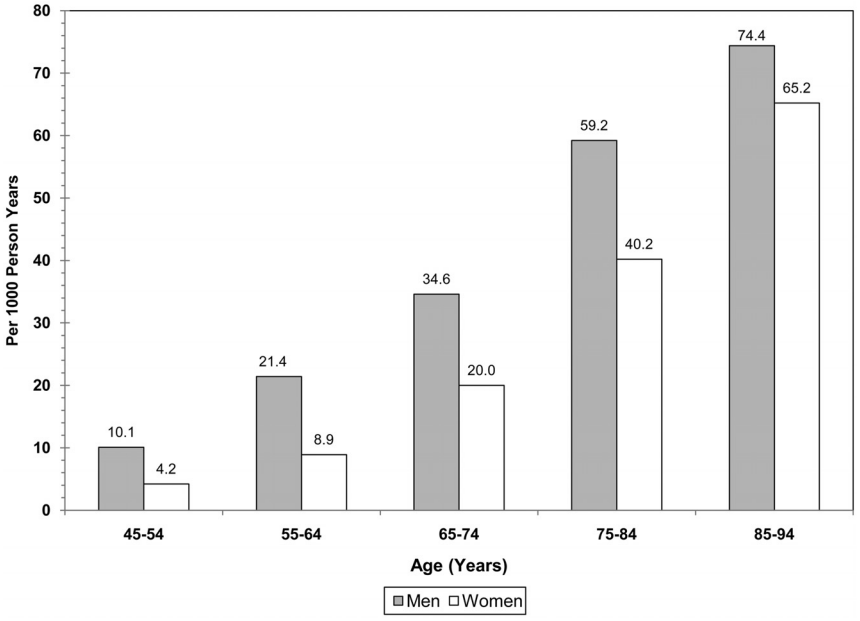


Chart 3-2. Incidence of cardiovascular disease* by age and sex (Framingham Heart Study, 1980–2003). *Coronary heart disease, heart failure, stroke, or intermittent claudication. Does not include hypertension alone. Source: National Heart, Lung, and Blood Institute.[4]

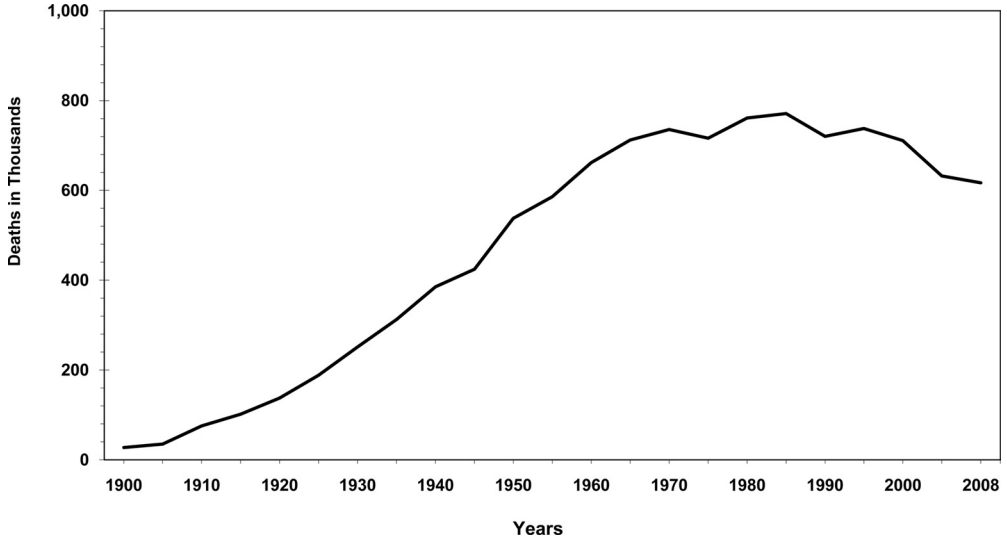


Chart 3-3. Deaths attributable to diseases of the heart (United States: 1900–2008). See Glossary (Chapter 25) for an explanation of "diseases of the heart." Note: In the years 1900–1920, the International Classification of Diseases codes were 77–80; for 1925, 87–90; for 1930–1945, 90–95; for 1950–1960, 402–404, 410–443; for 1965, 402–404, 410–443; for 1970–1975, 390–398, 404–429; for 1980–1995, 390–398, 402, 404–429; and for 2000–2008, I00–I09, I11, I13, I20–I51. Before 1933, data are for a death registration area and not the entire United States. In 1900, only 10 states were in the death registration area, and this increased over the years, so part of the increase in numbers of deaths is attributable to an increase in the number of states. Source: National Center for Health Statistics.

Chart 3-4. Deaths attributable to cardiovascular disease (United States: 1900–2008). Cardiovascular disease (International Classification of Diseases, 10th Revision codes I00–I99) does not include congenital. Before 1933, data are for a death registration area and not the entire United States. Source: National Center for Health Statistics.

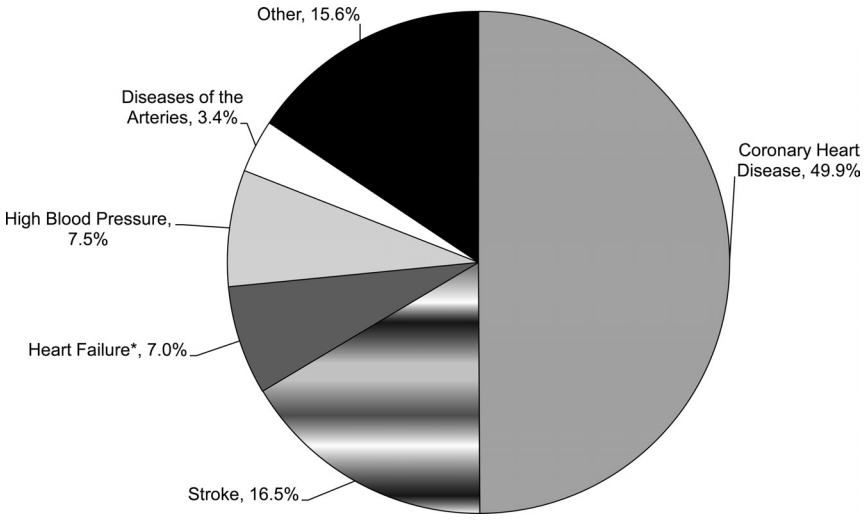


Chart 3-5. Percentage breakdown of deaths attributable to cardiovascular disease (United States: 2008). Source: National Heart, Lung, and Blood Institute from National Center for Health Statistics reports and data sets. *Not a true underlying cause. With any mention deaths, heart failure accounts for 35% of cardiovascular disease deaths. Total may not add to 100 because of rounding. Coronary heart disease includes International Classification of Diseases (ICD), 10th Revision codes I20–I25; stroke, I60–I69; heart failure, I50; high blood pressure, I10–I13; diseases of the arteries, I70–I78; and other, all remaining ICD I categories.

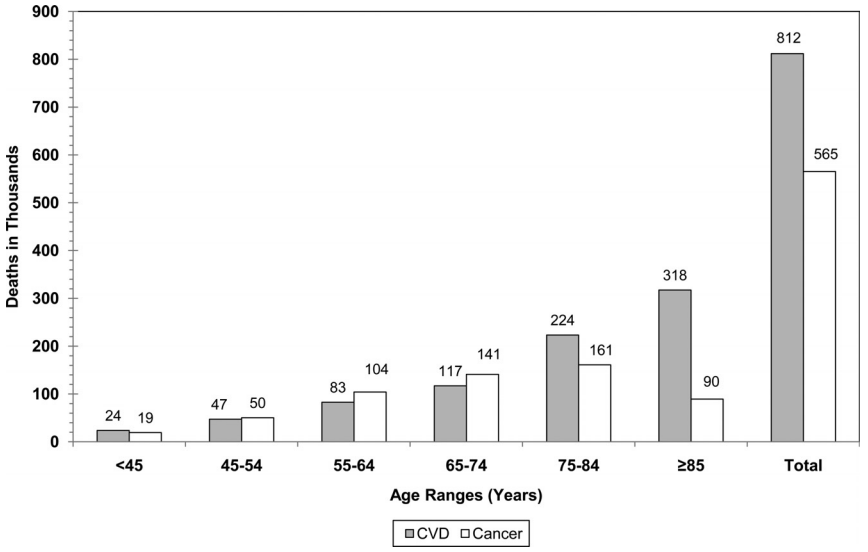


Chart 3-6. Cardiovascular disease (CVD) deaths vs cancer deaths by age (United States: 2008). Source: National Center for Health Statistics. CVD includes International Classification of Diseases, 10th Revision codes I00–I99, Q20–Q28; and cancer, C00–C97.

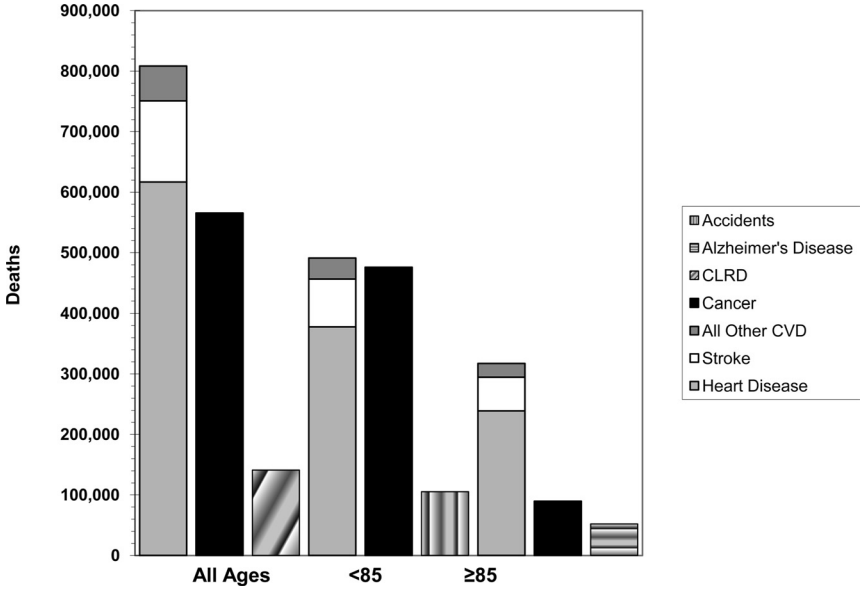


Chart 3-7. Cardiovascular disease (CVD) and other major causes of death: total, <85 years of age, and ≥85 years of age. Deaths among both sexes, United States, 2008. CLRD indicates chronic lower respiratory disease. Heart disease includes International Classification of Diseases, 10th Revision codes I00–I09, I11, I13, I20–I51; stroke, I60–I69; all other CVD, I10, I12, I15, I70–I99; cancer, C00–C97 ; CLRD, J40–J47; Alzheimer disease, G30; and accidents, V01–X59, Y85–Y86. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

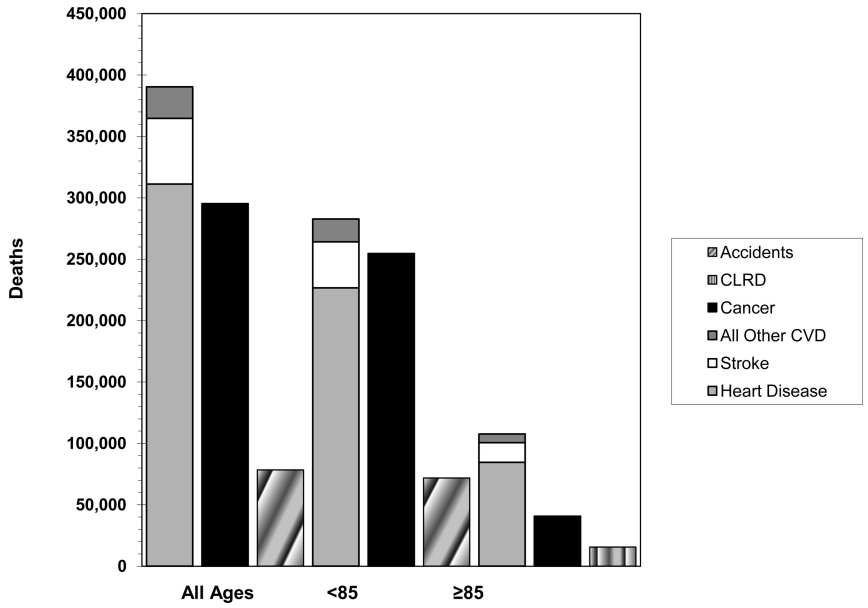


Chart 3-8. Cardiovascular disease (CVD) and other major causes of death in males: total, <85 years of age, and ≥85 years of age. Deaths among males, United States, 2008. CLRD indicates chronic lower respiratory disease. Heart disease includes International Classification of Diseases, 10th Revision codes I00–I09, I11, I13, I20–I51; stroke, I60–I69; all other CVD, I10, I12, I15, I70–I99; cancer, C00–C97; CLRD, J40–J47; and accidents, V01–X59, Y85–Y86. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

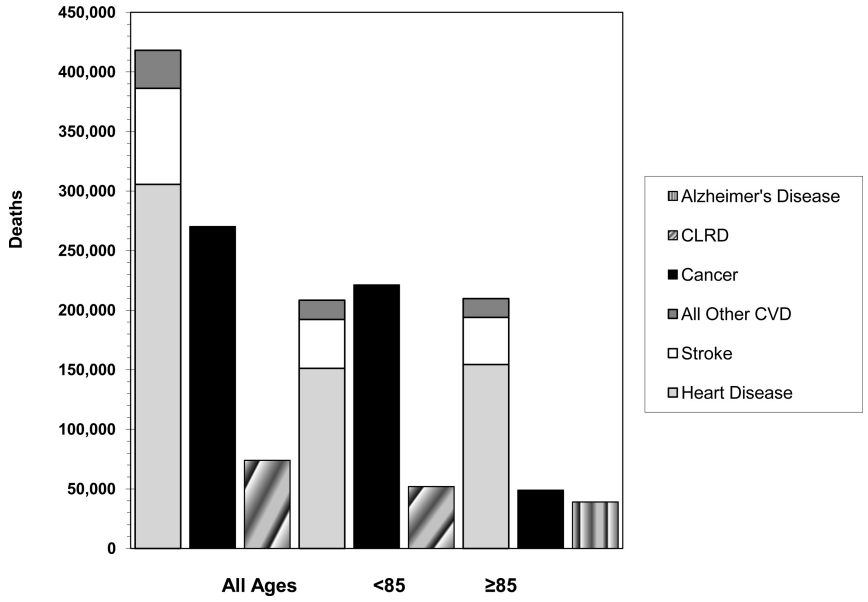


Chart 3-9. Cardiovascular disease (CVD) and other major causes of death in females: total, <85 years of age, and ≥85 years of age. Deaths among females, United States, 2008. CLRD indicates chronic lower respiratory disease. Heart disease includes International Classification of Diseases, 10th Revision codes I00–I09, I11, I13, I20–I51; stroke, I60–I69; all other CVD, I10, I12, I15, I70–I99; cancer, C00–C97; CLRD, J40–J47; and Alzheimer disease, G30. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

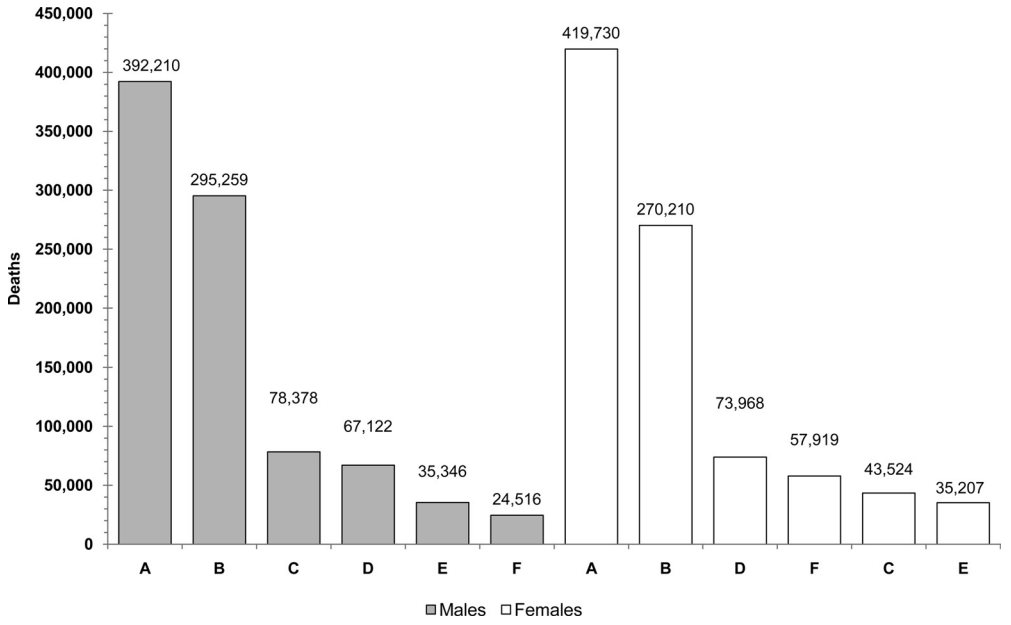


Chart 3-10. Cardiovascular disease and other major causes of death for all males and females (United States: 2008). A indicates cardiovascular disease plus congenital cardiovascular disease (International Classification of Diseases, 10th Revision codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, chronic lower respiratory disease (J40–J47); E, diabetes mellitus (E10–E14); and F, Alzheimer disease (G30). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

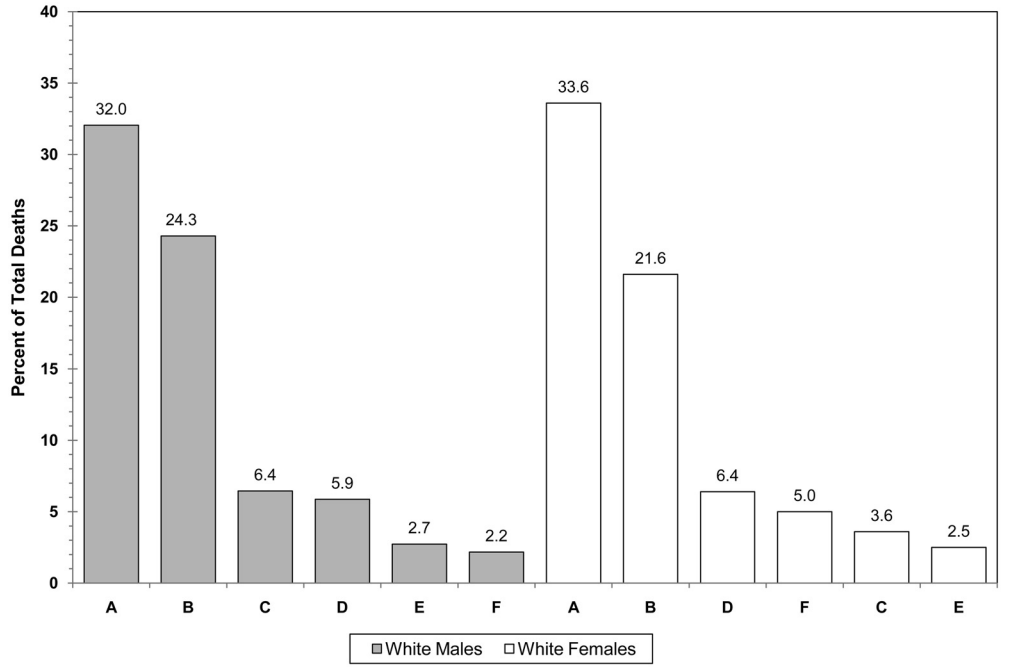


Chart 3-11. Cardiovascular disease and other major causes of death for white males and females (United States: 2008). A indicates cardiovascular disease plus congenital cardiovascular disease (International Classification of Diseases, 10th Revision codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, chronic lower respiratory disease (J40–J47); E, diabetes mellitus (E10–E14); and F, Alzheimer disease (G30). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

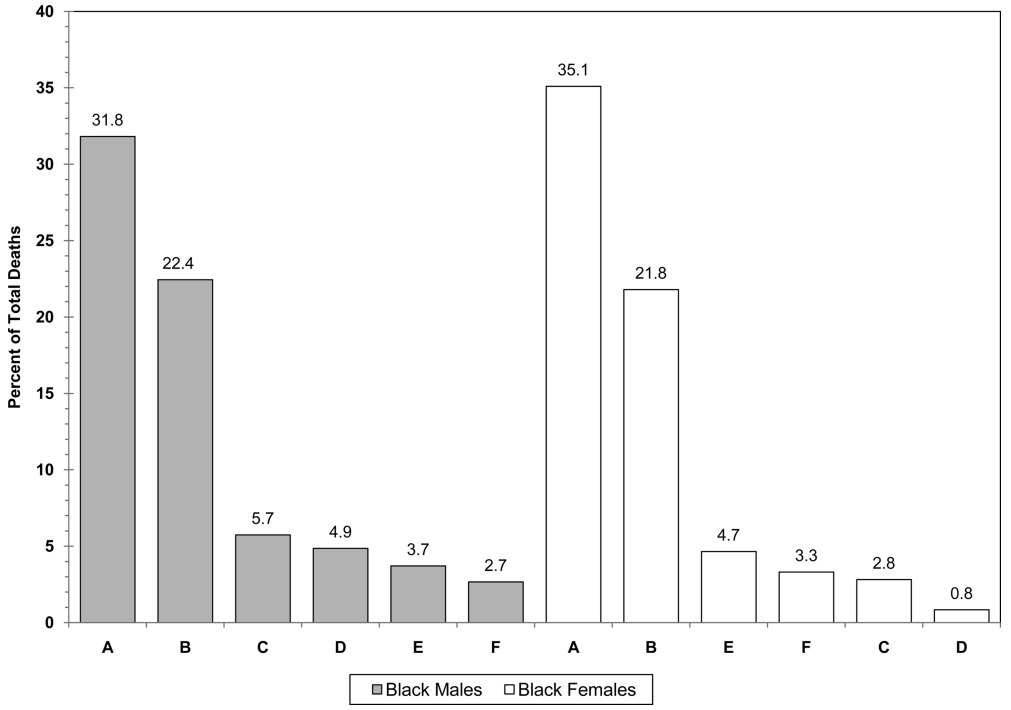


Chart 3-12. Cardiovascular disease and other major causes of death for black males and females (United States: 2008). A indicates cardiovascular disease plus congenital cardiovascular disease (International Classification of Diseases, 10th Revision codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, assaults (homicide) (U01–U02, X85–Y09, Y87.1); E, diabetes mellitus (E10–E14); and F, nephritis (N00–N07, N17–N19, N25–N27). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

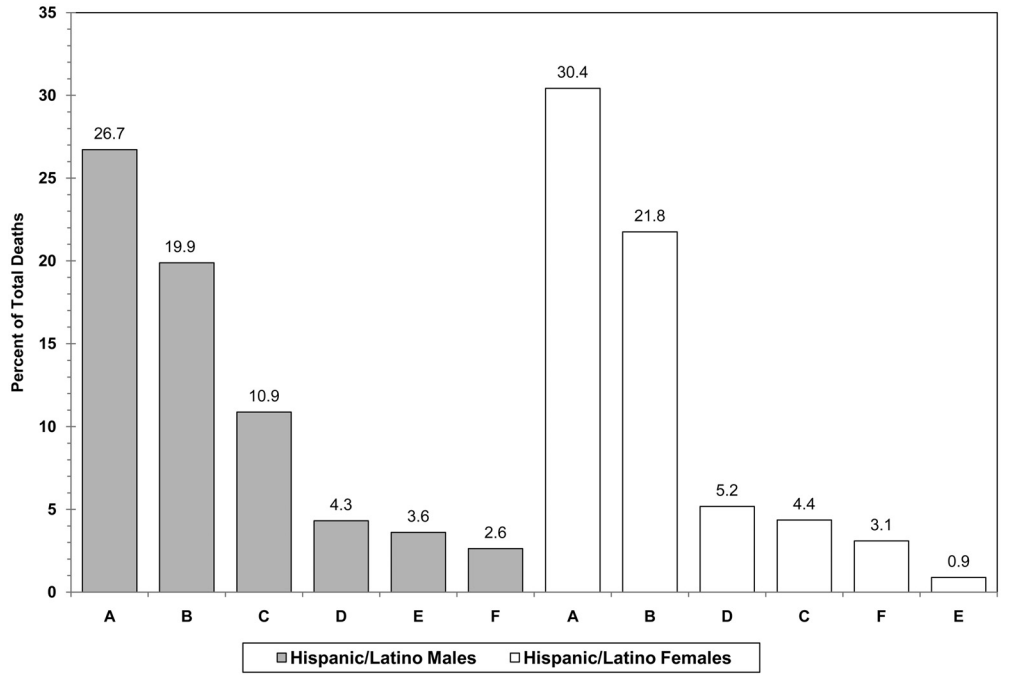


Chart 3-13. Cardiovascular disease and other major causes of death for Hispanic or Latino males and females (United States: 2008). A indicates cardiovascular disease plus congenital cardiovascular disease (International Classification of Diseases, 10th Revision codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, diabetes mellitus (E10–E14); E, assaults (homicide) (U01–U02, X85–Y09, Y87.1); and F, chronic lower respiratory disease (J40–J47). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

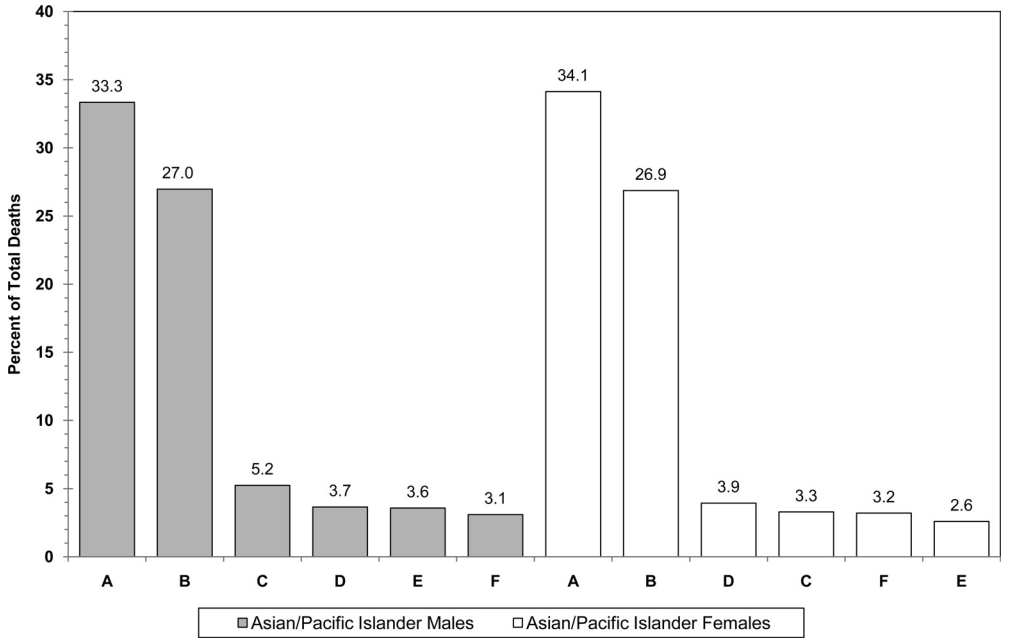


Chart 3-14. Cardiovascular disease and other major causes of death for Asian or Pacific Islander males and females (United States: 2008). "Asian or Pacific Islander" is a heterogeneous category that includes people at high cardiovascular disease risk (eg, South Asian) and people at low cardiovascular disease risk (eg, Japanese). More specific data on these groups are not available. A indicates cardiovascular disease plus congenital cardiovascular disease (International Classification of Diseases, 10th Revision codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, diabetes mellitus (E10–E14); E, chronic lower respiratory disease (J40–J47); and F, influenza and pneumonia (J09–J18). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

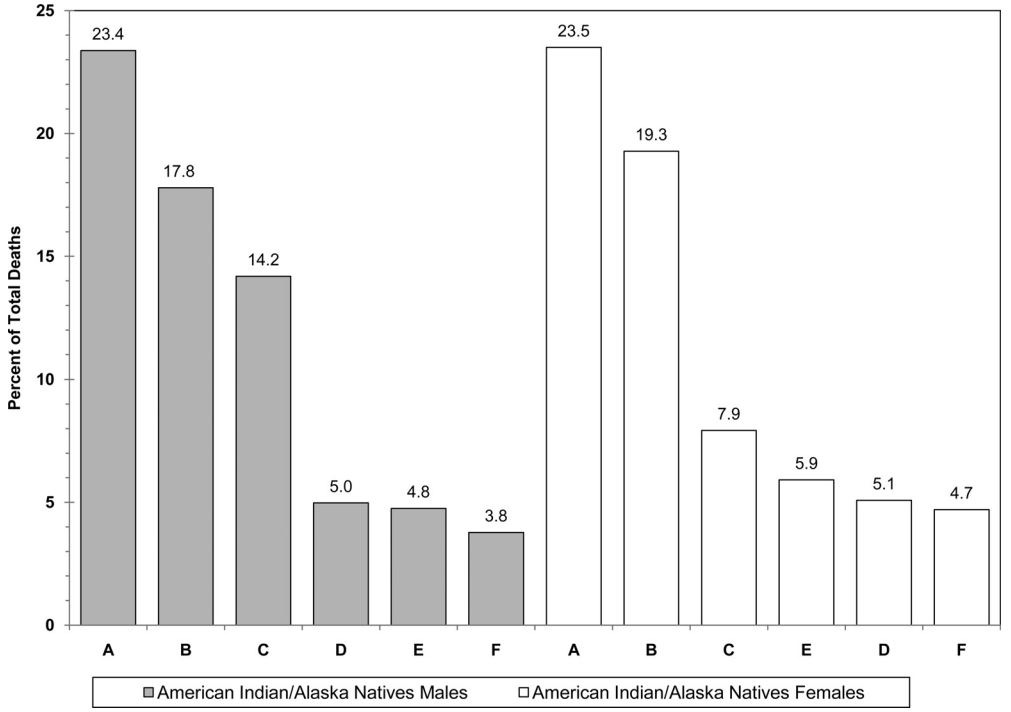


Chart 3-15. Cardiovascular disease and other major causes of death for American Indian or Alaska Native males and females (United States: 2008). A indicates cardiovascular disease plus congenital cardiovascular disease (International Classification of Diseases, 10th Revision codes I00–I99, Q20–Q28); B, cancer (C00–C97); C, accidents (V01–X59, Y85–Y86); D, chronic liver disease (K70, K73–K74); E, diabetes mellitus (E10–E14); and F, chronic lower respiratory disease (J40–J47). Source: National Center for Health Statistics.

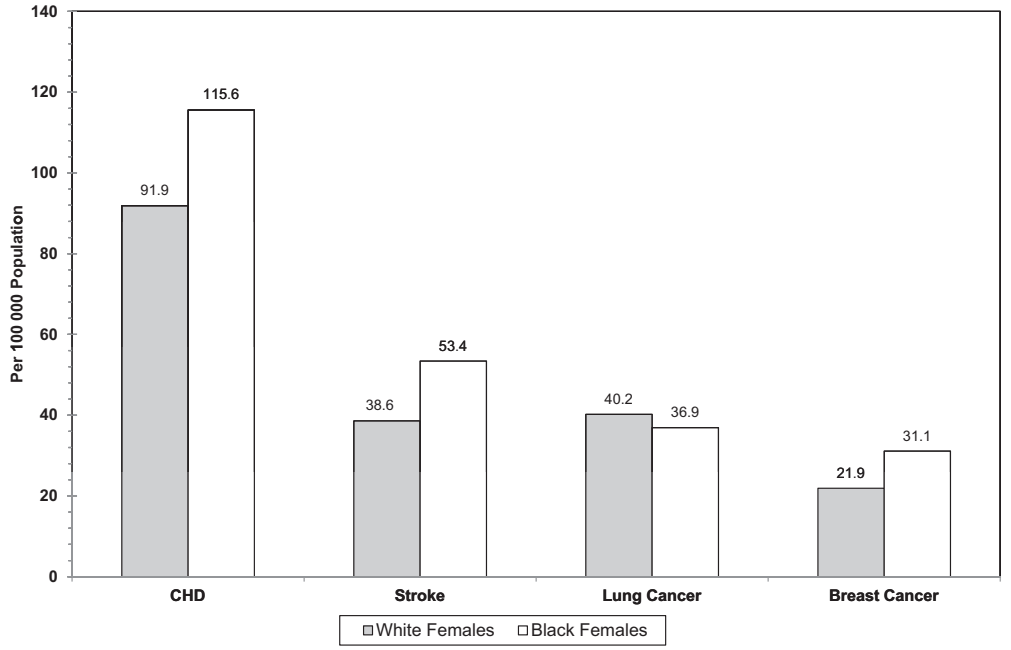


Chart 3-16. Age-adjusted death rates for coronary heart disease (CHD), stroke, and lung and breast cancer for white and black females (United States: 2008). CHD includes International Classification of Diseases, 10th Revision codes I20–I25; stroke, I60–I69; lung cancer, C33–C34; and breast cancer, C50. Source: National Center for Health Statistics.

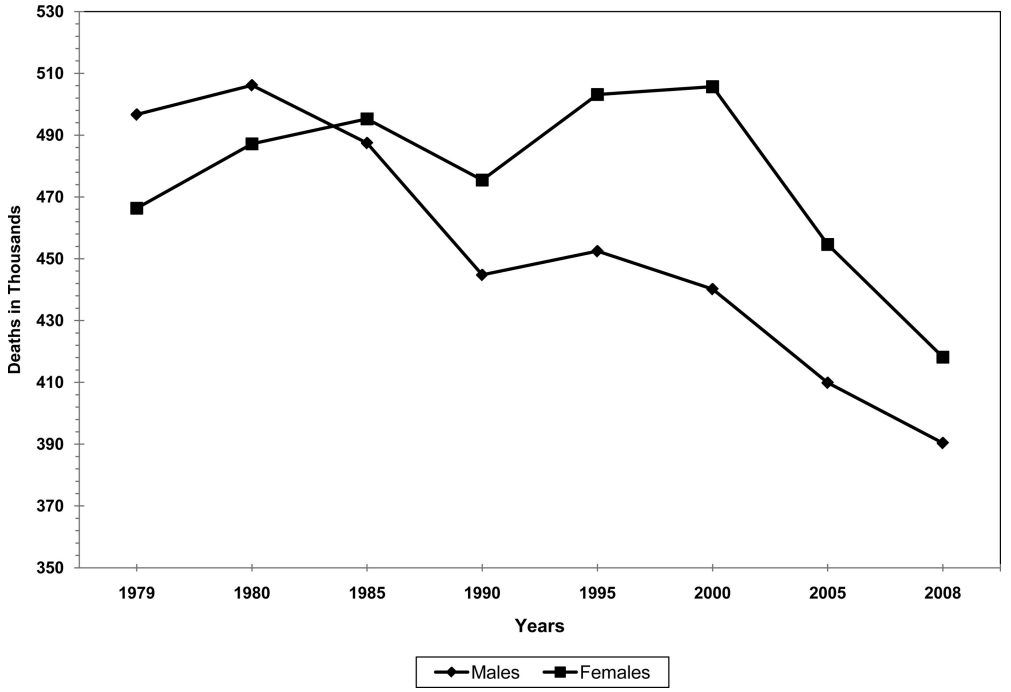


Chart 3-17. Cardiovascular disease mortality trends for males and females (United States: 1979–2008). Cardiovascular disease excludes congenital cardiovascular defects (International Classification of Diseases, 10th Revision [ICD-10] codes I00–I99). The overall comparability for cardiovascular disease between the International Classification of Diseases, 9th Revision (1979–1998) and ICD-10 (1999–2008) is 0.9962. No comparability ratios were applied. Source: National Center for Health Statistics.

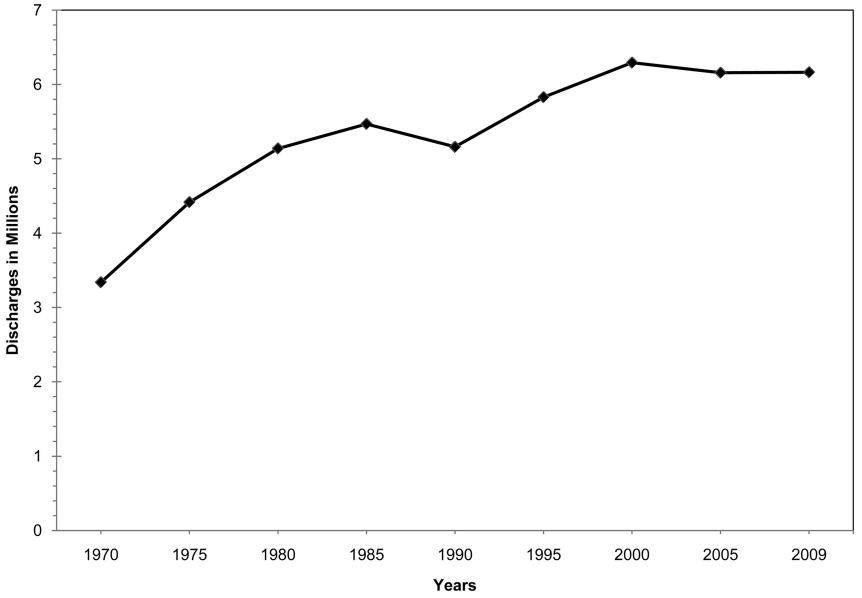


Chart 3-18. Hospital discharges for cardiovascular disease (United States: 1970–2009). Hospital discharges include people discharged alive, dead, and "status unknown." Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

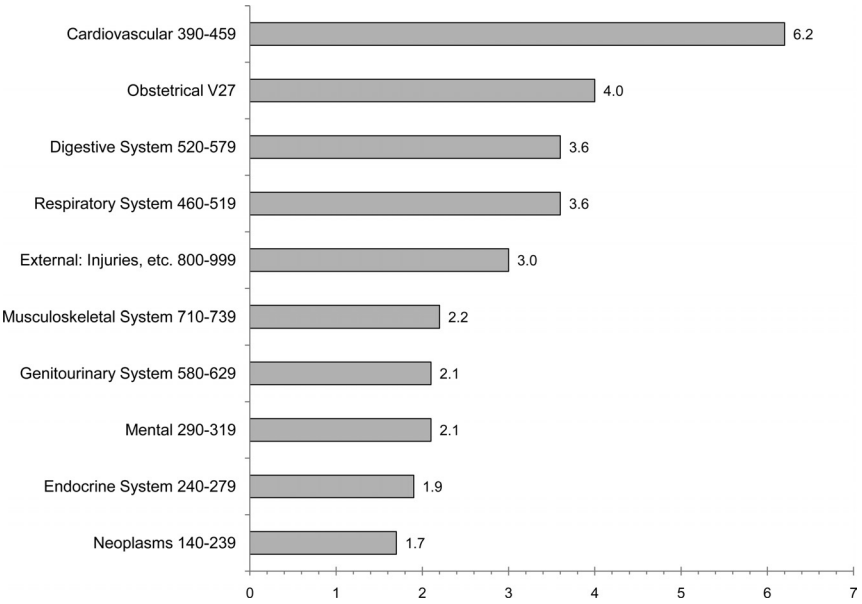
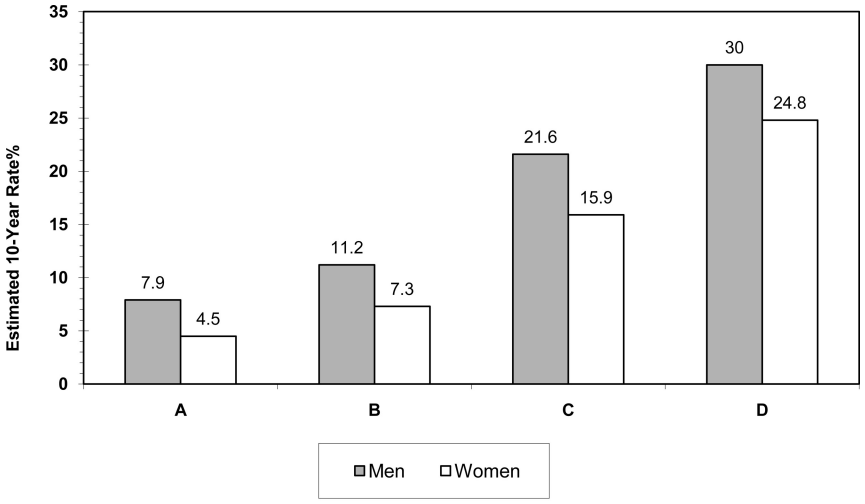


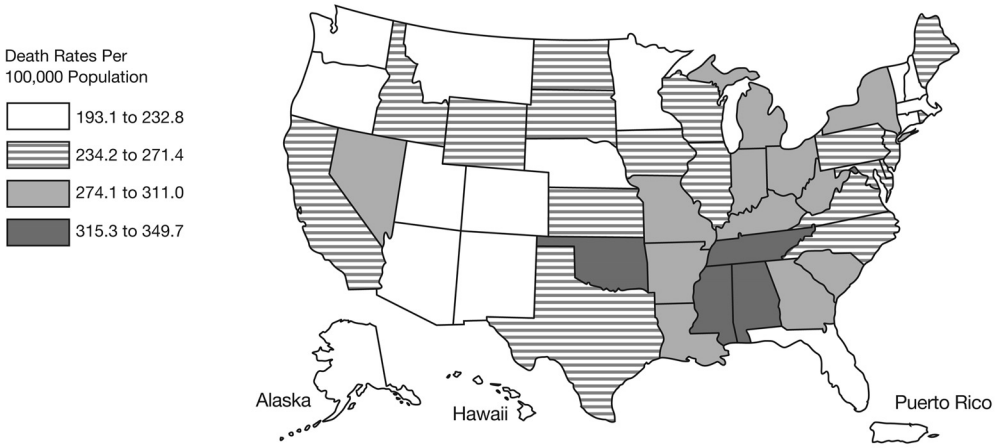
Chart 3-19. Hospital discharges for the 10 leading diagnostic groups (United States: 2009). Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.



	A	B	C	D
Age	50-54	50-54	50-54	50-54
HDL Cholesterol, mg/dL	45-49	45-49	35-44	35-44
Total Cholesterol, mg/dL	160-199	200-239	200-239	200-239
Systolic BP mm/Hg, no treatment	120-129	130-139	130-139	130-139
Smoker	No	No	No	Yes
Diabetes	No	No	Yes	Yes

Chart 3-20. Estimated average 10-year cardiovascular disease risk in adults 50 to 54 years of age according to levels of various risk factors (Framingham Heart Study). HDL indicates high-density lipoprotein; BP, blood pressure. Data derived from D'Agostino et al,[51] with permission of the publisher.

Major Cardiovascular Disease Age-Adjusted Death Rates by State



Coronary Heart Disease Age-Adjusted Death Rates by State

Stroke Age-Adjusted Death Rates by State

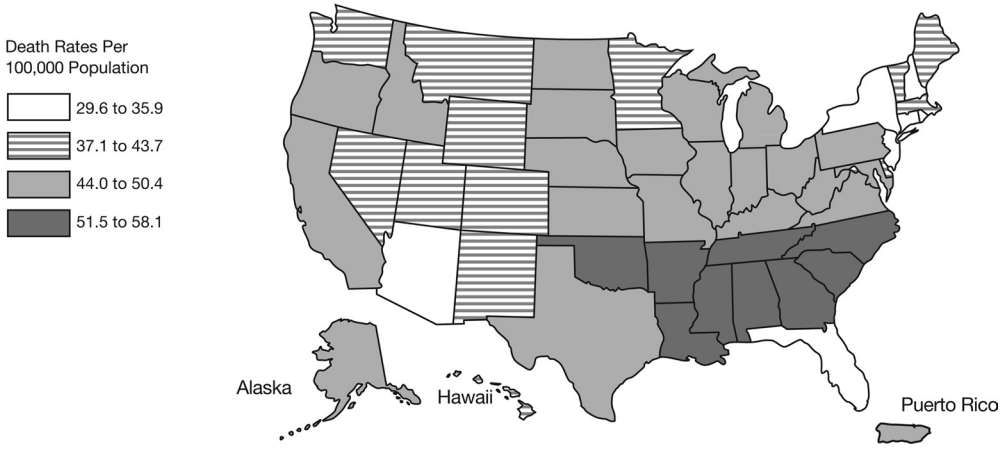


Chart 3-21. US maps corresponding to state death rates (including the District of Columbia).

4. Subclinical Atherosclerosis

See Table 4-1 and Charts 4-1 through 4-6.

Atherosclerosis, a systemic disease process in which fatty deposits, inflammation, cells, and scar tissue build up within the walls of arteries, is the underlying cause of the majority of clinical cardiovascular events. Individuals who develop atherosclerosis tend to develop it in a number of different types of arteries (large and small arteries and those feeding the heart, brain, kidneys, and extremities), although they may have much more in some parts of the body than others. In recent decades, advances in imaging technology have allowed for improved ability to detect and quantify atherosclerosis at all stages and in multiple different vascular beds. Two modalities, computed tomography (CT) of the chest for evaluation of coronary artery calcification (CAC) and B-mode ultrasound of the neck for evaluation of carotid artery intima-media thickness (IMT), have been used in large studies with outcomes data and may help define the burden of atherosclerosis in individuals before they develop clinical events such as heart attack or stroke. Another commonly used method for detecting and quantifying atherosclerosis in the peripheral arteries is the ankle-brachial index (ABI), which is discussed in Chapter 11. Data on cardiovascular outcomes are starting to emerge for additional modalities that measure anatomic and functional measures of subclinical disease, including brachial artery reactivity testing, aortic and carotid magnetic resonance imaging, and tonometric methods of measuring vascular compliance or microvascular reactivity. Further research may help to define the role of these techniques in cardiovascular risk assessment. Some guidelines have recommended screening for subclinical atherosclerosis, especially by CAC, or IMT may be appropriate in people at intermediate risk for HD (eg, 10-year estimated risk of 10% to 20%) but not for lower-risk general population screening or for people with preexisting HD or most other high-risk conditions.[1,2] However, a recent guideline notes those with DM who are ≥40 years of age may be suitable for screening of risk by coronary calcium. There are still limited data demonstrating whether screening with these and other imaging modalities can improve patient outcomes or whether it only increases downstream medical care costs. A recently published report in a large cohort randomly assigned to coronary calcium screening or not showed such screening to result in an improved risk factor profile without increasing downstream medical costs.[3]

Abbreviations Used in Chapter 4

ABI	ankle-brachial index
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
BP	blood pressure
CAC	coronary artery calcification
CAD	coronary artery disease
CARDIA	Coronary Artery Risk Development in Young Adults
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CT	computed tomography
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
FHS	Framingham Heart Study
FMD	flow-mediated dilation
FRS	Framingham Risk Score
HDL	high-density lipoprotein
HD	heart disease
HR	hazard ratio
IMT	intima-media thickness
LDL	low-density lipoprotein
MESA	Multi-Ethnic Study of Atherosclerosis
NHLBI	National Heart, Lung, and Blood Institute
RR	relative risk
SBP	systolic blood pressure

Coronary Artery Calcification

Background

- CAC is a measure of the burden of atherosclerosis in the heart arteries and is measured by CT. Other components of the atherosclerotic plaque, including fatty (eg, cholesterol-rich components) and fibrotic components, often accompany CAC and may be present even in the absence of CAC.
- The presence of any CAC, which indicates that at least some atherosclerotic plaque is present, is defined by an Agatston score >0. Clinically significant plaque, frequently an indication for more aggressive risk factor management, is often defined by an Agatston score ≥100 or a score ≥75th percentile for one's age and sex. An Agatston score ≥400 has been noted to be an indication for further diagnostic evaluation (eg, exercise testing or myocardial perfusion imaging) for CAD.

Prevalence

- The NHLBI's FHS reported CAC measured in 3238 white adults in age groups ranging from <45 years of age to ≥75 years of age.[4]
  - — Overall, 32.0% of women and 52.9% of men had prevalent CAC.
  - — Among participants at intermediate risk according to Framingham Risk Score (FRS), 58% of women and 67% of men had prevalent CAC.
- The NHLBI's CARDIA study measured CAC in 3043 black and white adults 33 to 45 years of age (at the CARDIA year 15 examination).[5]
  - — Overall, 15.0% of men and 5.1% of women, 5.5% of those 33 to 39 years of age and 13.3% of those 40 to 45 years of age, had prevalent CAC. Overall, 1.6% of participants had an Agatston score that exceeded 100.
  - — Chart 4-1 shows the prevalence of CAC by ethnicity and sex. The prevalence of CAC was lower in black men than in white men but was similar in black and white women at these ages.

- The NHLBI's MESA study measured CAC in 6814 participants 45 to 84 years of age, including white (n=2619), black (n=1898), Hispanic (n=1494), and Chinese (n=803) men and women.[6]
  - — Chart 4-2 shows the prevalence of CAC by sex and ethnicity.
  - — The prevalence and 75th percentile levels of CAC were highest in white men and lowest in black and Hispanic women. Significant ethnic differences persisted after adjustment for risk factors, with the RR of coronary calcium being 22% less in blacks, 15% less in Hispanics, and 8% less in Chinese than in whites.
  - — Table 4-1 shows the 75th percentile levels of CAC by sex and race at selected ages. These might be considered cut points above which more aggressive efforts to control risk factors (eg, elevated cholesterol or BP) could be implemented and/or at which treatment goals might be more aggressive (eg, LDL cholesterol <100 mg/dL instead of <130 mg/dL).
- The prevalence of CAC varies widely according to FRS. In a report from the MESA study,[7] the prevalence of CAC among individuals with very low FRS (10-year risk <5%) was low. These findings may have important implications for population screening for subclinical atherosclerosis.
- Investigators from the NHLBI's CARDIA study examined the association between neighborhood attributes and subclinical atherosclerosis in younger adult populations. Using 2000 US Census block-group-level data, among women, higher odds of CAC were associated with higher neighborhood deprivation and lower neighborhood cohesion. Among all men, neither neighborhood deprivation nor neighborhood cohesion was associated with CAC, whereas among men in deprived neighborhoods, low cohesion was associated with higher odds of CAC.[8]

CAC and Incidence of Coronary Events

- The NHLBI's MESA study recently reported on the association of CAC scores with first CHD events over a median follow-up of 3.9 years among a population-based sample of 6722 men and women (39% white, 27% black, 22% Hispanic, and 12% Chinese).[9]
  - — Chart 4-3 shows the HRs associated with CAC scores of 1 to 100, 101 to 300, and >300 compared with those without CAC (score=0), after adjustment for standard risk factors. People with CAC scores of 1 to 100 had ≈4 times greater risk and those with CAC scores >100 were 7 to 10 times more likely to experience a coronary event than those without CAC.
  - — CAC provided similar predictive value for coronary events in whites, Chinese, blacks, and Hispanics (HRs ranging from 1.15–1.39 for each doubling of coronary calcium).
- In another report of a community-based sample, not referred for clinical reasons, the South Bay Heart Watch examined CAC in 1461 adults (average age 66 years) with coronary risk factors, with a median of 7.0 years of follow-up.[10]
  - — Chart 4-4 shows the HRs associated with increasing CAC scores (relative to CAC=0 and <10% risk category) in low-risk (<10%), intermediate-risk (10% to 15% and 16% to 20%), and high-risk (>20%) FRS categories of estimated risk for CHD in 10 years. Increasing CAC scores further predicted risk in intermediate- and high-risk groups.
- In a study of healthy adults 60 to 72 years of age who were free of clinical CAD, predictors of the progression of CAC were assessed. Predictors tested included age, sex, race/ethnicity, smoking status, BMI, family history of CAD, C-reactive protein, several measures of DM, insulin levels, BP, and lipids. Insulin resistance, in addition to the traditional cardiac risk factors, independently predicts progression of CAC.[11] Clinically, however, it is not yet recommended to conduct serial scanning of CAC to measure effects of therapeutic interventions.
- A recent publication from MESA also used CAC, in particular, and carotid IMT to stratify CHD and CVD event risk in people with metabolic syndrome and DM; those with low levels of CAC or carotid IMT have CHD and CVD event rates as low as many people without metabolic syndrome and DM. Those with DM who have CAC scores <100 have annual CHD event rates of <1%.[12]
- It is noteworthy, as recently demonstrated in MESA in 5878 participants with a median of 5.8 years of follow-up, that the addition of CAC to standard risk factors resulted in significant improvement of classification of risk for incident CHD events, placing 77% of people in the highest or lowest risk categories compared with 69% based on risk factors alone. An additional 23% of those who experienced events were reclassified as high risk, and 13% with events were reclassified as low risk.[13]
- The contribution of CAC to risk prediction has also been observed in other cohorts, including both the Heinz Nixdorf Recall study[14] and the Rotterdam study.[15]

CAC Progression and Risk

A recent report in 4609 individuals who had baseline and repeat cardiac CT found that progression of CAC in predicting future all-cause mortality provided only incremental information over baseline score, demographics, and cardiovascular risk factors.[16]

Carotid IMT

Background

- Carotid IMT measures the thickness of 2 layers (the intima and media) of the wall of the carotid arteries, the largest conduits of blood going to the brain. Carotid IMT is thought to be an even earlier manifestation of atherosclerosis than CAC, because thickening precedes the development of frank atherosclerotic plaque. Carotid IMT methods are still being refined, so it is important to know which part of the artery was measured (common carotid, internal carotid, or bulb) and whether near and far walls were both measured. This information can affect the average-thickness measurement that is usually reported.
- Unlike CAC, everyone has some thickness to the layers of their arteries, but people who develop atherosclerosis have greater thickness. Ultrasound of the carotid arteries can also detect plaques and determine the degree of narrowing of the artery they may cause. Epidemiological data, including the data discussed below, have indicated that high-risk levels of thickening might be considered as those in the

highest quartile or quintile for one's age and sex, or ≥1 mm.
- Although ultrasound is commonly used to diagnose plaque in the carotid arteries in people who have had strokes or who have bruits (sounds of turbulence in the artery), guidelines are limited as to screening of asymptomatic people with carotid IMT to quantify atherosclerosis or predict risk. However, some organizations have recognized that carotid IMT measurement by B-mode ultrasonography may provide an independent assessment of coronary risk.[17]

Prevalence and Association With Incident Cardiovascular Events

- The Bogalusa Heart Study measured carotid IMT in 518 black and white men and women at a mean age of 32±3 years. These men and women were healthy but overweight.[18]
  - The mean values of carotid IMT for the different segments are shown in Chart 4-5 by sex and race. Men had significantly higher carotid IMT in all segments than women, and blacks had higher common carotid and carotid bulb IMTs than whites.
  - Even at this young age, after adjustment for age, race, and sex, carotid IMT was associated significantly and positively with waist circumference, SBP, diastolic BP (DBP), and LDL cholesterol. Carotid IMT was inversely correlated with high-density lipoprotein (HDL) cholesterol levels. Participants with greater numbers of adverse risk factors (0, 1, 2, 3, or more) had stepwise increases in mean carotid IMT levels.
- In a subsequent analysis, the Bogalusa investigators examined the association of risk factors measured since childhood with carotid IMT measured in these young adults.[19] Higher BMI and LDL cholesterol levels measured at 4 to 7 years of age were associated with increased risk for being >75th percentile for carotid IMT in young adulthood. Higher SBP and LDL cholesterol and lower HDL cholesterol in young adulthood were also associated with having high carotid IMT. These data highlight the importance of adverse risk factor levels in early childhood and young adulthood in the early development of atherosclerosis.
- Among both women and men in MESA, blacks had the highest common carotid IMT, but they were similar to whites and Hispanics in internal carotid IMT. Chinese participants had the lowest carotid IMT, in particular in the internal carotid, of the 4 ethnic groups (Chart 4-6).
- The NHLBI's CHS reported follow-up of 4476 men and women ≥65 years of age (mean age 72 years) who were free of CVD at baseline.[20]
  - Mean maximal common carotid IMT was 1.03±0.20 mm, and mean internal carotid IMT was 1.37±0.55 mm.
  - After a mean follow-up of 6.2 years, those with maximal combined carotid IMT in the highest quintile had a 4- to 5-fold greater risk for incident heart attack or stroke than those in the bottom quintile. After adjustment for other risk factors, there was still a 2- to 3-fold greater risk for the top versus the bottom quintile.
- A study of 441 individuals ≤65 years of age without a history of CAD, DM, or hyperlipidemia who were examined for carotid IMT found 42% had high-risk carotid ultrasound findings (carotid IMT ≥75th percentile adjusted for age, sex, and race or presence of plaque). Among those with an FRS ≤5%, 38% had high-risk carotid ultrasound findings.[21]
- Conflicting data have been reported on the contribution of carotid IMT to risk prediction. In 13 145 participants in the NHLBI's ARIC study, the addition of carotid IMT combined with identification of plaque presence or absence to traditional risk factors reclassified risk in 23% of individuals overall, with a net reclassification improvement of 9.9%. There was a modest but statistically significant improvement in the area under the receiver operating characteristic curve, from 0.742 to 0.755.[22] In contrast, data reported recently from the Carotid Atherosclerosis Progression Study observed a net reclassification improvement of −1.4% that was not statistically significant.[23]

CAC and Carotid IMT

- In the NHLBI's MESA study of white, black, Chinese, and Hispanic adults 45 to 84 years of age, carotid IMT and CAC were found to be commonly associated, but patterns of association differed somewhat by sex and race.[24]
  - Common and internal carotid IMT were greater in women and men who had CAC than in those who did not, regardless of ethnicity.
  - Overall, CAC prevalence and scores were associated with carotid IMT, but associations were somewhat weaker in blacks than in other ethnic groups.
  - In general, blacks had the thickest carotid IMT of all 4 ethnic groups, regardless of the presence of CAC.
  - Common carotid IMT differed little by race/ethnicity in women with any CAC, but among women with no CAC, IMT was higher among blacks (0.86 mm) than in the other 3 groups (0.76–0.80 mm).
- In a more recent analysis from the NHLBI's MESA study, the investigators reported on follow-up of 6698 men and women in 4 ethnic groups over 5.3 years and compared the predictive utility of carotid IMT and CAC.[25]
  - CAC was associated more strongly than carotid IMT with the risk of incident CVD.
  - After adjustment for each other (CAC score and IMT) and for traditional CVD risk factors, the HR for CVD increased 2.1-fold for each 1-standard deviation increment of log-transformed CAC score versus 1.3-fold for each 1-standard deviation increment of the maximum carotid IMT.
  - For CHD events, the HRs per 1-standard deviation increment increased 2.5-fold for CAC score and 1.2-fold for IMT.
  - A receiver operating characteristic curve analysis also suggested that CAC score was a better predictor of incident CVD than was IMT, with

areas under the curve of 0.81 versus 0.78, respectively.

— Investigators from the NHLBI's CARDIA and MESA studies examined the burden and progression of subclinical atherosclerosis among adults <50 years of age. Ten-year and lifetime risks for CVD were estimated for each participant, and the participants were stratified into 3 groups: (1) those with low 10-year (<10%) and low lifetime (<39%) predicted risk for CVD; (2) those with low 10-year (<10%) but high lifetime (≥39%) predicted risk; and (3) those with high 10-year risk (>10%). The latter group had the highest burden and greatest progression of subclinical atherosclerosis. Given the young age of those studied, ≈90% of participants were at low 10-year risk, but of these, half had high predicted lifetime risk. Compared with those with low short-term/low lifetime predicted risks, those with low short-term/high lifetime predicted risk had significantly greater burden and progression of CAC and significantly greater burden of carotid IMT, even at these younger ages. These data confirm the importance of early exposure to risk factors for the onset and progression of subclinical atherosclerosis.[26]

CT Angiography

CT angiography is widely used by cardiologists to aid in the diagnosis of CAD, particularly when other test results may be equivocal. It is also of interest because of its ability to detect and possibly quantitate overall plaque burden and certain characteristics of plaques that may make them prone to rupture, such as positive remodeling or low attenuation. However, because of the limited outcome data in asymptomatic people, as well as the associated expense and risk of CT angiography (including generally higher radiation levels than CT scanning to detect CAC), current guidelines do not recommend its use as a screening tool for assessment of cardiovascular risk in asymptomatic people.[2]

Measures of Vascular Function and Incident CVD Events

Background

- Measures of arterial tonometry (stiffness) are based on the concept that pulse pressure has been shown to be an important risk factor for CVD. Arterial tonometry offers the ability to directly and noninvasively measure central pulse wave velocity in the thoracic and abdominal aorta.
- Brachial flow-mediated dilation (FMD) is a marker for nitric oxide release from the endothelium that can be measured by ultrasound. Impaired FMD is an early marker of CVD.
- Recommendations have not been specific, however, as to which, if any, measures of vascular function may be useful for CVD risk stratification in selected patient subgroups. Because of the absence of significant prospective data relating these measures to outcomes, latest guidelines do not currently recommend measuring either FMD or arterial stiffness for cardiovascular risk assessment in asymptomatic adults.[2]

Arterial Tonometry and CVD

- The Rotterdam Study measured arterial stiffness in 2835 elderly participants (mean age 71 years[27]). They found that as aortic pulse wave velocity increased, the risk of CHD was 1.72 (second versus first tertile) and 2.45 (third versus first tertile). Results remained robust even after accounting for carotid IMT, ankle-brachial index (ABI), and pulse pressure.
- A study from Denmark measured 1678 individuals 40 to 70 years of age and found that aortic pulse wave velocity increased CVD risk by 16% to 20%.[28]
- The FHS measured several indices of arterial stiffness, including pulse wave velocity, wave reflection, and central pulse pressure.[29] They found that not only was higher pulse wave velocity associated with a 48% increased risk of incident CVD events, but pulse wave velocity additionally improved CVD risk prediction (integrated discrimination improvement of 0.7%, $P<0.05$).

FMD and CVD

- The MESA study measured FMD in 3026 participants (mean age 61 years) who were free of CVD. As FMD increased (ie, improved brachial function), the risk of CVD was 16% lower.[30] FMD also improved CVD risk prediction compared with the FRS by improving net reclassification by 29%.

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Table 4-1. CAC Scores for the 75th Percentile of Men and Women of Different Race/Ethnic Groups, at Specified Ages

	75th Percentile CAC Scores*			
Age, y	Black	Chinese	Hispanic	White
Women				
45	0	0	0	0
55	0	2	0	1
65	26	45	19	54
75	138	103	116	237
Men				
45	0	3	0	0
55	15	34	27	68
65	95	121	141	307
75	331	229	358	820

CAC indicates coronary artery calcification.

*The 75th percentile CAC score is the score at which 75% of people of the same age, sex, and race have a score at or below this level, and 25% of people of the same age, sex, and race have a higher score. (Source: Multi-Ethnic Study of Atherosclerosis CAC Tools Web site: http://www.mesa-nhlbi.org/Calcium/input.aspx.)

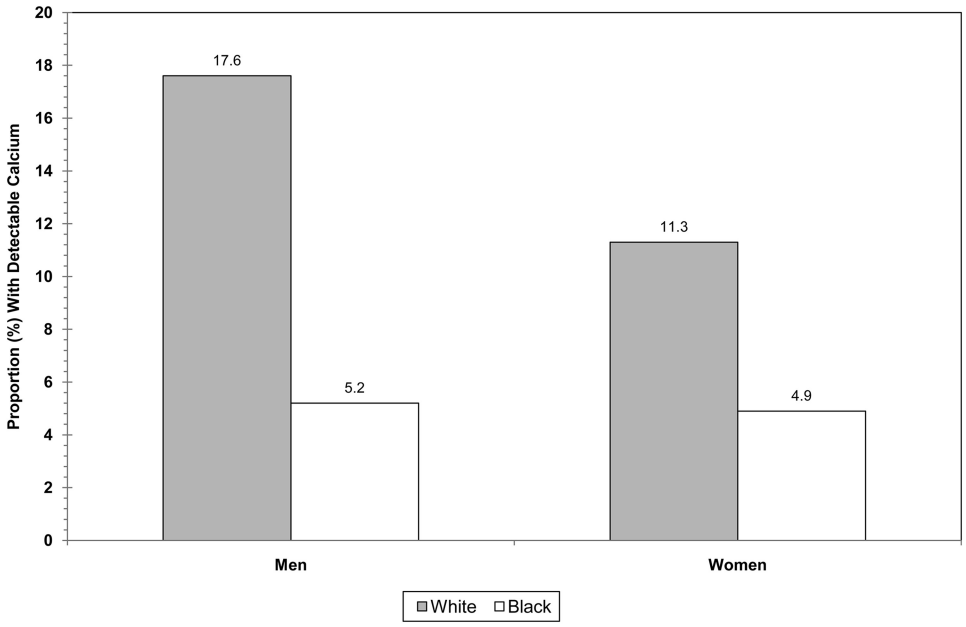

Chart 4-1. Prevalence (%) of coronary calcium: US adults 33 to 45 years of age. $P<0.0001$ across race-sex groups. Data derived from Loria et al.[5]

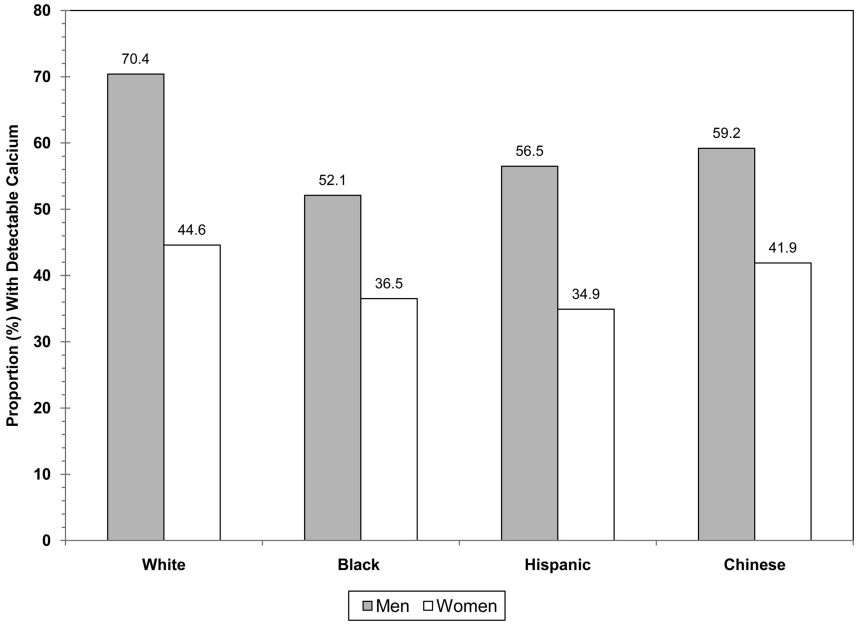

Chart 4-2. Prevalence (%) of coronary calcium: US adults 45 to 84 years of age. $P<0.0001$ across ethnic groups in both men and women. Data derived from Bild et al.[6]

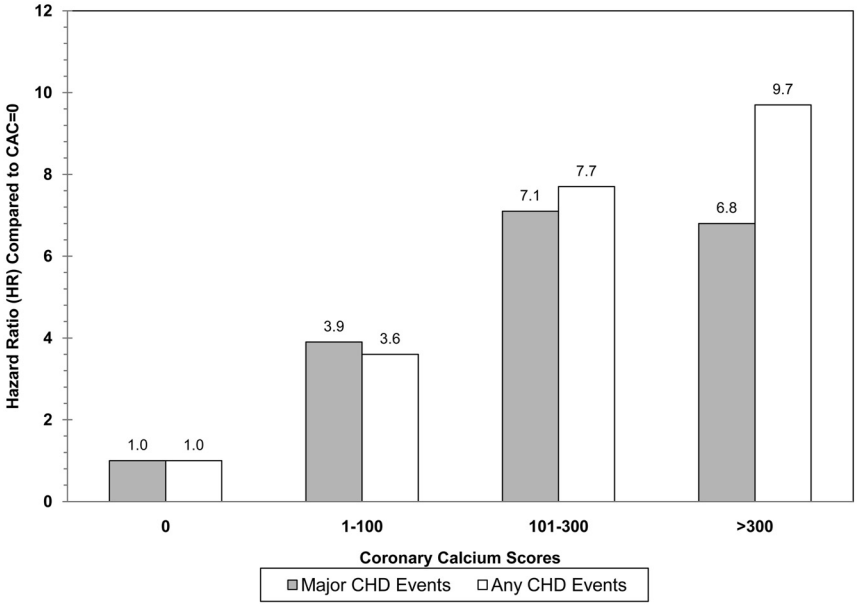

Chart 4-3. Hazard ratios (HRs) for coronary heart disease (CHD) events associated with coronary calcium scores: US adults 45 to 84 years of age (reference group: coronary artery calcification [CAC]=0). All HRs $P<0.0001$. Major CHD events included myocardial infarction and death attributable to CHD; any CHD events included major CHD events plus definite angina or definite or probable angina followed by revascularization. Data derived from Detrano et al.[9]

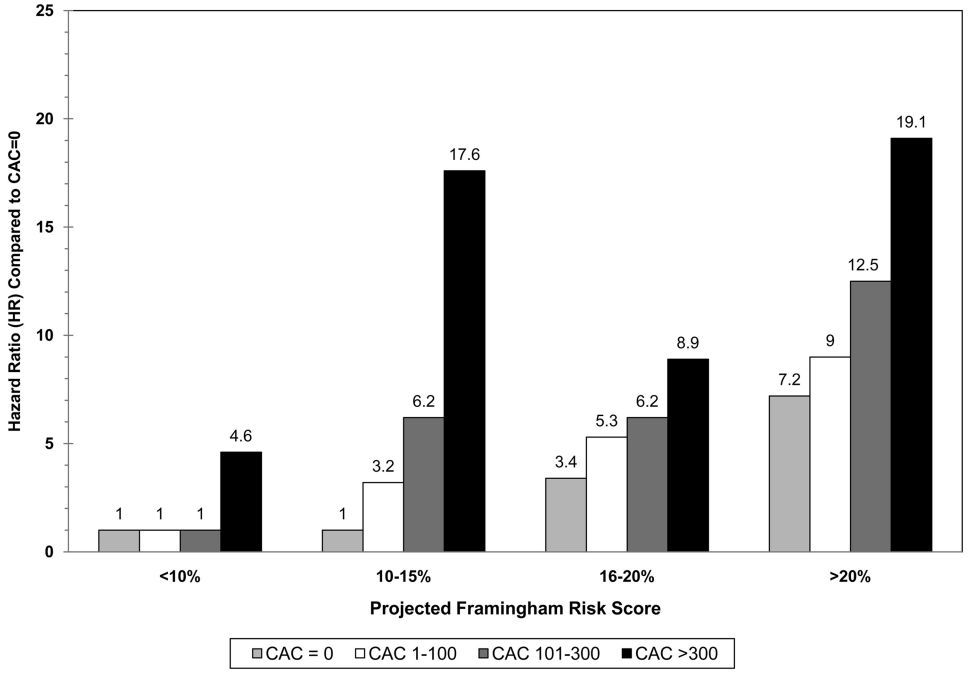

Chart 4-4. Hazard ratios (HRs) for coronary heart disease events associated with coronary calcium scores: US adults (reference group: coronary artery calcification [CAC]=0 and Framingham Risk Score <10%). Coronary heart disease events included nonfatal myocardial infarction and death attributable to coronary heart disease. Data derived from Greenland et al.[10]

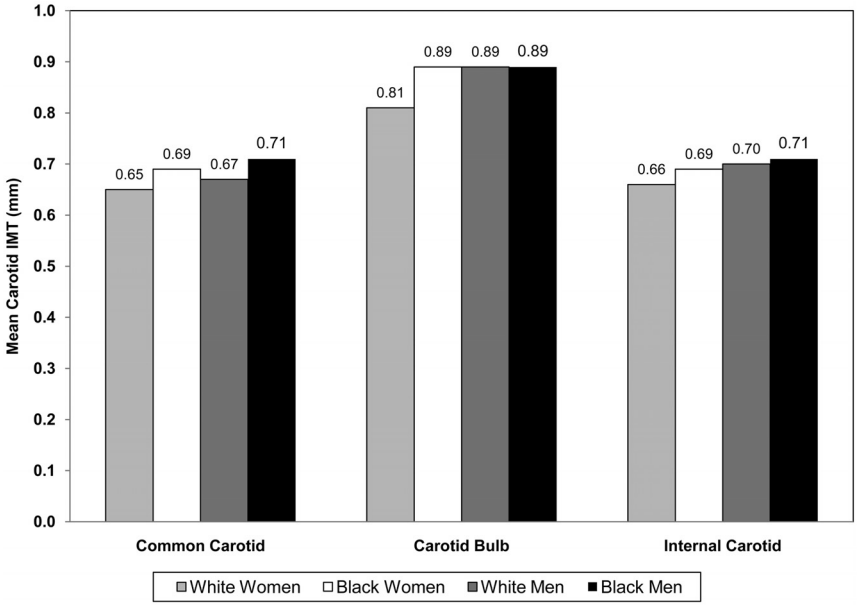

Chart 4-5. Mean values of carotid intima-media thickness (IMT) for different carotid artery segments in younger adults by race and sex (Bogalusa Heart Study). Data derived from Urbina et al.[18]

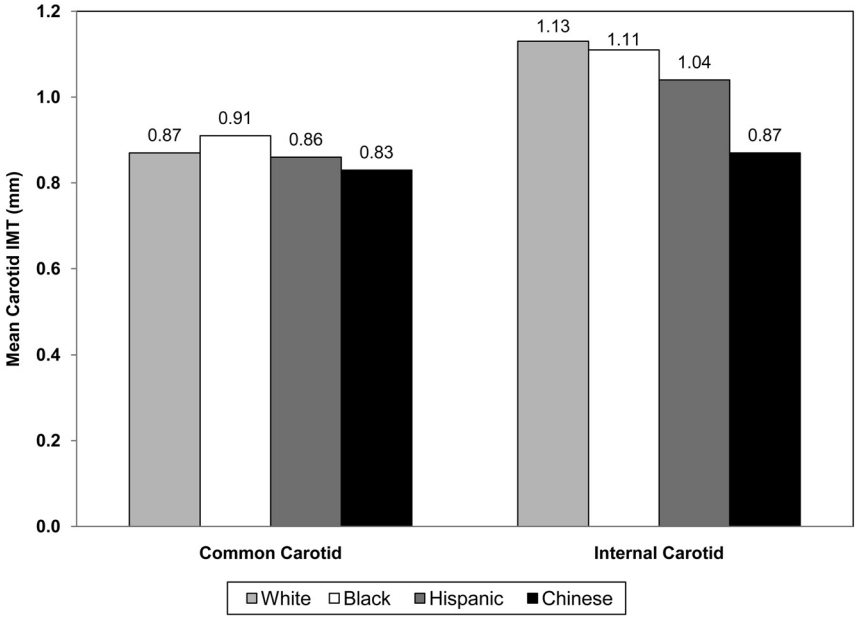

Chart 4-6. Mean values of carotid intima-media thickness (IMT) for different carotid artery segments in older adults, by race. Data derived from Manolio et al.[24]

5. Coronary Heart Disease, Acute Coronary Syndrome, and Angina Pectoris

Coronary Heart Disease

ICD-9 410 to 414, 429.2; ICD-10 I20 to I25; see Glossary (Chapter 25) for details and definitions. See Tables 5-1 and 5-2. See Charts 5-1 through 5-8.

Prevalence

- On the basis of data from NHANES 2005–2008 (NCHS; unpublished NHLBI tabulation; Table 5-1; Chart 5-1), an estimated 16.3 million Americans ≥20 years of age have CHD:
  - — Total CHD prevalence is 7.0% in US adults ≥20 years of age. CHD prevalence is 8.3% for men and 6.1% for women.
  - — Among non-Hispanic whites, CHD prevalence is 8.5% for men and 5.8% for women.
  - — Among non-Hispanic blacks, CHD prevalence is 7.9% for men and 7.6% for women.
  - — Among Mexican Americans, CHD prevalence is 6.3% for men and 5.6% for women.
- On the basis of data from the 2010 NHIS:
  - — Among Hispanic or Latino individuals ≥18 years of age, CHD prevalence is 5.2% (2010 NHIS, NCHS).[1]
  - — Among American Indian/Alaska Natives ≥18 years of age, it is estimated that 5.9% have CHD, and among Asians ≥18 years of age, the estimate is 4.9% (2010 NHIS, NCHS).[1]
- According to data from NHANES 2005–2008 (NCHS; unpublished NHLBI tabulation), the overall prevalence for MI is 3.1% in US adults ≥20 years of age. MI prevalence is 4.3% for men and 2.2% for women.
  - — Among non-Hispanic whites, MI prevalence is 4.3% for men and 2.1% for women.
  - — Among non-Hispanic blacks, MI prevalence is 4.3% for men and 2.2% for women.
  - — Among Mexican Americans, MI prevalence is 3.0% for men and 1.1% for women.
- Data from the BRFSS 2010 survey indicated that 4.2% of respondents had been told that they had an MI. The highest prevalence was in Arizona (6.7%) and West Virginia (6.3%). The lowest prevalence was in Alaska (2.6%) and Utah (2.8%). In the same survey, 4.1% of respondents were told that they had angina or CHD. The highest prevalence was in Arizona (6.8%), and the lowest was in Hawaii (2.3%).[2]
- Projections show that by 2030 an additional 8 million people could have CHD, a 16.6% increase in prevalence from 2010.[3]

Abbreviations Used in Chapter 5

ACC	American College of Cardiology	HDL	high-density lipoprotein
ACS	acute coronary syndrome	HF	heart failure
AHA	American Heart Association	ICD-9	International Classification of Diseases, 9th Revision
AMI	acute myocardial infarction	ICD-10	International Classification of Diseases, 10th Revision
AP	angina pectoris	LDL	low-density lipoprotein
ARIC	Atherosclerosis Risk in Communities study	MEPS	Medical Expenditure Panel Survey
BMI	body mass index	MI	myocardial infarction
BP	blood pressure	NAMCS	National Ambulatory Medical Care Survey
BRFSS	Behavioral Risk Factor Surveillance System	NCHS	National Center for Health Statistics
CABG	coronary artery bypass graft	NH	non-Hispanic
CAD	coronary artery disease	NHAMCS	National Hospital Ambulatory Medical Care Survey
CDC	Centers for Disease Control and Prevention	NHANES	National Health and Nutrition Examination Survey
CHD	coronary heart disease	NHDS	National Hospital Discharge Survey
CHS	Cardiovascular Health Study	NHIS	National Health Interview Study
CI	confidence interval	NHLBI	National Heart, Lung, and Blood Institute
CRUSADE	Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the ACC/AHA Guidelines	NRMI	National Registry of Myocardial Infarction
CVD	cardiovascular disease	NSTEMI	non–ST-segment–elevation myocardial infarction
DM	diabetes mellitus	OR	odds ratio
ECG	electrocardiogram/electrocardiographic	PA	physical activity
ED	emergency department	PCI	percutaneous coronary intervention
EMS	emergency medical services	PREMIER	Prospective Registry Evaluating Myocardial Infarction: Events and Recovery
FHS	Framingham Heart Study	SBP	systolic blood pressure
GRACE	Global Registry of Acute Coronary Events	STEMI	ST-segment–elevation myocardial infarction
GWTG	Get With The Guidelines	UA	unstable angina
HD	heart disease		

Incidence

- On the basis of unpublished data from the ARIC and CHS studies of the NHLBI:
  - This year, ≈785 000 Americans will have a new coronary attack, and ≈470 000 will have a recurrent attack. It is estimated that an additional 195 000 silent MIs occur each year. That assumes that ≈21% of the 935 000 first and recurrent MIs are silent.[4,5]
  - The estimated annual incidence of MI is 610 000 new attacks and 325 000 recurrent attacks.
  - Average age at first MI is 64.5 years for men and 70.3 years for women.
- On the basis of the NHLBI-sponsored FHS:
  - CHD makes up more than half of all cardiovascular events in men and women <75 years of age.[4]
  - The lifetime risk of developing CHD after 40 years of age is 49% for men and 32% for women.[6]
  - The incidence of CHD in women lags behind men by 10 years for total CHD and by 20 years for more serious clinical events such as MI and sudden death.[4]
- In the NHLBI-sponsored ARIC study, in participants 45 to 64 years of age, the average age-adjusted CHD incidence rates per 1000 person-years were as follows: white men, 12.5; black men, 10.6; white women, 4.0; and black women, 5.1. Incidence rates excluding revascularization procedures were as follows: white men, 7.9; black men, 9.2; white women, 2.9; and black women, 4.9.[7]
- Incidence rates for MI in the NHLBI-sponsored ARIC study are displayed in Charts 5-3 and 5-4, stratified by age, race, and sex. The annual age-adjusted rates per 1000 population of first MI (1987–2001) in ARIC Surveillance (NHLBI) were 4.2 in black men, 3.9 in white men, 2.8 in black women, and 1.7 in white women.[8]
- Analysis of more than 40 years of physician-validated AMI data in the FHS study of the NHLBI found that AMI rates diagnosed by electrocardiographic (ECG) criteria declined ≈50%, with a concomitant 2-fold increase in rates of AMI diagnosed by blood markers. These findings may explain the paradoxical stability of AMI rates in the United States despite concomitant improvements in CHD risk factors.[9]
- Among American Indians 65 to 74 years of age, the annual rates per 1000 population of new and recurrent MIs were 7.6 for men and 4.9 for women.[10] Analysis of data from NHANES III (1988–1994) and NHANES 1999–2002 (NCHS) showed that in adults 20 to 74 years of age, the overall distribution of 10-year risk of developing CHD changed little during this time. Among the 3 racial/ethnic groups, blacks had the highest proportion of participants in the high-risk group.[11]
- On the basis of data from the NHDS, since the mid-1990s, the rate of hospitalization for MI and in-hospital case fatality rates have decreased.[12]
- From 2002 to 2007, the rates of hospitalization for MI decreased among Medicare beneficiaries; however, the degree of reduction was more significant in whites than African Americans.[13]

Mortality

- CHD caused ≈1 of every 6 deaths in the United States in 2008. CHD mortality was 405 309.[14]
- CHD any-mention mortality was 571 366. MI mortality was 133 958. MI any-mention mortality was 172 733 (NHLBI tabulation; NCHS public-use data files).[14]
- In 2008, the overall CHD death rate was 122.7. From 1998 to 2008, the annual death rate due to CHD declined 28.7% and actual number of deaths declined 11.9%. The death rates were 161.7 for white males and 183.7 for black males; for white females, the rate was 91.9 and for black females it was 115.6 (NHLBI tabulation; NCHS public-use data files).[14]
- Approximately every 25 seconds, an American will experience a coronary event, and approximately every minute, someone will die of one.
- Approximately 34% of the people who experience a coronary attack in a given year will die of it, and ≈15% who experience a heart attack (MI) will die of it (AHA computation).
- Approximately every 34 seconds, an American will have an MI.
- The percentage of CHD deaths that occurred out of the hospital in 2008 was 70%. According to NCHS mortality data, 287 000 CHD deaths occur out of the hospital or in hospital EDs annually (2008, ICD-10 codes I20 to I25) (NHLBI tabulation of NCHS mortality data).
- A study of 1275 health maintenance organization enrollees 50 to 79 years of age who had cardiac arrest showed that the incidence of out-of-hospital cardiac arrest was 6.0/1000 subject-years in subjects with any clinically recognized HD compared with 0.8/1000 subject-years in subjects without HD. In subgroups with HD, incidence was 13.6/1000 subject-years in subjects with prior MI and 21.9/1000 subject-years in subjects with HF.[15]
- Approximately 81% of people who die of CHD are ≥65 years of age (NCHS; AHA computation).
- The estimated average number of years of life lost because of an MI is 16.6 (NHLBI tabulation of NCHS mortality data).
- On the basis of data from the FHS of the NHLBI[4]:
  - Fifty percent of men and 64% of women who die suddenly of CHD have no previous symptoms of this disease. Between 70% and 89% of sudden cardiac deaths occur in men, and the annual incidence is 3 to 4 times higher in men than in women; however, this disparity decreases with advancing age.
  - People who have had an MI have a sudden death rate 4 to 6 times that of the general population.
- Researchers investigating variation in hospital-specific 30-day risk-stratified mortality rates for patients with AMI found teaching status, number of hospital beds, AMI volume, cardiac facilities available, urban/rural location, geographic region, hospital ownership type, and socioeco-

nomic status profile of the patients were all significantly associated with mortality rates. However, a substantial proportion of variation in outcomes for patients with AMI between hospitals remains unexplained by measures of hospital characteristics.[16]

Temporal Trends in CHD Mortality

- An analysis of FHS data (NHLBI) from 1950 to 1999 showed that overall CHD death rates decreased by 59%. Nonsudden CHD death decreased by 64%, and sudden cardiac death fell by 49%. These trends were seen in men and women, in subjects with and without a prior history of CHD, and in smokers and nonsmokers.[17]
- The decline in CHD mortality rates in part reflects the shift in the pattern of clinical presentations of AMI. In the past decade, there has been a marked decline in ST-segment–elevation myocardial infarction (STEMI; from 133 to 50 cases per 100 000 person-years).[18]
- From 1997 to 2007, the annual death rate attributable to CHD declined 26.3%, and the actual number of deaths declined 12.9%. (Appropriate comparability ratios were applied.) In 2007, the overall CHD death rate was 126.0 per 100 000 population. The death rates were 165.6 for white males and 191.6 for black males; for white females, the rate was 94.2, and for black females, it was 121.5.[14] Age-adjusted death rates attributable to CHD were 122.3 for Hispanic or Latino males and 77.8 for females, 112.2 for American Indian or Alaska Native males and 65.6 for females, and 91.7 for Asian or Pacific Islander males and 55.0 for females.[14]
- According to data from the National Registry of Myocardial Infarction[19]:
  - From 1990 to 1999, in-hospital AMI mortality declined from 11.2% to 9.4%.
  - Mortality rate increases for every 30 minutes that elapse before a patient with ST-segment elevation is recognized and treated.
- Other studies also reported declining case fatality rates after MI:
  - In Olmsted County, Minnesota, the age- and sex-adjusted 30-day case fatality rate decreased by 56% from 1987 to 2006.[20]
  - In Worcester, MA, the hospital case fatality rates, 30-day postadmission case fatality rates, and 1-year postdischarge case fatality rates for STEMI were 11.1%, 13.2%, and 10.6%, respectively, in 1997 and 9.7%, 11.4%, and 8.4%, respectively, in 2005. The hospital case fatality rates, 30-day postadmission case fatality rates, and 1-year postdischarge case fatality rates for non–ST-segment MI (NSTEMI) were 12.9%, 16.0%, and 23.1%, respectively, in 1997 and 9.5%, 14.0%, and 18.7%, respectively, in 2005.[21]
  - Among enrollees of the Kaiser Permanente Northern California healthcare delivery system, the age- and sex-adjusted 30-day mortality rate for MI dropped from 10.5% in 1999 to 7.8% in 2008, and the 30-day mortality rate for NSTEMI dropped from 10.0% in 1999 to 7.6% in 2008.[18]
- CHD death rates have fallen from 1968 to the present. Analysis of NHANES (NCHS) data compared CHD death rates between 1980 and 2000 to determine how much of the decline in deaths attributable to CHD over that period could be explained by the use of medical and surgical treatments versus changes in CVD risk factors (resulting from lifestyle/behavior). After 1980 and 2000 data were compared, it was estimated that ≈47% of the decrease in CHD deaths was attributable to treatments, including the following[22]:
  - Secondary preventive therapies after MI or revascularization (11%).
  - Initial treatments for AMI or UA (10%).
  - Treatments for HF (9%).
  - Revascularization for chronic angina (5%).
  - Other therapies (12%), including antihypertensive and lipid-lowering primary prevention therapies.
- It was also estimated that a similar amount of the reduction in CHD deaths, ≈44%, was attributable to changes in risk factors, including the following[22]:
  - Lower total cholesterol (24%).
  - Lower SBP (20%).
  - Lower smoking prevalence (12%).
  - Decreased physical inactivity (5%).
  - Nevertheless, these favorable improvements in risk factors were offset in part by increases in BMI and in DM prevalence, which accounted for an increased number of deaths (8% and 10%, respectively).
- Between 1980 and 2002, death rates attributable to CHD among men and women ≥65 years of age fell by 52% in men and 49% in women. Among men, the death rate declined on average by 2.9% per year in the 1980s, 2.6% per year during the 1990s, and 4.4% per year from 2000 to 2002. Among women, death rates fell by 2.6%, 2.4%, and 4.4%, respectively. However, when stratified by age, among men 35 to 54 years of age, the average annual rate of death fell by 6.2%, 2.3%, and 0.5%, respectively. Among women 35 to 54 years of age, the average annual rate of death fell by 5.4% and 1.2% and then increased by 1.5%, respectively. This increase was not statistically significant; however, in even younger women (35–44 years of age), the rate of death has been increasing by an average of 1.3% annually between 1997 and 2002, which is statistically significant.[23]
- An analysis of 28 studies published from 1977 to 2007 found that revascularization by coronary bypass surgery or percutaneous intervention in conjunction with medical therapy in patients with nonacute CAD is associated with significantly improved survival compared with medical therapy alone.[24]
- A recent analysis of Centers for Medicare & Medicaid Services data suggests that between 1995 and 2006, the 30-day mortality rate attributable to MI decreased, as did hospital variation in mortality attributable to MI.[25]

- Data from the Nationwide Inpatient Sample database suggest that mortality attributable to MI has decreased since 1988.[26]

Risk Factors

- Risk factors for CHD act synergistically to increase CHD risk, as shown in the example in Chart 5-6.
- A study of men and women in 3 prospective cohort studies found that antecedent major CHD risk factor exposures were common among those who developed CHD. Approximately 90% of patients with CHD have prior exposure to at least 1 of these major risk factors, which include high total blood cholesterol levels or current medication with cholesterol-lowering drugs, hypertension or current medication with BP-lowering drugs, current cigarette use, and clinical report of DM.[27]
- According to a case-control study of 52 countries (INTERHEART), optimization of 9 easily measured and potentially modifiable risk factors could result in a 90% reduction in the risk of an initial AMI. The effect of these risk factors is consistent in men and women across different geographic regions and by ethnic group, which makes the study applicable worldwide. These 9 risk factors include cigarette smoking, abnormal blood lipid levels, hypertension, DM, abdominal obesity, a lack of PA, low daily fruit and vegetable consumption, alcohol overconsumption, and psychosocial index.[28]
- A study of >3000 members of the FHS (NHLBI) Offspring Cohort without CHD showed that among men with 10-year predicted risk for CHD of 20%, both failure to reach target heart rate and ST-segment depression more than doubled the risk of an event, and each metabolic equivalent increment in exercise capacity reduced risk by 13%.[29]
- An analysis of data from non-Hispanic white adults 35 to 74 years of age who participated in NHANES III (NCHS) showed that 26% of men and 41% of women had at least 1 borderline risk factor (smoking, blood pressure, LDL cholesterol, HDL cholesterol, or glucose intolerance). Additional analyses using data from the FHS (NHLBI) indicated that >90% of hard CHD events over a 10-year period were projected to occur in non-Hispanic white adults 35 to 74 years of age with at least 1 elevated risk factor and ≈8% in adults with only borderline levels of risk factors.[30]
- A recent analysis examined the number and combination of risk factors necessary to exceed Adult Treatment Panel III treatment thresholds. In this analysis, relatively high risk factor levels were required to exceed Adult Treatment Panel III treatment thresholds in men <45 years of age and women <65 years of age, which suggests that alternative means of risk prediction that focus on a longer time horizon than the 10 years captured by the traditional Framingham CHD risk score may be necessary to estimate risk in these individuals.[31]
- Analysis of data from the CHS study (NHLBI) among participants ≥65 years of age at entry into the study showed that subclinical CVD is prevalent among older individuals, is independently associated with risk of CHD (even over a 10-year follow-up period), and substantially increases the risk of CHD among participants with hypertension or DM.[32]
- On the basis of data from the CDC/BRFSS, it was found that patients with CHD are less likely to comply with PA recommendations than are subjects without CHD. Only 32% of CHD patients met moderate PA recommendations, 22% met vigorous PA recommendations, and 40% met total PA recommendations. In contrast, the percentage of subjects without CHD who met PA recommendations was significantly higher, and this percentage almost achieved the Healthy People 2010 objectives for PA.[33]
- Analysis of data from the PREMIER trial (Prospective Registry Evaluating Myocardial Infarction: Events and Recovery), sponsored by the NHLBI, found that in people with prehypertension or stage 1 hypertension, 2 multicomponent behavioral interventions significantly reduced estimated 10-year CHD risk by 12% and 14%, respectively, compared with advice only.[34]

Awareness of Warning Signs and Risk Factors for HD

- Data from the Women Veterans Cohort showed that 42% of women ≥35 years of age were concerned about HD. Only 8% to 20% were aware that CAD is the major cause of death for women.[35]
- Among people in 14 states and Washington, DC, participating in the 2005 BRFSS, only 27% were aware of 5 heart attack warning signs and symptoms (1, pain in jaw, neck, or back; 2, weak, lightheaded, or faint; 3, chest pain or discomfort; 4, pain or discomfort in arms or shoulder; and 5, shortness of breath) and indicated that they would first call 911 if they thought someone was having a heart attack or stroke. Awareness of all 5 heart attack warning signs and symptoms and the need to call 911 was higher among non-Hispanic whites (30.2%), women (30.8%), and those with a college education or more (33.4%) than among non-Hispanic blacks and Hispanics (16.2% and 14.3%, respectively), men (22.5%), and those with less than a high school education (15.7%), respectively. By state, awareness was highest in West Virginia (35.5%) and lowest in Washington, DC (16.0%).[36]
- A 2004 national study of physician awareness and adherence to CVD prevention guidelines showed that fewer than 1 in 5 physicians knew that more women than men die each year of CVD.[37] Women's awareness that CVD is their leading cause of death increased from 30% in 1997% to 54% in 2009.[38]
- A recent community surveillance study in 4 US communities reported that in 2000, the overall proportion of people with delays of ≥4 hours from onset of AMI symptoms to hospital arrival was 49.5%. The study also reported that from 1987 to 2000, there was no statistically significant change in the proportion of patients whose delays were ≥4 hours, which indicates that there has been little improvement in the speed at which patients with MI symptoms arrive at the hospital after symptom onset. Although the proportion of patients with MI who arrived at the hospital by emergency medical services (EMS) increased over this period, from 37% in 1987 to 55% in 2000, the total time

between onset and hospital arrival did not change appreciably.[39]

- According to 2003 data from the BRFSS (CDC), 36.5% of all women surveyed had multiple risk factors for HD and stroke. The age-standardized prevalence of multiple risk factors was lowest in whites and Asians. After adjustment for age, income, education, and health coverage, the odds for multiple risk factors were greater in black and Native American women and lower for Hispanic women than for white women. Prevalence estimates and odds of multiple risk factors increased with age; decreased with education, income, and employment; and were lower in those with no health coverage. Smoking was more common in younger women, whereas older women were more likely to have medical conditions and to be physically inactive.[40]
- Individuals with documented CHD have 5 to 7 times the risk of having a heart attack or dying as the general population. Survival rates improve after a heart attack if treatment begins within 1 hour; however, most patients are admitted to the hospital 2.5 to 3 hours after symptoms begin. More than 3500 patients surveyed with a history of CHD were asked to identify possible symptoms of heart attack. Despite their history of CHD, 44% had low knowledge levels. In this group, who were all at high risk of future AMI, 43% assessed their risk as less than or the same as others their age. More men than women perceived themselves as being at low risk, at 47% versus 36%, respectively.[41]
- Data from Worcester, MA, indicate that the average time from symptom onset to hospital arrival has not improved and that delays in hospital arrival are associated with less receipt of guidelines-based care. Mean and median prehospital delay times from symptom onset to arrival at the hospital were 4.1 and 2.0 hours in 1986 and 4.6 and 2.0 hours in 2005, respectively. Compared with those arriving within 2 hours of symptom onset, those with prolonged prehospital delay were less likely to receive thrombolytic therapy and PCI within 90 minutes of hospital arrival.[42]
- In an analysis from ARIC, low neighborhood household income (odds ratio [OR] 1.46, 95% confidence interval [CI] 1.09–1.96) and being a Medicaid recipient (OR 1.87, 95% CI 1.10–3.19) were associated with increased odds of having prolonged prehospital delays from symptom onset to hospital arrival for AMI compared with individuals with higher neighborhood household income and other insurance providers, respectively.[43]

Aftermath

- Depending on their sex and clinical outcome, people who survive the acute stage of an MI have a chance of illness and death 1.5 to 15 times higher than that of the general population. Among these people, the risk of another MI, sudden death, AP, HF, and stroke—for both men and women—is substantial (FHS, NHLBI).[4]
- On the basis of pooled data from the FHS, ARIC, and CHS studies of the NHLBI, within 1 year after a first MI:
  - At ≥45 years of age, 19% of men and 26% of women will die.
  - At 45 to 64 years of age, 5% of white men, 9% of white women, 14% of black men, and 8% of black women will die.
  - At ≥65 years of age, 25% of white men, 30% of white women, 25% of black men, and 30% of black women will die.
  - In part because women have MIs at older ages than men, they are more likely to die of MIs within a few weeks.
- Within 5 years after a first MI:
  - At ≥45 years of age, 36% of men and 47% of women will die.
  - At 45 to 64 years of age, 11% of white men, 18% of white women, 22% of black men, and 28% of black women will die.
  - At ≥65 years of age, 46% of white men, 53% of white women, 54% of black men, and 58% of black women will die.
- Of those who have a first MI, the percentage with a recurrent MI or fatal CHD within 5 years is:
  - At 45 to 64 years of age, 15% of men and 22 of women.
  - At ≥65 years of age, 22% of men and women.
  - At 45 to 64 years of age, 14% of white men, 18% of white women, 22% of black men, and 28% of black women.
  - At ≥65 years of age, 21% of white men and women, 33% of black men, and 26% of black women.
- The percentage of people with a first MI who will have HF in 5 years is:
  - At 45 to 64 years of age, 8% of men and 18% of women.
  - At ≥65 years of age, 20% of men and 23% of women.
  - At 45 to 64 years of age, 7% of white men, 15% of white women, 13% of black men, and 25% of black women.
  - At ≥65 years of age, 19% of white men, 23% of white women, 31% of black men, and 24% of black women.
- The percentage of people with a first MI who will have a stroke within 5 years is:
  - At 45 to 64 years of age, 2% of men and 6% of women.
  - At ≥65 years of age, 5% of men and 8% of women.
  - At 45 to 64 years of age, 2% of white men, 4% of white women, 3% of black men, and 10% of black women.
  - At ≥65 years of age, 5% of white men, 8% of white women, 9% of black men, and 10% of black women.
- The median survival time (in years) after a first MI is:
  - At 55 to 64 years of age, 17.0 for men and 13.3 for women.
  - At 65 to 74 years of age, 9.3 for men and 8.8 for women.
  - At ≥75 years of age, 3.2 for men and 3.2 for women.

- A Mayo Clinic study found that cardiac rehabilitation after an MI is underused, particularly in women and the elderly. Women were 55% less likely than men to participate in cardiac rehabilitation, and older study patients were less likely to participate than younger participants. Only 32% of men and women ≥70 years of age participated in cardiac rehabilitation compared with 66% of those 60 to 69 years of age and 81% of those <60 years of age.[44]
- Among survivors of an MI, in 2005, 34.7% of BRFSS respondents participated in outpatient cardiac rehabilitation. The prevalence of cardiac rehabilitation was higher among older age groups (≥50 years of age), among men versus women, among Hispanics, among those who were married, among those with higher education, and among those with higher levels of household income.[45]
- A recent analysis of Medicare claims data revealed that only 13.9% of Medicare beneficiaries enroll in cardiac rehabilitation after an AMI, and only 31% enroll after CABG. Older people, women, nonwhites, and individuals with comorbidities were less likely to enroll in cardiac rehabilitation programs.[46]

Hospital Discharges and Ambulatory Care Visits

(See Table 5-1 and Chart 5-7.)

- From 1999 to 2009, the number of inpatient discharges from short-stay hospitals with CHD as the first-listed diagnosis decreased from 2 270 000 to 1 537 000 (NHLBI tabulation of NHDS, NCHS).
- In 2009, there were 14 044 000 ambulatory care visits with CHD as the first-listed diagnosis (NCHS, NAMCS, NHAMCS). There were 12 816 000 physician office visits, 639 000 ED visits, and 589 000 outpatient department visits with a primary diagnosis of CHD (unpublished data, NCHS, NHAMCS, NHLBI tabulation). The majority of these visits (77.7%) were for coronary atherosclerosis.[47]
- Age-adjusted hospitalization rate for MI was 215 per 100 000 people in 1979 to 1981, increased to 342 in 1985 to 1987, stabilized for the next decade, and then declined after 1996 to 242 during the period from 2003 to 2005. Rates for men were almost twice those of women. Trends were similar for men and women. Hospitalization rates increased with age and were the highest among those ≥85 years of age.[12]
- Most hospitalized patients >65 years of age are women. For MI, 28.4% of hospital stays for people 45 to 64 years of age were for women, but 63.7% of stays for those ≥85 years of age were for women. Similarly, for coronary atherosclerosis, 32.7% of stays among people 45 to 64 years of age were for women; this figure increased to 60.7% of stays among those ≥85 years of age. For nonspecific chest pain, women were more numerous than men among patients <65 years of age. Approximately 54.4% of hospital stays among people 45 to 64 years of age were for women. Women constituted 73.9% of hospital stays for nonspecific chest pain among patients ≥85 years of age, higher than for any other condition examined. For AMI, one third more women than men died in the hospital: 9.3% of women died in the hospital compared with 6.2% of men.[48]

Operations and Procedures

- In 2009, an estimated 1 133 000 inpatient PCI procedures, 416 000 inpatient bypass procedures, 1 072 000 inpatient diagnostic cardiac catheterizations, 116 000 inpatient implantable defibrillator procedures, and 397 000 pacemaker procedures were performed for inpatients in the United States. (NHLBI, NCHS, unpublished tabulation).

Cost

- The estimated direct and indirect cost of heart disease in 2008 is $190.3 billion (MEPS, NHLBI tabulation).
- In 2006, $11.7 billion was paid to Medicare beneficiaries for in-hospital costs when CHD was the principal diagnosis ($14 009 per discharge for AMI, $12 977 per discharge for coronary atherosclerosis, and $10 630 per discharge for other ischemic HD).[41,49]
- Over the next 20 years, medical costs of CHD (real 2008$) are projected to increase by ≈200%:
  - Indirect costs for all CVD (real 2008$) are projected to increase 61% (from $171.7 billion to $275.8 billion) between 2010 and 2030. Of these indirect costs, CHD is projected to account for ≈40% and has the largest indirect costs.[3]

Acute Coronary Syndrome

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

The term acute coronary syndrome (ACS) is increasingly used to describe patients who present with either AMI or UA. (UA is chest pain or discomfort that is accelerating in frequency or severity and may occur while at rest but does not result in myocardial necrosis.) The discomfort may be more severe and prolonged than typical AP or may be the first time a person has AP. UA, NSTEMI, and STEMI share common pathophysiological origins related to coronary plaque progression, instability, or rupture with or without luminal thrombosis and vasospasm.

- A conservative estimate for the number of discharges with ACS from hospitals in 2009 is 683 000. Of these, an estimated 399 000 are males and 284 000 are females. This estimate is derived by adding the first-listed inpatient hospital discharges for MI (634 000) to those for UA (49 000; NHDS, NHLBI).
- When secondary discharge diagnoses in 2009 were included, the corresponding number of inpatient hospital discharges was 1 190 000 unique hospitalizations for ACS; 694 000 were males, and 496 000 were females. Of the total, 829 000 were for MI alone, 357 000 were for UA alone, and 4000 hospitalizations received both diagnoses (NHDS, NHLBI).

Decisions about medical and interventional treatments are based on specific findings noted when a patient presents with ACS. Such patients are classified clinically into 1 of 3 categories according to the presence or absence of ST-

segment elevation on the presenting ECG and abnormal ("positive") elevations of myocardial biomarkers, such as troponins, as follows:

- STEMI
- NSTEMI
- UA

The percentage of ACS or MI cases with ST-segment elevation varies in different registries/databases and depends heavily on the age of patients included and the type of surveillance used. According to the National Registry of Myocardial Infarction 4 (NRMI-4), ≈29% of patients with MI are patients with STEMI.[50] The AHA Get With The Guidelines (GWTG) project found that 32% of the patients with MI in the CAD module are patients with STEMI (personal communication from AHA GWTG staff, October 1, 2007). The Global Registry of Acute Coronary Events (GRACE) study, which includes US patient populations, found that 38% of ACS patients have STEMI, whereas the second Euro Heart Survey on ACS (EHS-ACS-II) reported that ≈47% of patients with ACS have STEMI.[51]

In addition, the percentage of ACS or MI cases with ST-segment elevation appears to be declining. In an analysis of 46 086 hospitalizations for ACS in the Kaiser Permanente Northern California study, the percentage of MI cases with ST-segment elevation decreased from 48.5% to 24% between 1999 and 2008.[18]

- Analysis of data from the GRACE multinational observational cohort study of patients with ACS found evidence of a change in practice for both pharmacological and interventional treatments in patients with either STEMI or non–ST-segment–elevation ACS. These changes have been accompanied by significant decreases in the rates of in-hospital death, cardiogenic shock, and new MI among patients with non–ST-segment–elevation ACS. The use of evidence-based therapies and PCI interventions increased in the STEMI population. This increase was matched with a statistically significant decrease in the rates of death, cardiogenic shock, and HF or pulmonary edema.[52]
- A study of patients with non–ST-segment–elevation ACS treated at 350 US hospitals found that up to 25% of opportunities to provide American College of Cardiology (ACC)/AHA guideline–recommended care were missed in current practice. The composite guideline adherence rate was significantly associated with in-hospital mortality.[52]
- A study of hospital process performance in 350 centers of nearly 65 000 patients enrolled in the CRUSADE (Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the ACC/AHA Guidelines) National Quality Improvement Initiative found that ACC/AHA guideline–recommended treatments were adhered to in 74% of eligible instances.[53]
- After adjustment for clinical differences and the severity of CAD by angiogram, 30-day mortality after ACS is similar in men and women.[54]

Angina Pectoris

ICD-9 413; ICD-10 I20.1 to I20.9. See Table 5-2 and Chart 5-5.

Prevalence

- A study of 4 national cross-sectional health examination studies found that among Americans 40 to 74 years of age, the age-adjusted prevalence of AP was higher among women than men. Increases in the prevalence of AP occurred for Mexican American men and women and African American women but were not statistically significant for the latter.[55]

Incidence

- Only 18% of coronary attacks are preceded by long-standing AP (NHLBI computation of FHS follow-up since 1986).
- The annual rates per 1000 population of new episodes of AP for nonblack men are 28.3 for those 65 to 74 years of age, 36.3 for those 75 to 84 years of age, and 33.0 for those ≥85 years of age. For nonblack women in the same age groups, the rates are 14.1, 20.0, and 22.9, respectively. For black men, the rates are 22.4, 33.8, and 39.5, and for black women, the rates are 15.3, 23.6, and 35.9, respectively (CHS, NHLBI).[8]
- On the basis of 1987 to 2001 data from the ARIC study of the NHLBI, the annual rates per 1000 population of new episodes of AP for nonblack men are 8.5 for those 45 to 54 years of age, 11.9 for those 55 to 64 years of age, and 13.7 for those 65 to 74 years of age. For nonblack women in the same age groups, the rates are 10.6, 11.2, and 13.1, respectively. For black men, the rates are 11.8, 10.6, and 8.8, and for black women, the rates are 20.8, 19.3, and 10.0, respectively.[8]

Mortality

A small number of deaths resulting from CHD are coded as being attributable to AP. These are included as a portion of total deaths attributable to CHD.

Cost

For women with nonobstructive CHD enrolled in the Women's Ischemia Syndrome Evaluation (WISE) study of the NHLBI, the average lifetime cost estimate was ≈$770 000 and ranged from $1.0 to $1.1 million for women with 1-vessel to 3-vessel CHD.[56]

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Table 5-1. Coronary Heart Disease

Population Group	Prevalence, CHD, 2008 Age ≥20 y	Prevalence, MI, 2008 Age ≥20 y	New and Recurrent MI and Fatal CHD, Age ≥35 y	New and Recurrent MI, Age ≥35 y	Mortality,* CHD, 2008, All Ages	Mortality,* MI, 2008, All Ages	Hospital Discharges, CHD, 2009, All Ages
Both sexes	16 300 000 (7.0%)	7 900 000 (3.1%)	1 255 000	935 000	405 309	133 958	1 537 000
Males	8 800 000 (8.3%)	4 800 000 (4.3%)	740 000	565 000	216 248 (53.4%)†	72 447 (54.1%)†	933 000
Females	7 500 000 (6.1%)	3 100 000 (2.2%)	515 000	370 000	189 061 (46.6%)†	61 511 (45.9%)†	604 000
NH white males	8.5%	4.3%	675 000‡	. . .	189 354	63 842	. . .
NH white females	5.8%	2.1%	445 000‡	. . .	164 485	53 276	. . .
NH black males	7.9%	4.3%	70 000‡	. . .	21 407	6883	. . .
NH black females	7.6%	2.2%	65 000‡	. . .	20 491	6908	. . .
Mexican American males	6.3%	3.0%	. . .	. . .	. . .	. . .	. . .
Mexican American females	5.6%	1.1%	. . .	. . .	. . .	. . .	. . .
Hispanic or Latino§	5.2%	. . .	. . .	. . .	. . .	. . .	. . .
Asian§	4.9%	. . .	. . .	. . .	7414	2448	. . .
American Indian/ Alaska Native§	55.9%	. . .	. . .	. . .	1777	601	. . .

CHD indicates coronary heart disease; MI, myocardial infarction; and NH, non-Hispanic.

CHD includes people who responded "yes" to at least 1 of the questions in "Has a doctor or other health professional ever told you had coronary heart disease, angina or angina pectoris, heart attack, or myocardial infarction?" Those who answered "no" but were diagnosed with Rose angina are also included (the Rose questionnaire is only administered to survey participants >40 years of age). Ellipses indicate data not available. Sources: Prevalence: National Health and Nutrition Examination Survey 2005–2008 (National Center for Health Statistics) and National Heart, Lung, and Blood Institute. Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2008 US population estimates. These data are based on self-reports. Incidence: Atherosclerosis Risk in Communities study (1987–2004), National Heart, Lung, and Blood Institute. Mortality: National Center for Health Statistics (these data represent underlying cause of death only). Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics (data include those inpatients discharged alive, dead, or status unknown).

*Mortality data for the white, black, Asian or Pacific Islander, and American Indian/Alaska Native populations include deaths of persons of Hispanic and non-Hispanic origin. Numbers of deaths for the American Indian/Alaska Native and Asian or Pacific Islander populations are known to be underestimated.

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

‡Estimates include Hispanics and non-Hispanics. Estimates for whites include other nonblack races.

§National Health Interview Survey, National Center for Health Statistics 2010; data are weighted percentages for Americans ≥18 years of age.[1]

Table 5-2. Angina Pectoris

Population Group	Prevalence, 2008 Age ≥20 y	Incidence of Stable AP, Age ≥45 y	Hospital Discharges, 2009,* All Ages
Both sexes	9 000 000 (3.9%)	500 000	34 000
Males	4 000 000 (3.8%)	320 000	19 000
Females	5 000 000 (4.0%)	180 000	15 000
NH white males	3.8%	. . .	. . .
NH white females	3.7%	. . .	. . .
NH black males	3.3%	. . .	. . .
NH black females	5.6%	. . .	. . .
Mexican American males	3.6%	. . .	. . .
Mexican American females	3.7%	. . .	. . .

AP indicates angina pectoris; NH, non-Hispanic; and ellipses, data not available.

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: National Health and Nutrition Examination Survey 2005–2008 (National Center for Health Statistics) and National Heart, Lung, and Blood Institute; percentages for racial/ethnic groups are age adjusted for US adults ≥20 years of age. AP includes persons who either answered "yes" to the question of ever having angina or AP or who were diagnosed with Rose angina (the Rose questionnaire is only administered to survey participants >40 years of age). Estimates from National Health and Nutrition Examination Survey 2005–2008 (National Center for Health Statistics) were applied to 2008 population estimates (≥20 years of age). Incidence: AP uncomplicated by a myocardial infarction or with no myocardial infarction (Framingham Heart Study 1980 to 2001–2003 of the original cohort and 1980 to 1998–2001 of the Offspring Cohort, National Heart, Lung, and Blood Institute). Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics; data include those inpatients discharged alive, dead, or status unknown.

*There were 166 000 days of care for discharges of patients with AP from short-stay hospitals in 2009.

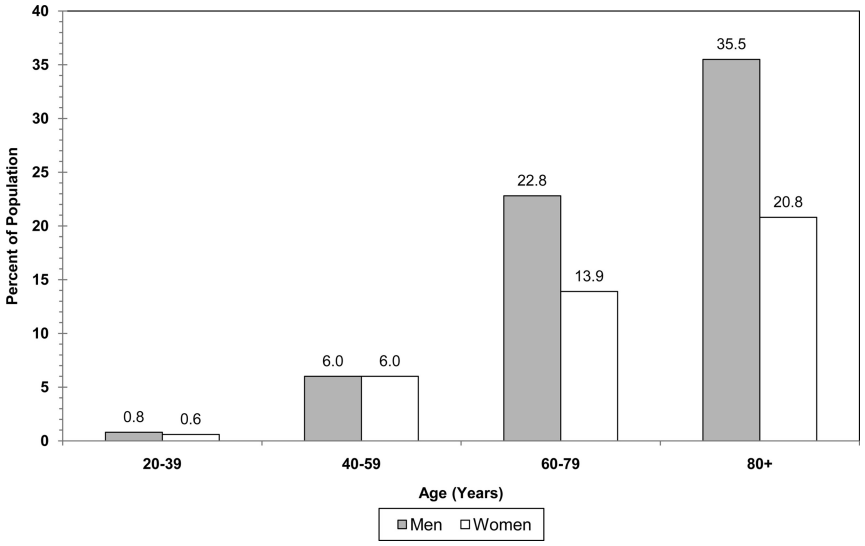


Chart 5-1. Prevalence of coronary heart disease by age and sex (National Health and Nutrition Examination Survey: 2005–2008). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

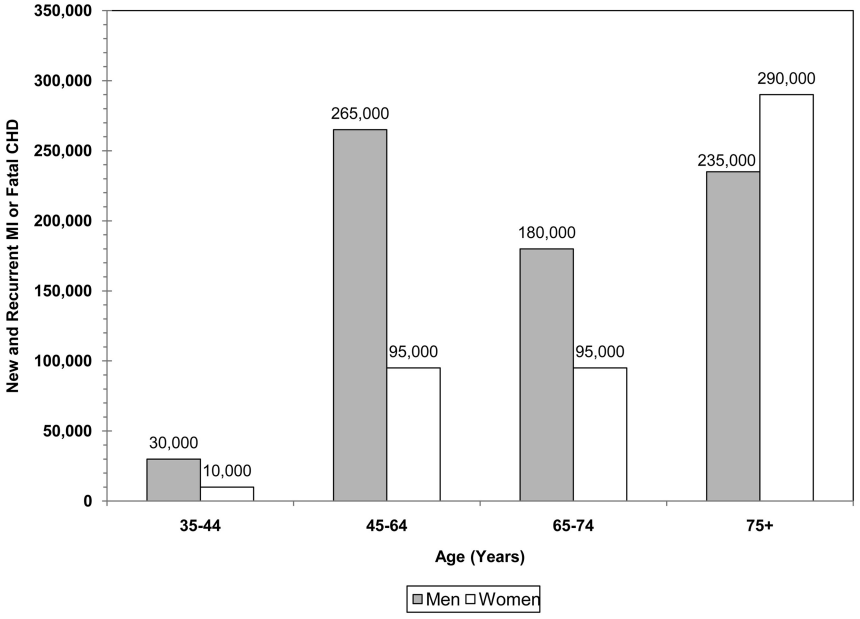


Chart 5-2. Annual number of adults having diagnosed heart attack or fatal coronary heart disease (CHD) by age and sex (Atherosclerosis Risk in Communities Surveillance: 1987–2004 and Cardiovascular Health Study: 1989–2004). These data include myocardial infarction (MI) and fatal coronary heart disease but not silent MI. Source: National Heart, Lung, and Blood Institute.

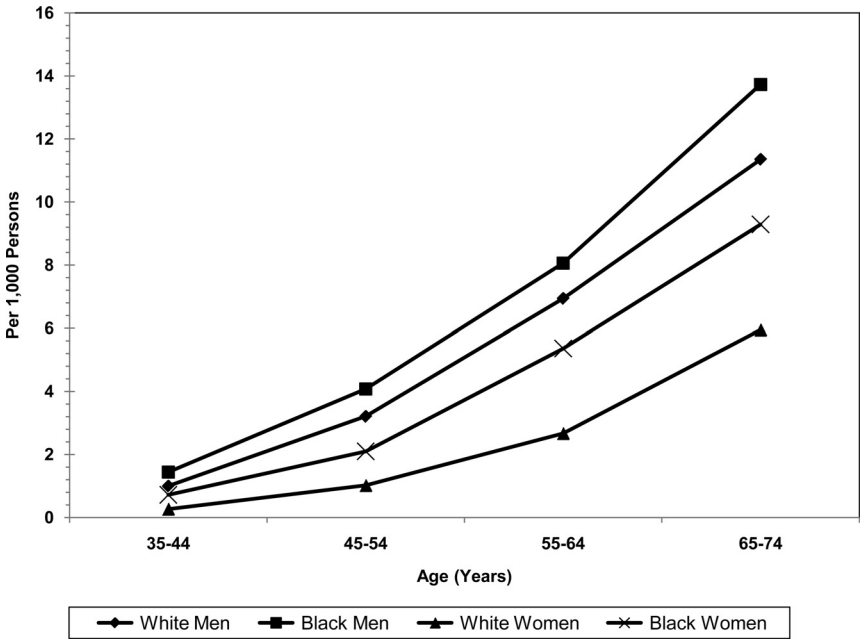


Chart 5-3. Annual rate of first heart attacks by age, sex, and race (Atherosclerosis Risk in Communities Surveillance: 1987–2004). Source: National Heart, Lung, and Blood Institute.

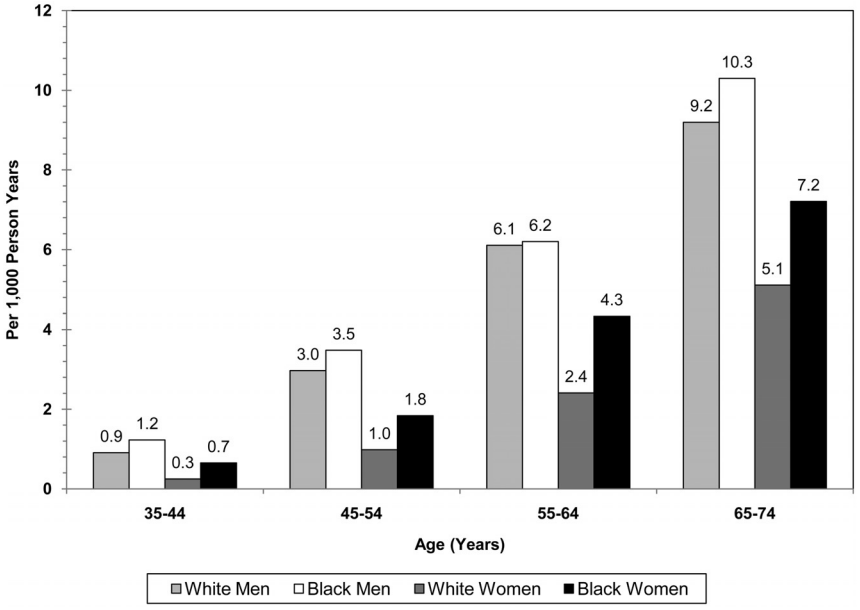


Chart 5-4. Incidence of myocardial infarction* by age, race, and sex (Atherosclerosis Risk in Communities Surveillance, 1987–2004). *Myocardial infarction diagnosis by expert committee based on review of hospital records. Source: Unpublished data from Atherosclerosis Risk in Communities study, National Heart, Lung, and Blood Institute.

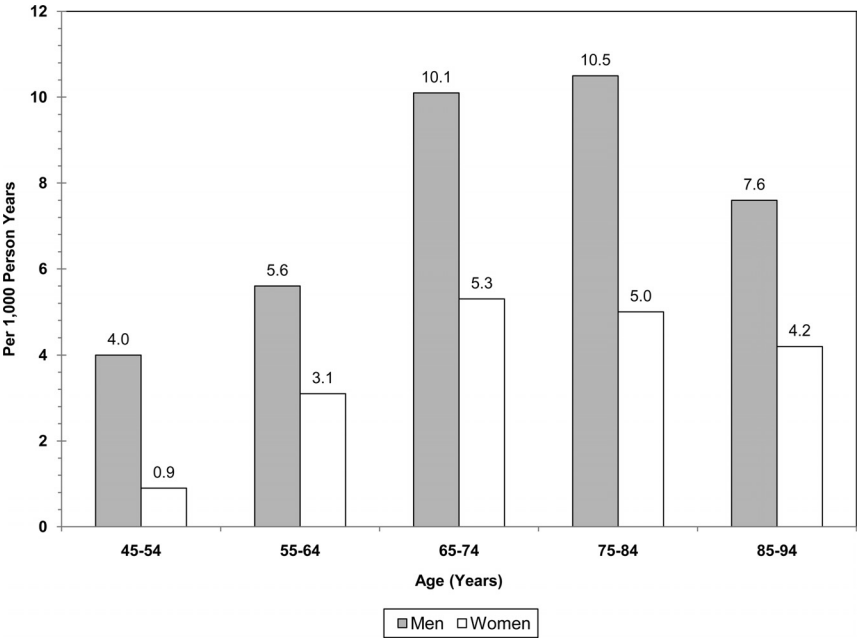
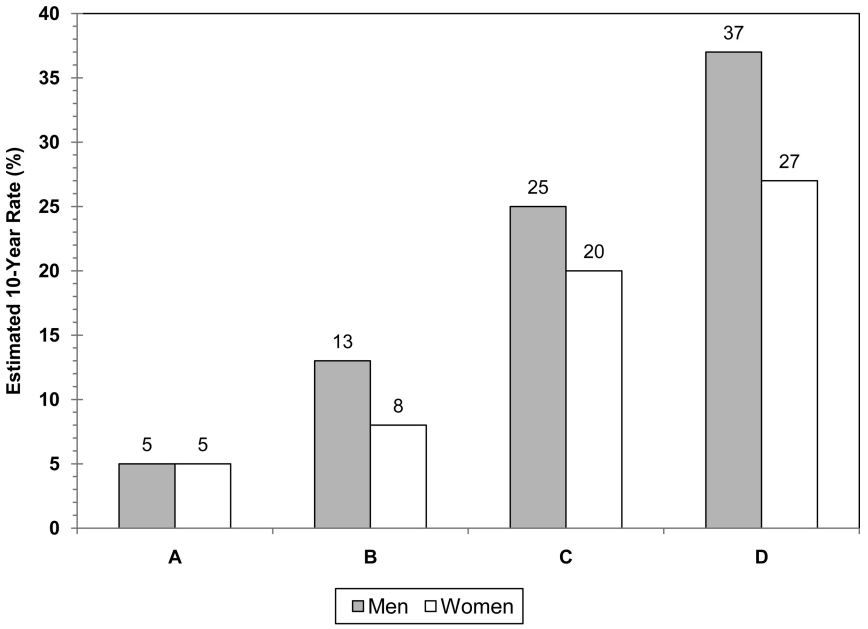


Chart 5-5. Incidence of angina pectoris* by age and sex (Framingham Heart Study 1980–2002/2003). *Angina pectoris considered uncomplicated on the basis of physician interview of patient. (Rate for women 45–54 years of age considered unreliable.) Data derived from National Heart, Lung, and Blood Institute.[8]



	A	B	C	D
Blood Pressure	120/80	140/90	140/90	140/90
Cholesterol	200	240	240	240
HDL-C	50	50	40	40
Diabetes	No	No	Yes	Yes
Cigarettes	No	No	No	Yes

Chart 5-6. Estimated 10-year coronary heart disease risk in adults 55 years of age according to levels of various risk factors (Framingham Heart Study). HDL-C indicates high-density lipoprotein cholesterol. Data derived from Wilson et al.[57]

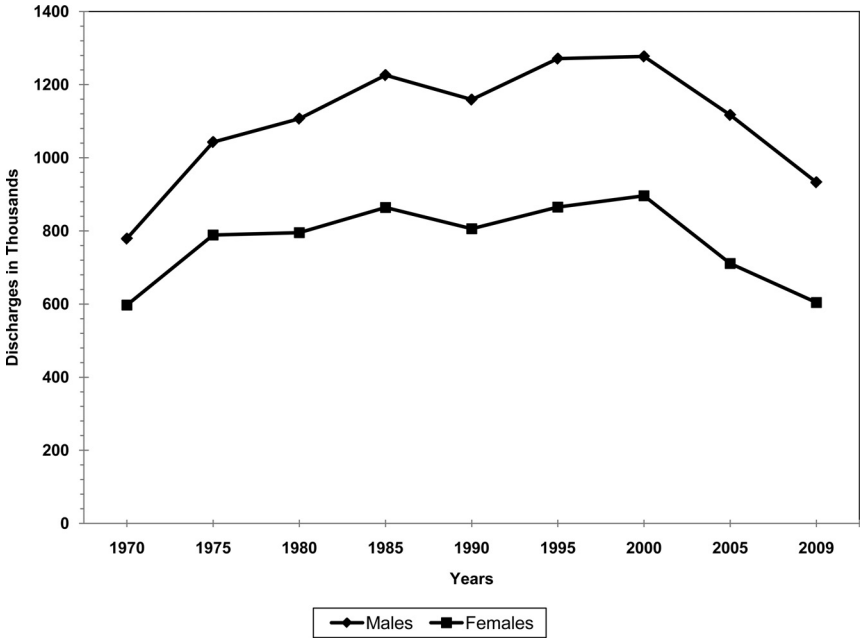


Chart 5-7. Hospital discharges for coronary heart disease by sex (United States: 1970–2009). Hospital discharges include people discharged alive, dead, and "status unknown." Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.

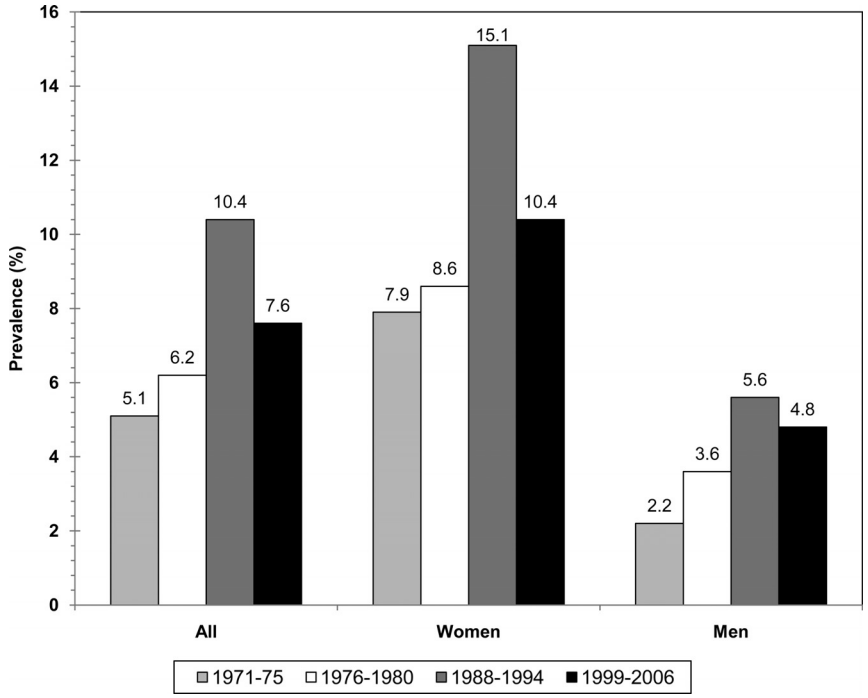


Chart 5-8. Prevalence of low coronary heart disease risk, overall and by sex (National Health and Nutrition Examination Survey: 1971–2006). Low risk is defined as systolic blood pressure <120 mm Hg and diastolic blood pressure <80 mm Hg; cholesterol <200 mg/dL; body mass index <25 kg/m^2; currently not smoking cigarettes; and no prior myocardial infarction or diabetes mellitus. Source: Personal communication with the National Heart, Lung, and Blood Institute, June 28, 2007.

6. Stroke (Cerebrovascular Disease)

ICD-9 430 to 438, ICD-10 I60 to I69. See Tables 6-1 and 6-2 and Charts 6-1 through 6-13.

Stroke Prevalence

- An estimated 7 000 000 Americans ≥20 years of age have had a stroke (extrapolated to 2008 using NCHS/NHANES 2005–2008 data). Overall stroke prevalence during this period is an estimated 3.0% (Table 6-1).
- According to data from the 2010 BRFSS (CDC), 2.6% of men and 2.6% of women ≥18 years of age had a history of stroke; 2.4% of non-Hispanic whites, 4.0% of non-Hispanic blacks, 1.4% of Asian/Pacific Islanders, 2.5% of Hispanics (of any race), 5.8% of American Indian/Alaska Natives, and 4.1% of other races or multiracial people had a history of stroke (NHLBI tabulation of BRFSS).
- The prevalence of silent cerebral infarction is estimated to range from 6% to 28%, with higher prevalence with increasing age.[1–3] The prevalence estimates also vary depending on the population studied (eg, ethnicity, sex, risk factor profile), definition of silent cerebral infarction, and imaging technique. It has been estimated that 13 million people had prevalent silent stroke in the 1998 US population.[4,5]
- The prevalence of stroke-related symptoms was found to be relatively high in a general population free of a prior diagnosis of stroke or transient ischemic attack (TIA). On the basis of data from 18 462 participants enrolled in a national cohort study, 17.8% of the population >45 years of age reported at least 1 symptom. Stroke symptoms were more likely among blacks than whites, among those with lower income and lower educational attainment, and among those with fair to poor perceived health status. Symptoms also were more likely in participants with higher Framingham stroke risk score (Reasons for Geographic and Racial Differences in Stroke study [REGARDS], NINDS).[6]
- Projections show that by 2030, an additional 4 million people will have had a stroke, a 24.9% increase in prevalence from 2010.[7]

Stroke Incidence

- Each year, ≈795 000 people experience a new or recurrent stroke. Approximately 610 000 of these are first attacks, and 185 000 are recurrent attacks (GCNKSS, NINDS, and NHLBI; GCNKSS and NINDS data for 1999 provided July 9, 2008; estimates compiled by NHLBI). Of all strokes, 87% are ischemic and 10% are intracerebral hemorrhagic strokes, whereas 3% are subarachnoid hemorrhage strokes (GCNKSS, NINDS, 1999).
- On average, every 40 seconds, someone in the United States has a stroke (AHA computation based on the latest available data).
- Each year, ≈55 000 more women than men have a stroke (GCNKSS, NINDS).[8]
- Women have a higher lifetime risk of stroke than men. In the FHS, lifetime risk of stroke among those 55 to75 years of age was 1 in 5 for women (20% to 21%) and approximately 1 in 6 for men (14% to 17%).[9]
- Women have lower age-adjusted stroke incidence than men; however, sex differences in stroke risk may be modified by age.[10] Data from FHS demonstrate that compared with white men, white women 45 to 84 years of age have lower stroke risk than men, but this association is reversed in older ages such that women >85 years of age

Abbreviations Used in Chapter 6

AF	atrial fibrillation
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities study
BASIC	Brain Attack Surveillance in Corpus Christi
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CI	confidence interval
CLRD	chronic lower respiratory disease
CREST	Carotid Revascularization Endarterectomy versus Stenting Trial
CVD	cardiovascular disease
DM	diabetes mellitus
ED	emergency department
EMS	emergency medical services
FHS	Framingham Heart Study
FRS	Framingham Risk Score
GCNKSS	Greater Cincinnati/Northern Kentucky Stroke Study
HD	heart disease
HDL	high-density lipoprotein
HERS	Heart and Estrogen/Progestin Replacement Study
HR	hazard ratio
ICD-9	International Classification of Diseases, 9th Revision
ICD-10	International Classification of Diseases, 10th Revision
MEPS	Medical Expenditure Panel Survey
MI	myocardial infarction
NCHS	National Center for Health Statistics
NH	Non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NIHSS	National Institutes of Health Stroke Scale
NINDS	National Institutes of Neurological Disorders and Stroke
NOMAS	Northern Manhattan Study
OR	odds ratio
PA	physical activity
REGARDS	Reasons for Geographic and Racial Differences in Stroke study
RR	relative risk
TIA	transient ischemic attack
WEST	Women's Estrogen for Stroke Trial
WHI	Women's Health Initiative

have elevated risk compared with men.[11] Similarly, a population-based study in Sweden found stroke incidence to be lower for women than men at ages 55 to 64 years, but at 75 to 85 years of age, this association reversed, and women had a higher incidence than men.[12] Other studies report an excess risk of stroke in men compared with women that persists throughout the life course or diminishes but does not reverse with age.[13–15]

- On average, women are older at stroke onset than men (≈75 years compared with 71 years).[11]
- Blacks have a risk of first-ever stroke that is almost twice that of whites.[16]
- In the national REGARDS cohort, in 27 744 participants followed up over 4.4 years (2003–2010), the overall age- and sex-adjusted black/white incidence rate ratio was 1.51, but for ages 45 to 54 years, it was 4.02, whereas for those ≥85 years of age, it was 0.86.[17] Similar trends for decreasing incidence rate ratio were seen in the GCNKSS.[18]
- Analysis of data from the FHS suggests that stroke incidence is declining over time in this largely white cohort. Data from 1950 to 1977, 1978 to 1989, and 1990 to 2004 showed that the age-adjusted incidence of first stroke per 1000 person-years in each of the 3 periods was 7.6, 6.2, and 5.3 in men and 6.2, 5.8, and 5.1 in women, respectively. Lifetime risk for incident stroke at 65 years of age decreased significantly in the latest data period compared with the first, from 19.5% to 14.5% in men and from 18.0% to 16.1% in women.[19]
- In a similar fashion, data from the most recent GCNKSS show that compared with the 1990s, when incidence rates of stroke were stable, stroke incidence in 2005 was decreased for whites. Unfortunately, a similar decline was not seen in blacks. These changes for whites were driven by a decline in ischemic strokes for whites. There were no changes in incidence of ischemic stroke for blacks or for hemorrhagic strokes in blacks or whites.[8]
- The BASIC (Brain Attack Surveillance in Corpus Christi) project (NINDS) demonstrated an increased incidence of stroke among Mexican Americans compared with non-Hispanic whites in a community in southeast Texas. The crude 3-year cumulative incidence (2000–2002) was 16.8 per 1000 in Mexican Americans and 13.6 per 1000 in non-Hispanic whites. Specifically, Mexican Americans had a higher cumulative incidence for ischemic stroke at younger ages (45–59 years of age: RR 2.04, 95% CI 1.55–2.69; 60–74 years of age: RR 1.58, 95% CI 1.31–1.91) but not at older ages (≥75 years of age: RR 1.12, 95% CI 0.94–1.32). Mexican Americans also had a higher incidence of intracerebral hemorrhage and subarachnoid hemorrhage than non-Hispanic whites, adjusted for age.[20]
- The age-adjusted incidence of first ischemic stroke per 1000 was 0.88 in whites, 1.91 in blacks, and 1.49 in Hispanics according to data from the Northern Manhattan Study (NOMAS; NINDS) for 1993 to 1997. Among blacks, compared with whites, the relative rate of intracranial atherosclerotic stroke was 5.85; of extracranial atherosclerotic stroke, 3.18; of lacunar stroke, 3.09; and of cardioembolic stroke, 1.58. Among Hispanics (primarily Cuban and Puerto Rican), compared with whites, the relative rate of intracranial atherosclerotic stroke was 5.00; of extracranial atherosclerotic stroke, 1.71; of lacunar stroke, 2.32; and of cardioembolic stroke, 1.42.[21]
- Among 4507 American Indian participants without a prior stroke in the Strong Heart Study in 1989 to 1992, the age- and sex-adjusted incidence of stroke through 2004 was 6.79 per 100 person-years, with 86% of incident strokes being ischemic.[22]
- A review of published studies and data from clinical trials found that hospital admissions for intracerebral hemorrhage have increased by 18% in the past 10 years, probably because of increases in the number of elderly people, many of whom lack adequate BP control, as well as the increasing use of anticoagulants, thrombolytics, and antiplatelet agents. Mexican Americans, Latin Americans, blacks, Native Americans, Japanese people, and Chinese people have higher incidences than do white Americans.[23]
- In the GCNKSS, the annual incidence of anticoagulant-associated intracerebral hemorrhage per 100 000 people increased from 0.8 (95% CI 0.3–1.3) in 1988 to 1.9 (95% CI 1.1–2.7) in 1993/1994 and 4.4 (95% CI 3.2–5.5) in 1999 ($P<0.001$ for trend). Among people ≥80 years of age, the rate of anticoagulant-associated intracerebral hemorrhage increased from 2.5 (95% CI 0–7.4) in 1988 to 45.9 (95% CI 25.6–66.2) in 1999 ($P<0.001$ for trend). Over this period of time, incidence rates of cardioembolic ischemic stroke were similar, whereas warfarin distribution in the United States quadrupled on a per capita basis. The increase in incidence is therefore attributable to prescribing behavior and patterns of care.[24]

TIA: Prevalence and Incidence

- In a nationwide survey of US adults, the estimated prevalence of self-reported physician-diagnosed TIA was 2.3%, which translates into ≈5 million people. The true prevalence of TIA is greater, because many patients who experience neurological symptoms consistent with a TIA fail to report it to their healthcare provider.[25]
- In the GCNKS, using data from 1993 and 1994, the age-, sex-, and race-adjusted incidence rates for TIA were 0.83 per 10 000. [26] Age- and sex-adjusted incidence rates for TIA in Rochester, MN, were estimated at 0.68 per 1000 for the years 1985 through 1989.[27]
- The prevalence of physician-diagnosed TIA increases with age.[25] Incidence of TIA increases with age and varies by sex and race/ethnicity. Men, blacks, and Mexican Americans have higher rates of TIA than their female and non-Hispanic white counterparts.[20,26]
- Approximately 15% of all strokes are heralded by a TIA.[28]
- TIAs confer a substantial short-term risk of stroke, hospitalization for CVD events, and death. Of 1707 TIA patients evaluated in the ED of Kaiser Permanente Northern California, a large, integrated healthcare delivery system, 180 (10%) experienced a stroke within 90 days. Ninety-one patients (5%) had a stroke within 2 days. Predictors of stroke included age >60 years, DM, focal symptoms of weakness or speech impairment, and TIA that lasted >10 minutes.[29]

- Meta-analyses of cohorts of patients with TIA have shown the short-term risk of stroke after TIA is significant. Risk has been shown to be as high as 10% at 2 days and as high as 17% at 90 days.[30,31]
- Individuals who have a TIA and survive the initial high-risk period have a 10-year stroke risk of roughly 19% and a combined 10-year stroke, MI, or vascular death risk of 43% (4% per year).[32]
- Within 1 year of TIA, ≈12% of patients will die.[26]
- It is estimated that one third of episodes characterized as TIA according to the classic definition (ie, focal neurological deficits that resolve within 24 hours) would be considered infarctions on the basis of diffusion-weighted magnetic resonance imaging findings.[33]

Stroke Mortality

- On average, every 4 minutes, someone dies of a stroke (NCHS, NHLBI).[33a]
- Stroke accounted for ≈1 of every 18 deaths in the United States in 2008.[33a]
- When considered separately from other CVDs, stroke ranks No. 4 among all causes of death, behind diseases of the heart, cancer, and CLRD (NCHS mortality data). Stroke mortality in 2008 was 134 148; any-mention mortality in 2008 was 223 841 and the death rate was 40.7.[33a] See Chart 6-6 for sex and race comparisons.
- From 1998 to 2008, the annual stroke death rate decreased 34.8%, and the actual number of stroke deaths declined 19.4% (NHLBI tabulation) (appropriate comparability ratios were applied).[33a,34]
- Conclusions about changes in stroke death rates from 1980 to 2005 are as follows:
  - — There was a greater decline in stroke death rates in men than in women, with a male-to-female ratio decreasing from 1.11 to 1.03 (age adjusted).
  - — There were greater declines in stroke death rates in men than in women among people ≥65 years of age than among younger ages.[34]
- Approximately 54% of stroke deaths in 2008 occurred out of the hospital (unpublished NHLBI tabulation of NCHS 2008 Mortality Data Set).
- Among people 45 to 64 years of age, 8% to 12% of ischemic strokes and 37% to 38% of hemorrhagic strokes result in death within 30 days, according to 1987 to 2001 data from the ARIC study of the NHLBI.[35]
- In a study of people ≥65 years of age recruited from a random sample of Health Care Financing Administration Medicare Part B eligibility lists in 4 US communities (CHS), over the time period 1989 to 2000, the 1-month case fatality rate was 12.6% for all strokes, 8.1% for ischemic strokes, and 44.6% for hemorrhagic strokes.[36]
- More women than men die of stroke each year because of the larger number of elderly women. Women accounted for 60.1% of US stroke deaths in 2008.
- From 1995 to 1998, age-standardized mortality rates for ischemic stroke, subarachnoid hemorrhage, and intracerebral hemorrhage were higher among blacks than whites. Death rates attributable to intracerebral hemorrhage also were higher among Asians/Pacific Islanders than among whites. All minority populations had higher death rates attributable to subarachnoid hemorrhage than did whites. Among adults 25 to 44 years of age, blacks and American Indian/Alaska Natives had higher risk ratios than did whites for all 3 stroke subtypes.[37]
- Age-adjusted stroke mortality rates began to level off in the 1980s and stabilized in the 1990s for both men and women, according to the Minnesota Heart Study. Women had lower rates of stroke mortality than did men throughout the period. Some of the improvement in stroke mortality may be the result of improved acute stroke care, but most is thought to be the result of improved detection and treatment of hypertension.[38]
- In 2002, death certificate data showed that the mean age at stroke death was 79.6 years; however, males had a younger mean age at stroke death than females. Blacks, American Indian/Alaska Natives, and Asian/Pacific Islanders had younger mean ages than whites, and the mean age at stroke death was also younger among Hispanics than non-Hispanics.[39]
- A report released by the CDC in collaboration with the Centers for Medicare & Medicaid Services, the *Atlas of Stroke Hospitalizations Among Medicare Beneficiaries*, found that in Medicare beneficiaries over the time period 1995 to 2002, 30-day mortality rate varied by age: 9% in patients 65 to 74 years of age, 13.1% in those 74 to 84 years of age, and 23% in those ≥85 years of age.[40]
- The black/white disparity in stroke mortality varies by age in a similar fashion to stroke incidence as described above.
- There are substantial geographic disparities in stroke mortality, with higher rates in the southeastern United States, known as the "stroke belt" (Chart 6-7). This area is usually defined to include the 8 southern states of North Carolina, South Carolina, Georgia, Tennessee, Mississippi, Alabama, Louisiana, and Arkansas. These geographic differences have existed since at least 1940,[41] and despite some minor shifts,[42] they persist.[43–45] Within the stroke belt, a "buckle" region along the coastal plain of North Carolina, South Carolina, and Georgia has been identified with even a higher stroke mortality rate than the remainder of the stroke belt.[46] The overall average stroke mortality is ≈20% higher in the stroke belt than in the rest of the nation and ≈40% higher in the stroke buckle.
- Racial and regional patterns in stroke incidence have been shown to be similar to patterns for stroke mortality, which suggests that disparities in incidence play a substantial role in mortality disparities. However, incidence only partly explains mortality disparities, and differences in case fatality or other factors likely contribute to racial and geographic disparities in stroke mortality.[47]

Stroke Risk Factors

For prevalence and other information on any of these specific risk factors, refer to the specific risk factor chapters:

- High blood pressure: Chapter 7
- Disorders of heart rhythm (including atrial fibrillation): Chapter 10

- Smoking/Tobacco Use: Chapter 13
- High blood cholesterol and other lipids: Chapter 14
- Physical inactivity: Chapter 15
- Diabetes mellitus: Chapter 17
- End-stage renal disease and chronic kidney disease: Chapter 18

(See Table 6-2 for data on modifiable stroke risk factors.)

- BP is a powerful determinant of risk for both ischemic stroke and intracranial hemorrhage. Subjects with BP <120/80 mm Hg have approximately half the lifetime risk of stroke of subjects with hypertension. The treatment and lowering of BP among hypertensive individuals was associated with a significant reduction in stroke risk.[48]
- In REGARDS (NINDS), black participants were more aware than whites of their hypertension and more likely to be undergoing treatment if aware of their diagnosis, but among those treated for hypertension, they were less likely than whites to have their BP controlled.[49]
- REGARDS (NINDS) also showed no evidence of a difference between the stroke belt and other regions in awareness of hypertension, but there was a trend for better treatment and BP control in the stroke belt region. The lack of substantial geographic differences in hypertension awareness and the trend toward better treatment and control in the stroke belt suggest that differences in hypertension management may not be a major contributor to the geographic disparity in stroke mortality.[49]
- Impaired glucose tolerance nearly doubled the stroke risk compared with patients with normal glucose levels and tripled the risks for patients with DM.[50]
- Age-specific incidence rates and rate ratios show that DM increases ischemic stroke incidence at all ages, but this risk is most prominent before 55 years of age in blacks and before 65 years of age in whites. Ischemic stroke patients with DM are younger, more likely to be black, and more likely to have hypertension, MI, and high cholesterol than nondiabetic patients.[51]
- Atrial fibrillation (AF) is a powerful risk factor for stroke, independently increasing risk ≈5-fold throughout all ages. The percentage of strokes attributable to AF increases steeply from 1.5% at 50 to 59 years of age to 23.5% at 80 to 89 years of age.[52,53]
- Because AF is often asymptomatic[54,55] and likely frequently clinically undetected,[56] the stroke risk attributed to AF may be substantially underestimated.[57] Therefore, although AF is an important stroke risk factor, both patients and treating physicians may be unaware of its presence. A related point is that no strategy to pursue normal sinus rhythm, including cardioversion, antiarrhythmic drug therapy, and/or ablation, has definitively been shown to reduce the risk of stroke.
- Data from the Honolulu Heart Program/NHLBI found that in Japanese men 71 to 93 years of age, low concentrations of HDL cholesterol were more likely to be associated with a future risk of thromboembolic stroke than were high concentrations.[58]
- In the FHS, a documented parental ischemic stroke by the age of 65 years was associated with a 300% increase in documented ischemic stroke risk in offspring, even after adjustment for other known stroke risk factors. The absolute magnitude of the increased risk was greatest in those in the highest quintile of the FRS. By age 65 years, people in the highest FRS quintile with an early parental ischemic stroke had a 25% risk of stroke compared with a 7.5% risk of ischemic stroke for those without such a history.[59]
- The CHS (NHLBI) showed people with creatinine ≥1.5 mg/dL were at increased risk for stroke, with an adjusted HR of 1.77 (95% CI 1.08–2.91).[60] Participants in REGARDS with a reduced estimated glomerular filtration rate (eGFR) were also shown to have increased risk of incident stroke symptoms.[61]

Risk Factor Issues Specific to Women

- Analysis of data from the FHS found that women with natural menopause before 42 years of age had twice the ischemic stroke risk of women with natural menopause after age 42.[62] Investigators from the Nurse's Health Study, however, did not find an association between age at natural menopause and risk of ischemic or hemorrhagic stroke.[63]
- Overall, randomized clinical trial data indicate that the use of estrogen plus progestin, as well as estrogen alone, increases stroke risk in postmenopausal, generally healthy women and provides no protection for postmenopausal women with established HD.[64,65]
- Among postmenopausal women who were generally healthy, the Women's Health Initiative (WHI), a randomized trial of 16 608 women (95% of whom had no preexisting CVD), found that estrogen plus progestin increased ischemic stroke risk by 44%, with no effect on hemorrhagic stroke.[64]
- In the WHI trial, among 10 739 women with hysterectomy, it was found that conjugate equine estrogen alone increased the risk of ischemic stroke by 55% and that there was no significant effect on hemorrhagic stroke.[66]
- In postmenopausal women with known CHD, the Heart and Estrogen/Progestin Replacement Study (HERS), a secondary CHD prevention trial, found that estrogen plus progestin hormone therapy did not reduce stroke risk.[67]
- The Women's Estrogen for Stroke Trial (WEST) found that estrogen alone in postmenopausal women with a recent stroke or TIA had no significant overall effect on recurrent stroke or fatality.[68]
- Analysis of data from the FHS found that women with menopause at 42 to 54 years of age and at ≥55 years of age had lower stroke risk than those with menopause at <42 years of age, even after adjustment for potential confounders. Women with menopause before 42 years of age had twice the stroke risk of all other women in different age groups.[62]
- The risk of ischemic stroke or intracerebral hemorrhage during pregnancy and the first 6 weeks after giving birth was 2.4 times greater than for nonpregnant women of similar age and race, according to the Baltimore-Washington Cooperative Young Stroke Study. The risk of ischemic stroke during pregnancy was not increased

during pregnancy per se but was increased 8.7-fold during the first 6 postpartum weeks. Intracerebral hemorrhage showed a small RR of 2.5 during pregnancy but increased dramatically to an RR of 28.3 in the first 6 postpartum weeks. The excess risk of stroke (all types except subarachnoid hemorrhage) attributable to the combined pregnancy/postpregnancy period was 8.1 per 100 000 pregnancies.[69]

- In the US Nationwide Inpatient Sample from 2000 to 2001, the rate of events per 100 000 pregnancies was 9.2 for ischemic stroke, 8.5 for intracerebral hemorrhage, 0.6 for cerebral venous thrombosis, and 15.9 for the ill-defined category of pregnancy-related cerebrovascular events, for a total rate of 34.2 per 100 000, not including subarachnoid hemorrhage. The risk was increased in blacks and among older women. Death occurred during hospitalization in 4.1% of women with these events and in 22% of survivors after discharge to a facility other than home.[70]
- Analyses of the US Nationwide Inpatient Sample from 1994 to 1995 and from 2006 to 2007 show a temporal increase in the proportion of pregnancy hospitalizations that were associated with a stroke, with a 47% increase for antenatal hospitalizations and 83% increase for postpartum hospitalizations, but no increase for delivery hospitalizations. Increases in the prevalence of heart disease and hypertensive disorders accounted for almost all the increase in postpartum stroke hospitalizations but not the antenatal stroke hospitalizations.[71]
- Preeclampsia is a risk factor for ischemic stroke remote from pregnancy.[72] The subsequent stroke risk of preeclampsia may be mediated by a 3.6- to 6.1-fold higher later risk of hypertension and a 3.1- to 3.7-fold higher later risk of DM, depending on whether the preeclampsia was mild or severe.[73]

Physical Inactivity as a Risk Factor for Stroke

- In NOMAS, a prospective cohort that included white, black, and Hispanic men and women in an urban setting followed up for a median of 9 years, baseline PA was associated with an overall 35% reduction in risk of ischemic stroke.[74]
- The NOMAS study found that only moderate- to vigorous-intensity exercise was associated with reduced stroke incidence, whereas light exercise (such as walking) showed no benefit.[75]
- Timing of PA in relation to stroke onset has also been examined in several studies. In a hospital-based case-control study from Heidelberg, Germany, recent activity (within the prior months) was associated with reduced odds of having a stroke or TIA, whereas sports activity during young adulthood that was not continued showed no benefit.[76] In a Danish case-control study, ischemic stroke patients were less physically active in the week preceding the stroke than age- and sex-matched control subjects, with the highest activity scores associated with the greatest reduction in odds of stroke.[77]

Smoking

(See Chapter 13 for more information.)

- Cigarette smoking is one of the well-established modifiable risk factors for stroke. This includes ischemic, intracerebral, and subarachnoid hemorrhage, but the data for intracerebral hemorrhage are less consistent.[78,79]
- Smoking is perhaps the most important modifiable risk factor in preventing subarachnoid hemorrhage, with the highest population attributable risk of any subarachnoid hemorrhage risk factor.[80]
- Current smokers have a 2 to 4 times increased risk of stroke compared with nonsmokers or those who have quit for more than 10 years.[78,79]
- Data also support a dose-response relationship across old and young age groups.[78,81]
- Discontinuation of smoking has been shown to reduce stroke risk across sex, race, and age groups.[81]

Sleep Apnea

- Sleep apnea is an independent risk factor for stroke, increasing the risk of stroke or death 2-fold.[82–85]
- Worsening sleep apnea severity is associated with greater stroke risk; patients with severe sleep apnea have 3- to 4-fold increased odds of stroke.[82,84,85]
- Continuous positive airway pressure improves a variety of outcomes after stroke.[86–88] For example, continuous positive airway pressure reduces the risk of recurrent vascular events among patients with stroke (relative risk reduction of 81.4%; number needed to treat of 3.4).[87]
- Sleep apnea is common after stroke, occurring in 60% to 96% of poststroke patients.[89–98]

Awareness of Stroke Warning Signs and Risk Factors

- Correct knowledge of at least 1 stroke warning sign increased from 48% in 1995% to 68% in 2000, with no significant improvement to 2005 (68%) on the basis of a telephone survey conducted in a biracial population in the greater Cincinnati/Northern Kentucky region. Knowledge of 3 correct warning signs was low but increased over time: 5.4% in 1995, 12.0% in 2000, and 15.7% in 2005. Knowledge of at least 1 stroke risk factor increased from 59% in 1995% to 71% in 2000, but there was no improvement to 2005 (71%). Only 3.6% of those surveyed were able to independently identify tissue-type plasminogen activator as an available drug therapy, and only 9% of these were able to identify a window of $<$3 hours for treatment.[99]
- In the 2005 BRFSS, among respondents in 14 states, 38.1% were aware of 5 stroke warning symptoms and would first call 9-1-1 if they thought that someone was having a heart attack or stroke. Awareness of all 5 stroke warning symptoms and calling 9-1-1 was higher among whites than blacks and Hispanics (41.3%, 29.5%, and 26.8%, respectively), women than men (41.5% versus 34.5%), and people with higher versus lower educational attainment (47.6% for people with a college degree or more versus

22.5% for those who had not received a high school diploma).[100]

- A study was conducted of patients admitted to an ED with possible stroke to determine their knowledge of the signs, symptoms, and risk factors of stroke. Of the 163 patients able to respond, 39% did not know a single sign or symptom. Patients ≥65 years of age were less likely than those <65 years old to know a sign or symptom of stroke (28% versus 47%), and 43% did not know a single risk factor. Overall, almost 40% of patients did not know the signs, symptoms, and risk factors of stroke.[101]
- In 2004, 800 adults ≥45 years of age were surveyed to assess their perceived risk for stroke and their history of stroke risk factors. Overall, 39% perceived themselves to be at risk. Younger age, current smoking, a history of DM, high BP, high cholesterol, HD, and stroke/TIA were independently associated with perceived risk for stroke. Respondents with AF were no more likely to report being at risk than were respondents without AF. Perceived risk for stroke increased as the number of risk factors increased; however, 46% of those with ≥3 risk factors did not perceive themselves to be at risk.[102]
- A study of patients who have had a stroke found that only 60.5% were able to accurately identify 1 stroke risk factor and that 55.3% were able to identify 1 stroke symptom. Patients' median delay time from onset of symptoms to admission in the ED was 16 hours, and only 31.6% accessed the ED in <2 hours. Analysis showed that the appearance of nonmotor symptoms as the primary symptom and nonuse of the 9-1-1 system were significant predictors of delay >2 hours. Someone other than the patient made the decision to seek treatment in 66% of the cases.[103]
- Spanish-speaking Hispanics are less likely to know all stroke symptoms than English-speaking Hispanics, non-Hispanic blacks, and non-Hispanic whites. Lack of English proficiency is strongly associated with lack of stroke knowledge among Hispanics.[104]

Aftermath

- Stroke is a leading cause of serious long-term disability in the United States (Survey of Income and Program Participation, a survey of the US Bureau of the Census).[105]
- In the NHLBI's FHS, among ischemic stroke survivors who were ≥65 years of age, these disabilities were observed at 6 months after stroke[106]:
  - 50% had some hemiparesis
  - 30% were unable to walk without some assistance
  - 26% were dependent in activities of daily living
  - 19% had aphasia
  - 35% had depressive symptoms
  - 26% were institutionalized in a nursing home
- Data from the BRFSS (CDC) 2005 survey on stroke survivors in 21 states and the District of Columbia found that 30.7% of stroke survivors received outpatient rehabilitation. The findings indicated that the prevalence of stroke survivors receiving outpatient stroke rehabilitation was lower than would be expected if clinical practice guideline recommendations for all stroke patients had been followed.[107]
- Black stroke survivors had greater limitations in ambulation than did white stroke survivors, after adjustment for age, sex, and educational attainment but not stroke subtype, according to data from the NHIS (2000–2001, NCHS) as analyzed by the CDC.[108] A national study of inpatient rehabilitation after first stroke found that blacks were younger, had a higher proportion of hemorrhagic stroke, and were more disabled on admission. Compared with non-Hispanic whites, blacks and Hispanics also had a poorer functional status at discharge but were more likely to be discharged to home rather than to another institution even after adjustment for age and stroke subtype. After adjustment for the same covariates, compared with non-Hispanic whites, blacks also had less improvement in functional status per inpatient day.[109]
- After stroke, women have greater disability than men. A cross-sectional analysis of 5888 community-living elderly people (>65 years of age) in the CHS who were ambulatory at baseline found that women were half as likely to be independent in activities of daily living after stroke, even after controlling for age, race, education, and marital status.[110] A prospective study from a Michigan-based stroke registry found that women had a 63% lower probability of achieving independence in activities of daily living 3 months after discharge, even after controlling for age, race, subtype, prestroke ambulatory status, and other patient characteristics.[111]

Hospital Discharges/Ambulatory Care Visits

- From 1999 to 2009, the number of inpatient discharges from short-stay hospitals with stroke as the first-listed diagnosis remained about the same, with discharges of 961 000 and 971 000, respectively (NHLBI tabulation, NHDS, NCHS).
- Data from 2009 from the Hospital Discharge Survey of the NCHS showed that the average length of stay for discharges with stroke as the first-listed diagnosis was 5.3 days.
- In 2003, men and women accounted for roughly the same number of hospital stays for stroke in the 18- to 44-year-old age group. After 65 years of age, women were the majority. Among people 65 to 84 years of age, 54.5% of stroke patients were women, whereas among the oldest age group, women constituted 69.7% of all stroke patients.[112]
- A first-ever county-level *Atlas of Stroke Hospitalizations Among Medicare Beneficiaries* was released in 2008 by the CDC in collaboration with the Centers for Medicare & Medicaid Services. It found that the stroke hospitalization rate for blacks was 27% higher than for the US population in general, 30% higher than for whites, and 36% higher than for Hispanics. In contrast to whites and Hispanics, the highest percentage of strokes in blacks (42.3%) occurred in the youngest Medicare age group (65–74 years of age).[40]
- In 2009, there were 768 000 ED visits and 127 000 outpatient department visits with stroke as the first-listed

diagnosis. In 2009, physician office visits for a first-listed diagnosis of stroke totaled 3 327 000 (unpublished data, NCHS, NHAMCS, NHLBI tabulation).[113]

Stroke in Children

- On the basis of pathogenic differences, pediatric strokes are typically classified as either perinatal, occurring at ≤28 days of life and including in utero strokes, or (later) childhood.
- Recent estimates of the overall annual incidence of stroke in US children are 6.4 per 100 000 children (0–15 years) in 1999 in GCNKSS[114] and 4.6 per 100 000 children (0–19 years) from 1997 to 2003 according to Kaiser Permanente of Northern California, a large, integrated healthcare delivery system.[115] Approximately half of all incident childhood strokes are hemorrhagic.[114–116]
- The prevalence of perinatal strokes is 29 per 100 000 live births, or 1 per 3500 live births in the 1997 to 2003 Kaiser Permanente of Northern California population.[115]
- A history of infertility, preeclampsia, prolonged rupture of membranes, and chorioamnionitis were found to be independent risk factors for perinatal arterial ischemic stroke in the Kaiser Permanente of Northern California population. The RR of perinatal stroke increased ≈25-fold, with an absolute risk of 1 per 200 deliveries, when ≥3 antenatally determined risk factors were present.[117]
- Although children with sickle cell disease and congenital HD are at high risk for ischemic stroke, the most common cause in a previously healthy child is a cerebral arteriopathy, found in approximately two thirds of cases.[118]
- Congenital HD accounted for 25% of pediatric arterial ischemic strokes in a population based study in Utah, Wyoming, Idaho, and Nevada; it increased the odds of stroke 16-fold compared with the general population.[119]
- Thrombophilias (genetic and acquired) are risk factors for childhood stroke, with summary ORs ranging from 1.6 to 8.8 in a recent meta-analysis.[120]
- From 1979 to 1998 in the United States, childhood mortality resulting from stroke declined by 58% overall, with reductions in all major subtypes.[121]
- The incidence of stroke in children has been stable over the past 10 years, whereas the 30-day case fatality rates declined from 18% in 1988–1989 to 9% in 1993–1994 and 9% in 1999 in the GCNKSS population.[114]
- Compared with girls, boys have a 1.28-fold higher risk of stroke.[122] Compared with white children, black children have a 2-fold risk of both incident stroke and death attributable to stroke.[121,122] The increased risk among blacks is not fully explained by the presence of sickle cell disease, nor is the excess risk among boys fully explained by trauma.[122]
- Strokes in children can be mistaken for a postictal Todd's paresis: 22% of children with acute arterial ischemic stroke have a seizure on presentation; younger age predicts presentation with seizures.[123]
- At a median follow-up time of 6.3 years, half of 53 childhood ischemic stroke survivors and two thirds of 80 neonatal ischemic stroke survivors had at least 1 neurological deficit; only 10% to 20% had mild deficits, whereas the remainder had moderate or severe deficits.[124] Involvement of deep structures (basal ganglia, posterior limb of the internal capsule) as opposed to pure cortical lesions predicts motor deficits.[125]
- Despite current treatment, 1 of 10 children with ischemic or hemorrhagic stroke will have a recurrence within 5 years.[126,127] The 5-year recurrence risk is as high as 60% among children with cerebral arteriopathy. The recurrence risk after perinatal stroke, however, is negligible.[128]

Barriers to Stroke Care

- On the basis of NHIS data, the inability to afford medications among stroke survivors increased significantly from 8.1% to 12.7% between 1997 and 2004, totaling 76 000 US stroke survivors in 2004. Compared with stroke survivors able to afford medications, those unable to afford them more frequently reported lack of transportation, no health insurance, no usual place of care, income <$20 000, and out-of-pocket medical expenses ≥$2000.[129]
- In 2002, ≈21% of US counties did not have a hospital, 31% lacked a hospital with an ED, and 77% did not have a hospital with neurological services.[130]
- Of patients with ischemic stroke in the California Acute Stroke Pilot Registry, 23.5% arrived at the ED within 3 hours of symptom onset, and 4.3% received thrombolysis. If all patients had called 9-1-1 immediately, the expected overall rate of thrombolytic treatment within 3 hours would have increased to 28.6%. If all patients with known onset had arrived within 1 hour and had been treated optimally, 57% could have received thrombolytic treatment.[131]
- Data from the Paul Coverdell National Acute Stroke Registry were analyzed from the 142 hospitals that participated in the 4 registry states. More patients were transported by ambulance than by other means (43.6%). Time of stroke symptom onset was recorded for 44.8% of the patients. Among these patients, 48% arrived at the ED within 2 hours of symptom onset. Significantly fewer blacks (42.4%) arrived within 2 hours of symptom onset than did whites (49.5%), and significantly fewer nonambulance patients (36.2%) arrived within 2 hours of symptom onset than did patients transported by ambulance (58.6%).[132]
- NHIS data from 1998 to 2002 found that younger stroke survivors (45–64 years) self-reported worse access to physician care and medication affordability than older stroke survivors. Compared with older patients, younger stroke survivors were more likely to be male (52% versus 47%), to be black (19% versus 10%), and to lack health insurance (11% versus 0.4%). Lack of health insurance was associated with reduced access to care.[133]
- Data from 142 hospitals participating in the Paul Coverdell National Acute Stroke Registry found that fewer than 48% of stroke patients arrived at the ED within 2 hours of symptom onset in 2005 to 2006. Blacks were less likely to arrive within the 2-hour window than whites (42.4% versus 49.5%). Among those arriving within 2 hours, 65.2% received imaging within 1 hour of ED arrival; significantly fewer women

received imaging within 1 hour than men (62.9% versus 67.6%), but no differences were observed by racial group.[132]
- Results from the BASIC project found that women were less likely to arrive at the ED within 3 hours of stroke symptom onset than men (OR 0.7, 95% CI 0.5–0.9). Mexican Americans were 40% less likely to arrive by EMS than non-Hispanic whites, even after adjustment for age, National Institutes of Health Stroke Scale score, education, history of stroke, and insurance status. Language fluency was not associated with time to hospital arrival or use of EMS. The receipt of tissue-type plasminogen activator was low (1.5%) but did not differ by sex or race.[134]
- A national study of academic medical centers found no change in the proportion of patients with stroke arriving at hospitals within 2 hours of symptom onset between 2001 and 2004 (37% versus 38%); however, the rate of intravenous tissue-type plasminogen activator use increased over this time period (14% to 38%), which suggests system-level improvements in the organization of in-hospital care. In risk-adjusted analyses, black patients were 45% less likely to arrive within 2 hours than white patients.[135]

Operations and Procedures

Among stroke or TIA patients with high-grade carotid stenosis, carotid endarterectomy has been the recommended treatment for the prevention of stroke, whereas carotid stenting has been proposed as a therapeutic option for patients at high risk for surgical revascularization.

- In 2009, an estimated 93 000 inpatient endarterectomy procedures were performed in the United States. Carotid endarterectomy is the most frequently performed surgical procedure to prevent stroke (NHDS, NCHS, NHLBI tabulation).
- Although rates of carotid endarterectomy in the Medicare population decreased slightly between 1998 and 2004, the use of carotid artery stenting increased dramatically[136] (Chart 6-12).
- The randomized Carotid Revascularization Endarterectomy versus Stenting Trial (CREST) compared carotid endarterectomy and stenting for symptomatic and asymptomatic carotid stenosis. There was no overall difference in the primary end point of stroke, MI, or death; however, carotid endarterectomy showed superiority with increasing age, with the crossover point at approximately age 70, and was associated with fewer strokes, which had a greater impact on quality of life than MI.[137]

Cost

The direct and indirect cost of stroke in 2008 was $34.3 billion (MEPS, NHLBI tabulation).

- The estimated direct medical cost of stroke for 2008 is $18.8 billion. This includes hospital outpatient or office-based provider visits, hospital inpatient stays, ED visits, prescribed medicines, and home health care.[138]
- The mean expense per person for stroke care in the United States in 2007 was estimated at $7657.[138]
- The mean lifetime cost of ischemic stroke in the United States is estimated at $140 048. This includes inpatient care, rehabilitation, and follow-up care necessary for lasting deficits. (All numbers were converted to 1999 dollars by use of the medical component of the Consumer Price Index.)[139]
- The estimated cost of acute pediatric stroke in the United States was $42 million in 2003. The mean cost of short-term hospital care was $20 927 per discharge.[140]
- After adjustment for routine healthcare costs, the average 5-year cost of a neonatal stroke was $51 719 and that of a childhood stroke was $135 161. Costs among children with stroke continued to exceed those in age-matched control children even in the fifth year by an average of $2016.[141]
- In a study of stroke costs within 30 days of an acute event between 1987 and 1989 in the Rochester Stroke Study, the average cost was $13 019 for mild ischemic strokes and $20 346 for severe ischemic strokes (4 or 5 on the Rankin Disability Scale).[142]
- Inpatient hospital costs for an acute stroke event account for 70% of first-year poststroke costs.[115]
- The largest components of short-term care costs were room charges (50%), medical management (21%), and diagnostic costs (19%).[143]
- Death within 7 days, subarachnoid hemorrhage, and stroke while hospitalized for another condition are associated with higher costs in the first year. Lower costs are associated with mild cerebral infarctions or residence in a nursing home before the stroke.[142]
- Demographic variables (age, sex, and insurance status) are not associated with stroke cost. Severe strokes (National Institutes of Health Stroke Scale score >20) cost twice as much as mild strokes, despite similar diagnostic testing. Comorbidities such as ischemic HD and AF predict higher costs.[143,144]
- The total cost of stroke from 2005 to 2050, in 2005 dollars, is projected to be $1.52 trillion for non-Hispanic whites, $313 billion for Hispanics, and $379 billion for blacks. The per capita cost of stroke estimates is highest in blacks ($25 782), followed by Hispanics ($17 201) and non-Hispanic whites ($15 597). Loss of earnings is expected to be the highest cost contributor in each race/ethnic group.[145]

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Table 6-1. Stroke

Population Group	Prevalence, 2008: Age ≥20 y	New and Recurrent Attacks All Ages	Mortality, 2008: All Ages*	Hospital Discharges, 2009: All Ages	Cost, 2008
Both sexes	7 000 000 (3.0%)	795 000	134 148	971 000	$34.3 billion
Males	2 800 000 (2.7%)	370 000 (46.5%)†	53 525 (39.9%)†	467 000	
Females	4 200 000 (3.3%)	425 000 (53.5%)†	80 623 (60.1%)†	504 000	
NH white males	2.4%	325 000‡	44 457		
NH white females	3.3%	365 000‡	68 787		
NH black males	4.5%	45 000‡	7222		
NH black females	4.4%	60 000‡	9488		
Mexican-American males	2.0%				
Mexican-American females	2.7%				
Hispanic or Latino	2.6%§				
Asian	2.0%§				
Hawaiian and other Pacific Islander	10.6%§				
American Indian/Alaska Native	5.9% §‖				

NH indicates non-Hispanic; ellipses (. . .) indicate data not available.

*Mortality data for the white, black, Asian or Pacific Islander, and American Indian/Alaska Native populations include deaths of persons of Hispanic and non-Hispanic origin. Numbers of deaths for the American Indian/Alaska Native and Asian or Pacific Islander populations are known to be underestimated.

†These percentages represent the portion of total stroke incidence or mortality that applies to males vs females.

‡Estimates include Hispanics and non-Hispanics. Estimates for whites include other nonblack races.

§National Health Interview Survey (2010), National Center for Health Statistics (NCHS); data are weighted percentages for Americans >18 years of age.[146]

‖This estimate has a relative standard error of >30% but <50%.

Sources: Prevalence: National Health and Nutrition Examination Survey 2005 to 2008, NCHS and National Heart, Lung, and Blood Institute (NHLBI). Percentages for racial/ethnic groups are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2008 US population.

Incidence: Greater Cincinnati/Northern Kentucky Stroke Study/National Institutes of Neurological Disorders and Stroke data for 1999 provided on August 1, 2007. US estimates compiled by NHLBI. Data include children. Mortality: NCHS. These data represent underlying cause of death only. Mortality data for white and black males and females include Hispanics. Hospital discharges: National Hospital Discharge Survey, NCHS. Data include those inpatients discharged alive, dead, or status unknown. Cost: NHLBI. Data include estimated direct and indirect costs for 2008.

Table 6-2. Modifiable Stroke Risk Factors

Factor	Prevalence, %	Population Attributable Risk, %*	RR
Cigarette smoking			
Overall	19.8	12–14†	1.9 (ischemic stroke)
Men	22.3		2.9 (SAH)
Women	17.4		
Hypertension		‡	
Ages 20–34 y			8
Men	13.4	99	
Women	6.2	98	
Ages 35–44 y			
Men	23.2	99	
Women	16.5	106	
Ages 45–54 y			
Men	36.2	100	
Women	35.9	103	
Ages 55–64 y			
Men	53.7	100	
Women	55.8	102	
Ages 65–74 y			
Men	64.7	100	
Women	69.6	101	
Age ≥75 y			
Men	64.1	100	
Women	76.4	101	
Diabetes	7.3	5–27	1.8–6.0
High total cholesterol	Data calculated for highest quintile (20%) vs lowest quintile	9.1 (5.7–13.8)	1.5 (95% CI 1.3–1.8)
	Continuous risk for ischemic stroke	. . .	1.25 per 1-mmol/L (38.7 mg/dL) increase
Low HDL cholesterol			
<40 mg/dL			
Men	35		
Women	15		
	Data calculated for highest quintile (20%) vs lowest quintile	23.7	0.4
<35 mg/dL	26	20.6 (10.1–30.7)	2.00 (95% CI 1.43–2.70)
	Continuous risk for ischemic stroke		≈0.5–0.6 for each 1-mmol/L increase
Atrial fibrillation (nonvalvular)			
Age 50–59 y	0.5	1.5	4.0
Age 60–69 y	1.8	2.8	2.6
Age 70–79 y	4.8	9.9	3.3
Age 80–89 y	8.8	23.5	4.5
Asymptomatic carotid stenosis	2–8	2–7§	2.0
Sickle cell disease	0.25 (of blacks)	. . .	200–400 ‖
Postmenopausal hormone therapy	25 (Women 50–74 y of age)	9	1.4
Oral contraceptive use	13 (Women 25–44 y)	9.4	2.3

(*Continued*)

Table 6-2. Continued

Factor	Prevalence, %	Population Attributable Risk, %*	RR
Dietary factors			
Na intake >2300 mg	75–90	Unknown	Unknown
K intake <4700 mg	90–99	Unknown	Unknown
Physical inactivity	25	30	2.7
Obesity			1.39 Stroke death per increase of 5 kg/m^2
Men	33.3		
Women	35.3		
CHD			
Men	8.4	5.8	1.73 (1.68–1.78)
Women	5.6	3.9¶	1.55 (1.17–2.07)
Heart failure			
Men	2.6	1.4¶	
Women	2.1	1.1¶	
Peripheral arterial disease	4.9	3.0¶	

RR indicates relative risk; SAH, subarachnoid hemorrhage; CI, confidence interval; HDL, high-density lipoprotein; and CHD, coronary heart disease.

*Population attributable risk is the proportion of ischemic stroke in the population that can be attributed to a particular risk factor (see Goldstein et al[78] for formula).

†Population attributable risk is for stroke deaths, not ischemic stroke incidence.

‡Population attributable risk percent=100[(prevalence)(RR−1)/((prevalence)(RR−1)+1)].

§Calculated on the basis of point estimates of referenced data provided in the table. For peripheral arterial disease, calculation was based on average relative risk for men and women.

‖Calculated based on referenced data provided in the table or text.

¶Relative to stroke risk in children without sickle cell disease.

Adapted from Goldstein et al.[78]

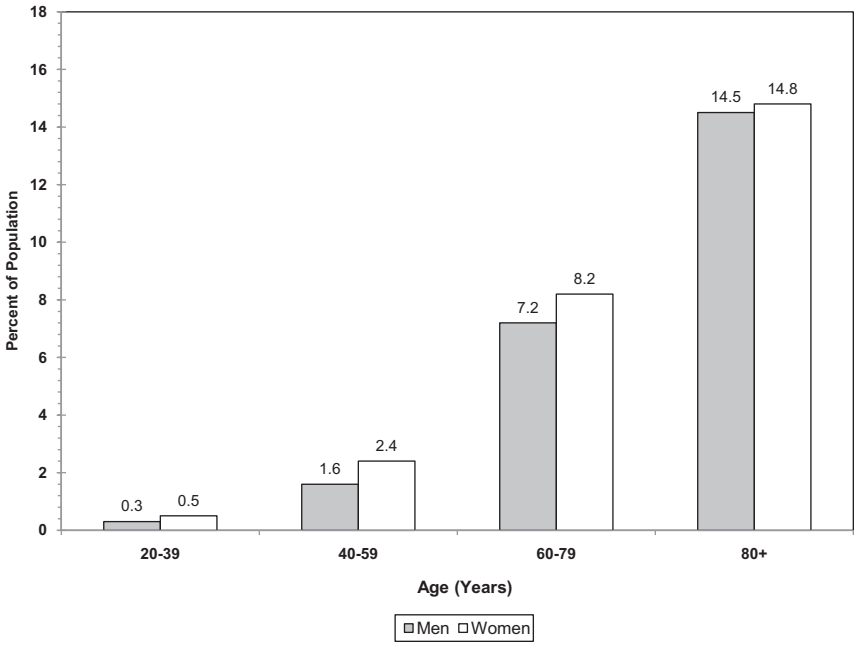


Chart 6-1. Prevalence of stroke by age and sex (National Health and Nutrition Examination Survey: 2005–2008). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

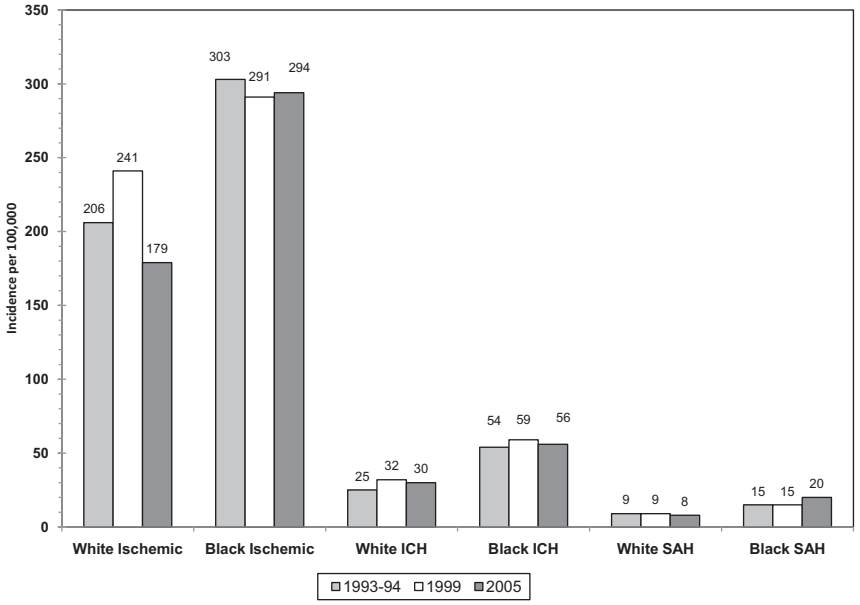


Chart 6-2. Annual age-adjusted incidence of first-ever stroke by race. Hospital plus out-of-hospital ascertainment, 1993–1994, 1999 and 2005. Data derived from Kleindorfer et al.[8]

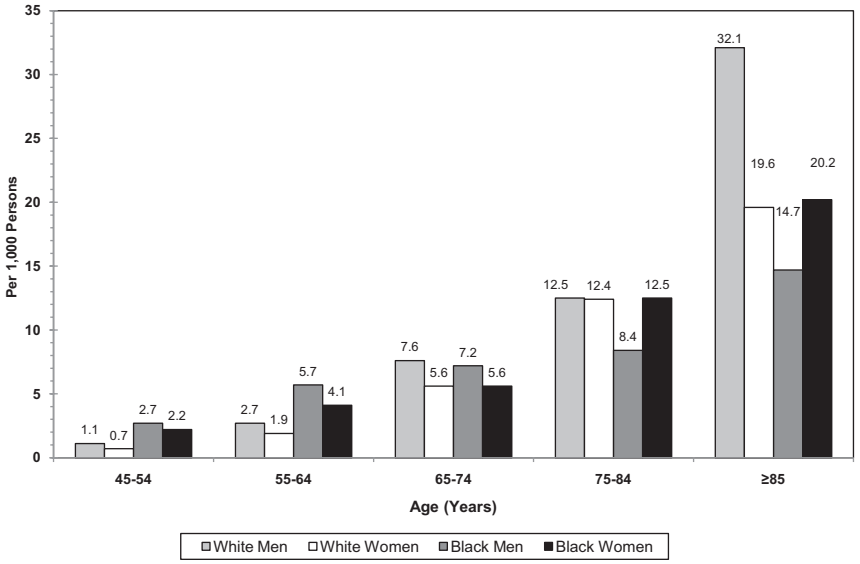


Chart 6-3. Annual rate of first cerebral infarction by age, sex, and race (Greater Cincinnati/Northern Kentucky Stroke Study: 1999). Rates for black men and women 45 to 54 years of age and for black men ≥75 years of age are considered unreliable. Source: Unpublished data from the Greater Cincinnati/Northern Kentucky Stroke Study.

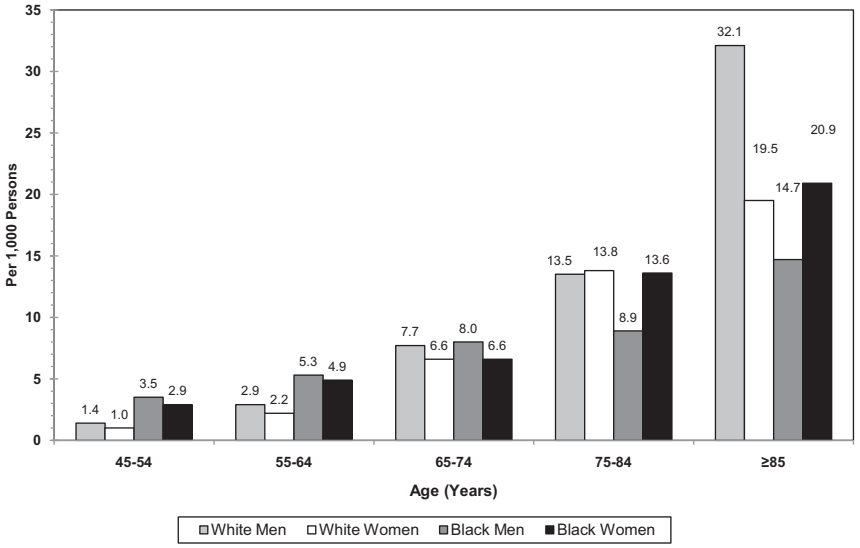


Chart 6-4. Annual rate of all first-ever strokes by age, sex, and race (Greater Cincinnati/Northern Kentucky Stroke Study: 1999). Rates for black men and women 45 to 54 years of age and for black men ≥75 years of age are considered unreliable.

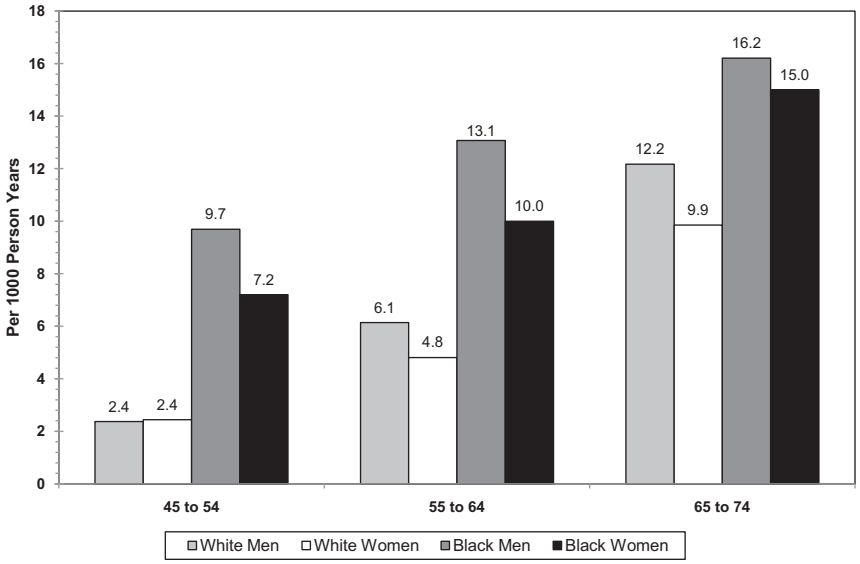


Chart 6-5. Age-adjusted incidence of stroke/transient ischemic attack by race and sex, ages 45–74 Atherosclerosis Risk in Communities study cohort, 1987–2001. Data derived from National Heart, Lung, and Blood Institute, Incidence and Prevalence Chart Book, 2006.[16]

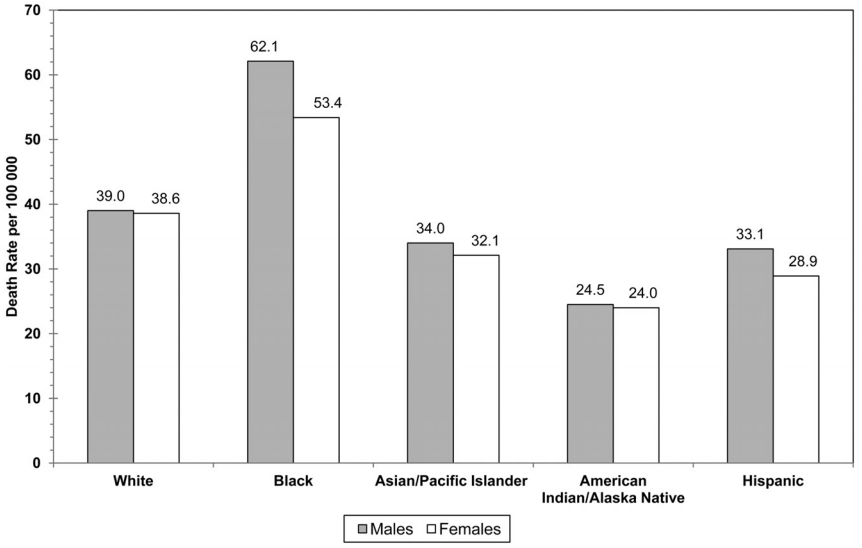


Chart 6-6. Age-adjusted death rates for stroke by sex and race/ethnicity, 2008. Death rates for the American Indian/Alaska Native and Asian or Pacific Islander populations are known to be underestimated. Stroke includes *International Classification of Diseases, 10th Revision* codes I60 to I69 (cerebrovascular disease). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

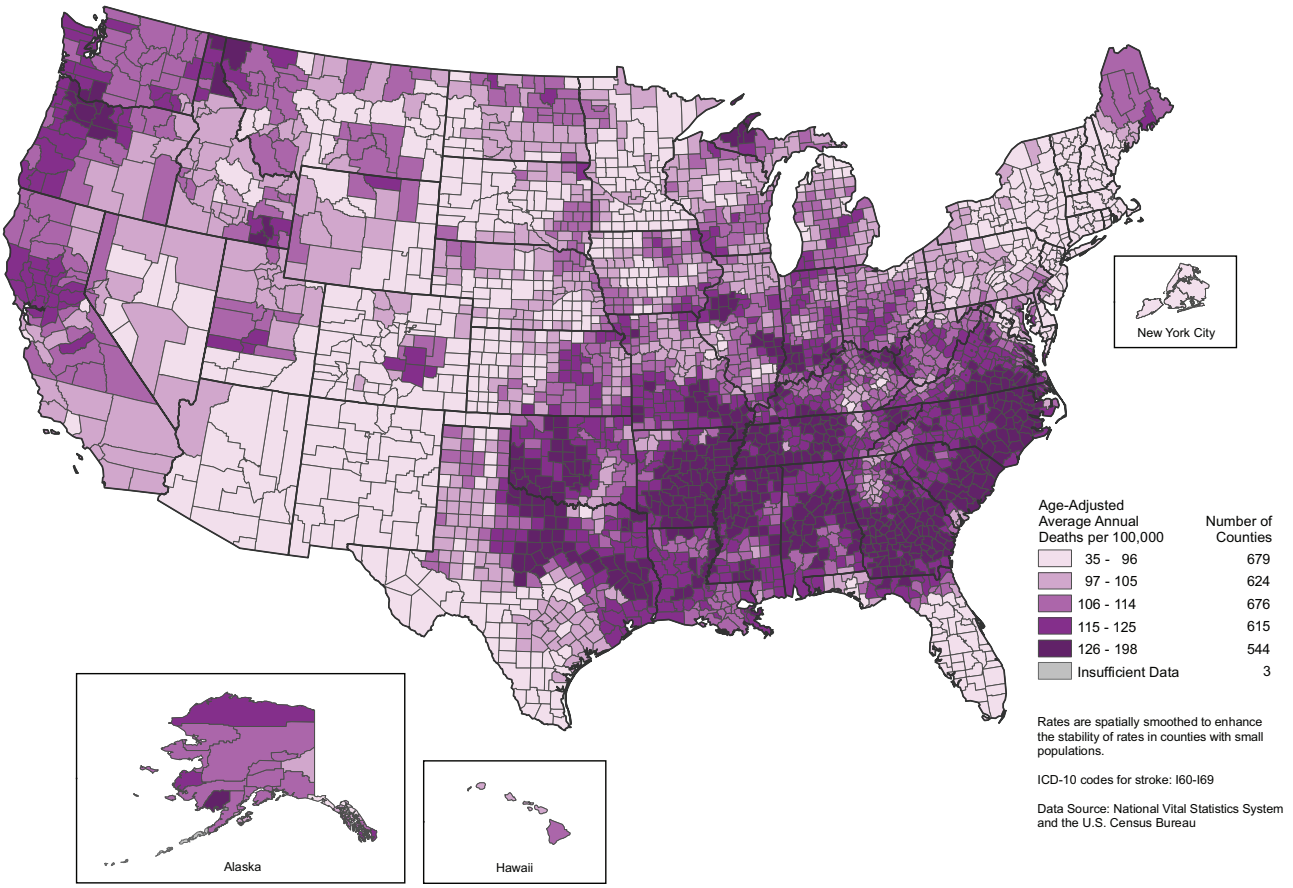
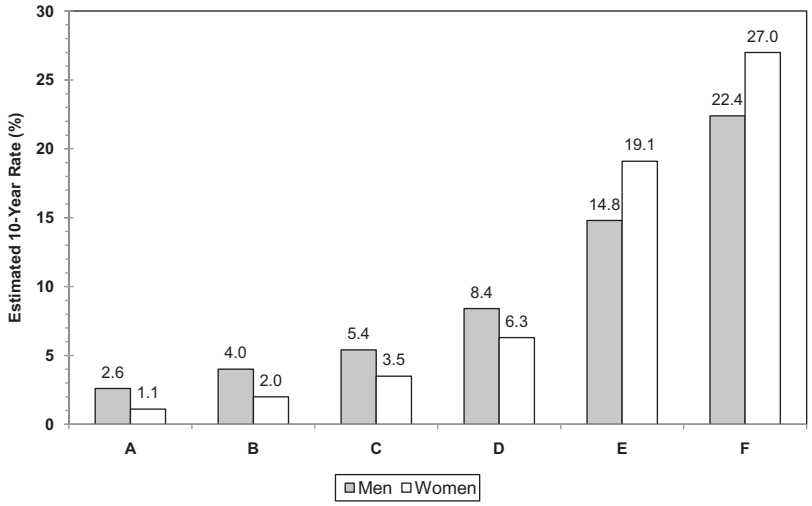


Chart 6-7. Stroke death rates, 2000–2006: adults ≥35 years of age, by county. Rates are spatially smoothed to enhance the stability of rates in counties with small populations. *International Classification of Diseases, 10th Revision* codes for stroke: I60–I69. Data source: National Vital Statistics System and the US Census Bureau.



	A	B	C	D	E	F
Blood Pressure*	95-105	138-148	138-148	138-148	138-148	138-148
Diabetes	No	No	Yes	Yes	Yes	Yes
Cigarette Smoking	No	No	No	Yes	Yes	Yes
Prior AF	No	No	No	No	Yes	Yes
Prior CVD	No	No	No	No	No	Yes

* - Closest ranges for women are : 95-104 and 115-124.

Chart 6-8. Estimated 10-year stroke risk in adults 55 years of age according to levels of various risk factors (Framingham Heart Study). AF indicates atrial fibrillation; CVD, cardiovascular disease. Data derived from Wolf et al[147] with permission of the publisher. Copyright © 1991, American Heart Association.

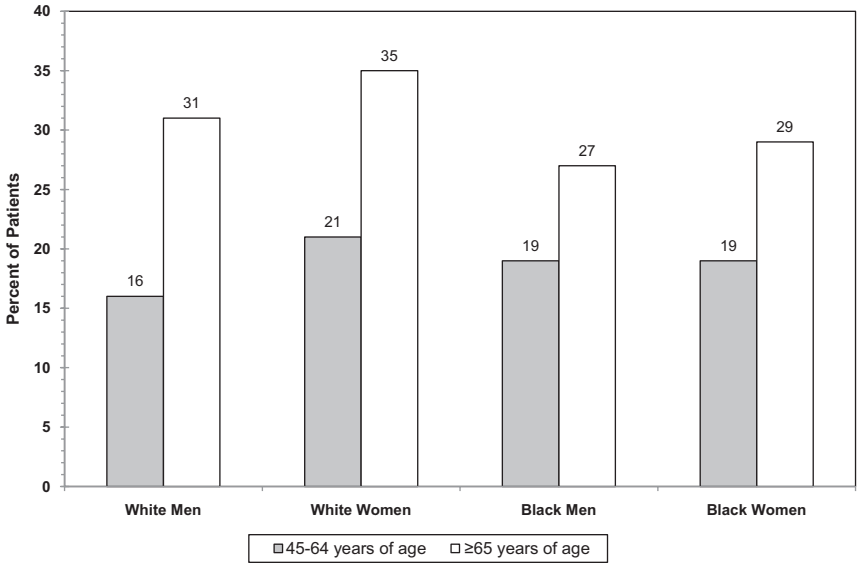


Chart 6-9. Proportion of patients dead 1 year after first stroke. Source: pooled data from the Framingham Heart Study, Atherosclerosis Risk in Communities study, and Cardiovascular Health Study of the National Heart, Lung, and Blood Institute.

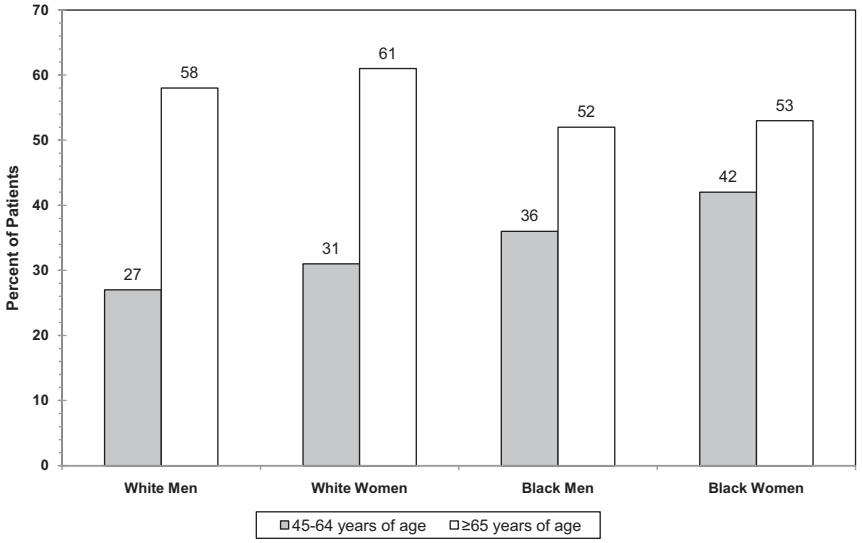


Chart 6-10. Proportion of patients dead within 5 years after first stroke. Source: pooled data from the Framingham Heart Study, Atherosclerosis Risk in Communities study, and Cardiovascular Health Study of the National Heart, Lung, and Blood Institute.

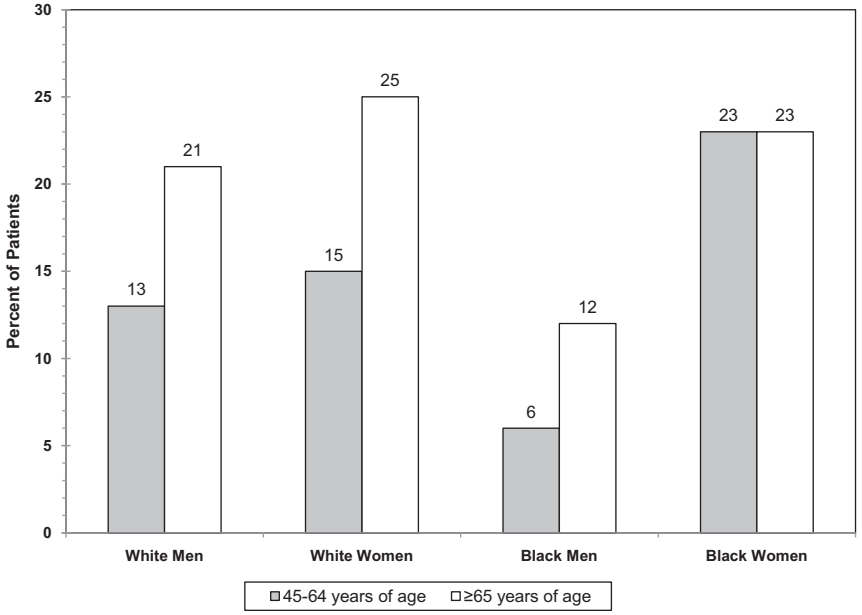


Chart 6-11. Proportion of patients with recurrent stroke in 5 years after first stroke. Source: pooled data from the Framingham Heart Study, Atherosclerosis Risk in Communities study, and Cardiovascular Health Study of the National Heart, Lung, and Blood Institute.

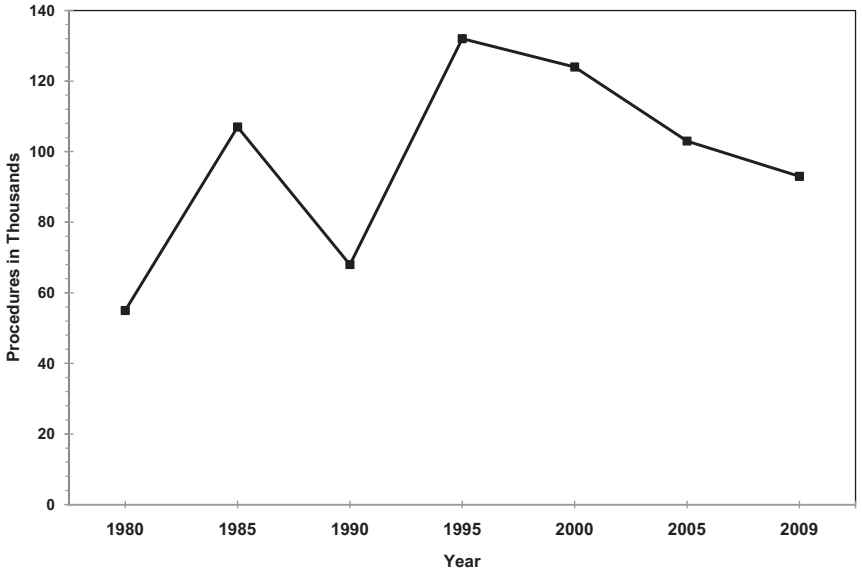


Chart 6-12. Trends in carotid endarterectomy procedures (United States: 1980–2009). Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.

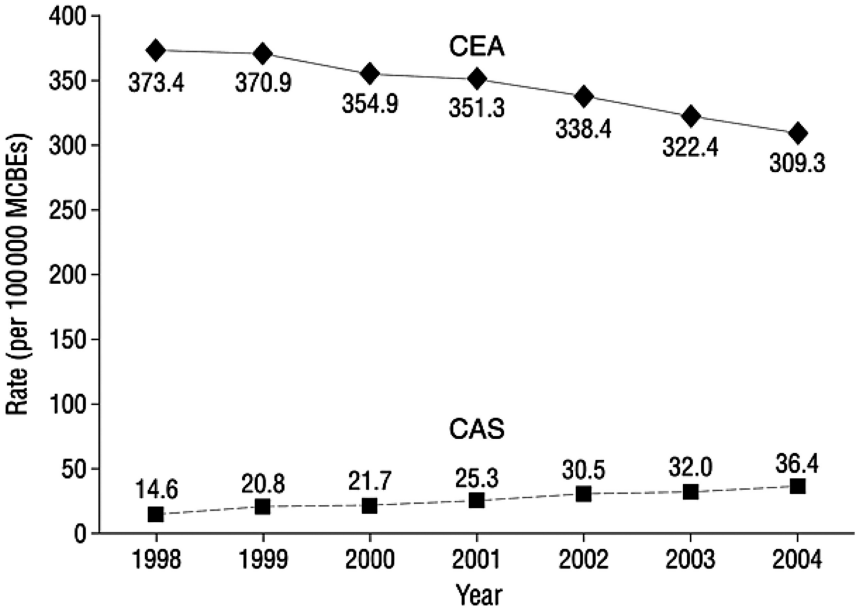


Chart 6-13. Trends in carotid revascularization procedures. MCBEs indicates Medicare beneficiaries; CEA, carotid endarterectomy; and CAS, carotid artery stenting. Reproduced with permission from Goodney et al.[136] Copyright © 2008, American Medical Association. All rights reserved.

7. High Blood Pressure

ICD-9 401 to 404, ICD-10 I10 to I15. See Tables 7-1 and 7-2 and Charts 7-1 through 7-5.

Prevalence

- HBP is defined as:
  - SBP ≥140 mm Hg or DBP ≥90 mm Hg or taking antihypertensive medicine, or
  - Having been told at least twice by a physician or other health professional that one has HBP.
- One in 3 US adults has HBP.[1]
- Data from NHANES 1999–2006 found that ≈8% of US adults have undiagnosed hypertension.[2]
- An estimated 76 400 000 adults ≥20 years of age have HBP, extrapolated to 2008 with NHANES 2005–2008 data (Table 7-1).

Abbreviations Used in Chapter 7

ARIC	Atherosclerosis Risk in Communities Study
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHF	congestive heart failure
CHS	Cardiovascular Health Study
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
FHS	Framingham Heart Study
HBP	high blood pressure
HD	heart disease
HHANES	Hispanic Health and Nutrition Examination Survey
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-9-CM	*International Classification of Diseases, Clinical Modification, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
LDL	low-density lipoprotein
MEPS	Medical Expenditure Panel Survey
MESA	Multi-Ethnic Study of Atherosclerosis
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHES	National Health Examination Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NINDS	National Institute of Neurological Disorders and Stroke
PA	physical activity
REGARDS	REasons for Geographic And Racial Differences in Stroke study
SBP	systolic blood pressure
SEARCH	Search for Diabetes in Youth Study

- NHANES data show that a higher percentage of men than women have hypertension until 45 years of age. From 45 to 54 and from 55 to 64 years of age, the percentages of men and women with hypertension are similar. After that, a higher percentage of women have hypertension than men.[3]
- HBP is 2 to 3 times more common in women taking oral contraceptives, especially among obese and older women, than in women not taking them.[4]
- Data from NHANES 2005–2006 found that 29% of US adults ≥18 years of age were hypertensive. The prevalence of hypertension was nearly equal between men and women; 7% of adults had HBP but had never been told that they had hypertension. Among hypertensive adults, 78% were aware of their condition, 68% were using antihypertensive medication, and 64% of those treated had their hypertension controlled.[5]
- Data from the 2009 BRFSS/CDC indicate that the percentage of adults ≥18 years of age who had been told that they had HBP ranged from 21.6% in Minnesota to 37.6% in West Virginia. The median percentage was 28.7%.[6]
  - According to NHANES data 2003–2008, among US adults with hypertension, 8.9% met the criteria for resistant hypertension (BP was ≥140/90 mm Hg, and they reported using antihypertensive medications from 3 different drug classes or drugs from ≥4 antihypertensive drug classes regardless of BP). This represents 12.8% of the population taking antihypertensive medication.[7]
- According to data from NHANES from 1988–1994 and 2007–2008, HBP control rates improved from 27.3% to 50.1%, treatment improved from 54.0% to 73.5%, and the control/treated rates improved from 50.6% to 72.3%.[8]
- Projections show that by 2030, an additional 27 million people could have hypertension, a 9.9% increase in prevalence from 2010.[9]

Older Adults

- In 2007 to 2008, diagnosed chronic conditions that were more prevalent among older (≥65 years of age) women than men included hypertension (58% for women, 53% for men). Ever-diagnosed conditions that were more prevalent among older men than older women included HD (38% for men, 27% for women) and DM (20% for men, 18% for women) on the basis of data from NHIS/NCHS.[10]
- The age-adjusted prevalence of hypertension (both diagnosed and undiagnosed) in 2003 to 2006 was 75% for older women and 65% for older men on the basis of data from NHANES/NCHS.[11]

Children and Adolescents

- Analysis of the NHES, the Hispanic Health and Nutrition Examination Survey, and the NHANES/NCHS surveys of the NCHS (1963–2002) found that the BP, pre-HBP, and HBP trends in children and adolescents 8 to 17 years of age moved downward from 1963 to 1988 and upward thereafter. Pre-HBP and HBP increased 2.3% and 1%, respec-

tively, between 1988 and 1999. Increased obesity (more so abdominal obesity than general obesity) partially explained the HBP and pre-HBP rise from 1988 to 1999. BP and HBP reversed their downward trends 10 years after the increase in the prevalence of obesity. In addition, an ethnic and sex gap appeared in 1988 for pre-HBP and in 1999 for HBP: Non-Hispanic blacks and Mexican Americans had a greater prevalence of HBP and pre-HBP than non-Hispanic whites, and the prevalence was greater in boys than in girls. In that study, HBP in children and adolescents was defined as SBP or DBP that was, on repeated measurement, ≥95th percentile.[12]

- A study in Ohio of >14 000 children and adolescents 3 to 18 years of age who were observed at least 3 times between 1999 and 2006 found that 3.6% had hypertension. Of these, 26% had been diagnosed and 74% were undiagnosed. In addition, 3% of those with hypertension had stage 2 hypertension, and 41% of those with stage 2 hypertension were undiagnosed. Criteria for prehypertension were met by 485 children. Of these, 11% were diagnosed. In this study, HBP in children and adolescents was defined as SBP or DBP that was, on repeated measurement, ≥95th percentile.[13]
- A study from 1988–1994 through 1999–2000 of children and adolescents 8 to 17 years of age showed that among non-Hispanic blacks, mean SBP levels increased by 1.6 mm Hg among girls and by 2.9 mm Hg among boys compared with non-Hispanic whites. Among Mexican Americans, girls' SBP increased 1.0 mm Hg and boys' SBP increased 2.7 mm Hg compared with non-Hispanic whites.[14]
- Analysis of data from the Search for Diabetes in Youth Study (SEARCH), which included children 3 to 17 years of age with type 1 and type 2 DM, found the prevalence of elevated BP among those with type 1 DM to be 5.9% and the prevalence of elevated BP among those with type 2 DM to be 23.7%.[15]

Race/Ethnicity and HBP

- The prevalence of hypertension in blacks in the United States is among the highest in the world, and it is increasing. From 1988 to 1994 through 1999 to 2002, the prevalence of HBP in adults increased from 35.8% to 41.4% among blacks, and it was particularly high among black women at 44.0%. Prevalence among whites also increased, from 24.3% to 28.1%.[16]
- Compared with whites, blacks develop HBP earlier in life, and their average BPs are much higher. As a result, compared with whites, blacks have a 1.3-times greater rate of nonfatal stroke, a 1.8-times greater rate of fatal stroke, a 1.5-times greater rate of death attributable to HD, and a 4.2-times greater rate of end-stage kidney disease (fifth and sixth reports of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure).
- Within the black community, rates of hypertension vary substantially[16,17]:
  - — Those with the highest rates are more likely to be middle-aged or older, less educated, overweight or obese, and physically inactive and are more likely to have DM.
  - — Those with the lowest rates are more likely to be younger but also overweight or obese.
  - — Those with uncontrolled HBP who are not taking antihypertensive medication tend to be male, to be younger, and to have infrequent contact with a physician.
- Analysis from the REGARDS study of the NINDS suggests that efforts to raise awareness of prevalent hypertension among blacks apparently have been successful (31% greater odds in blacks relative to whites), and efforts to communicate the importance of receiving treatment for hypertension have been successful (69% greater odds among blacks relative to whites); however, substantial racial disparities remain with regard to the control of BP (SBP <140 mm Hg, DBP <90 mm Hg), with the odds of control being 27% lower in blacks than in whites. In contrast, geographic disparities in hypertension awareness, treatment, and control were minimal.[18]
- Data from the 2010 NHIS showed that black adults ≥18 years of age were more likely (33.8%) to have been told on ≥2 occasions that they had hypertension than white adults (23.6%) or Asian adults (20.5%); there was no significant difference between the estimates for American Indian/Alaska Native adults (30.0%) and black adults.[19]
- The CDC analyzed death certificate data from 1995 to 2002 (any-mention mortality; ICD-9 codes 401–404 and ICD-10 codes I10–I13). The results indicated that Puerto Rican Americans had a consistently higher hypertension-related death rate than all other Hispanic subpopulations and non-Hispanic whites. The age-standardized hypertension-related mortality rate was 127.2 per 100 000 population for all Hispanics, similar to that of non-Hispanic whites (135.9). The age-standardized rate for Hispanic females (118.3) was substantially lower than that observed for Hispanic males (135.9). Hypertension-related mortality rates for males were higher than rates for females for all Hispanic subpopulations. Puerto Rican Americans had the highest hypertension-related death rate among all Hispanic subpopulations (154.0); Cuban Americans had the lowest (82.5).[20]
- Some studies suggest that Hispanic Americans have rates of HBP similar to or lower than those of non-Hispanic white Americans. Findings from a new analysis of combined data from the NHIS of 2000 to 2002 point to a health disparity between black and white adults of Hispanic descent. Black Hispanics were at slightly greater risk than white Hispanics, although non-Hispanic black adults had by far the highest rate of HBP. The racial disparity among Hispanics also was evident in the fact that higher-income, better-educated black Hispanics still had a higher rate of HBP than lower-income, less-educated white Hispanics.[21] Data from the NHLBI's ARIC study found that hypertension was a particularly powerful risk factor for CHD in black people, especially black women.[22]
- Data from MESA found that being born outside the United States, speaking a language other than English at home, and living fewer years in the United States were each associated with a decreased prevalence of hypertension.[23]
- Filipino (27%) and Japanese (25%) adults were more likely than Chinese (17%) or Korean (17%) adults to have ever been told that they had hypertension.[24]

Mortality

HBP mortality in 2008 was 61 005. Any-mention mortality in 2008 was 347 689 (NHLBI tabulation of NCHS mortality data). The 2008 death rate was 18.3.[25]

- From 1998 to 2008, the death rate caused by HBP increased 20.2%, and the actual number of deaths rose 49.7% (NCHS and NHLBI; appropriate comparability ratios were applied).[25,25a]
- The 2008 overall death rate resulting from HBP was 18.3. Death rates were 16.5 for white males, 50.3 for black males, 14.5 for white females, and 38.6 for black females. When any-mention mortality for 2008 was used, the overall death rate was 108.5. Death rates were 108.6 for white males, 228.8 for black males, 90.7 for white females, and 174.8 for black females (NHLBI tabulation of NCHS mortality data).
- Analysis of NHANES I and II comparing hypertensive and nonhypertensive individuals found a reduction in age-adjusted mortality rate of 4.6/1000 person-years among people with hypertension compared with a reduction of 4.2/1000 person-years among those without hypertension.[26]
- Assessment of 30-year follow-up of the Hypertension Detection and Follow-Up Program identified the long-term benefit of stepped care, and the increased survival for hypertensive African Americans.[27]
- Assessment of the Charleston Heart Study and Evans County Heart Study identified the excess burden of elevated BP for African Americans and its effect on long-term health outcomes.[28]

Risk Factors

- Numerous risk factors and markers for development of hypertension, including age, ethnicity, family history of hypertension and genetic factors, lower education and socioeconomic status, greater weight, lower PA, tobacco use, psychosocial stressors, sleep apnea, and dietary factors (including dietary fats, higher sodium intake, lower potassium intake, and excessive alcohol intake), have been identified.
- A study of related individuals in the NHLBI's FHS suggested that different sets of genes regulate BP at different ages.[29]
- Recent data from the Nurses' Health Study suggest that a large proportion of incident hypertension in women can be prevented by controlling dietary and lifestyle risk factors.[30]
- A meta-analysis identified the benefit of a goal BP of 130/80 mm Hg for individuals with hypertension and type 2 DM but less evidence for treatment below this value.[31]

Aftermath

- Approximately 69% of people who have a first heart attack, 77% of those who have a first stroke, and 74% of those who have CHF have BP >140/90 mm Hg (NHLBI unpublished estimates from ARIC, CHS, and FHS Cohort and Offspring studies).
- Data from FHS/NHLBI indicate that recent (within the past 10 years) and remote antecedent BP levels may be an important determinant of risk over and above the current BP level.[32]
- Data from the FHS/NHLBI indicate that hypertension is associated with shorter overall life expectancy, shorter life expectancy free of CVD, and more years lived with CVD.[33]
  - — Total life expectancy was 5.1 years longer for normotensive men and 4.9 years longer for normotensive women than for hypertensive people of the same sex at 50 years of age.
  - — Compared with hypertensive men at 50 years of age, men with untreated BP <140/90 mm Hg survived on average 7.2 years longer without CVD and spent 2.1 fewer years of life with CVD. Similar results were observed for women.

Hospital Discharges/Ambulatory Care Visits

- From 1999 to 2009, the number of inpatient discharges from short-stay hospitals with HBP as the first-listed diagnosis increased from 439 000 to 579 000 (no significant difference; NCHS, NHDS). The number of all-listed discharges increased from 7 629 000 to 11 591 000 (NHLBI, unpublished data from the NHDS, 2009).
- Data from ambulatory medical care utilization estimates for 2009 showed that the number of visits for essential hypertension was 55 148 000. Of these, 49 966 000 were physician office visits, 1 000 000 were ED visits, and 4 182 000 were outpatient department visits (NCHS, NAMCS and NHAMCS, NHLBI tabulation).
- In 2009, there were 372 000 hospitalizations with a first-listed diagnosis of essential hypertension (ICD-9-CM code 401), but essential hypertension was listed as either a primary or a secondary diagnosis 9 317 000 times for hospitalized inpatients (NHLBI, unpublished data from the NHDS, 2009).

Awareness, Treatment, and Control

- Data from NHANES/NCHS 2005–2008 showed that of those with hypertension who were ≥20 years of age, 79.6% were aware of their condition, 70.9% were under current treatment, 47.8% had their hypertension under control, and 52.2% did not have it controlled (NHLBI tabulation, NCHS, NHANES data).
- Data from NHANES 1999–2006 showed that 11.2% of adults ≥20 years of age had treated and controlled BP levels.[34]
- Analysis of NHANES/NCHS data from 1999–2004 through 2005–2006 found that there were substantial increases in awareness and treatment rates of hypertension. The control rates increased in both sexes, in non-Hispanic blacks, and in Mexican Americans. Among the group ≥60 years of age, awareness, treatment, and control rates of hypertension increased significantly.[5,35]
- In NHANES/NCHS 2005–2006, rates of control were lower in Mexican Americans (35.2%) than in non-Hispanic whites (46.1%) and non-Hispanic blacks (46.5%).[5]
- The awareness, treatment, and control of HBP among those ≥65 years of age in the CHS/NHLBI improved during the 1990s. The percentages of those aware of and treated for

HBP were higher among blacks than among whites. Prevalence rates with HBP under control were similar. For both groups combined, the control of BP to <140/90 mm Hg increased from 37% in 1990 to 49% in 1999. Improved control was achieved by an increase in antihypertensive medications per person and by an increase in the proportion of the CHS population treated for hypertension from 34.5% to 51.1%.[36]

- Data from the FHS of the NHLBI show that:
  - Among those ≥80 years of age, only 38% of men and 23% of women had BPs that met targets set forth in the National High Blood Pressure Education Program's clinical guidelines. Control rates in men <60, 60 to 79, and ≥80 years of age were 38%, 36%, and 38%, respectively; for women in the same age groups, they were 38%, 28%, and 23%, respectively.[37]
- Data from the WHI observational study of nearly 100 000 postmenopausal women across the country enrolled between 1994 and 1998 indicate that although prevalence rates ranged from 27% of women 50 to 59 years of age to 41% of women 60 to 69 years of age to 53% of women 70 to 79 years of age, treatment rates were similar across age groups: 64%, 65%, and 63%, respectively. Despite similar treatment rates, hypertension control is especially poor in older women, with only 29% of hypertensive women 70 to 79 years of age having clinic BPs <140/90 mm Hg compared with 41% and 37% of those 50 to 59 and 60 to 69 years of age, respectively.[38]
- Among a cohort of postmenopausal women taking hormone replacement, hypertension was the most common comorbidity, with a prevalence of 34%.[39]
- A study of >300 women in Wisconsin showed a need for significant improvement in BP and LDL levels. Of the screened participants, 35% were not at BP goal, 32.4% were not at LDL goal, and 53.5% were not at both goals.[40]
- In 2005, a survey of people in 20 states conducted by the BRFSS of the CDC found that 19.4% of respondents had been told on ≥2 visits to a health professional that they had HBP. Of these, 70.9% reported changing their eating habits; 79.5% reduced the use of or were not using salt; 79.2% reduced the use of or eliminated alcohol; 68.8% were exercising; and 73.4% were taking antihypertensive medication.[41]
- On the basis of NHANES 2003–2004 data, it was found that nearly three fourths of adults with CVD comorbidities have hypertension. Poor control rates of systolic hypertension remain a principal problem that further compromises their already high CVD risk.[42]
- According to data from NHANES 2001–2006, non-Hispanic blacks had 90% higher odds of poorly controlled BP than non-Hispanic whites. Among those who were hypertensive, non-Hispanic blacks and Mexican Americans had 40% higher odds of uncontrolled BP than non-Hispanic whites.[43]

Cost

- The estimated direct and indirect cost of HBP for 2008 is $50.6 billion (MEPS, NHLBI tabulation).

Prehypertension

- Prehypertension is untreated SBP of 120 to 139 mm Hg or untreated DBP of 80 to 89 mm Hg and not having been told on 2 occasions by a physician or other health professional that one has hypertension.
- Data from NHANES 1999–2006 estimate that 29.7% of adults ≥20 years of age have prehypertension.[34]
- Follow-up of 9845 men and women in the FHS/NHLBI who attended examinations from 1978 to 1994 revealed that at 35 to 64 years of age, the 4-year incidence of hypertension was 5.3% for those with baseline BP <120/80 mm Hg, 17.6% for those with SBP of 120 to 129 mm Hg or DBP of 80 to 84 mm Hg, and 37.3% for those with SBP of 130 to 139 mm Hg or DBP of 85 to 89 mm Hg. At 65 to 94 years of age, the 4-year incidences of hypertension were 16.0%, 25.5%, and 49.5% for these BP categories, respectively.[44]
- Data from FHS/NHLBI also reveal that prehypertension is associated with elevated relative and absolute risks for CVD outcomes across the age spectrum. Compared with normal BP (<120/80 mm Hg), prehypertension was associated with a 1.5- to 2-fold increased risk for major CVD events in those <60, 60 to 79, and ≥80 years of age. Absolute risks for major CVD associated with prehypertension increased markedly with age: 6-year event rates for major CVD were 1.5% in prehypertensive people <60 years of age, 4.9% in those 60 to 79 years of age, and 19.8% in those ≥80 years of age.[37]
- In a study of NHANES 1999–2000 (NCHS), people with prehypertension were more likely than those with normal BP levels to have above-normal cholesterol levels, overweight/obesity, and DM, whereas the probability of currently smoking was lower. People with prehypertension were 1.65 times more likely to have 1 or more of these adverse risk factors than were those with normal BP.[45]
- Assessment of the REGARDS data identified high risk of prehypertension to be associated with increased age and black race.[46]

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Table 7-1. High Blood Pressure

Population Group	Prevalence, 2008: Age ≥20 y	Mortality,* 2008: All Ages	Hospital Discharges, 2009: All Ages	Estimated Cost, 2008
Both sexes	76 400 000 (33.5%)	61 005	579 000	$50.6 billion
Males	36 500 000 (34.1%)	26 776 (43.9%)†	260 000	. . .
Females	39 900 000 (32.7%)	34 229 (56.1%)†	319 000	. . .
NH white males	33.9%	19 576	. . .	. . .
NH white females	31.3%	26 342	. . .	. . .
NH black males	43.0%	6370	. . .	. . .
NH black females	45.7%	7002	. . .	. . .
Mexican American males	27.8%	. . .	. . .	. . .
Mexican American females	28.9%	. . .	. . .	. . .
Hispanic or Latino‡	24.7%	. . .	. . .	. . .
Asian‡	20.5%		. . .	. . .
American Indian/Alaska Native‡	30.0%		. . .	. . .

Ellipses (. . .) indicate data not available; NH, non-Hispanic.

*Mortality data for the white, black, Asian or Pacific Islander, and American Indian/Alaska Native populations include deaths among persons of Hispanic and non-Hispanic origin. Numbers of deaths for the American Indian/Alaska Native and Asian or Pacific Islander populations are known to be underestimated.

†These percentages represent the portion of total high blood pressure mortality that is for males vs females.

‡National Health Interview Survey (2010), National Center for Health Statistics; data are weighted percentages for Americans ≥18 years of age. Source: Schiller et al.[19]

Sources: Prevalence: National Health and Nutrition Examination Survey (2005–2008, National Center for Health Statistics) and National Heart, Lung, and Blood Institute. Percentages for racial/ethnic groups are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2008 US population estimates. Mortality: National Center for Health Statistics. These data represent underlying cause of death only. Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics; data include those discharged alive, dead, or status unknown. Cost: Medical Expenditure Panel Survey data include estimated direct costs for 2007; indirect costs calculated by National Heart, Lung, and Blood Institute for 2007.

Hypertension is defined in terms of National Health and Nutrition Examination Survey blood pressure measurements and health interviews. A subject was considered hypertensive if systolic blood pressure was ≥140 mm Hg or diastolic blood pressure was ≥90 mm Hg, if the subject said "yes" to taking antihypertensive medication, or if the subject was told on 2 occasions that he or she had hypertension.

Table 7-2. Hypertension Awareness, Treatment, and Control: NHANES 1988–1994 and 1999–2008, by Race and Sex

	Awareness, %		Treatment, %		Control, %	
	1988–1994	1999–2008	1988–1994	1999–2008	1988–1994	1999–2008
NH white male	63.0	73.5	46.2	63.8	22.0	44.1
NH white female	74.7	78.2	61.6	70.0	32.2	42.7
NH black male	62.5	70.8	42.3	60.3	16.6	35.2
NH black female	77.8	85.8	64.6	77.0	30.0	45.3
Mexican American male	47.8	59.5	30.9	46.1	13.5	30.3
Mexican American female	69.3	70.1	47.8	59.9	19.4	34.2

NHANES indicates National Health and Nutrition Examination Survey; NH, non-Hispanic.

Sources: NHANES (1988–1994, 1999–2008) and National Heart, Lung, and Blood Institute.

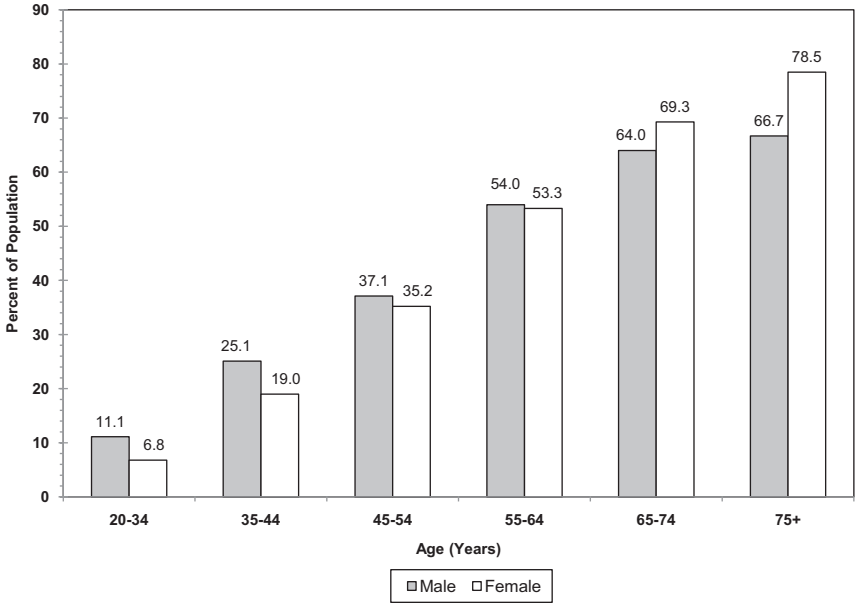

Chart 7-1. Prevalence of high blood pressure in adults ≥20 years of age by age and sex (National Health and Nutrition Examination Survey: 2005–2008). Hypertension is defined as systolic blood pressure >140 mm Hg or diastolic blood pressure >90 mm Hg, taking antihypertensive medication, or being told twice by a physician or other professional that one has hypertension. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

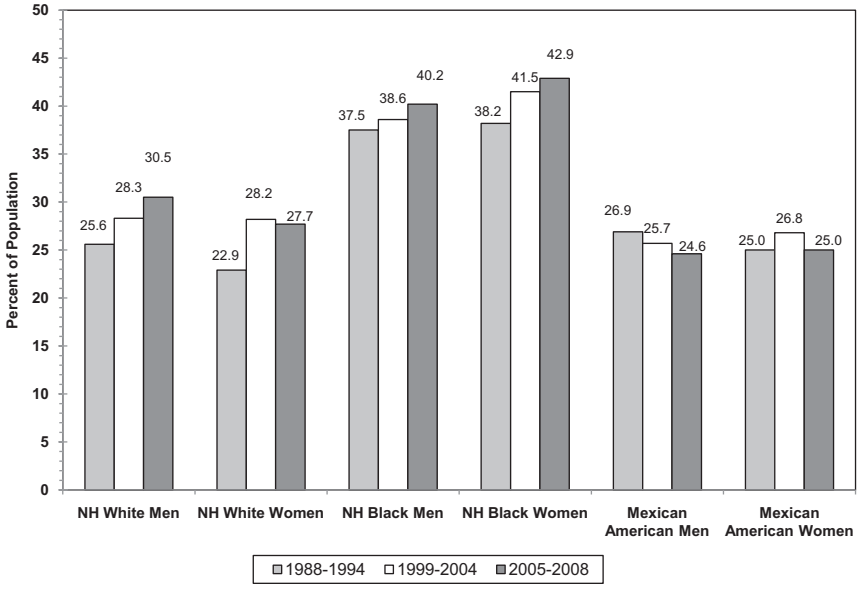

Chart 7-2. Age-adjusted prevalence trends for high blood pressure in adults ≥20 years of age by race/ethnicity, sex, and survey (National Health and Nutrition Examination Survey: 1988–1994, 1999–2004, and 2005–2008). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

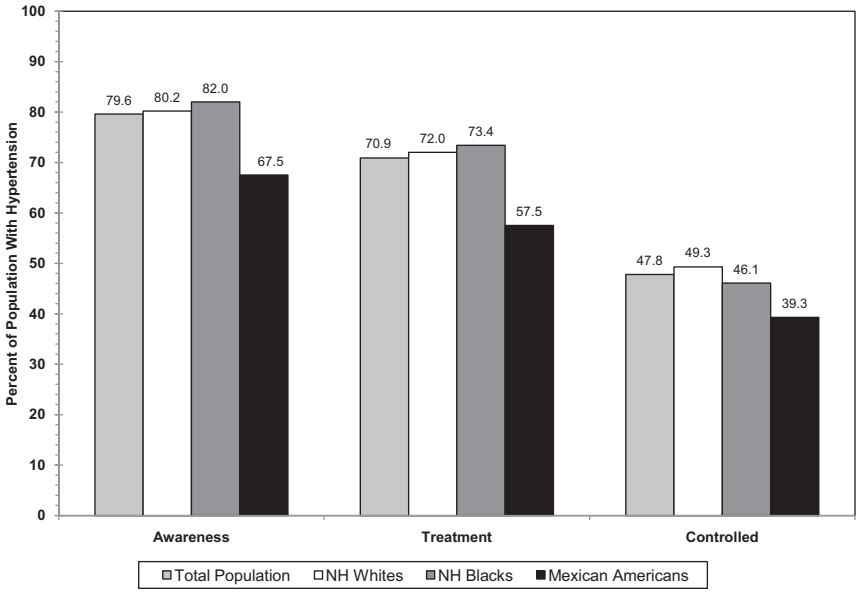

Chart 7-3. Extent of awareness, treatment, and control of high blood pressure by race/ethnicity (National Health and Nutrition Examination Survey: 2005–2008). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

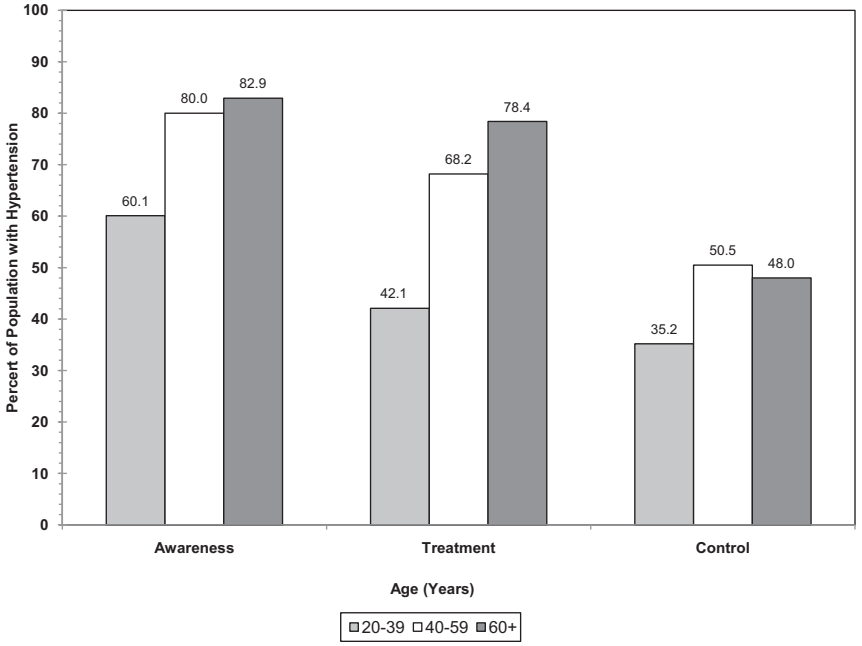

Chart 7-4. Extent of awareness, treatment, and control of high blood pressure by age (National Health and Nutrition Examination Survey: 2005–2008). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

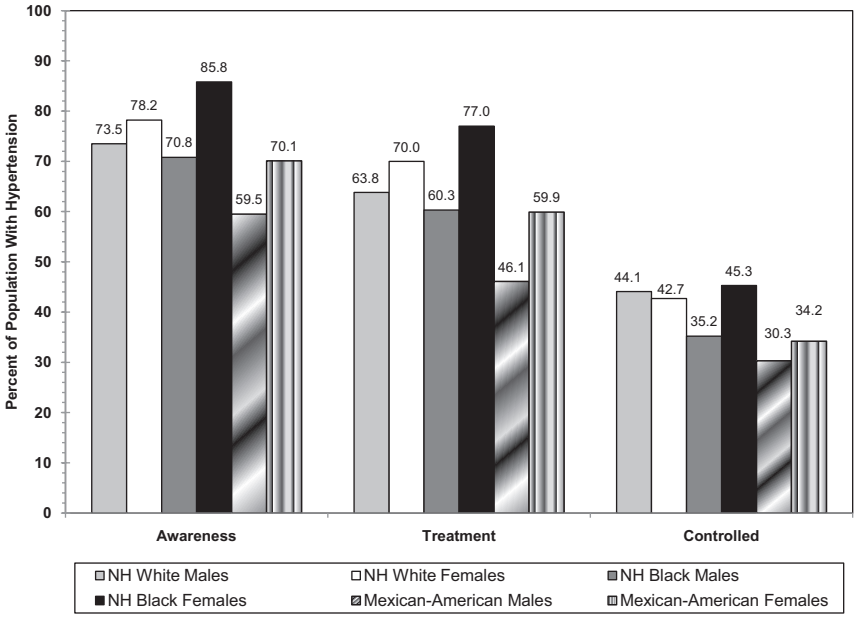

Chart 7-5. Extent of awareness, treatment, and control of high blood pressure by race/ethnicity and sex (National Health and Nutrition Examination Survey: 1999–2008). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

8. Congenital Cardiovascular Defects

ICD-9 745 to 747, ICD-10 Q20 to Q28. See Tables 8-1 through 8-4.

Congenital cardiovascular defects, also known as congenital heart defects, are structural problems that arise from abnormal formation of the heart or major blood vessels. ICD-9 lists 25 congenital heart defects codes, of which 21 designate specified anatomic or hemodynamic lesions.

Defects range in severity from tiny pinholes between chambers that may resolve spontaneously to major malformations that can require multiple surgical procedures before school age and may result in death in utero, in infancy, or in childhood. The common complex defects include the following:

- Tetralogy of Fallot (TOF)
- Transposition of the great arteries (TGA)
- Atrioventricular (AV) septal defects
- Coarctation of the aorta
- Hypoplastic left heart syndrome

Congenital heart defects are serious and common conditions that have significant impact on morbidity, mortality, and healthcare costs in children and adults.[1–4]

Incidence

The most commonly reported incidence of congenital heart defects in the United States is between 4 and 10 per 1000, clustering around 8 per 1000 live births.[5,6] Variations in reported number of incident cases are largely accounted for by the age at detection and the method of diagnosis. Major defects may be apparent in the prenatal or neonatal period, but minor defects may not be detected until adulthood. Detection rates have increased since the advent of cardiac ultrasound.[4] Thus, true measures of the incidence of congenital HD would need to record new cases of defects that present from fetal life onward. Because most estimates are available for new cases detected between birth and the first year of life, birth prevalence is the best proxy for incident congenital heart defects. These are typically reported as cases per 1000 live births per year and do not distinguish between tiny defects that resolve without treatment and major malformations. To distinguish more serious defects, some studies also report new cases of sufficient severity to require an invasive procedure or that result in death within the first year of life. Despite the absence of true incidence figures, some data are available and are provided in Table 8-2.

- Using population-based data from the Metropolitan Atlanta Congenital Defects Program (MACDP) in metropolitan Atlanta, GA, congenital heart defects occurred in 1 of every 111 to 125 births (live, still, or >20 weeks' gestation) from 1995 to 1997 and from 1998 to 2005, with variations in sex and racial distribution of some lesions.[4,5]
- Analysis of contemporary birth cohorts with MACDP data revealed that the most common defects at birth were ventricular septal defect (VSD; 4.2/1000), atrial septal defect (ASD; 1.3/1000), valvar pulmonic stenosis (0.6/1000); TOF (0.5/1000), aortic coarctation (0.4/1000), AV septal defect (0.4/1000), and TGA (0.2/1000).[5,7]
- An estimated minimum of 32 000 infants are expected to be affected each year in the United States. Of these, an approximate 25%, or 2.4 per 1000 live births, require invasive treatment in the first year of life.[1]
- Estimates also are available for bicuspid aortic valves, which occur in 13.7 per 1000 people; these defects may not require treatment in infancy but can cause problems later in adulthood.[8]
- Data collected by the National Birth Defects Prevention Network from 11 states from 1999 to 2001 showed the average prevalence of 18 selected major birth defects. These data indicated that there are >6500 estimated annual cases of 5 cardiovascular defects: truncus arteriosus, TGA, TOF, AV septal defect, and hypoplastic left heart syndrome.[9]

Prevalence

The 32nd Bethesda Conference estimated that the total number of adults living with congenital HD in the United States in 2000 was 800 000.[2,3] In the United States, 1 in 150 adults are expected to have some form of congenital HD.[3] Nearly 2 decades ago, the estimated number of children with congenital heart defects in the United States was 600 000.[1] In population data from Canada, the measured prevalence of congenital cardiac defects in the general population was 11.89 per 1000 children and 4.09 per 1000 adults in the year 2000.[10] Extrapolated to the US population in the same year, this yields published estimates of 859 000 children and 850 000 adults over a decade ago,[7] with expected growth rates of the congenital heart defects population varying from 1% to 5% per year depending on the age and distribution of lesions.[2,10]

Estimates of the distribution of lesions in the congenital heart defects population using available data vary with assumptions made. If all those born were treated, there would be 750 000 survivors with simple lesions, 400 000 with moderate lesions, and 180 000 with complex lesions; in addition, there would be 3 000 000 subjects alive with bicus-

Abbreviations Used in Chapter 8

ASD	atrial septal defect
AV	atrioventricular
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CI	confidence interval
DM	diabetes mellitus
HD	heart disease
HPLHS	hypoplastic left heart syndrome
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
MACDP	Metropolitan Atlanta Congenital Defects Program
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHLBI	National Heart, Lung, and Blood Institute
TGA	transposition of the great arteries
TOF	tetralogy of Fallot
VSD	ventricular septal defect

pid aortic valves.[11] Without treatment, the number of survivors in each group would be 400 000, 220 000, and 30 000, respectively. The actual numbers surviving are projected to be between these 2 sets of estimates as of 1 decade ago.[11] Using measurements from population data in Canada, the prevalence of severe forms of congenital heart defects increased 85% in adults and 22% in children from 1985 to 2000.[10] The most common types of defects in children are (at a minimum) VSD, 620 000 people; ASD, 235 000 people; valvular pulmonary stenosis, 185 000 people; and patent ductus arteriosus, 173 000 people.[11] The most common lesions seen in adults are ASD and TOF.[2]

Risk Factors

- Numerous intrinsic and extrinsic nongenetic risk factors contribute to CHD.[12]
- Attributable risks or fractions have been shown to include paternal anesthesia in TOF (3.6%), sympathomimetic medication for coarctation of the aorta (5.8%), pesticides for VSD (5.5%), and solvents for hypoplastic left heart syndrome (4.6%).[13]
- A study of infants born with heart defects unrelated to genetic syndromes who were included in the National Birth Defects Prevention Study found that women who reported smoking in the month before becoming pregnant or in the first trimester were more likely to give birth to a child with a septal defect. Compared with the infants of mothers who did not smoke during pregnancy, infants of mothers who were heavy smokers (≥25 cigarettes daily) were twice as likely to have a septal defect.[14]
- Data from the Baltimore-Washington Infant Study reported that maternal smoking during the first trimester of pregnancy was associated with at least a 30% increased risk of the following lesions in the fetus: ASD, pulmonary valvar stenosis, truncus arteriosus, and TGA.[15]
- Associations between exposure to air pollutants during first-trimester pregnancy and risks of congenital heart defects were documented from 1986 to 2003 by the MACDP that related carbon monoxide, nitrogen dioxide, and sulfur dioxide measurements to the risk of ASD, VSD, TGA, and TOF.[16]
- The results of a population-based study examining pregnancy obesity found a weak to moderate positive association of maternal obesity with 7 of 16 categories of birth defects, including heart defects.[17]
- Although folic acid supplementation is recommended during pregnancy to potentially reduce the risk of congenital heart defects,[12] there has been only 1 US population-based case-control study, performed with the Baltimore-Washington Infant Study between 1981 and 1989, that showed an inverse relationship between folic acid use and the risk of TGA.[18] A study from Quebec, Canada, that analyzed 1.3 million births from 1990 to 2005 found a significant 6% per year reduction in severe congenital heart defects using a time-trend analysis before and after public health measures were instituted that mandated folic acid fortification of grain and flour products in Canada.[19]
- Pregestational DM was significantly associated with cardiac defects, both isolated and multiple. Gestational DM was associated with a limited group of birth defects.[20]

Mortality

Mortality related to congenital cardiovascular defects in 2008 was 3415. Any-mention mortality related to congenital cardiovascular defects in 2008 was 5359.[21]

- Congenital cardiovascular defects are the most common cause of infant death resulting from birth defects; >24% of infants who die of a birth defect have a heart defect.[21]
- The mortality rate attributable to congenital heart defects in the United States has continued to decline from 1979 to 1997 and from 1999 to 2006. Age-adjusted death rates attributable to all congenital heart defects declined 21% to 39%, and deaths tended to occur at progressively older ages. Nevertheless, mortality in infants <1 year of age continues to account for nearly half of the deaths, with persistence of ethnicity differences revealing higher mortality rates in non-Hispanic blacks.[15,22]
- When CDC death registry data were used to examine mortality in cyanotic and acyanotic lesions between 1979 and 2005, all-age mortality rates had declined by 60% for VSD and 40% for TOF.[23]
- In population-based data from Canada, 8123 deaths occurred among 71 686 congenital HD patients followed up for nearly 1 million patient-years. Overall mortality decreased by 31%, and the median age of death increased from 2 to 23 years between 1987 and 2005.[24]
- The 2008 death rate attributable to congenital cardiovascular defects was 1.1. Death rates were 1.2 for white males, 1.5 for black males, 1.0 for white females, and 1.2 for black females. Infant mortality rates (<1 year of age) were 34.9 for white infants and 46.5 for black infants.[21]
- According to CDC multiple-cause death data, from 1999 to 2006, sex differences in mortality over time varied with age. Between the ages of 18 and 34 years, mortality over time decreased significantly in females but not in males.[25]
- On the basis of data from the Healthcare Cost and Utilization Project's Kids' Inpatient Database from 2000, 2003, and 2006, male children had more congenital heart defect surgeries in infancy, more high-risk surgeries, and more procedures to correct multiple congenital heart defects. Female infants with high risk congenital heart defects had a 39% higher adjusted mortality.[26]
- In 2007, 189 000 life-years were lost before 55 years of age because of deaths attributable to congenital cardiovascular defects. This is almost as many as life-years as were lost from leukemia and asthma combined (NHLBI tabulation of NCHS mortality data).
- Data from the Pediatric Heart Network conducted in 15 North American centers revealed that even in lesions associated with the highest mortality among congenital lesions, such as hypoplastic left heart syndrome, aggressive palliation can lead to an increase in the 12-month survival rate from 64% to 74%.[27]
- Data analysis from the Society of Thoracic Surgeons, a voluntary registry with self-reported data for a 4-year cycle (2006–2009) from 68 centers performing congenital heart surgery (67 from the United States and 1 from Canada), showed that for 88 989 total operations, the overall aggregate hospital discharge mortality rate was 3.6%[28]; specifi-

cally, for neonates (0–30 days of age), the mortality rate was 10.2%[29]; for infants (31 days to 1 year of age), it was 2.8%[30]; for children (>1 year to 18 years of age), it was 1.1%[31]; and for adults (>18 years of age), it was 1.8%.[32]

- Using the Nationwide Inpatient Sample 1988–2003, mortality was examined for 12 congenital heart defects procedures. A total of 30 250 operations were identified, which yielded a national estimate of 152 277±7875 operations. Of these, 27% were performed in patients ≥18 years of age. The overall in-hospital mortality rate for adult congenital heart defect patients was 4.71% (95% CI 4.19% to 5.23%), with a significant reduction in mortality observed when surgery was performed on adult congenital heart defect patients by pediatric versus nonpediatric heart surgeons (1.87% versus 4.84%; $P<0.0001$).[33]

Hospitalizations

In 2004, birth defects accounted for >139 000 hospitalizations, representing 47.4 stays per 100 000 people. Cardiac and circulatory congenital anomalies accounted for 34% of all hospital stays for birth defects. Although the most common congenital lesions were shunts, including patent ductus arteriosus, VSDs, and ASDs, TOF accounted for a higher proportion of in-hospital death than any other birth defect. Between 1997 and 2004, hospitalization rates increased by 28.5% for cardiac and circulatory congenital anomalies.[34]

Cost

- From 2003 data from the Healthcare Cost and Utilization Project 2003 Kids' Inpatient Database and information on birth defects in the Congenital Malformations Surveillance Report, it was found that the most expensive average neonatal hospital charges were for 2 congenital heart defects: hypoplastic left heart syndrome ($199 597) and common truncus arteriosus ($192 781). Two other cardiac defects, coarctation of the aorta and TGA, were associated with average hospital charges in excess of $150 000. For the 11 selected cardiovascular congenital defects (of 35 birth defects considered), there were 11 578 hospitalizations in 2003 and 1550 in-hospital deaths (13.4%). Estimated total hospital charges for these 11 conditions were $1.4 billion.[35]
- In 2004, hospital costs for congenital cardiovascular defect conditions totaled $2.6 billion. The highest aggregate costs were for stays related to cardiac and circulatory congenital anomalies, which accounted for ≈$1.4 billion, more than half of all hospital costs for birth defects.[34]

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Table 8-1. Congenital Cardiovascular Defects

Population Group	Estimated Prevalence, 2002: All Ages	Mortality, 2008: All Ages	Hospital Discharges, 2009: All Ages
Both sexes	650 000 to 1.3 million[11]	3415	52 000
Males	. . .	1839 (53.9%)*	25 000
Females	. . .	1576 (46.1%)*	27 000
NH white males	. . .	1427	. . .
NH white females	. . .	1236	. . .
NH black males	. . .	335	. . .
NH black females	. . .	270	. . .

Ellipses (. . .) indicate data not available; NH, non-Hispanic.

*These percentages represent the portion of total congenital cardiovascular mortality that is for males vs females.

Sources: Mortality: National Center for Health Statistics (NCHS). These data represent underlying cause of death only; data for white and black males and females include Hispanics. Hospital discharges: National Hospital Discharge Survey, NCHS; data include those inpatients discharged alive, dead, or status unknown.

Table 8-2. Annual Birth Prevalence of Congenital Cardiovascular Defects in the United States[1,4,6,8,36,37]

Type of Presentation	Rate per 1000 Live Births	Estimated N (Variable With Yearly Birth Rate)
Fetal loss	Unknown	Unknown
Invasive procedure during the first year	2.4	9200
Detected during first year*	8	36 000
Bicuspid aortic valve	13.7	54 800

*Includes stillbirths and pregnancy termination at <20 weeks' gestation; includes some defects that resolve spontaneously or do not require treatment.

Table 8-3. Estimated Prevalence of Congenital Cardiovascular Defects and Percent Distribution by Type, United States, 2002* (in Thousands)

	Prevalence, n			Percent of Total		
Type	Total	Children	Adults	Total	Children	Adults
Total	994	463	526	100	100	100
VSD†	199	93	106	20.1	20.1	20.1
ASD	187	78	109	18.8	16.8	20.6
Patent ductus arteriosus	144	58	86	14.2	12.4	16.3
Valvular pulmonic stenosis	134	58	76	13.5	12.6	14.4
Coarctation of aorta	76	31	44	7.6	6.8	8.4
Valvular aortic stenosis	54	25	28	5.4	5.5	5.2
TOF	61	32	28	6.1	7	5.4
Atrioventricular septal defect	31	18	13	3.1	3.9	2.5
TGA	26	17	9	2.6	3.6	1.8
Hypoplastic right heart syndrome	22	12	10	2.2	2.5	1.9
Double-outlet right ventricle	9	9	0	0.9	1.9	0.1
Single ventricle	8	6	2	0.8	1.4	0.3
Anomalous pulmonary venous connection	9	5	3	0.9	1.2	0.6
Truncus arteriosus	9	6	2	0.7	1.3	0.5
HPLHS	3	3	0	0.3	0.7	0
Other	22	12	10	2.1	2.6	1.9

VSD indicates ventricular septal defect; ASD, atrial septal defect; TOF, tetralogy of Fallot; TGA, transposition of the great arteries; and HPLHS, hypoplastic left heart syndrome.

*Excludes an estimated 3 million bicuspid aortic valve prevalence (2 million in adults and 1 million in children).

†Small VSD, 117 000 (65 000 adults and 52 000 children); large VSD, 82 000 (41 000 adults and 41 000 children).

Source: Reprinted from Hoffman et al,[11] with permission from Elsevier. Average of the low and high estimates, two thirds from low estimate.[11]

Table 8-4. Surgery for Congenital Heart Disease

	Sample	Population, Weighted
Surgery for congenital heart disease	14 888	25 831
Deaths	736	1253
Mortality rate, %	4.9	4.8
By sex (81 missing in sample)		
Males	8127	14 109
Deaths	420	714
Mortality rate, %	5.2	5.1
Females	6680	11 592
Deaths	315	539
Mortality rate, %	4.7	4.6
By type of surgery		
ASD secundum surgery	834	1448
Deaths	3	6
Mortality rate, %	0.4	0.4
Norwood procedure for HPLHS	161	286
Deaths	42	72
Mortality rate, %	26.1	25.2

ASD indicates atrial septal defect; HPLHS, hypoplastic left heart syndrome.

In 2003, 25 000 cardiovascular operations for congenital cardiovascular defects were performed on children <20 years of age. Inpatient mortality rate after all types of cardiac surgery was 4.8%. Nevertheless, mortality risk varies substantially for different defect types, from 0.4% for ASD repair to 25.2% for first-stage palliation for HPLHS. Fifty-five percent of operations were performed in males. In unadjusted analysis, mortality after cardiac surgery was somewhat higher for males than for females (5.1% vs 4.6%).

Source: Analysis of 2003 Kids' Inpatient Database[38] and personal communication with Kathy Jenkins, MD, Children's Hospital of Boston, MA, October 1, 2006.

9. Cardiomyopathy and Heart Failure

See Table 9-1 and Charts 9-1 through 9-3.

Cardiomyopathy

ICD-9 425; ICD-10 I42.

Mortality—24 703. Any-mention mortality—48 579. Hospital discharges—52 000.

- Since 1996, the NHLBI-sponsored Pediatric Cardiomyopathy Registry has collected data on all children with newly diagnosed cardiomyopathy in New England and the Central Southwest (Texas, Oklahoma, and Arkansas).[1]
  - The overall incidence of cardiomyopathy is 1.13 cases per 100 000 among children <18 years of age.
  - Among children <1 year of age, the incidence is 8.34, and among children 1 to 18 years of age, it is 0.70 per 100 000.
  - The annual incidence is lower in white than in black children, higher in boys than in girls, and higher in New England (1.44 per 100 000) than in the Central Southwest (0.98 per 100 000).
- Hypertrophic cardiomyopathy (HCM) is the most common inherited heart defect, occurring in 1 of 500 individuals. In the United States, ≈500 000 people have HCM, yet most are unaware of it.[2] See Chapter 10, Disorders of Heart Rhythm, for statistics regarding sudden death in HCM.
- In a recent report of the Pediatric Cardiomyopathy Registry, the overall annual incidence of HCM in children was 4.7 per 1 million children. There was a higher incidence in the New England than in the Central Southwest region, in boys than in girls, and in children diagnosed at <1 year of age than in older children.[3]
- Dilated cardiomyopathy is the most common form of cardiomyopathy. The Pediatric Cardiomyopathy Registry recently reported an annual incidence of dilated cardiomyopathy in children <18 years of age of 0.57 per 100 000 overall. The annual incidence was higher in boys than in girls (0.66 versus 0.47 cases per 100 000), in blacks than in whites (0.98 versus 0.46 cases per 100 000), and in infants (<1 year of age) than in children (4.40 versus 0.34 cases per 100 000). The majority of children (66%) had idiopathic disease. The most common known causes were myocarditis (46%) and neuromuscular disease (26%).[4]
- Tachycardia-induced cardiomyopathy develops slowly and appears reversible, but recurrent tachycardia causes rapid decline in left ventricular function and development of HF. Sudden death is possible.[5]

Abbreviations for Chapter 9

ABC	Aging, Body and Composition
ARIC	Atherosclerosis Risk in Communities Study
BP	blood pressure
CARDIA	Coronary Artery Risk Development in Young Adults Study
CHS	Cardiovascular Health Study
CVD	cardiovascular disease
DM	diabetes mellitus
EF	ejection fraction
FHS	Framingham Heart Study
HbA_{1c}	hemoglobin A_{1c}
HCM	Hypertrophic cardiomyopathy
HF	heart failure
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
MESA	Multi-Ethnic Study of Atherosclerosis
MI	Myocardial infarction
NCHS	National Center for Health Statistics
NH	Non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHLBI	National Heart, Lung, and Blood Institute
PAR	Population-attributable risk

Heart Failure

ICD-9 428; ICD-10 I50.

Prevalence

- On the basis of data from NHANES 2005–2008, an estimated 5 700 000 Americans ≥20 years of age have HF (NCHS, unpublished NHLBI tabulation; Table 9-1; Chart 9-1).
- Projections of crude prevalence show that in 2010, ≈6.6 million US adults ≥18 years of age (2.8%) had HF.[6]
- It is estimated that by 2030, an additional 3 million people will have HF, a 25.0% increase in prevalence from 2010.[6]

Incidence

- Data from the NHLBI-sponsored FHS[7] indicate the following:
  - HF incidence approaches 10 per 1000 population after 65 years of age.
  - Seventy-five percent of HF cases have antecedent hypertension.
  - At 40 years of age, the lifetime risk of developing HF for both men and women is 1 in 5. At 80 years of age, remaining lifetime risk for development of new HF remains at 20% for men and women, even in the face of a much shorter life expectancy.
  - At 40 years of age, the lifetime risk of HF occurring without antecedent MI is 1 in 9 for men and 1 in 6 for women.
  - The lifetime risk for people with BP >160/90 mm Hg is double that of those with BP <140/90 mm Hg.
- The annual rates per 1000 population of new HF events for white men are 15.2 for those 65 to 74 years of age, 31.7 for those 75 to 84 years of age, and 65.2 for those ≥85 years of age. For white women in the same age groups, the rates are 8.2, 19.8, and 45.6, respectively. For black men, the rates are 16.9, 25.5, and 50.6,* and for black women, the estimated rates are 14.2, 25.5, and 44.0,* respectively (CHS, NHLBI).[8]

*Unreliable estimate.

- In MESA, African Americans had the highest risk of developing HF, followed by Hispanic, white, and Chinese Americans (4.6, 3.5, 2.4, and 1.0 per 1000 person-years, respectively). This higher risk reflected differences in the prevalence of hypertension, DM, and socioeconomic status. African Americans had the highest proportion of incident HF not preceded by clinical MI (75%).[9]
- In Olmsted County, Minnesota, the incidence of HF did not decline between 1979 and 2000.[10]
- In the ARIC study of the NHLBI, the age-adjusted incidence rate per 1000 person-years was 3.4 for white women, less than for all other groups; that is, white men (6.0), black women (8.1), and black men (9.1). The 30-day, 1-year, and 5-year case fatality rates after hospitalization for HF were 10.4%, 22%, and 42.3%, respectively. Blacks had a greater 5-year case fatality rate than whites ($P<0.05$). HF incidence rates in black women were more similar to those of men than of white women. The greater HF incidence in blacks than in whites is explained largely by blacks' greater levels of atherosclerotic risk factors.[11]
- Data from Kaiser Permanente indicated an increase in the incidence of HF among the elderly, with the effect being greater in men.[12]
- Data from hospitals in Worcester, MA, indicate that during 2000, the incidence and attack rates for HF were 219 per 100 000 and 897 per 100 000, respectively. HF was more frequent in women and the elderly. The hospital fatality rate was 5.1%.[13]
- In the CARDIA study, HF before 50 years of age was more common among blacks than whites. Hypertension, obesity, and systolic dysfunction are important risk factors that may be targets for prevention.[14]

Mortality

- In 2008, HF any-mention mortality was 281 437 (124 598 males and 156 839 females). HF was the underlying cause in 56 830 of those deaths in 2008 (NCHS, NHLBI). Table 9-1 shows the numbers of these deaths that are coded for HF as the underlying cause.
- The 2008 overall any-mention death rate for HF was 84.6. Any-mention death rates were 98.9 for white males, 102.7 for black males, 75.9 for white females, and 78.8 for black females (NCHS, NHLBI).
- One in 9 deaths has HF mentioned on the death certificate (NCHS, NHLBI).
- The number of any-mention deaths from HF was approximately as high in 1995 (287 000) as it was in 2008 (283 000; NCHS, NHLBI).
- Survival after HF diagnosis has improved over time, as shown by data from the FHS[15] and the Olmsted County Study.[10] However, the death rate remains high: ≈50% of people diagnosed with HF will die within 5 years.[10,16]
- In the elderly, data from Kaiser Permanente indicate that survival after the onset of HF has also improved.[12]
- In the CHS, depression and amino-terminal pro-B-type natriuretic peptide were independent risk factors for CVD-related and all-cause mortality.[17]

Risk Factors

- In the NHLBI-sponsored FHS, hypertension is a common risk factor for HF, followed closely by antecedent MI.[18] B-type natriuretic peptide, urinary albumin-to creatinine ratio, and elevated serum γ-glutamyl transferase were also identified as risk factors for HF.[18,19]
- In the Framingham Offspring Study, among 2739 participants, increased circulating concentrations of resistin were associated with incident HF independent of prevalent coronary disease, obesity, insulin resistance, and inflammation.[20]
- Among 20 900 male physicians in the Physicians Health Study, the lifetime risk of HF was higher in men with hypertension; healthy lifestyle factors (normal weight, not smoking, regular exercise, moderate alcohol intake, consumption of breakfast cereals, and consumption of fruits and vegetables) were related to lower risk of HF.[21]
- Among 2934 participants in the Health Aging, Body and Composition (ABC) study, the incidence of HF was 13.6 per 1000 person-years. Men and black participants were more likely to develop HF. Coronary disease (population attributable risk 23.9% for white participants, 29.5% for black participants) and uncontrolled BP (population attributable risk 21.3% for white participants, 30.1% for black participants) had the highest population attributable risks in both races. There was a higher overall proportion of HF attributable to modifiable risk factors in black participants than white participants (67.8% versus 48.9%). Hospitalizations were higher among black participants.[22] Inflammatory markers (interleukin-6 and tumor necrosis factor-α) and serum albumin levels were also associated with HF risk.[23,24]
- In the CHS, baseline cardiac troponin and changes in cardiac troponin levels measured by a sensitive assay were significantly associated with incident HF.[25]
- In the ARIC study, albuminuria, hemoglobin A_{1c} (HbA_{1c}) among individuals without DM, cardiac troponin measured with a sensitive assay, and socioeconomic position over the life course were all identified as risk factors for HF.[15,26–28]

Left Ventricular Function

- Data from Olmsted County, Minnesota, indicate that:
  - Among asymptomatic individuals, the prevalence of left ventricular diastolic dysfunction was 21% for mild diastolic dysfunction and 7% for moderate or severe diastolic dysfunction. The prevalence of systolic dysfunction was 6%. The presence of any left ventricular dysfunction (systolic or diastolic) was associated with an increased risk of developing overt HF, and diastolic dysfunction was predictive of all-cause death.[29]
  - Among individuals with symptomatic HF, the prevalence of left ventricular diastolic dysfunction was 6% for mild diastolic dysfunction and 75% for moderate or severe diastolic dysfunction.[30] The proportion of people with HF and preserved ejection fraction (EF) increased over time. Survival improved over time among individuals with reduced EF but not among those with preserved EF.[31]

Hospital Discharges/Ambulatory Care Visits

- Hospital discharges for HF were essentially unchanged from 1999 to 2009, with first-listed discharges of 975 000 and 1 094 000, respectively (unpublished data from the NHDS 2009, NCHS, NHLBI).
- In 2009, there were 3 041 000 physician office visits with a primary diagnosis of HF. In 2009, there were 668 000 ED

visits and 293 000 outpatient department visits for HF (NCHS, NHAMCS, NHLBI tabulation).

- Among 1077 patients with HF in Olmsted County, Minnesota, hospitalizations were common after HF diagnosis, with 83% patients hospitalized at least once and 43% hospitalized at least 4 times. More than one half of all hospitalizations were related to noncardiovascular causes.[32]

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Table 9-1. Heart Failure

Population Group	Prevalence, 2008: Age ≥20 y	Incidence (New Cases): Age ≥45 y	Mortality 2008: All Ages*	Hospital Discharges, 2009: All Ages
Both sexes	5 700 000 (2.4%)	670 000	56 830	1 094 000
Males	3 100 000 (3.0%)	350 000	23 017 (40.5%)†	531 000
Females	2 600 000 (2.0%)	320 000	33 813 (59.5%)†	563 000
NH white males	2.7%	. . .	20 278	. . .
NH white females	1.8%	. . .	30 244	. . .
NH black males	4.5%	. . .	2391	. . .
NH black females	3.8%	. . .	3068	. . .
Mexican American males	2.3%	. . .	. . .	. . .
Mexican American females	1.3%	. . .	. . .	. . .

NH indicates non-Hispanic; ellipses (. . .), data not available.

Heart failure includes persons who answered "yes" to the question of ever having congestive heart failure.

*Mortality data are for whites and blacks and include Hispanics.

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

Sources: Prevalence: National Health and Nutrition Examination Survey 2005–2008 (National Center for Health Statistics) and National Heart, Lung, and Blood Institute. Percentages are age adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolated to the 2008 US population estimates. These data are based on self-reports. Incidence: Framingham Heart Study, 1980–2003 from National Heart, Lung, and Blood Institute Incidence and Prevalence Chart Book, 2006. Mortality: National Center for Health Statistics.

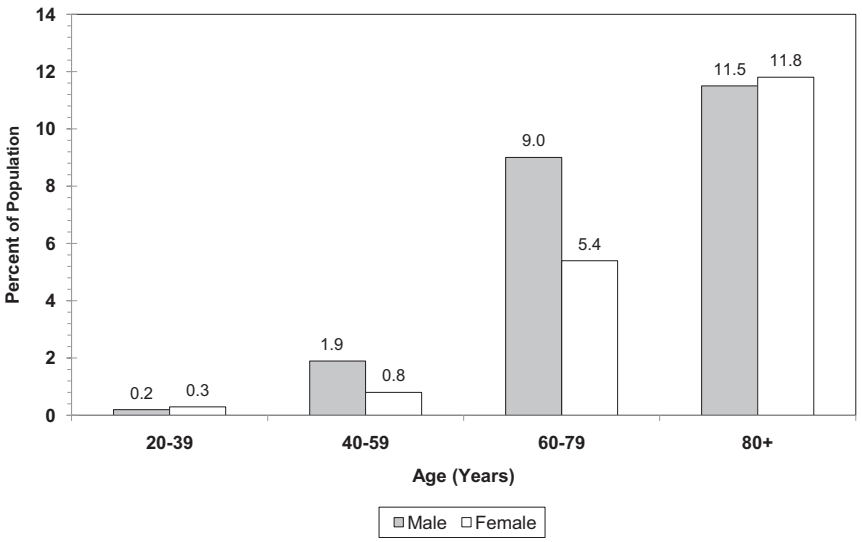

Chart 9-1. Prevalence of heart failure by sex and age (National Health and Nutrition Examination Survey: 2005–2008). Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

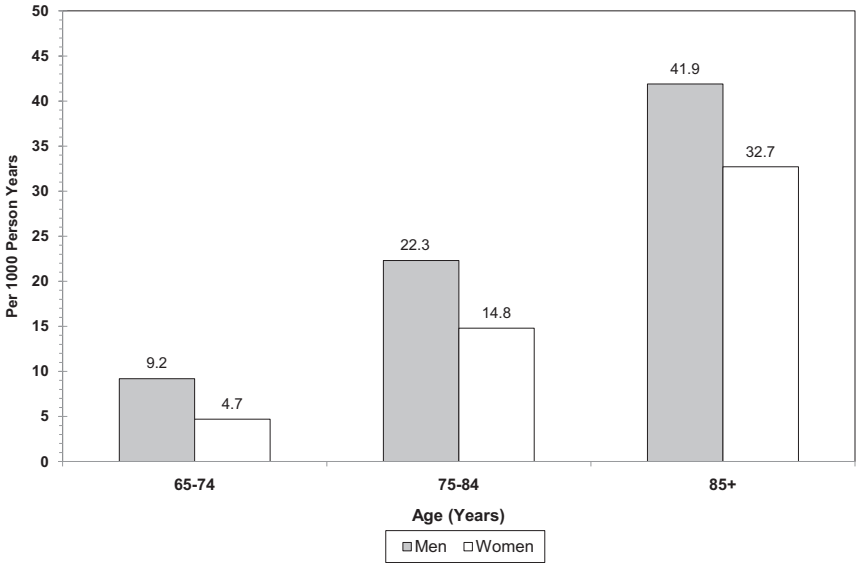

Chart 9-2. Incidence of heart failure (heart failure based on physician review of medical records and strict diagnostic criteria) by age and sex (Framingham Heart Study: 1980–2003). Source: National Heart, Lung, and Blood Institute.

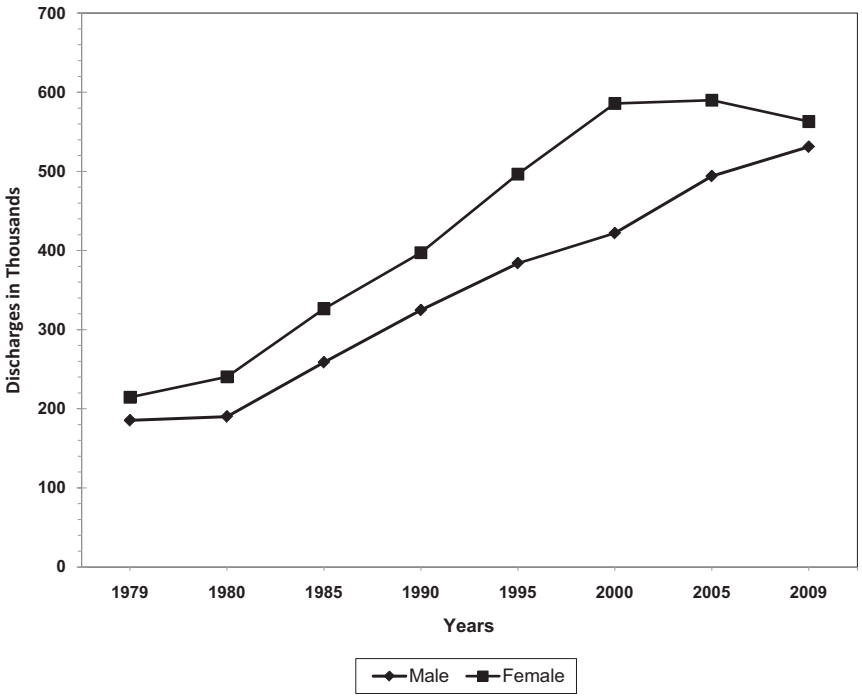

Chart 9-3. Hospital discharges for heart failure by sex (United States: 1979–2009). Note: Hospital discharges include people discharged alive, dead, and status unknown. Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.

10. Disorders of Heart Rhythm

See Table 10-1.

Bradyarrhythmias

ICD-9 426.0, 426.1, 427.81; ICD-10 I44.0 to I44.3, I49.5. Mortality—835. Any-mention mortality—4818. Hospital discharges—120 000.

AV Block

Prevalence and Incidence

- The prevalence of first-degree AV block in NHANES III is 3.7% (313 of 8434 participants with ECG data readable for PR interval).[1]

Abbreviations Used in Chapter 10

AHA	American Heart Association
AF	Atrial fibrillation
AMI	Acute myocardial infarction
ARIC	Atherosclerosis Risk in Communities study
AV	Atrioventricular
BMI	Body mass index
BP	Blood pressure
CAD	Coronary artery disease
CARDIA	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
CHD	Coronary heart disease
CHS	Cardiovascular Health Study
CI	Confidence interval
CVD	Cardiovascular disease
DM	Diabetes mellitus
ECG	Electrocardiogram
ED	Emergency department
EMS	Emergency medical services
FHS	Framingham Heart Study
GWTG	Get With the Guidelines
HCM	Hypertrophic cardiomyopathy
HD	Heart disease
HF	Heart failure
HR	Hazard ratio
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
MI	Myocardial infarction
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHLBI	National Heart, Lung, and Blood Institute
OR	Odds ratio
PVT	Polymorphic ventricular tachycardia
RR	Relative risk
SBP	Systolic blood pressure
SVT	Supraventricular tachycardia
TdP	Torsade de pointes
VF	Ventricular fibrillation
VT	Ventricular tachycardia

- In a healthy sample of subjects from the ARIC study (mean age 53 years), the prevalence of first-degree AV block was 7.8% in black men, 3.0% in black women, 2.1% in white men, and 1.3% in white women.[2] Smaller prevalence estimates were noticed in the relatively younger population (mean age 45 years) of the CARDIA study at its year-20 follow-up examination: 2.6% in black men, 1.9% in black women, 1.2% in white men, and 0.1% in white women.[3]
- Mobitz II second-degree AV block is rare in healthy individuals (≈0.003%), whereas Mobitz I (Wenckebach) is observed in 1% to 2% of healthy young people, especially during sleep.[4]
- The prevalence of third-degree AV block in the general adult population is ≈0.02% to 0.04%.[5,6]
- Third-degree AV block is very rare in apparently healthy individuals. Johnson et al[7] found only 1 case among >67 000 symptom-free individuals; Rose et al,[8] in their study of >18 000 civil servants, did not find any cases. On the other hand, among 293 124 patients with DM and 552 624 with hypertension enrolled with Veterans Health Administration hospitals, third-degree AV block was present in 1.1% and 0.6% of those patients, respectively.[9]
- Congenital complete AV block is estimated to occur in 1 of 15 000 to 25 000 live births.[4]

Risk Factors

- Although first-degree AV block and Mobitz type I second-degree AV block can occur in apparently healthy individuals, presence of Mobitz II second-degree or third-degree AV block usually indicates underlying HD, including CHD and HF.[4]
- Reversible causes of AV block include electrolyte abnormalities, drug-induced AV block, perioperative AV block attributable to hypothermia, or inflammation near the AV conduction system after surgery in this region. Some conditions may warrant pacemaker implantation because of the possibility of disease progression even if the AV block reverses transiently (eg, sarcoidosis, amyloidosis, and neuromuscular diseases).[10]
- Long sinus pauses and AV block can occur during sleep apnea. In the absence of symptoms, these abnormalities are reversible and do not require pacing.[11]

Prevention

- Detection and correction of reversible causes of acquired AV block could be of potential importance in preventing symptomatic bradycardia and other complications of AV block.[10]
- In utero detection of congenital AV block is possible by use of echocardiography.[12]

Aftermath

- In the FHS, PR interval prolongation (>200 ms) was associated with an increased risk of AF (HR 2.06, 95% CI 1.36–3.12),[13,14] pacemaker implantation (HR 2.89, 95% CI 1.83–4.57),[14] and all-cause mortality (HR 1.44, 95% CI 1.09–1.91).[14] Compared with individuals with a PR interval ≤200 ms, individuals with a PR interval >200 ms had an

absolute increased risk per year of 1.04% for AF, 0.55% for pacemaker implantation, and 2.05% for death.
- Patients with abnormalities of AV conduction may be asymptomatic or may experience serious symptoms related to bradycardia, ventricular arrhythmias, or both.
- Decisions about the need for a pacemaker are influenced by the presence or absence of symptoms directly attributable to bradycardia. Permanent pacing improves survival in patients with third-degree AV block, especially if syncope has occurred.[10] Nevertheless, the overall prognosis depends to a large extent on the underlying HD.
- Although there is little evidence to suggest that pacemakers improve survival in patients with isolated first-degree AV block,[15] it is now recognized that marked first-degree AV block (PR >300 ms) can lead to symptoms even in the absence of higher degrees of AV block.[16]

Sinus Node Dysfunction

Prevalence and Incidence

- The prevalence of sinus node dysfunction has been estimated to be between 403 to 666 per million, with an incidence rate of 63 per million per year requiring pacemaker therapy.[17]
- Sinus node dysfunction occurs in 1 of every 600 cardiac patients >65 years of age and accounts for ≈50% of implantations of pacemakers in the United States.[18,19]
- Sinus node dysfunction is commonly present with other causes of bradyarrhythmias (carotid sinus hypersensitivity in 33% of patients and advanced AV conduction abnormalities in 17%).[20,21]

Risk Factors

- The causes of sinus node dysfunction can be classified as intrinsic (secondary to pathological conditions involving the sinus node) or extrinsic (cause by depression of sinus node function by external factors such as drugs or autonomic influences).[22]
- Sinus node dysfunction may occur at any age but is primarily a disease of the elderly, with the average age being ≈68 years.[18]
- Idiopathic degenerative disease is probably the most common cause of sinus node dysfunction.[23]
- Collected data from 28 different studies on atrial pacing for sinus node dysfunction showed a median annual incidence of complete AV block of 0.6% (range 0%–4.5%) with a total prevalence of 2.1% (range 0%–11.9%). This suggests that the degenerative process also affects the specialized conduction system, although the rate of progression is slow and does not dominate the clinical course of disease.[24]
- Ischemic HD can be responsible for one third of cases of sinus node dysfunction. Transient sinus node dysfunction can complicate MI, which is common during inferior MI, and is caused by autonomic influences. Cardiomyopathy, long-standing hypertension, infiltrative disorders (eg, amyloidosis and sarcoidosis), collagen vascular disease, and surgical trauma can also result in sinus node dysfunction.[25,26]

Aftermath

- The course of sinus node dysfunction is typically progressive, with 57% of patients experiencing symptoms over a 4-year period if untreated, and a 23% prevalence of syncope over the same time frame.[27]
- Approximately 50% of patients with sinus node dysfunction develop tachy-brady syndrome over a lifetime; such patients have a higher risk of stroke and death. The survival of patients with sinus node dysfunction appears to depend primarily on the severity of underlying cardiac disease and is not significantly changed by pacemaker therapy.[28–30]
- In a retrospective study,[31] patients with sinus node dysfunction who had pacemaker therapy were followed up for 12 years; at 8 years, mortality among those with ventricular pacing was 59% compared with 29% among those with atrial pacing. This discrepancy may well be a result of selection bias. For instance, the physiological or anatomic disorder (eg, fibrosis of conductive tissue) that led to the requirement for the particular pacemaker may have influenced prognosis, rather than the type of pacemaker used.
- The incidence of sudden death is extremely low, and sinus node dysfunction does not appear to affect survival whether untreated or treated with pacemaker therapy.[10]
- Supraventricular tachycardia (SVT) including AF occurs in 47% to 53% of patients with sinus node dysfunction.[30,32]
- On the basis of records from the NHDS, age-adjusted pacemaker implantation rates increased progressively from 370 per million in 1990 to 612 per million in 2002. This escalating implantation rate is attributable to increasing implantation for isolated sinus node dysfunction; implantation for sinus node dysfunction increased by 102%, whereas implantation for all other indications did not increase.[33]

SVT (Excluding AF and Atrial Flutter)

ICD-9 427.0; ICD-10 I47.1.

Mortality—132. Any-mention mortality—1174. Hospital discharges—23 000.

Prevalence and Incidence

- Data from the Marshfield Epidemiological Study Area in Wisconsin suggested the incidence of documented paroxysmal SVT is 35 per 100 000 person-years. The mean age at SVT onset was 57 years, and both female sex and age >65 years were significant risk factors.[34]
- A review of ED visits from 1993 to 2003 revealed that 550 000 visits were for SVT (0.05% of all visits, 95% CI 0.04%–0.06%), or ≈50 000 visits per year. Of these patients, 24% (95% CI 15%–34%) were admitted, and 44% (95% CI 32%–56%) were discharged without specific follow-up.[35]
- The prevalence of SVT that is clinically undetected is likely much greater than the estimates from ED visits and electrophysiology procedures would suggest. For example, among a random sample of 604 participants in Finland, 7 (1.2%) fulfilled the diagnostic criteria for inappropriate sinus tachycardia.[36]

- Of 1383 participants in the Baltimore Longitudinal Study of Aging undergoing maximal exercise testing, 6% exhibited SVT during the test; increasing age was a significant risk factor. Only 16% exhibited >10 beats of SVT, only 4% were symptomatic, and the SVT participants were more likely to develop spontaneous SVT or AF.[37]
- From the surface ECG, the prevalence of atrial tachycardia is estimated to be 0.34% in asymptomatic patients and 0.46% in symptomatic patients.[38]

Aftermath

- The primary consequence of SVT for the majority of patients is a decline in quality of life.[39] However, rare cases of incessant SVT can lead to a tachycardia-induced cardiomyopathy,[40] and rare cases of sudden death attributed to SVT as a trigger have been described.[41]

Specific Types

- Among those presenting for invasive electrophysiological study and ablation, AV nodal reentrant tachycardia (a circuit requiring 2 AV nodal pathways) is the most common mechanism of SVT[42,43] and usually represents the majority of cases (56% of 1 series of 1754 cases from Loyola University Medical Center).[43]
- AV reentrant tachycardia (an arrhythmia requiring the presence of an extranodal connection between the atria and ventricles or specialized conduction tissue) is the second most common[42,43] (27% in the Loyola series),[43] and atrial tachycardia is the third most common (17% in the Loyola series).[43]
- In the pediatric population, AV reentrant tachycardia is the most common SVT mechanism, followed by AV nodal reentrant tachycardia and then atrial tachycardia.[44]
- AV reentrant tachycardia prevalence decreases with age, whereas AV nodal reentrant tachycardia and atrial tachycardia prevalences increase with age.[43]
- The majority of AV reentrant tachycardia patients in the Loyola series were men (55%), whereas the majority of patients with AV nodal reentrant tachycardia (70%) or atrial tachycardia (62%) were women.[43]

Wolff-Parkinson-White Syndrome

- Wolff-Parkinson White syndrome, a diagnosis reserved for those with both ventricular preexcitation (evidence of an anterograde conducting AV accessory pathway on a 12-lead ECG) and tachyarrhythmias,[39] deserves special attention because of the associated risk of sudden death. Sudden death is generally attributed to rapid heart rates in AF conducting down an accessory pathway and leading to ventricular fibrillation (VF).[45,46] Of note, AF is common in Wolff-Parkinson White patients, and surgical or catheter ablation of the accessory pathway often results in elimination of the AF.[47]
- Ventricular preexcitation was observed in 0.11% of 47 358 ECGs in adults participating in 4 large Belgian epidemiological studies[48] and in 0.17% of 32 837 Japanese high school students in ECGs obtained by law before the students entered school.[49]
- Asymptomatic adults with ventricular preexcitation appear to be at low risk of sudden death or potentially at no increased risk compared with the general population,[50–53] although certain characteristics found during invasive electrophysiological study (including inducibility of AV reentrant tachycardia or AF, accessory pathway refractory period, and the shortest R-R interval during AF) can help risk stratify these patients.[46,54]
- Symptomatic adult patients with the Wolff-Parkinson White syndrome are at a higher risk of sudden death. In a study of 60 symptomatic patients in Olmsted County, Minnesota, including some who underwent curative surgery, 2 (3.3%) experienced sudden death over a 13-year period. Of 690 Wolff-Parkinson White syndrome patients referred to a single hospital in The Netherlands, 15 (2.2%) had aborted sudden death, and VF was the first manifestation of the disease in 8 patients.[55]
- Although some studies in asymptomatic children with ventricular preexcitation suggest a benign prognosis,[52,56] others suggest that electrophysiological testing can identify a group of asymptomatic children with a risk of sudden death or VF as high as 11% over 19 months of follow-up.[57]

AF and Atrial Flutter

ICD-9 427.3; ICD-10 I48.

Prevalence

- Estimates of the prevalence of AF in the United States range from ≈2.7 to 6.1 million in 2010, and AF prevalence is expected to rise to between ≈5.6 and 12 million in 2050.[58,59]
- Data from a California health plan suggest that compared with whites, blacks (OR 0.49, 95% CI 0.47–0.52), Asians (OR 0.68, 95% CI 0.64–0.72), and Hispanics (OR 0.58, 95% CI 0.55–0.61) have significantly lower adjusted prevalences of AF.[60]
- Data from the NHDS/NCHS (1996–2001) on cases that included AF as a primary discharge diagnosis found the following:
  - Approximately 44.8% of patients were men.
  - The mean age for men was 66.8 years versus 74.6 years for women.
  - The racial breakdown for admissions was 71.2% white, 5.6% black, and 2.0% other races (20.8% were not specified).
  - Black patients were much younger than patients of other races.
- Among Medicare patients ≥65 years of age, AF prevalence increased from 3.2% in 1992 to 6.0% in 2002, with higher prevalence in older patients.[61]

Incidence

- Data from the NHDS/NCHS (1996–2001) on cases that included AF as a primary discharge diagnosis found the following:

— The incidence in men ranged from 20.6 per 100 000 people per year for patients between 15 and 44 years of age to 1077.4 per 100 000 people per year for patients ≥85 years of age.
— In women, the incidence ranged from 6.6 per 100 000 people per year for patients between 15 and 44 years of age to 1203.7 per 100 000 people per year for those ≥85 years of age.

- In Olmsted County, Minnesota:

— The age-adjusted incidence of clinically recognized AF in a white population increased by 12.6% between 1980 and 2000.[59,62]
— The incidence of AF was greater in men (incidence ratio for men over women 1.86) and increased markedly with older age.[59]

Mortality

- In 2008, AF was mentioned on 99 294 US death certificates and was the underlying cause in 15 383 of those deaths (NCHS, NHLBI). In adjusted analyses from the FHS, AF was associated with an increased risk of death in both men (OR 1.5, 95% CI 1.2–1.8) and women (OR 1.9, 95% CI 1.5–2.2).[63] Furthermore, there was an interaction with sex, such that AF appeared to diminish the survival advantage typically observed in women.
- In data from the Nurse's Health Study, the death rates per 1000 person-years among women without and with AF were 3.1 (95% CI 2.9–3.2) and 10.8 (95% CI 8.1–13.5).[64]
- In 1999, the CDC analyzed data from national and state multiple-cause mortality statistics and Medicare hospital claims for people with AF. The most common disease listed as the primary diagnosis for people hospitalized with AF was HF (11.8%), followed by AF (10.9%), CHD (9.9%), and stroke (4.9%).[65]
- A study of >4600 patients diagnosed with first AF showed that risk of death within the first 4 months after the AF diagnosis was high. The most common causes of CVD death were CAD, HF, and ischemic stroke, accounting for 22%, 14%, and 10%, respectively, of the early deaths (within the first 4 months) and 15%, 16%, and 7%, respectively, of the late deaths.[62]

Lifetime Risk and Cumulative Risk

- Participants in the NHLBI-sponsored FHS study were followed up from 1968 to 1999. At 40 years of age, remaining lifetime risks for AF were 26.0% for men and 23.0% for women. At 80 years of age, lifetime risks for AF were 22.7% for men and 21.6% for women. In further analysis, counting only those who had development of AF without prior or concurrent HF or MI, lifetime risk for AF was ≈16%.[66]
- By 80 years of age, investigators from the NHLBI-sponsored ARIC study observed that the cumulative risk of AF was 21% in white men, 17% in white women, and 11% in African Americans of both sexes.[67]

Risk Factors

- Standard risk factors

— Both ARIC[68] and FHS (http://www.framinghamheartstudy.org/risk/atrial.html)[13,69] have developed risk prediction models to predict new-onset AF. Predictors of increased risk of new-onset AF include advancing age, European ancestry, body size (higher height and BMI), electrocardiography features (left ventricular hypertrophy, left atrial enlargement), DM, BP (SBP and hypertension treatment), and presence of CVD (CHD, HF, valvular HD).
— Clinical and subclinical hyperthyroidism[70,71] and heavy alcohol consumption also have been identified as risk factors for AF.[72]

- Family history

— Although unusual, early-onset familial lone AF has long been recognized as a risk factor.[73,74]
— In the past decade, the heritability of AF in the community has been appreciated. In studies from the FHS:

○ Adjusted for coexistent risk factors, having at least 1 parent with AF was associated with a 1.85-fold increased risk of AF in the adult offspring (multivariable-adjusted 95% CI 1.12–3.06; $P=0.02$).[75]
○ A history of a first-degree relative with AF also was associated with an increased risk of AF (HR 1.40, 95% CI 1.13–1.74).[76] The risk was greater if the first-degree relative's age of onset was ≤65 years (HR 2.01, 95% CI 1.49–2.71) and with each additional affected first-degree relative (HR 1.24, 95% CI 1.05–1.46).[76]

- Genetics

— Mutations in genes coding channels (sodium and potassium), gap junction proteins, and signaling have been described, often in lone AF or familial AF series, but they are responsible for few cases of AF in the community.[77]
— Meta-analyses of genome-wide association studies have revealed single-nucleotide polymorphisms on chromosomes 4q25 (upstream of *PITX2*),[78–80] 16q22 (*ZFHX3*),[79,81] and 1q21 (*KCNN3*).[80] Although an area of intensive inquiry, the causative single-nucleotide polymorphisms and the functional basis of the associations have not been revealed.

Awareness

- In a US national biracial study of individuals with AF, compared with whites, blacks had approximately one third the likelihood (OR 0.32, 95% CI 0.20–0.52) of being aware that they had AF.[82]

Prevention

- Data from the ARIC study indicated that having at least 1 elevated risk factor explained 50% and having at least 1 borderline risk factor explained 6.5% of incident AF cases. The estimated overall incidence rate per 1000 person-years

at a mean age of 54.2 years was 2.19 for those with optimal risk, 3.68 for those with borderline risk, and 6.59 for those with elevated risk factors.[83]
- Hypertension accounted for ≈14%[84] to 22%[83] of AF cases.
- Observational data from the CHS suggested that moderate-intensity exercise (such as regular walking) was associated with a lower risk of AF (HR 0.72).[85] However, data from many studies suggested that vigorous-intensity exercise 5 to 7 days a week was associated with a slightly increased risk of AF (HR 1.20, $P=0.04$).[86]
- Secondary end-point analyses from randomized controlled studies have suggested that the treatment of hypertension[87] might prevent the onset of AF.
- Although heterogeneous in their findings, modest-sized short-term studies suggested that the use of statins might prevent AF; however, larger longer-term studies do not provide support that statins are effective in AF prevention.[88]
- The NHLBI sponsored a workshop highlighting important research areas to advance the prevention of AF.[89]

Aftermath

- Hospitalization
  - Hospital discharges—467 000
    - From 1996 to 2001, hospitalizations with AF as the first-listed diagnosis increased by 34%.[90]
    - On the basis of Medicare and MarketScan databases, annually, individuals with AF (37.5%) are approximately twice as likely to be hospitalized as age- and sex-matched control subjects (17.5%).[91]
- Stroke
  - Stroke rates per 1000 patient-years declined in AF patients taking anticoagulants, from 46.7 in 1992 to 19.5 in 2002, for ischemic stroke but remained fairly steady for hemorrhagic stroke (1.6–2.9).[61]
  - When standard stroke risk factors were accounted for, AF was associated with a 4- to 5-fold increased risk of ischemic stroke.[92]
  - Although the RR of stroke associated with AF did not vary (≈3–5-fold increased risk) substantively with advancing age, the proportion of strokes attributable to AF increased significantly. In FHS, AF accounted for ≈1.5% of strokes in individuals 50 to 59 years of age, and ≈23.5% in those 80 to 89 years of age.[92]
  - Paroxysmal, persistent, and permanent AF all appeared to increase the risk of ischemic stroke to a similar degree.[93]
  - AF was also an independent risk factor for ischemic stroke severity, recurrence and mortality.[94] In one study, people who had AF and were not treated with anticoagulants had a 2.1-fold increase in risk for recurrent stroke and a 2.4-fold increase in risk for recurrent severe stroke.[95]
- Cognition
  - Individuals with AF have an adjusted 2-fold increased risk of dementia.[96]
  - In individuals with AF in Olmsted County, Minnesota, the cumulative rate of dementia at 1 and 5 years was 2.7% and 10.5%, respectively.[97]
- Heart failure
  - AF and HF share many antecedent risk factors, and ≈40% of individuals with either AF or HF will develop the other condition.[98]
  - In the community, estimates of the incidence of HF in individuals with AF ranged from ≈3.3[98] to 4.4[99] per 100 person-years of follow-up.

Cost

Investigators examined Medicare and MarketScan databases (2004–2006) to estimate costs attributed to AF in 2008 US dollars:

- Annual total direct costs for AF patients were ≈$20 670 versus ≈$11 965 in the control group, for an incremental per-patient cost of $8705.[91]
- Extrapolating to the US population, it is estimated that the incremental cost of AF was ≈$26 billion, of which $6 billion was attributed to AF, $9.9 billion to other cardiovascular expenses, and $10.1 billion to noncardiovascular expenses.[91]

Tachycardia

ICD-9 427.0, 1, 2; ICD-10 I47.0, I47.1, I47.2, I47.9.

Mortality—621. Any-mention mortality—5863. Hospital discharges—86 000.

Monomorphic VT

Prevalence and Incidence

- Of 150 consecutive patients with wide-complex tachycardia subsequently studied by invasive electrophysiological study, 122 (80%) had ventricular tachycardia (VT; the remainder had SVT).[100]
- Of patients with ventricular arrhythmias presenting for invasive electrophysiological studies, 11% to 21% had no structural HD, and the majority of those with structural HD had CAD.[101,102]
- In 634 patients with implantable cardioverter-defibrillators who had structural HD (including both primary and secondary prevention patients) followed up for a mean 11±3 months, ≈80% of potentially clinically relevant ventricular tachyarrhythmias were attributable to VT amenable to antitachycardia pacing (implying a stable circuit and therefore monomorphic VT).[103] Because therapy may have been delivered before spontaneous resolution occurred, the proportion of these VT episodes with definite clinical relevance is not known.
- Of those with VT in the absence of structural HD, right ventricular outflow tract VT is the most common form.[104]

Aftermath

- Although the prognosis of those with VT or frequent premature ventricular contractions in the absence of structural HD is good,[101,104] a potentially reversible cardiomy-

opathy may develop in patients with very frequent premature ventricular contractions,[105,106] and some cases of sudden death attributable to short-coupled premature ventricular contractions have been described.[107,108]

Polymorphic VT

Prevalence and Incidence

- The true prevalence and incidence of polymorphic VT (PVT) in the US general population is not known.
- During ambulatory cardiac monitoring, PVT prevalence ranged from 0.01% to 0.15%[109,110]; however, among patients who developed sudden cardiac death during ambulatory cardiac monitoring, PVT was detected in 30% to 43%.[110–112]
- A prevalence range of 15% to 19% was reported during electrophysiological study in patients resuscitated from cardiac arrest.[112–114]
- In the setting of AMI, the prevalence of PVT ranged from 1.2% to 2%.[115,116]
- Out-of-hospital PVT is estimated to be present in ≈25% to 26% of all cardiac arrest cases involving VT.[117,118]

Risk Factors

- PVT in the setting of a normal QT interval is most frequently seen in the context of acute ischemia or MI.[116,119]
- Less frequently, PVT with a normal QT interval can occur in patients without apparent structural HD. Catecholaminergic PVT, which is discussed under inherited arrhythmic syndromes, is one such disorder.
- A prolonged QT, whether acquired (drug induced) or congenital, is a common cause of PVT. Drug-induced prolongation of QT causing PVT is discussed under torsade de pointes (TdP), whereas congenital prolonged QT is discussed under inherited arrhythmic syndromes.

Aftermath

- The presentation of PVT can range from a brief, asymptomatic, self-terminating episode to recurrent syncope or sudden cardiac death.[120]
- The overall hospital discharge rate (survival) of PVT has been estimated to be ≈28%.[121]
- In the out-of-hospital setting, the existing literature suggests that PVT has a variable response to the standard antiarrhythmic medications used in such situations.[122]

Prevention

- Prompt detection and correction of myocardial ischemia would potentially minimize the risk of PVT with normal QT in the setting of AMI.

Torsade de Pointes

Prevalence and Incidence

- The true incidence and prevalence of drug-induced TdP in the US general population is largely unknown.
- By extrapolating data from non-US registries,[123] it has been estimated that 12 000 cases of drug-induced TdP occur annually in the United States.[124]
- The prevalence of drug-induced prolongation of QT and TdP is 2 to 3 times higher in women than in men.[125]
- With the majority of QT-prolonging drugs, drug-induced TdP may occur in 3% to 15% of patients.[126]
- Antiarrhythmic drugs with QT-interval–prolonging potential carry a 1% to 3% risk of TdP over 1 to 2 years of exposure.[127]

Risk Factors

- TdP is usually related to administration of QT-prolonging drugs.[128] An up-to-date list of drugs with the potential to cause TdP may be found at http://www.azcert.org/medical-pros/drug-lists/drug-lists.cfm, a Web site maintained by the University of Arizona Center for Education and Research on Therapeutics.
- Specific risk factors for drug-induced TdP include prolonged QT, female sex, advanced age, bradycardia, hypokalemia, hypomagnesemia, left ventricular systolic dysfunction, and conditions that lead to elevated plasma concentrations of causative drugs, such as kidney disease, liver disease, drug interactions, or some combination of these.[124,129,130]
- Predisposition was also noted in patients who had a history of ventricular arrhythmia and who experienced a recent symptomatic increase in the frequency and complexity of ectopy.[131]
- Drug-induced TdP rarely occurs in patients without concomitant risk factors. An analysis of 144 published articles describing TdP associated with noncardiac drugs revealed that 100% of the patients had at least 1 risk factor, and 71% had at least 2 risk factors.[132]

Aftermath

- Drug-induced TdP may result in morbidity that requires hospitalization and in mortality attributable to sudden cardiac death in up to 31% of patients.[124,126]
- Patients with advanced HF with a history of drug-induced TdP had a significantly higher risk of sudden cardiac death during therapy with amiodarone than amiodarone-treated patients with no history of drug-induced TdP (55% versus 15%).[133] Current use of antipsychotic drugs was associated with a significant increase in the risk of sudden cardiac death attributable to TdP (OR 3.3, 95% CI 1.8–6.2).[134]
- Hospitalization was required in 47% and death occurred in 8% of patients with QT prolongation and TdP caused by administration of methadone.[135]

Prevention

- Keys to reducing the incidence of drug-induced cardiac arrhythmias include increased awareness among the medical, pharmaceutical, and nursing professions of the potential problems associated with the use of certain agents.
- Appropriate monitoring when a QT-prolonging drug is administered is essential. Also, prompt withdrawal of the offending agent should be initiated.[136]

VF and Ventricular Flutter

ICD-9 427.4; ICD-10 I49.0.

Mortality—1056. Any-mention mortality—9325.

Out-of-Hospital Cardiac Arrest: Adults

Out-of-hospital cardiac arrest is defined as a sudden and unexpected pulseless condition attributable to cessation of cardiac mechanical activity.[137] There are wide variations in the reported incidence of and outcomes for out-of-hospital cardiac arrest. These differences are due in part to differences in definition and ascertainment of cardiac arrest data, as well as differences in treatment after the onset of cardiac arrest.

Incidence

- The incidence of nontraumatic EMS-treated cardiac arrest and bystander-witnessed VF among individuals of any age during 2010 in the United States is best characterized by an ongoing registry from the Resuscitation Outcomes Consortium. See Table 10-1.
- The total resident population of the United States in 2010 was 308 745 538 individuals (www.census.gov). Extrapolation of the mortality rate reported by the Resuscitation Outcomes Consortium (Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2011) to the total population of the United States suggests that each year, 382 800 (quasi CI 375 400–390 300) people experience EMS-assessed out-of-hospital cardiac arrests in the United States.
- Approximately 60% of out-of-hospital cardiac arrests are treated by EMS personnel.[138]
- Only 33% of those with EMS-treated out-of-hospital cardiac arrest have symptoms within 1 hour of death.[139]
- Among EMS-treated out-of-hospital cardiac arrests, 23% have an initial rhythm of VF or VT or are shockable by an automated external defibrillator.[140]
- The incidence of cardiac arrest with an initial rhythm of VF is decreasing over time; however, the incidence of cardiac arrest with any initial rhythm is not decreasing.[141]

Risk Factors

- A study conducted in New York City found the age-adjusted incidence of out-of-hospital cardiac arrest per 10 000 adults was 10.1 among blacks, 6.5 among Hispanics, and 5.8 among whites.[142]
- Prior HD is a major risk factor for cardiac arrest. A study of 1275 health maintenance organization enrollees 50 to 79 years of age who had cardiac arrest showed that the incidence of out-of-hospital cardiac arrest was 6.0 per 1000 person-years in subjects with any clinically recognized HD compared with 0.8 per 1000 person-years in subjects without HD. In subgroups with HD, incidence was 13.6 per 1000 person-years in subjects with prior MI and 21.9 per 1000 person-years in subjects with HF.[143]
- A family history of cardiac arrest in a first-degree relative is associated with an ≈2-fold increase in risk of cardiac arrest.[144,145]

Aftermath

- Survival to hospital discharge, in 2010, of EMS-treated nontraumatic cardiac arrest was 11.4% (95% CI 10.5–12.2%; Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2011) and that of bystander-witnessed VF was 32.0% (95% CI 28.5–35.5%).
- A study conducted in New York City found the age-adjusted survival to 30 days after discharge was more than twice as poor for blacks as for whites, and survival among Hispanics was also lower than among whites.[142]
- Seventy-nine percent of the lay public are confident that they know what actions to take in a medical emergency; 98% recognize an automated external defibrillator as something that administers an electric shock to restore a normal heart beat among victims of sudden cardiac arrest; and 60% are familiar with cardiopulmonary resuscitation (Harris Interactive survey conducted on behalf of the AHA among 1132 US residents ≥18 years of age, January 8, 2008, through January 21, 2008).

Out-of-Hospital Cardiac Arrest: Children

- The incidence of nontraumatic EMS-treated cardiac arrest and bystander-witnessed VF among individuals <18 years of age in the United States are best characterized by an ongoing registry (Table 10-1). Survival to hospital discharge among children with EMS-treated, non-traumatic cardiac arrest: 8.6% (95% CI, 4.9% to 12.2%) (Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2011) and of bystander-witnessed VF: 62.5% (95% CI, 29.0% to 96.0%).
- Most sudden deaths in athletes were attributable to CVD (56%). Of the cardiovascular deaths that occurred, 29% occurred in blacks, 54% in high school students, and 82% with physical exertion during competition/training, and only 11% occurred in females, although this proportion has increased over time.[148]
- A longitudinal study of students 17 to 24 years of age participating in National Collegiate Athletic Association sports showed that the incidence of nontraumatic out-of-hospital cardiac arrest was 1 per 22 903 athlete participant-years. The incidence of cardiac arrest tended to be higher among blacks than whites and among men than women.[149]

In-Hospital Cardiac Arrest

- Extrapolation of the incidence of in-hospital cardiac arrest reported by GWTG-Resuscitation to the total population of hospitalized patients in the United States suggests that each year, 209 000 (quasi CIs 192 000–211 000) people are treated for in-hospital cardiac arrest.[150]
- 35.0% of children and 23.1% of adults who experience in-hospital cardiac arrest survive to discharge (GWTG-Resuscitation unpublished data).
- 17.1% of adults (14.3% of children) had VF or pulseless VT as the first recorded rhythm. Of these, 43.3% (41.4% of children) survived to discharge (GWTG-Resuscitation unpublished data).

For additional details on out-of-hospital and in-hospital arrest treatment and outcomes, please refer to Chapter 21, Quality of Care.

Monogenic Inherited Syndromes Associated With Sudden Cardiac Death

Long-QT Syndrome

- The hereditary long-QT syndrome is a genetic channelopathy characterized by prolongation of the QT interval (typically >460 ms) and susceptibility to ventricular tachyarrhythmias that lead to syncope and sudden cardiac death. Investigators have identified mutations in 13 genes leading to this phenotype (LQT1 through LQT13). LQT1 (*KCNQ1*), LQT2 (*KCNH2*), and LQT3 (*SCN5A*) mutations account for the majority (≈80%) of the typed mutations.[151,152]
- Prevalence of long-QT syndrome is estimated at 1 per 2000 live births from ECG-guided molecular screening of ≈44 000 mostly white infants born in Italy.[153] A similar prevalence was found among nearly 8000 Japanese school children screened by use of an ECG-guided molecular screening approach.[154]
- Long-QT syndrome has been reported among those of African descent, but its prevalence is not well assessed.[155]
- There is variable penetrance and a sex-time interaction for long-QT syndrome symptoms. Risk of cardiac events is higher among boys than girls (21% among boys and 14% among girls by age 12 years). Risk of events during adolescence is equivalent between sexes (≈25% for both sexes from ages 12–18 years). Conversely, risk of cardiac events in young adulthood is higher among women than men (39% among women from ages 18–40 years and 16% among men).[152]
- In addition to age and sex, the clinical course is influenced by prior syncope or aborted cardiac arrest, family history, QT-interval duration, genotype, number of mutations, and congenital deafness.[151,152,156]
- Risk of cardiac events is decreased during pregnancy but increased during the 9-month postpartum period.[157]
- The mainstay of therapy and prevention is β-blockade treatment.[152,156] Implantable defibrillators are considered for high-risk individuals.[158]

Short-QT Syndrome

- Short-QT syndrome is a recently described inherited mendelian condition characterized by shortening of the QT interval (typically QT <320 ms) and predisposition to AF and ventricular tachyarrhythmias and sudden death. Mutations in 5 ion channel genes have been described (SQT1–SQT5).[159]
- In a population of 41 767 young, predominantly male Swiss transcripts, 0.02% of the population had a QT interval shorter than 320 ms.[160]
- Among 53 patients from the European Short QT Syndrome Registry (75% males, median age 26 years), a familial or personal history of cardiac arrest was present in 89%. Twenty-four patients received an implantable cardioverter-defibrillator, and 12 received long-term prophylaxis with hydroquinidine. During a median follow-up of 64 months, 2 patients received an appropriate implantable cardioverter-defibrillator shock, and 1 patient experienced syncope. Nonsustained polymorphic VT was recorded in 3 patients.[161]

The Brugada Syndrome

- The Brugada syndrome is an inherited channelopathy characterized by persistent ST-segment elevation in the precordial leads (V_1–V_3), right bundle-branch block, and susceptibility to ventricular arrhythmias and sudden cardiac death.[162]
- Mutations in several ion channel–related genes have been identified that lead to Brugada syndrome.[162]
- Prevalence is estimated at 1 to 5 per 10 000 individuals. Prevalence is higher in South East Asian countries, including Thailand and Philippines. There is a strong male predominance (80% male).[162–167]
- Cardiac event rates for Brugada syndrome patients followed up prospectively in northern Europe (31.9 months) and Japan (48.7 months) were similar: 8% to 10% in patients with prior aborted sudden death, 1% to 2% in those with history of syncope, and 0.5% in asymptomatic patients.[168,169] Predictors of poor outcome included family history of sudden death and early repolarization patter on ECG.[168,169]

Catecholaminergic PVT

- Catecholaminergic PVT is a familial condition characterized by adrenergically induced ventricular arrhythmias associated with syncope and sudden death. It is associated with frequent ectopy, bidirectional VT, and polymorphic VT with exercise or catecholaminergic stimulation (such as emotion, or medicines such as isoproterenol).
- Mutations in genes encoding the ryanodine type 2 receptor (*RYR2*)[170,171] are found in the majority, and mutations in genes encoding calsequestrin 2 (*CASQ2*)[172,173] are found in a small minority.[174] However, a substantial proportion of individuals with catecholaminergic PVT do not have an identified mutation.
- Statistics regarding catecholaminergic PVT are primarily from case series. Of 101 patients with catecholaminergic PVT, the majority had experienced symptoms before 21 years of age.[174]
- In small series (n=27 to n=101) of patients followed up over a mean of 6.8 to 7.9 years, 27% to 62% experienced cardiac symptoms, and fatal or near-fatal events occurred in 13% to 31%.[174–176]
- Risk factors for cardiac events included younger age of diagnosis and absence of β-blocker therapy. A history of aborted cardiac arrest and absence of β-blocker therapy were risk factors for fatal or near-fatal events.[174]

Arrhythmogenic Right Ventricular Cardiomyopathy

- Arrhythmogenic right ventricular cardiomyopathy is a form of genetically inherited structural HD that presents with fibrofatty replacement of the myocardium, with clin-

ical presentation of palpitations, syncope, and sudden death.[177]

- Twelve arrhythmogenic right ventricular cardiomyopathy loci have been described (ARVC1–ARVC12). Disease-causing genes for 8 of these loci have been identified, the majority of which are in desmosomally related proteins.[177]
- Prevalence is estimated at 2 to 10 per 10 000 individuals.[177,178] Of 100 patients reported on from the Johns Hopkins Arrhythmogenic Right Ventricular Dysplasia Registry, 51 were men, and 95 were white, with the rest being of black, Hispanic, or Middle Eastern origin. Twenty-two percent of index cases had evidence of the familial form of arrhythmogenic right ventricular cardiomyopathy.[179]
- The most common presenting symptoms were palpitations (27%), syncope (26%), and sudden cardiac death (23%).[179]
- During a median follow-up of 6 years, 47 patients received an implantable cardioverter-defibrillator, 29 of whom received appropriate implantable cardioverter-defibrillator shocks. At the end of follow-up, 66 patients were alive. Twenty-three patients died at study entry, and 11 died during follow-up (91% of deaths were attributable to sudden cardiac arrest).[179] Similarly, the annual mortality rate was 2.3% for 130 patients with arrhythmogenic right ventricular cardiomyopathy from Paris, France, who were followed up for a mean of 8.1 years.[180]

Hypertrophic Cardiomyopathy

(Please refer to Chapter 9, Cardiomyopathy and Heart Failure, for statistics regarding the general epidemiology of HCM.)

- Over a mean follow-up of 8±7 years, 6% of HCM patients experienced sudden cardiac death.[181]
- Among 1866 sudden deaths in athletes between 1980 and 2006, HCM was the most common cause of cardiovascular sudden death (in 251 cases, or 36% of the 690 deaths that could be reliably attributed to a cardiovascular cause).[148]
- The risk of sudden death increases with increasing maximum left ventricular wall thickness,[182,183] and the risk for those with wall thickness ≥30 mm is 18.2 per 1000 patient-years (95% CI 7.3–37.6),[182] or approximately twice that of those with maximal wall thickness <30 mm.[182,183] Of note, an association between maximum wall thickness and sudden death has not been found in every HCM population.[184]
- Nonsustained VT is a risk factor for sudden death,[185,186] particularly in younger patients. Nonsustained VT in those ≤30 years of age is associated with a 4.35-greater odds of sudden death (95% CI 1.5–12.3).[185]
- A history of syncope is also a risk factor for sudden death in these patients,[187] particularly if the syncope was recent before the initial evaluation and not attributable to a neurally mediated event.[188]
- The presence of left ventricular outflow tract obstruction ≥30 mm Hg appears to increase the risk of sudden death by ≈2-fold.[189,190] The presence of left ventricular outflow tract obstruction has a low positive predictive value (7%–8%) but a high negative predictive value (92%–95%) for predicting sudden death.[189,191]
- The rate of malignant ventricular arrhythmias detected by implantable cardioverter-defibrillators appears to be similar between those with a family history of sudden death in ≥1 first-degree relatives and those with at least 1 of the risk factors described above.[192]
- The risk of sudden death increases with the number of risk factors.[193,194]

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Table 10-1. Incidence and Outcome of Out-of-Hospital Cardiac Arrest in United States

	Incidence per 100 000 Resident Population, Mean (95% CI)		
	Overall	Adults	Children
EMS-assessed	124.0 (121.6, 126.4)	140.9 (138.0, 143.8)	13.8 (12.2, 15.4)
EMS treated, non-traumatic cardiac arrest	67.2 (65.5, 68.9)	85.2 (82.9, 87.5)	10.5 (9.1, 11.9)
Bystander-witnessed VF	8.0 (7.4, 8.6)	10.5 (9.7, 11.3)	0.4 (0.1, 0.6)

CI indicates confidence interval; EMS, emergency medical services; VF, ventricular fibrillation.
Source: Resuscitation Outcomes Consortium Investigators, unpublished data, June 20, 2011.

11. Other Cardiovascular Diseases

See Table 11-1.

Mortality and any-mention mortality in this section are for 2008. "Mortality" is the number of deaths in 2008 for the given underlying cause. Prevalence data are for 2006. Hospital discharge data are from the NHDS/NCHS; data include inpatients discharged alive, dead, or status unknown. Hospital discharge data for 2009 are based on *ICD-9* codes.

Valvular Heart Disease

ICD-9 424; ICD-10 I34 to I38.

Mortality—21 824. Any-mention mortality—45 062. Hospital discharges—92 000.

Abbreviations Used in Chapter 11

AAA	abdominal aortic aneurysm
ABI	ankle-brachial index
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
CARDIA	Coronary Artery Risk Development in Young Adults
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CKD	chronic kidney disease
CI	confidence interval
CT	computed tomography
CVD	cardiovascular disease
DM	diabetes mellitus
DVT	deep vein thrombosis
ECG	electrocardiogram/electrocardiographic
FHS	Framingham Heart Study
FRS	Framingham Risk Score
HD	heart disease
HLA	human leukocyte antigen
ICD	*International Classification of Diseases*
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
IE	infective endocarditis
MESA	Multi-Ethnic Study of Atherosclerosis
MI	myocardial infarction
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHLBI	National Heart, Lung, and Blood Institute
OR	odds ratio
PA	physical activity
PAD	peripheral arterial disease
PE	pulmonary embolism
RR	relative risk
VTE	venous thromboembolism
VHD	valvular heart disease

- Three important factors have contributed to the changing epidemiology of valvular heart disease (VHD) in the United States and other industrialized countries over the past 3 decades: the aging population, the increase in degenerative VHD, and the increased ability to ascertain VHD by cardiac ultrasound before it becomes clinically manifest.
- Epidemiological challenges, including uniform definitions of VHD-linked wide variations in disease severity and latency between disease onset on clinical presentation, make estimation of the growing burden of VHD difficult.[1]
- A large population-based epidemiological study performed with cardiac ultrasound in a representative US population of 11 911 patients showed an overall age-adjusted prevalence of VHD of 2.5% (95% CI 2.2–2.7%).[2,3]
- Echocardiographic data from the CARDIA study (4351), the ARIC study (2435), and the CHS (5125) were pooled to assess the age-dependent prevalence of VHD. The prevalence increased from 0.7% (95% CI 0.5–1.0) in participants 18 to 44 years of age to 13.3% (95% CI 11.7–15.0) in participants ≥75 years of age ($P<0.0001$).[2]
- The adjusted mortality risk ratio associated with valve disease was 1.36 (95% CI 1.15–1.62; $P=0.0005$).[1]
- Doppler echocardiography data in 1696 men and 1893 women (54±10 years of age) attending a routine examination of the FHS were used to assess the prevalence of valvular regurgitation. Mitral regurgitation and tricuspid regurgitation of more than or equal to mild severity were seen in 19.0% and 14.8% of men and 19.1% and 18.4% of women, respectively. Aortic regurgitation of more than or equal to trace severity was present in 13.0% of men and 8.5% of women.[3]

Aortic Valve Disorders

ICD-9 424.1; ICD-10 I35.

Mortality—14 337. Any-mention mortality—29 246. Hospital discharges—60 000.

- The prevalence of moderate aortic stenosis in patients aged 70 to 80 years is estimated to be 2%.[2,4,5]
- Calcific aortic stenosis on a trileaflet valve or bicuspid aortic valve is the most common cause of aortic stenosis.[6]
- In the MESA study of 5880 participants aged 45 to 84 years, aortic valve calcium was quantified with serial CT images. During a mean follow-up of 2.4 years, 210 subjects (4.1%) of the 5142 with no aortic valve calcium had a mean incidence rate of progression of 1.7% per year, which increased significantly with age. The incident aortic valve calcium risk was associated with several traditional cardiovascular risk factors, specifically age, male sex, BMI, and smoking.[7]
- In the Euro Heart Survey, which included 4910 patients in >25 countries, aortic stenosis was the most frequent lesion, accounting for 43% of all patients who had VHD.[8]
- Among men and women ≥65 years of age enrolled in the CHS who underwent echocardiography, the aortic valve was normal in 70% of cases, sclerotic without outflow obstruction in 29%, and stenotic in 2%. Aortic sclerosis was associated with an increase of ≈50% in the risk of death of cardiovas-

cular causes and the risk of MI.[9] Clinical factors associated with aortic sclerosis and stenosis were similar to risk factors for atherosclerosis.[4] These data largely exclude patients with congenital HD, a group that is expected to increasingly contribute to the prevalence of valve disease.

- Degenerative disease of the aortic valve and root is the most common cause of aortic regurgitation in industrialized countries.[5]
- The congenital bicuspid aortic valve is more often associated with aortic stenosis than regurgitation but was found to be the most common cause of aortic regurgitation in the Euro Heart Survey.[10]

Mitral Valve Disorders

ICD-9 424.0; ICD-10 I34.

Mortality—2372. Any-mention mortality—5477. Hospital discharges—27 000.

Prevalence

- In pooled data from the CARDIA, ARIC, and CHS studies, mitral valve disease was the most common valvular lesion. At least moderate mitral regurgitation occurred at a frequency of 1.7% as adjusted to the US adult population of 2000, increasing from 0.5% to 9.3% in those between 18 and ≥75 years of age.[1]
- Isolated mitral stenosis is more common in women and occurs in 40% of all patients presenting with rheumatic HD.[11]
- The NHLBI-sponsored FHS reports that among people 26 to 84 years of age, prevalence of mitral valve disorders is ≈1% to 2% and equal between women and men.[12]
- The prevalence of mitral valve prolapse in the general population was evaluated with the use of echocardiograms of 1845 women and 1646 men who participated in the fifth examination of the Offspring Cohort of the FHS. The prevalence of mitral valve prolapse was 2.4%. The frequencies of chest pain, dyspnea, and ECG abnormalities were similar among subjects with and those without prolapse.[12]

Pulmonary Valve Disorders

ICD-9 424.3; ICD-10 I37.

Mortality—12. Any-mention mortality—38.

Tricuspid Valve Disorders

ICD-9 424.2; ICD-10 I36.

Mortality—14. Any-mention mortality—70.

Rheumatic Fever/Rheumatic HD

ICD-9 390 to 398; ICD-10 I00 to I09.

Mortality—3141. Any-mention mortality—5881. Hospital discharges—38 000.

- Rheumatic HD is most common in developing countries, where estimates vary from 2% to 3% with use of cardiac ultrasound screening.[5]
- The incidence of acute rheumatic fever has decreased in the United States.[13]
- Although localized outbreaks have occurred, the overall incidence of acute rheumatic fever remains very low in most areas of the United States.[14,15]
- The incidence of rheumatic fever remains high in blacks, Puerto Ricans, Mexican Americans, and American Indians.[16]
- In 1950, ≈15 000 Americans (adjusted for changes in ICD codes) died of rheumatic fever/rheumatic HD compared with ≈3100 today (NCHS/NHLBI).
- From 1996 to 2006, the death rate attributable to rheumatic fever/rheumatic HD fell 8.3%, and actual deaths declined 26.2% (NCHS/NHLBI).
- The 2007 overall death rate for rheumatic fever/rheumatic HD was 1.0. Death rates were 0.8 for white males, 0.7 for black males, 1.2 for white females, and 0.9 for black females.[17]
- Immune risk factors have been linked with rheumatic HD. Human leukocyte antigen (HLA) typing was performed in 120 black patients with severe chronic rheumatic HD requiring cardiac surgery; HLA-DR 1 antigen was present in 12.6% of patients compared with 2.7% of normal control subjects, and the HLA-DRw6 antigen was present in 31.1% of patients compared with 15% of control subjects, which suggests that genetically determined immune response factors may play a role in the pathogenesis of severe chronic rheumatic HD.[18]

Bacterial Endocarditis

ICD-9 421.0; ICD-10 I33.0.

Mortality—1143. Any-mention mortality—2420. Hospital discharges—28 000, primary plus secondary diagnoses.

- The 2007 AHA guidelines on prevention of infective endocarditis (IE)[15] state that IE is thought to result from the following sequence of events: (1) Formation of nonbacterial thrombotic endocarditis on the surface of a cardiac valve or elsewhere that endothelial damage occurs; (2) bacteremia; and (3) adherence of the bacteria in the bloodstream to nonbacterial thrombotic endocarditis and proliferation of bacteria within a vegetation. Viridans group streptococci are part of the normal skin, oral, respiratory, and gastrointestinal tract flora, and they cause ≥50% of cases of community-acquired native valve IE not associated with intravenous drug use.[19]
- The best estimates of the incidence of IE in the general population come from a prospective study of 16 million people in France conducted in 1999. The annual age- and sex-standardized incidence was 31 cases per million.[20]
- In studies comparing the exposure to bacteremia from various sources, the cumulative exposure during 1 year from routine daily activities such as tooth brushing and food chewing may be as much as 5.6 million times greater that that occurring as a result of a single tooth extraction, the procedure associated with the highest risk of bacteremia.[21]
- Although the absolute risk for IE from a dental procedure is impossible to measure precisely, the best available estimates are as follows: If dental treatment causes 1% of

all cases of viridans group streptococcal IE annually in the United States, the overall risk in the general population is estimated to be as low as 1 case of IE per 14 million dental procedures. The estimated absolute risk rates for IE from a dental procedure in patients with underlying cardiac conditions are as follows[21]:

— Mitral valve prolapse: 1 per 1.1 million procedures;
— CHD: 1 per 475 000;
— Rheumatic HD: 1 per 142 000;
— Presence of a prosthetic cardiac valve: 1 per 114 000; and
— Previous IE: 1 per 95 000 dental procedures

- Although these calculations of risk are estimates, it is likely that the number of cases of IE that result from a dental procedure is exceedingly small. Therefore, the number of cases that could be prevented by antibiotic prophylaxis, even if prophylaxis were 100% effective, is similarly small. One would not expect antibiotic prophylaxis to be near 100% effective, however, because of the nature of the organisms and choice of antibiotics.[21]
- Patients with congenital HD present a particular set of risk factors related to the presence of cyanosis, the use of prosthetic material for repair of HD, and the presence of residual defects at the site of previous repair.[10] In adults with congenital HD, the presence of multiple heart defects and previous endocarditis are significant predictors of endocarditis.[22]
- Although IE occurs less often in children than in adults, the incidence of IE in children is increasing with the increasing numbers of children with repaired congenital HD. IE accounts for 1 of every 1280 pediatric admissions per year.[23]

Endocarditis, Valve Unspecified

ICD-9 424.9; ICD-10 I38.

Mortality—5089. Any-mention mortality—10 443.

Kawasaki Disease

ICD-9 446.1; ICD-10 M30.3.

Mortality—6. Any-mention mortality—7.

- Kawasaki disease is more prevalent in the United States than in Japan, where outbreaks occurred in 1979, 1982, and 1986, and where the majority of cases occurred in those under the age of 2 years and predominantly in males.[24]
- An estimated 4248 hospitalizations for Kawasaki disease occurred in the United States in 2000, with a median patient age of 2 years. Race-specific incidence rates indicate that Kawasaki disease is most common among Americans of Asian and Pacific Island descent (32.5/100 000 children <5 years of age), occurs with intermediate frequency in non-Hispanic blacks (16.9/100 000 children <5 years of age) and Hispanics (11.1/100 000 children <5 years of age), and is least common in whites (9.1/100 000 children <5 years of age).[25] In the United States, Kawasaki disease is more common during the winter and early spring months; boys outnumber girls by ≈1.5:1 to 1.7:1; and 76% of children with Kawasaki disease are <5 years of age.[26]

Venous Thromboembolism Epidemiology (Including Deep Vein Thrombosis and Pulmonary Embolism)[27]

Pulmonary Embolism

ICD-9 415.1; ICD-10 I26.

Mortality—7158. Any-mention mortality—28 852. Hospital discharges—158 000.

Deep Vein Thrombosis

ICD-9 451.1; ICD-10 I80.2.

Mortality—2352. Any-mention mortality—12 296.

Incidence

- Venous thromboembolism (VTE) consists of deep vein thrombosis (DVT; typically involving deep veins of the leg or pelvis) and its complication, pulmonary embolism (PE).
- VTE average annual incidence among whites is 108 per 100 000 person-years, with ≈250 000 incident cases occurring annually among US whites.
- VTE incidence appears to be similar or higher among African-Americans and lower among Asian and Native Americans.
- After adjustment for the different age and sex distribution of African Americans, VTE incidence is ≈78 per 100 000, which suggests 27 000 incident VTE cases occur annually among African Americans.
- Modeling suggests that >900 000 incident or recurrent VTE events occur annually in the United States, of which approximately one third are fatal.
- VTE incidence has not changed significantly over the past 25 years.
- Incidence rates increase exponentially with age for both men and women and for both DVT and PE.
- Incidence rates are higher in women during childbearing years, whereas incidence rates after 45 years of age are higher in men.
- PE accounts for an increasing proportion of VTE with increasing age for both sexes.

Survival

- Observed survival after VTE is significantly worse than expected survival for age and sex, and survival after PE is much worse than after DVT alone.
- For almost one quarter of PE patients, the initial clinical presentation is sudden death.
- Thirty-day VTE survival is 74.8% (DVT alone, 96.2%; PE with or without DVT, 59.1%).[28]
- PE is an independent predictor of reduced survival for up to 3 months.
- Because most PE deaths are sudden and usually attributed to underlying disease (eg, cancer, other chronic heart, lung, or renal disease), secular trends in VTE survival are confounded by autopsy rates.

Recurrence

- VTE is a chronic disease with episodic recurrence; ≈30% develop recurrence within the next 10 years.

- The hazard of recurrence varies with the time since the incident event and is highest within the first 6 to 12 months.
- Independent predictors of recurrence include increasing patient age and BMI; neurological disease with leg paresis; active cancer; lupus anticoagulant or antiphospholipid antibody; antithrombin, protein C, or protein S deficiency; and persistently increased plasma fibrin D-dimer.
- Idiopathic incident VTE, incident PE, and male sex may predict a higher risk of recurrence, but reports are conflicting.[29–31]

Complications

- VTE complications include venous stasis syndrome (or postthrombotic syndrome) and venous ulcer, as well as chronic thromboembolic pulmonary hypertension.
- The 20-year incidence of cumulative venous stasis syndrome and venous ulcer after proximal DVT is ≈40% and 3.7%, respectively.
- Chronic thromboembolic pulmonary hypertension incidence is 6.5 per million person-years; ≈1400 incident chronic thromboembolic pulmonary hypertension cases occur annually among US whites.

Risk Factors

- Independent VTE risk factors include increasing patient age, surgery, trauma/fracture, hospital or nursing home confinement, active cancer, central vein catheterization or transvenous pacemaker, prior superficial vein thrombosis, varicose veins and neurological disease with leg paresis, and among women, oral contraceptives, pregnancy/postpartum, and hormone therapy.
- Together, these risk factors account for >75% of all incident VTE occurring in the community.
- Compared with residents in the community, hospitalized residents have more than a 130-fold higher VTE incidence (71 versus 9605 per 100 000 person-years).[32]
- Hospitalization and nursing home residence together account for almost 60% of incident VTE events that occur in the community.
- Among cancer patients beginning chemotherapy, tumor site, BMI, hemoglobin, platelet and white blood cell count, and plasma D-dimer and soluble P-selectin levels are predictors of VTE in the next 6 months.[33]
- Physical inactivity is a risk factor for PE among women.[34]
- Use of injectable depot-medroxyprogesterone acetate as contraception is a risk factor for DVT.[35]
- Pregnancy-associated VTE incidence is 200 per 100 000 woman-years; compared with nonpregnant women of childbearing age, the relative risk is increased ≈4-fold.
- VTE risk during the postpartum period is ≈5-fold higher than during pregnancy.
- VTE is highly heritable and follows a complex mode of inheritance that involves environmental interaction.
- Inherited thrombophilias (eg, inherited antithrombin, protein C, or protein S deficiency; factor V Leiden; prothrombin G20210A; ABO blood type non-O) interact with such clinical risk factors (ie, environmental "exposures") as oral contraceptives, pregnancy, hormone therapy, and surgery to compound VTE risk.
- Similarly, genetic interaction compounds the risk of incident and recurrent VTE.

Arteries, Diseases of

ICD-9 440 to 448; ICD-10 I70 to I79. Includes PAD.

Mortality—27 765. Any-mention mortality—89 924. Hospital discharges—331 000.

Aortic Aneurysm

ICD-9 441; ICD-10 I71.

Mortality—11 079. Any-mention mortality—17 816. Hospital discharges—84 000.

- Although the definition varies somewhat by age and body surface area, generally an abdominal aortic aneurysm (AAA) is considered to be present when the anteroposterior diameter of the aorta reaches 3.0 cm.[36]
- The prevalence of AAAs 2.9 to 4.9 cm in diameter ranges from 1.3% in men 45 to 54 years of age to 12.5% in men 75 to 84 years of age. For women, the prevalence ranges from 0% in the youngest to 5.2% in the oldest age group.[36]
- Factors associated with increased prevalence of AAA include older age, male sex, family history of AAA, tobacco use, hypertension, and manifest atherosclerotic disease in other vascular beds, including the coronary and peripheral arteries.[36,37] The association of dyslipidemia with AAA is mixed.[38]
- Patients with DM are approximately half as likely as patients without DM to have an AAA.[39,40]
- Male sex, older age, and smoking are important risk factors for incident AAA in the next 7 years.[41]
- Large AAAs tend to expand more rapidly than small AAAs, and large AAAs are at substantially higher risk for rupture.[36]
  - Average annual expansion rates are ≈1 to 4 mm for aneurysms <4.0 cm in diameter, 4 to 5 mm for AAAs 4.0 to 6.0 cm in diameter, and as much as 7 to 8 mm for AAAs >6.0 cm in diameter.
  - Absolute risk for eventual rupture is ≈20% for AAAs >5.0 cm, ≈40% for AAAs >6.0 cm, and >50% for AAAs >7.0 cm in diameter.
  - Rupture of an AAA may be associated with death rates as high as 90%.

Peripheral Arterial Disease

ICD-9: 440.20 to 440.24, 440.30 to 440.32, 440.4, 440.9, 443.9, 445.02; ICD-10: I70.2, I70.9, I73.9, I74.3, I74.4.

Mortality—14 501. Any-mention mortality—68 849. Hospital discharges—166 000.

- PAD affects ≈8 million Americans and is associated with significant morbidity and mortality.[42] Prevalence increases dramatically with age, and PAD disproportionately affects blacks.[42]

- PAD affects 12% to 20% of Americans ≥65 years of age.[43] Despite its prevalence and cardiovascular risk implications, only ≈70% to 80% of patients with PAD undergo recommended antiplatelet therapy or lipid-lowering therapy.[44]
- In the general population, only ≈10% of people with PAD have the classic symptom of intermittent claudication. Approximately 40% do not complain of leg pain, whereas the remaining 50% have a variety of leg symptoms different from classic claudication.[45,46] In an older, disabled population of women, however, as many as two thirds of individuals with PAD had no exertional leg symptoms.[47]
- The risk factors for PAD are similar but not identical to those for CHD. DM and cigarette smoking are stronger risk factors for PAD than for CHD.[36] ORs for associations of DM and smoking with symptomatic PAD are ≈3.0 to 4.0. Most studies suggest that the prevalence of PAD is similar in men and women.[48]
- Pooled data from 11 studies in 6 countries found that PAD is a marker for systemic atherosclerotic disease. The age- and sex-adjusted RR of all-cause death was 2.35; for CVD mortality, it was 3.34; and for CHD fatal and nonfatal events combined, it was 2.13. The findings for stroke were slightly weaker but still significant, with a pooled RR of 1.86 for fatal and nonfatal events combined.[49]
- A recent meta-analysis of 24 955 men and 23 339 women demonstrated that the association of the ABI with mortality has a reverse J-shaped distribution in which participants with an ABI of 1.11 to 1.40 are at lowest risk for mortality.[50] Furthermore, an ABI <0.90 added meaningfully to the FRS in predicting 10-year risk of total mortality, cardiovascular mortality, and major coronary events. An ABI <0.90 approximately doubled the risk of total mortality, cardiovascular mortality, and major coronary events in each FRS category.[50]
- Among 508 patients (449 men) identified from 2 vascular laboratories in San Diego, CA, a decline in ABI of >0.15 within a 10-year period was associated with a subsequent increased risk of all-cause mortality (RR 2.4) and CVD mortality (RR of 2.8) at 3 years' follow-up.[51]
- Among 440 patients with PAD, male sex and smoking were more associated with aortoiliac (proximal) disease than with infrailiac (distal) disease. In addition, aortoiliac disease was associated with an increased risk of mortality or cardiovascular events compared with infrailiac disease (adjusted HR 3.28, 95% CI 1.87–5.75).[52]
- Men and women with PAD have higher levels of inflammatory biomarkers than individuals without PAD. Elevated levels of C-reactive protein were associated with an increased risk of developing PAD among men in the Physicians' Health Study.[53] The OR for developing PAD 5 years after C-reactive protein measurement was 2.1 for those in the highest versus lowest baseline quartile of C-reactive protein. Among participants in the Women's Health Study, 12 years after soluble intercellular adhesion molecule-1 measurement, women in the highest baseline tertile for levels of soluble intercellular adhesion molecule-1 had a 2-fold increased risk of developing PAD compared with women in the lowest baseline tertile.[54] Among individuals with PAD, higher levels of inflammatory biomarkers are associated with increased all-cause and cardiovascular mortality rates and increased risk of failure of lower-extremity revascularization procedures.[55–57]
- Data from the NHANES 1999–2004 cohort demonstrated an inverse association between bilirubin levels and prevalence of PAD. A 0.1-mg/dL higher level of bilirubin was associated with a 6% reduction in the odds of PAD (OR 0.94, 95% CI 0.90–0.98) after adjustment for PAD risk factors.[58]
- People with PAD have impaired function and quality of life. This is true even for people who do not report leg symptoms. Furthermore, patients with PAD, including those who are asymptomatic, experience a significant decline in lower-extremity functioning over time.[59–61]
- Data from NHANES 1999–2000 (NCHS) show that high blood levels of lead and cadmium are associated with an increased prevalence of PAD. Exposure to these 2 metals can occur through cigarette smoke. The risk was 2.8 for high levels of cadmium and 2.9 for high levels of lead. The OR of PAD for current smokers was 4.13 compared with people who had never smoked.[62]
- Results from NHANES 1999–2000 (NCHS) and the CHS showed a remarkably high prevalence of PAD among patients with renal insufficiency.[63,64] In addition, chronic kidney disease (CKD) is common among community-dwelling older men and women with a high ABI.[64]
- Available evidence suggests that the prevalence of PAD in people of Hispanic origin is similar to or slightly higher than that in non-Hispanic whites.[42,65]
- Among patients with established PAD, higher PA levels during daily life are associated with better overall survival rate, a lower risk of death because of CVD, and slower rates of functional decline.[66,67] In addition, better 6-minute walk performance and faster walking speed are associated with lower rates of all-cause mortality, cardiovascular mortality, and mobility loss.[68,69]
- A cross-sectional, population-based telephone survey of >2500 adults ≥50 years of age, with oversampling of blacks and Hispanics, found that 26% expressed familiarity with PAD. Of these, half were not aware that DM and smoking increase the risk of PAD. One in 4 knew that PAD is associated with increased risk of heart attack and stroke, and only 14% were aware that PAD could lead to amputation. All knowledge domains were lower in individuals with lower income and education levels.[70]

Other Diseases of Arteries

ICD-9 440 to 448, excluding AAA and PAD; ICD-10 I70 to I79, excluding AAA and PAD.

Mortality—8850. Any-mention mortality—30 290. Hospital discharges—81 000.

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Table 11-1. Rheumatic Fever/Rheumatic Heart Disease

Population Group	Mortality, 2008: All Ages*	Hospital Discharges, 2009: All Ages
Both sexes	3141	38 000
Males	1025 (32.6%)†	16 000
Females	2116 (67.4%)†	22 000
NH white males	882	. . .
NH white females	1873	. . .
NH black males	97	. . .
NH black females	166	. . .

NH indicates non-Hispanic; ellipses (. . .), data not available.

*Mortality data are for whites and blacks and include Hispanics.

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

Sources: Mortality: National Center for Health Statistics; data represent underlying cause of death only. Hospital discharges: National Hospital Discharge Survey, National Center for Health Statistics, and National Heart, Lung, and Blood Institute; data include those inpatients discharged alive, dead, or of unknown status.

12. Risk Factor: Family History and Genetics

Biologically related first-degree relatives (siblings, offspring and parents) share roughly 50% of their genetic variation with one another. This constitutes much greater sharing of genetic variation than with a randomly selected person from the population, and thus, when a trait aggregates within a family, this lends evidence for a genetic risk factor for the trait. Similarly, racial/ethnic minorities are more likely to share their genetic variation within their demographic than with other demographics. Familial aggregation of CVD may be related to aggregation of specific behaviors (eg, smoking, alcohol use) or risk factors (eg, hypertension, DM, obesity) that may themselves have environmental and genetic contributors. Unlike classic mendelian genetic risk factors, whereby usually 1 mutation directly causes 1 disease, a complex trait's genetic contributors may increase risk without necessarily always causing the condition. The effect size of any specific contributor to risk may be small but widespread throughout a population, or may be large but affect only a small population, or may have an enhanced risk when an environmental contributor is present. Although the breadth of all genetic research into CVD is beyond the scope of this chapter, we present a summary of evidence that a genetic risk for CVD is likely, as well as a summary of evidence on the most consistently replicated genetic markers for HD identified to date.

Prevalence

Family History of HD

- Among adults ≥20 years of age, 13.3% reported a parent or sibling with a heart attack or angina before the age of 50 years. The racial/ethnic breakdown is as follows (NHANES 2007–2008; tabulation by Donald Lloyd-Jones, MD, Northwestern University, Chicago, IL):
  - — For non-Hispanic whites, 14.9% for men, 16.7% for women.
  - — For non-Hispanic blacks, 10.0% for men, 12.4% for women.
  - — For Mexican Americans, 8.8% for men, 12.3% for women.
  - — For other races, 11.4% for men, 13.6% for women.
- HD occurs as people age, and those without a family history of HD may survive longer, so the prevalence of family history will vary depending on the age at which it is assessed. The breakdown of reported family history of heart attack by age in the US population as measured by NHANES is as follows (NHANES 2007–2008; tabulation by Donald Lloyd-Jones, MD, Northwestern University, Chicago, IL):
  - — Age 20 to 39 years, 10.3% for men, 11.6% for women.
  - — Age 40 to 59 years, 14.1% for men, 18.4% for women.
  - — Age 60 to 79 years, 12.4% for men, 15.5% for women.
  - — Age ≥80 years, 11.2% for men, 9.2% for women.
- In the multigenerational FHS, only 75% of participants with a documented parental history of a heart attack before age 55 years reported that history when asked.[1]

Abbreviations Used in Chapter 12

ABI	ankle-brachial index
BMI	body mass index
CAC	coronary artery calcification
CARDIoGRAM	Coronary ARtery DIsease Genome-wide Replication And Meta-analysis
CI	confidence interval
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
FHS	Framingham Heart Study
HbA_{1c}	glycosylated hemoglobin
HD	heart disease
HDL	high-density lipoprotein
LDL	low-density lipoprotein
MI	myocardial infarction
NHANES	National Health and Nutrition Examination Survey
OR	odds ratio
SBP	systolic blood pressure
SNP	single-nucleotide polymorphism

Impact of Family History

- Premature paternal history of a heart attack has been shown to approximately double the risk of a heart attack in men and increase the risk in women by ≈70%.[2,3]
- History of a heart attack in both parents increases the risk of heart attack, especially when 1 parent has had a premature heart attack[4] (Table 12-2).
- Sibling history of HD has been shown to increase the odds of HD in men and women by ≈50%.[5]

Genetics

- The increased risk of HD seen in people with a family history of a heart attack is likely caused in part by shared genetics. The full genetic basis for CVD has not yet been determined, and genetic markers discovered thus far have not been shown to add to cardiovascular risk prediction tools beyond current models that incorporate family history.[6]
- Heritability is the ratio of genetically caused variation to the total variation of a trait or measure. Table 12-2 presents heritability estimates for standard CVD risk factors using data generated from the FHS. These data suggest that most CVD risk factors have at least moderate heritability.
- Genome-wide association is a robust technique to identify associations between genotypes and phenotypes. Table 12-3 presents results from the CARDIoGRAM (Coronary ARtery DIsease Genome-wide Replication And Meta-analysis) consortium, which represents the largest genetic study of MI to date, with 22 233 MI case subjects and 64 762 control subjects and with independent validation in an

additional 56 682 individuals.[7] Altogether, there are 23 well-replicated loci for MI. The ORs are modest, ranging from 1.06 to 1.51 per copy of the risk allele (individuals may harbor up to 2 copies of a risk allele). However, these are common alleles, which suggests that the attributable risk may be substantial.

- The most consistently replicated genetic marker for HD in European-derived populations is located at 9p21.3. At this single-nucleotide polymorphism, ≈27% of the white population is estimated to have 0 risk alleles, 50% is estimated to have 1 risk allele, and the remaining 23% is estimated to have 2 risk alleles.[8]
- The 10-year HD risk for a 65-year-old man with 2 risk alleles at 9p21.3 and no other traditional risk factors is ≈13.2%, whereas a similar man with 0 alleles would have a 10-year risk of ≈9.2%. The 10-year HD risk for a 40-year-old woman with 2 alleles and no other traditional risk factors is ≈2.4%, whereas a similar woman with 0 alleles would have a 10-year risk of ≈1.7%.[8]
- Variation at the same 9p21.3 region also increases the risk of stroke,[9] as well as the risk of aortic aneurysms,[10–12] intracranial aneurysms,[11] heart failure,[13] and sudden death.[14] Associations have also been observed between the 9p21.3 region and CAC.[15,16] Additionally, stronger associations have been found between variation at 9p21.3 and earlier[17,18] and more severe[19] heart attacks. The biological mechanism underpinning the association of genetic variation in the 9p21 region with disease outcomes is still under investigation.

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Table 12-1. OR for Combinations of Parental Heart Attack History

Parental Heart Attack History	OR (95% CI)
No family history	1.00
One parent with heart attack at ≥50 y of age	1.67 (1.55–1.81)
One parent with heart attack at <50 y of age	2.36 (1.89–2.95)
Both parents with heart attack at ≥50 y of age	2.90 (2.30–3.66)
Both parents with heart attack, 1 at <50 y of age	3.26 (1.72–6.18)
Both parents with heart attack, both at <50 y of age	6.56 (1.39–30.95)

OR indicates odds ratio; CI, confidence interval.
Data derived from Chow et al.[4]

Table 12-2. Heritability of CVD Risk Factors From the FHS

Trait	Heritability
ABI	0.21[20]
SBP	0.42[21]
DBP	0.39[21]
Left ventricular mass	0.24 to 0.32[22]
BMI	0.37 (mean age 40 y) to 0.52 (mean age 60 y)[23]
Waist circumference	0.41[24]
Visceral abdominal fat	0.36[25]
Subcutaneous abdominal fat	0.57[25]
Fasting glucose	0.34[26]
HbA_{1c}	0.27[26]
Triglycerides	0.48[27]
HDL cholesterol	0.52[27]
Total cholesterol	0.57[27]
LDL cholesterol	0.59[27]
Estimated glomerular filtration rate	0.33[28]

CVD indicates cardiovascular disease; FHS, Framingham Heart Study; ABI, ankle-brachial index; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; HbA_{1c}, glycosylated hemoglobin; HDL, high-density lipoprotein; and LDL, low-density lipoprotein.

Table 12-3. Validated SNPs for MI, the Nearest Gene, and the OR From the CARDIoGRAM Consortium

SNP	Chromosomal Region	Gene	Effect Size (OR)	Minor Allele Frequency
rs599839	1p13.3	*SORT1*	1.11	0.22
rs17465637	1q41	*MIA3*	1.14	0.26
rs17114036	1p32.2	*PPAP2B*	1.17	0.09
rs11206510	1p32.3	*PCSK9*	1.08	0.18
rs6725887	2q33	*WDR12*	1.14	0.15
rs2306374	3q22.3	*MRAS*	1.12	0.18
rs17609940	6p21.31	*ANKS1A*	1.07	0.25
rs12526453	6p24.1	*PHACTR1*	1.10	0.33
rs12190287	6q23.2	*TCF21*	1.08	0.38
rs798220	6q25	*LPA*	1.51	0.02
rs11556924	7q32.2	*ZC3HC1*	1.09	0.38
rs4977574	9p21.3	*CDKN2A, CDKN2B*	1.29	0.46
rs579459	9q34.2	*ABO*	1.10	0.21
rs1746048	10q11	*CXCL12*	1.09	0.13
rs12413409	10q24.32	*CYP17A1-CNNM2-NT5C2*	1.12	0.11
rs964184	11q23.3	*ZNF259-APOA5-A4-C3-A1*	1.13	0.13
rs3184504	12q24	*Sh2b3*	1.07	0.44
rs4773144	13q34	*COL4A1-COL4A2*	1.07	0.44
rs2895811	14q32.2	*HHIPL1*	1.08	0.43
rs3825807	15q25.1	*ADAMTS7*	1.07	0.43
rs216172	17p13.3	*SMG6-SRR*	1.07	0.37
rs12936587	17p11.2	*RASD1-SMCR3-PEMT*	1.07	0.44
rs46522	17q21.32	*UBE2Z-GIP-ATP5G1-SNF8*	1.06	0.47
rs1122608	19q13.2	*LDLR*	1.14	0.23
rs9982601	21q22.11	*MRPS6*	1.18	0.15

SNPs indicates single-nucleotide polymorphisms; MI, myocardial infarction; OR, odds ratio; and CARDIoGRAM Consortium, Coronary ARtery DIsease Genome-wide Replication And Meta-analysis Consortium.

Data derived from Schunkert et al.[7]

13. Risk Factor: Smoking/Tobacco Use

See Table 13-1 and Charts 13-1 and 13-2.

Prevalence

Youth

- In 2009, in grades 9 through 12, 19.5% of students reported current cigarette use (on at least 1 day during the 30 days before the survey), 14.0% of students reported current cigar use, and 8.9% of students reported current smokeless tobacco use. Overall, 26.0% of students reported any current tobacco use (YRBSS).[1]
- In 2009, in grades 9 to 12, male and female students were equally likely to report current cigarette use (19.8% compared with 19.1%); however, male students were more likely than female students to report current cigar use (18.6% compared with 8.8%) and current smokeless tobacco use (15.0% compared with 2.2%; YRBSS).[1]
- In 2009, in grades 9 through 12, non-Hispanic white students were more likely than Hispanic or non-Hispanic black students to report any current tobacco use, which includes cigarettes, cigars, or smokeless tobacco (30.3% compared with 20.8% for Hispanic students and 16.2% for non-Hispanic black students; YRBSS).[1]
- Among youths 12 to 17 years of age in 2009, 2.9 million (11.6%) used a tobacco product (cigarettes, cigars, or smokeless tobacco) in the past month, and 2.2 million (8.9%) used cigarettes. Cigarette use in the past month in this age group declined from 13.0% in 2002 to 8.9% in 2009 (National Survey on Drug Use and Health [NSDUH]).[2,3]
- Data from the YRBSS[4,5] for students in grades 9 to 12 indicated the following:
  - — The percentage of students who reported ever trying cigarettes remained stable from 1991 to 1999 and then declined from 70.4% in 1999 to 46.3% in 2009.
  - — The percentage who reported current cigarette use (on at least 1 day in the 30 days before the survey) increased between 1991 and 1997 and then declined from 36.4% in 1997 to 19.5% in 2009.
  - — The percentage who reported current frequent cigarette use (smoked on ≥20 of the 30 days before the survey) increased from 1991 to 1999 and then declined from 16.8% in 1999 to 7.3% in 2009.
- In 2009, 50.8% of students in grades 9 to 12 who currently smoked cigarettes had tried to quit smoking cigarettes during the previous 12 months. The prevalence of this behavior was higher among female student smokers (54.2%) than among male student smokers (48.0%) and among white males (47.0%) and Hispanic males (52.2%) than among black males (36.5%; YRBSS).[1]

Abbreviations Used in Chapter 13

AMI	acute myocardial infarction
BRFSS	Behavioral Risk Factor Surveillance System
CHD	coronary heart disease
CVD	cardiovascular disease
MEPS	Medical Expenditure Panel Survey
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
NSDUH	National Survey on Drug Use and Health
YRBSS	Youth Risk Behavior Surveillance System

Adults

- From 1998 to 2010, the percentage of US adults ≥18 years of age who were current cigarette smokers declined from 24.1% to 19.3%. The percentage who were current smokers did not change significantly between 2005 and 2009, but there was a small but significant decline between 2009 and 2010 (NHIS).[6–8]
- In 2010, among Americans ≥18 years of age, 21.2% of men and 17.5% of women were current cigarette smokers (NHIS).
- From 1998 to 2007, cigarette smoking prevalence among adults ≥18 years of age decreased in 44 states and the District of Columbia. Six states had no substantial changes in prevalence after controlling for age, sex, and race/ethnicity (BRFSS).[9]
- In 2010, among adults ≥18 years of age, the states with the highest percentage of current cigarette smokers were West Virginia (26.8%), Kentucky (24.8%), and Oklahoma (23.7%). Utah, the state with the lowest percentage of smokers (9.1%), has met the Healthy People 2010 target for reducing adult smoking prevalence to 12%, and California has almost met the target, with a 2010 smoking rate of 12.1% (BRFSS).[10]
- In 2007 to 2009, among adults ≥18 years of age, Asian men (15.4%) and Hispanic men (17.9%) were less likely to be current cigarette smokers than non-Hispanic black men (23.8%), non-Hispanic white men (24.1%), and American Indian or Alaska Native men (26.8%) on the basis of age-adjusted estimates (NHIS). Similarly, in 2007 to 2009, Asian women (5.4%) and Hispanic women (9.3%) were less likely to be current cigarette smokers than non-Hispanic black women (17.2%), non-Hispanic white women (21.0%), and American Indian or Alaska Native women (19.9%).[11]
- In 2004 to 2006 data, adult cigarette smoking varied among Asian subgroups. Most Asian adults had never smoked, with rates ranging from 65% of Korean adults to 84% of Chinese adults. Korean adults (22%) were approximately 2 to 3 times as likely to be current smokers as Japanese (12%), Asian Indian (7%), or Chinese (7%) adults on the basis of age-adjusted estimates (NHIS).[12]
- In 2007 to 2009, among people ≥65 years of age, 9.3% of men and 8.6% of women were current smokers. In this age group, men were more likely than women to be former smokers (54.7% compared with 29.6%) on the basis of age-adjusted estimates (NHIS).[11]
- In 2008 to 2009, among women 15 to 44 years of age, past-month cigarette use was lower for those who were

pregnant (15.3%) than among those who were not pregnant (27.4%). This pattern was found for women 18 to 25 years of age (22.0% versus 32.0% for pregnant and nonpregnant women, respectively) and for women 26 to 44 years of age (10.8% versus 27.7%, respectively). Among adolescents 15 to 17 years of age, past-month cigarette use was higher for those who were pregnant (20.6%) than for those who were not pregnant (13.9%; NSDUH).[2]

- In 2009, an estimated 69.7 million Americans ≥12 years of age were current (past month) users of a tobacco product (cigarettes, cigars, smokeless tobacco, or tobacco in pipes). The rate of current use of any tobacco product in this age range declined from 2007 to 2009 (from 28.6% to 27.7%; NSDUH).[2]

Incidence

- In 2009, ≈2.5 million people ≥12 years of age smoked cigarettes for the first time within the past 12 months, which was similar to the estimate in 2008 (2.4 million). The 2009 estimate averages out to ≈6900 new cigarette smokers every day. Most new smokers (58.8%) in 2009 were <18 years of age when they first smoked cigarettes (NSDUH).[2]
- In 2009, among people ages 12 to 49 years who had started smoking within the past 12 months, the average age of first cigarette use was 17.5 years, similar to the average in 2008 (17.4 years).[2]
- Data from 2002 to 2004 suggest that ≈1 in 5 nonsmokers 12 to 17 years of age is likely to start smoking. Youths in the Mexican subpopulations were significantly more susceptible (28.8%) to start smoking than those in non-Hispanic white (20.8%), non-Hispanic black (23.0%), Cuban (16.4%), Asian Indian (15.4%), Chinese (15.3%), and Vietnamese (13.8%) subpopulations. There was no significant difference in susceptibility to start smoking between boys and girls in any of the major populations or subpopulations (NSDUH).[13]

Mortality

- During 2000 to 2004, cigarette smoking resulted in an estimated 443 000 premature deaths each year caused by smoking-related illnesses, and ≈49 000 of these deaths were attributable to secondhand smoke. In adults ≥35 years of age, a total of 32.7% of these deaths were related to CVD.[14]
- Each year from 2000 to 2004, smoking caused 3.1 million years of potential life lost for males and 2.0 million years for females, excluding deaths attributable to smoking-attributable residential fires and adult deaths attributable to secondhand smoke.[14]
- From 2000 to 2004, smoking during pregnancy resulted in an estimated 776 infant deaths annually.[14]
- During 2000 to 2004, cigarette smoking resulted in an estimated 269 655 deaths annually among males and 173 940 deaths annually among females.[14]
- On average, male smokers die 13.2 years earlier than male nonsmokers, and female smokers die 14.5 years earlier than female nonsmokers.[15]
- Current cigarette smoking is a powerful independent predictor of cardiac arrest in patients with CHD.[16]

Secondhand Smoke

- The national prevalence of households with smoke-free home rules increased from 43.2% during 1992 to 1993 to 72.2% in 2003 on the basis of data from the "Tobacco Use Supplement" to the Current Population Survey (a continuing monthly survey of the Bureau of Labor Statistics conducted by the US Census Bureau). During this period, the prevalence of such rules increased from 9.6% to 31.8% among households with at least 1 smoker and from 56.8% to 83.5% among households with no smokers. Approximately 126 million children and nonsmoking adults were still exposed to secondhand smoke in the United States as of 1999 to 2002.[17]
- In 2008, data from 11 states showed that the majority of people surveyed in each state reported having smoke-free home rules, ranging from 68.8% in West Virginia to 85.6% in Arizona (BRFSS).[18]
- As of December 31, 2010, 25 states and the District of Columbia had laws that prohibited smoking in indoor areas of worksites, restaurants, and bars; no states had such laws in 2000. As of December 31, 2010, an additional 10 states had laws that prohibited smoking in 1 or 2 but not all 3 venues.[19]
- The percentage of the US nonsmoking population with detectable serum cotinine declined from 52.5% in 1999 to 2000 to 40.1% in 2007 to 2008, with declines occurring for children and adults. During 2007 to 2008, the percentage of nonsmokers with detectable serum cotinine was higher for those 3 to 11 years of age (53.6%) and those 12 to 19 years of age (46.5%) than for those ≥20 years of age (36.7%); the percentage was also higher for non-Hispanic blacks (55.9%) than for non-Hispanic whites (40.1%) and Mexican Americans (28.5%; NHANES).[20]
- Data from a 2006 report of the US Surgeon General on the consequences of involuntary exposure to tobacco smoke[21] indicate the following:
  - Nonsmokers who are exposed to secondhand smoke at home or at work increase their risk of developing CHD by 25% to 30%.
  - Short exposures to secondhand smoke can cause blood platelets to become stickier, damage the lining of blood vessels, and decrease coronary flow velocity reserves, potentially increasing the risk of an AMI.

Aftermath

- A 2010 report of the US Surgeon General on how tobacco causes disease summarizes an extensive body of literature on smoking and CVD and the mechanisms through which smoking is thought to cause CVD. Among its conclusions are the following:
  - There is a sharp increase in CVD risk with low levels of exposure to cigarette smoke, including secondhand

smoke, and a less rapid further increase in risk as the number of cigarettes per day increases.
— Smoking cessation reduces the risk of cardiovascular morbidity and mortality for smokers with and without CHD.
— There is no evidence to date that reducing the amount smoked by smoking fewer cigarettes per day reduces the risk of CVD.[22]

- In 2007, 66.0% of adult current smokers 18 to 64 years of age with a checkup during the preceding year reported that they had been advised to quit, which was not significantly different from 2002 (62.6%; MEPS).[23]

Cost

Direct medical costs ($96 billion) and lost productivity costs ($97 billion) associated with smoking totaled an estimated $193 billion per year between 2000 and 2004.[14]

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Table 13-1. Cigarette Smoking

Population Group	Prevalence, 2010: Age ≥18 y*	Cost[24]
Both sexes	44 114 000 (19.3%)	$193 Billion per year
Males	23 725 000 (21.2%)	. . .
Females	20 389 000 (17.5%)	. . .
NH white males	23.0%	. . .
NH white females	20.5%	. . .
NH black males	23.4%	. . .
NH black females	16.7%	. . .
Hispanic or Latino males	15.2%	. . .
Hispanic or Latino females	9.0%	. . .
Asian only (both sexes)	9.3%	. . .
American Indian/Alaska Native only (both sexes)	26.6%	. . .

Ellipses (. . .) indicate data not available; NH, non-Hispanic.

Percentages are age adjusted. Estimates for Asian only and American Indian/Alaska Native only include non-Hispanic and Hispanic persons.

*Centers for Disease Control and Prevention/National Center for Health Statistics/National Health Interview Survey.[7]

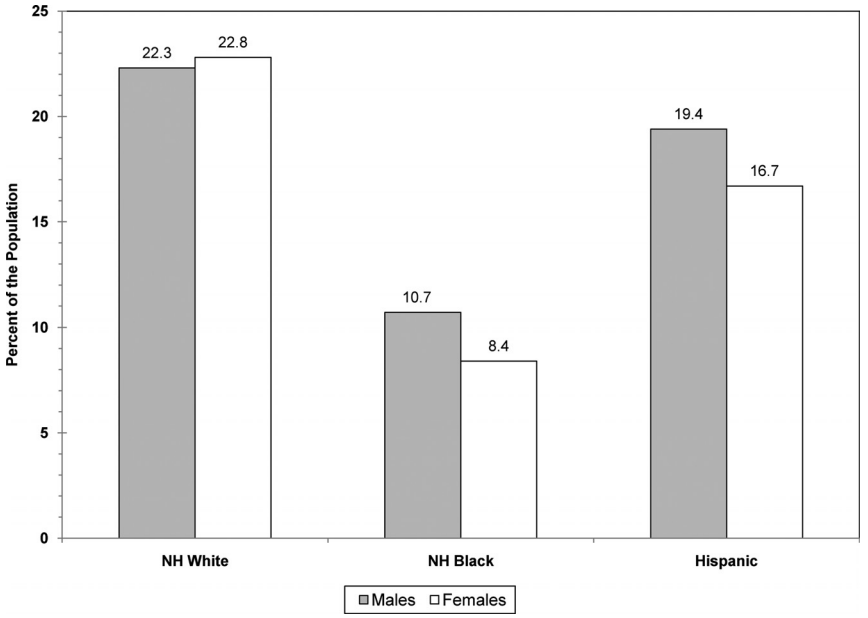

Chart 13-1. Prevalence (%) of students in grades 9 to 12 reporting current cigarette use by sex and race/ethnicity (Youth Risk Behavior Surveillance System, 2009). NH indicates non-Hispanic. Data derived from *MMWR: Morbidity and Mortality Weekly Report*.[1]

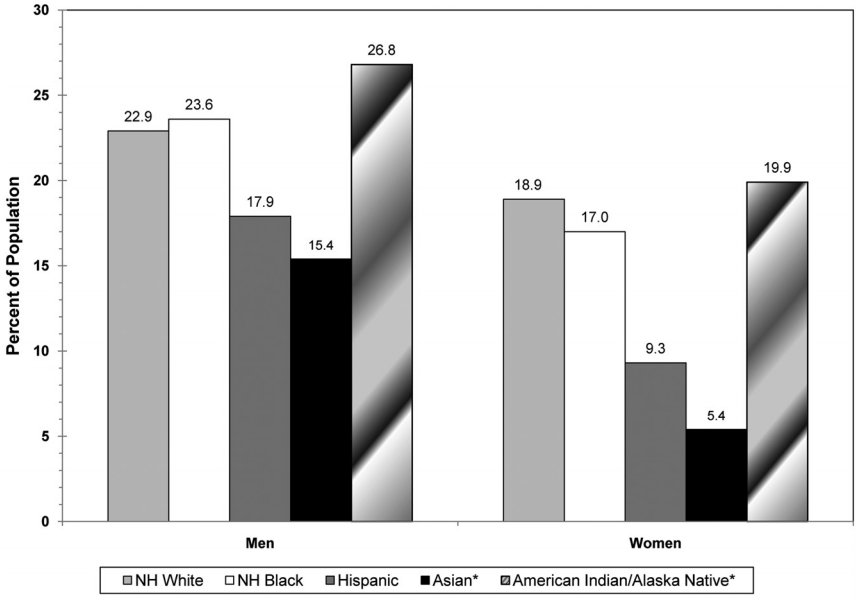

Chart 13-2. Prevalence (%) of current smoking for adults >18 years of age by race/ethnicity and sex (National Health Interview Survey: 2007–2009). All percentages are age adjusted. NH indicates non-Hispanic. *Includes both Hispanics and non-Hispanics. Data derived from Centers for Disease Control and Prevention/National Center for Health Statistics, Health Data Interactive.[11]

14. Risk Factor: High Blood Cholesterol and Other Lipids

See Table 14-1 and Charts 14-1 through 14-3.

Prevalence

For information on dietary cholesterol, total fat, saturated fat, and other factors that affect blood cholesterol levels, see Chapter 20 (Nutrition).

Youth

- Among children 4 to 11 years of age, the mean total blood cholesterol level is 164.5 mg/dL. For boys, it is 163.8 mg/dL; for girls, it is 165.2 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis):
  - — For non-Hispanic whites, 163.9 mg/dL for boys and 165.6 mg/dL for girls.
  - — For non-Hispanic blacks, 165.7 mg/dL for boys and 162.3 mg/dL for girls.
  - — For Mexican Americans, 160.7 mg/dL for boys and 161.5 mg/dL for girls.
- Among adolescents 12 to 19 years of age, the mean total blood cholesterol level is 159.2 mg/dL. For boys, it is 156.3 mg/dL; for girls, it is 162.3 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis):
  - — For non-Hispanic whites, 155.9 mg/dL for boys and 162.3 mg/dL for girls.
  - — For non-Hispanic blacks, 157.7 mg/dL for boys and 163.6 mg/dL for girls.
  - — For Mexican Americans, 156.9 mg/dL for boys and 161.3 mg/dL for girls.
- The prevalence of abnormal lipid levels among youths 12 to 19 years of age is 20.3%; 14.2% of normal-weight youths, 22.3% of overweight youths, and 42.9% of obese youths have at least 1 abnormal lipid level (NHANES 1999–2006, NCHS).[1]

Abbreviations Used in Chapter 14

BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	Coronary heart disease
CVD	Cardiovascular disease
DM	Diabetes mellitus
HD	Heart disease
HDL	High-density lipoprotein
LDL	Low-density lipoprotein
Mex. Am.	Mexican American
NCHS	National Center for Health Statistics
NH	Non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart, Lung, and Blood Institute

- Approximately 8.5% of adolescents 12 to 19 years of age have total cholesterol levels ≥200 mg/dL (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis).
- Fewer than 1% of adolescents are eligible for pharmacological treatment.[1,2]

Adults

- An estimated 33.5 million adults ≥20 years of age have total serum cholesterol levels ≥240 mg/dL (extrapolated to 2008 by use of NCHS/NHANES 2005–2008 data), with a prevalence of 16.2% (Table 14-1; NCHS and NHLBI, unpublished analysis).
- Data from NHANES 1999–2006 showed that ≈8% of adults ≥20 years of age have undiagnosed hypercholesterolemia.[3]
- Data from the BRFSS study of the CDC in 2009 showed that the percentage of adults who had been screened for high blood cholesterol in the preceding 5 years ranged from 67.5% in Utah to 85.3% in the District of Columbia. The median percentage among all 50 states was 77.0%.[4]
- The percentage of adults who reported having had a cholesterol check increased from 68.6% during 1999 to 2000 to 74.8% during 2005 to 2006.[5]
- Data from NHANES 1999–2002 (NCHS) showed that overall, 63.3% of participants whose test results indicated high blood cholesterol or who were taking a cholesterol-lowering medication had been told by a professional that they had high cholesterol. Women were less likely than men to be aware of their condition; blacks and Mexican Americans were less likely to be aware of their condition than were whites. Fewer than half of Mexican Americans with high cholesterol were aware of their condition.[6]
- Between the periods 1988 to 1994 and 1999 to 2002 (NHANES/NCHS), the age-adjusted mean total serum cholesterol level of adults ≥20 years of age decreased from 206 to 203 mg/dL, and LDL cholesterol levels decreased from 129 to 123 mg/dL.[7]
- Data from NHANES 2003–2008 (NCHS) showed the serum total crude mean cholesterol level in adults ≥20 years of age was 195 mg/dL for men and 201 mg/dL for women.[8]
- Data from the Minnesota Heart Survey (1980–1982 to 2000–2002) showed a decline in age-adjusted mean total cholesterol concentrations from 5.49 and 5.38 mmol/L for men and women, respectively, in 1980 to 1982 to 5.16 and 5.09 mmol/L, respectively, in 2000 to 2002; however, the decline was not uniform across all age groups. Middle-aged to older people have shown substantial decreases, but younger people have shown little overall change and recently had increased total cholesterol values. Lipid-lowering drug use rose significantly for both sexes among those 35 to 74 years of age. Awareness, treatment, and control of hypercholesterolemia have increased; however, more than half of those at borderline-high risk remain unaware of their condition.[9]
- Data from the BRFSS (CDC) survey in 2009 showed that among adults screened for high blood cholesterol, the percentage who had been told that they had high blood cholesterol ranged from 32.9% in Tennessee to 41.8% in

South Carolina. The median percentage among states was 37.5%.[4]

- Among adults with hypercholesterolemia, the percentage who had been told that they had high cholesterol increased from 42.0% during 1999 to 2000 to 50.4% during 2005 to 2006.[5]
- According to data from NHANES 2005–2006, between the periods 1999 to 2000 and 2005 to 2006, mean serum total cholesterol levels in adults ≥20 years of age declined from 204 to 199 mg/dL. This decline was observed for men ≥40 years of age and for women ≥60 years of age. There was little change over this time period for other sex/age groups. In 2005 to 2006, ≈65% of men and 70% of women had been screened for high cholesterol in the past 5 years, and 16% of adults had serum total cholesterol levels of 240 mg/dL or higher.[10]
- Self-reported use of cholesterol-lowering medications increased from 8.2% during 1999 to 2000 to 14.0% during 2005 to 2006.[5]
- According to data from NHANES, from 1999 to 2006, the prevalence of elevated LDL cholesterol levels in adults >20 years of age has decreased by ≈33%.[11]
- From 1999 to 2006, 26.0% of adults had hypercholesterolemia, 9% of adults had both hypercholesterolemia and hypertension, 1.5% of adults had DM and hypercholesterolemia, and 3% of adults had all 3 conditions.[3]

Adherence

Youth

The American Academy of Pediatrics recommends screening for dyslipidemia in children and adolescents who have a family history of dyslipidemia or premature CVD, those whose family history is unknown, and those youths with risk factors for CVD, such as being overweight or obese, having hypertension or DM, or being a smoker.[1]

Analysis of data from NHANES 1999–2006 showed that the overall prevalence of abnormal lipid levels among youths 12 to 19 years of age was 20.3%.[1]

Adults

- On the basis of data from the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults[12]:
  - — Fewer than half of all people who qualify for any kind of lipid-modifying treatment for CHD risk reduction are receiving it.
  - — Fewer than half of even the highest-risk people (those with symptomatic CHD) are receiving lipid-lowering treatment.
  - — Only approximately one third of treated patients are achieving their LDL goal; <20% of patients with CHD are at their LDL goal.
- Data from NHANES 2005–2006 indicate that among those with elevated LDL cholesterol levels, 35.5% had not been screened previously, 24.9% were screened but not told they had elevated cholesterol, and 39.6% were treated inadequately.[11]
- NHANES data on the treatment of high LDL cholesterol showed an increase from 28.4% of individuals during 1999 to 2002 to 48.1% during 2005 to 2008.[13]
- There were 33.2% of adults overall during 2005 to 2008 in NHANES who achieved LDL cholesterol goals. Among adults without health insurance, only 22.6% achieved LDL cholesterol goals; however, 82.8% of those adults with uncontrolled LDL cholesterol did have some form of health insurance.[13]

Lipid Levels

LDL (Bad) Cholesterol

Youth

- There are limited data available on LDL cholesterol for children 4 to 11 years of age.
- Among adolescents 12 to 19 years of age, the mean LDL cholesterol level is 88.5 mg/dL. For boys, it is 87.1 mg/dL, and for girls, it is 89.9 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis):
  - — Among non-Hispanic whites, 87.6 mg/dL for boys and 89.8 mg/dL for girls.
  - — Among non-Hispanic blacks, 88.8 mg/dL for boys and 92.6 mg/dL for girls.
  - — Among Mexican Americans, 88.4 mg/dL for boys and 88.8 mg/dL for girls.
- High levels of LDL cholesterol occurred in 8.4% of male adolescents and 6.8% of female adolescents during 1999 to 2006.[1]

Adults

- The mean level of LDL cholesterol for American adults ≥20 years of age was 115.2 mg/dL in 2008.[11] Levels of 130 to 159 mg/dL are considered borderline high, levels of 160 to 189 mg/dL are classified as high, and levels of ≥190 mg/dL are considered very high.
- According to NHANES 2005–2008 (NCHS and NHLBI; unpublished data):
  - — Among non-Hispanic whites, mean LDL cholesterol levels were 114.5 mg/dL for men and 115.8 mg/dL for women.
  - — Among non-Hispanic blacks, mean LDL cholesterol levels were 114.6 mg/dL for men and 111.5 mg/dL for women.
  - — Among Mexican Americans, mean LDL cholesterol levels were 121.2 mg/dL for men and 113.6 mg/dL for women.
- The age-adjusted prevalence of high LDL cholesterol in US adults was 26.6% in 1988 to 1994 and 25.3% in 1999 to 2004 (NHANES/NCHS). Between 1988 to 1994 and 1999 to 2004, awareness increased from 39.2% to 63.0%, and use of pharmacological lipid-lowering treatment increased from 11.7% to 40.8%. LDL cholesterol control increased from 4.0% to 25.1% among those with high LDL choles-

terol. In 1999 to 2004, rates of LDL cholesterol control were lower among adults 20 to 49 years of age than among those ≥65 years of age (13.9% versus 30.3%, respectively), among non-Hispanic blacks and Mexican Americans than among non-Hispanic whites (17.2% and 16.5% versus 26.9%, respectively), and among men than among women (22.6% versus 26.9%, respectively).[14]

- Mean levels of LDL cholesterol decreased from 126.1 mg/dL during 1999 to 2000 to 114.8 mg/dL during 2005 to 2006. The prevalence of high LDL cholesterol decreased from 31.5% during 1999 to 2000 to 21.2% during 2005 to 2006.[11]

HDL (Good) Cholesterol

Youth

- Among children 4 to 11 years of age, the mean HDL cholesterol level is 54.7 mg/dL. For boys, it is 55.6 mg/dL, and for girls, it is 53.6 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis):
  - Among non-Hispanic whites, 54.7 mg/dL for boys and 52.8 mg/dL for girls.
  - Among non-Hispanic blacks, 61.4 mg/dL for boys and 58.1 mg/dL for girls.
  - Among Mexican Americans, 53.6 mg/dL for boys and 51.1 mg/dL for girls.
- Among adolescents 12 to 19 years of age, the mean HDL cholesterol level is 51.6 mg/dL. For boys, it is 49.3 mg/dL, and for girls, it is 54.0 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis):
  - Among non-Hispanic whites, 48.1 mg/dL for boys and 53.3 mg/dL for girls.
  - Among non-Hispanic blacks, 54.6 mg/dL for boys and 56.9 mg/dL for girls.
  - Among Mexican Americans, 48.3 mg/dL for boys and 53.5 mg/dL for girls.
- Low levels of HDL cholesterol occurred in 11% of male adolescents and 4% of female adolescents during 1999 to 2006.[1]

Adults

- An HDL cholesterol level below 40 mg/dL in adult males and below 50 mg/dL in adult females is considered low and is a risk factor for HD and stroke. The mean level of HDL cholesterol for American adults ≥20 years of age is 53.3 mg/dL (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis).
- According to NHANES 2005–2008 (NCHS and NHLBI; unpublished analysis):
  - Among non-Hispanic whites, mean HDL cholesterol levels were 47.2 mg/dL for men and 58.8 mg/dL for women.
  - Among non-Hispanic blacks, mean HDL cholesterol levels were 52.3 mg/dL for men and 61.3 mg/dL for women.
  - Among Mexican Americans, mean HDL cholesterol levels were 46.0 mg/dL for men and 54.2 mg/dL for women.

Triglycerides

Youth

- There are limited data available on triglycerides for children 4 to 11 years of age.
- Among adolescents 12 to 19 years of age, the mean triglyceride level is 87.8 mg/dL. For boys, it is 87.2 mg/dL, and for girls, it is 88.5 mg/dL. The racial/ethnic breakdown is as follows (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis):
  - Among non-Hispanic whites, 92.7 mg/dL for boys and 90.9 mg/dL for girls.
  - Among non-Hispanic blacks, 68.8 mg/dL for boys and 63.0 mg/dL for girls.
  - Among Mexican Americans, 94.5 mg/dL for boys and 90.2 mg/dL for girls.
- High levels of triglycerides occurred in 11.4% of male adolescents and 8.8% of female adolescents during 1999 to 2006.[1]

Adults

- A fasting triglyceride level >150 mg/dL in adults is considered elevated and is a risk factor for HD and stroke. The mean level of triglycerides for American adults ≥20 years of age is 137.6 mg/dL (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis).
  - Among men, the mean triglyceride level is 149.9 mg/dL (NHANES 2005–2008, NCHS and NHLBI; unpublished analysis). The racial/ethnic breakdown is as follows:
    - 150.2 mg/dL for white men.
    - 120.1 mg/dL for black men.
    - 169.4 mg/dL for Mexican American men.
  - Among women, the mean triglyceride level is 125.5 mg/dL, with the following racial/ethnic breakdown:
    - 128.8 mg/dL for white women.
    - 97.0 mg/dL for black women.
    - 139.0 mg/dL for Mexican American women.
- Approximately 33% of adults ≥20 years of age had a triglyceride level ≥150 mg/dL during 1999 to 2004.[15]
- Fewer than 3% of adults with a triglyceride level ≥150 mg/dL received pharmacological treatment during 1999 to 2004.[15]

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Table 14-1. High Total and LDL Cholesterol and Low HDL Cholesterol

Population Group	Prevalence of Total Cholesterol ≥200 mg/dL, 2008: Age ≥20 y	Prevalence of Total Cholesterol ≥240 mg/dL, 2008: Age ≥20 y	Prevalence of LDL Cholesterol ≥130 mg/dL, 2008: Age ≥20 y	Prevalence of HDL Cholesterol <40 mg/dL, 2008: Age ≥20 y
Both sexes*	98 800 000 (44.4%)	33 600 000 (15.0%)	71 300 000 (31.9%)	41 800 000 (18.9%)
Males*	45 000 000 (41.8%)	14 600 000 (13.5%)	35 300 000 (32.5%)	30 800 000 (28.6%)
Females*	53 800 000 (46.3%)	19 000 000 (16.2%)	36 000 000 (31.0%)	11 000 000 (9.7%)
NH white males, %	41.2	13.7	30.5	29.5
NH white females, %	47.0	16.9	32.0	10.1
NH black males, %	37.0	9.7	34.4	16.6
NH black females, %	41.2	13.3	27.7	6.6
Mexican-American males, %	50.1	16.9	41.9	31.7
Mexican-American females, %	46.5	14.0	31.6	12.2

LDL indicates low-density lipoprotein; HDL, high-density lipoprotein; and NH, non-Hispanic.

Prevalence of total cholesterol ≥200 mg/dL includes people with total cholesterol ≥240 mg/dL. In adults, levels of 200 to 239 mg/dL are considered borderline high. Levels of ≥240 mg/dL are considered high.

*Total data for total cholesterol are for Americans ≥20 y of age. Data for LDL cholesterol, HDL cholesterol, and all racial/ethnic groups are age adjusted for age ≥20 y.

Source for total cholesterol ≥200 mg/dL, total cholesterol ≥240 mg/dL, LDL, and HDL: National Health and Nutrition Examination Survey (2005–2008), National Center for Health Statistics, and National Heart, Lung, and Blood Institute. Estimates from National Health and Nutrition Examination Survey 2005–2008 (National Center for Health Statistics) applied to 2008 population estimates.

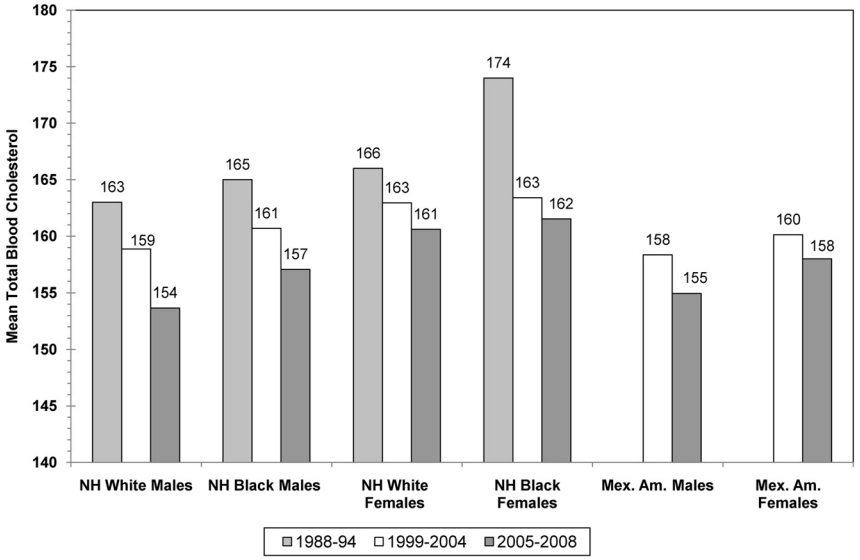

Chart 14-1. Trends in mean total serum cholesterol among adolescents 12 to 17 years of age by race, sex, and survey year (National Health and Nutrition Examination Survey: 1988–1994,* 1999–2004, and 2005–2008). Values are in mg/dL. NH indicates non-Hispanic; Mex. Am., Mexican American. *Data for Mexican Americans not available. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

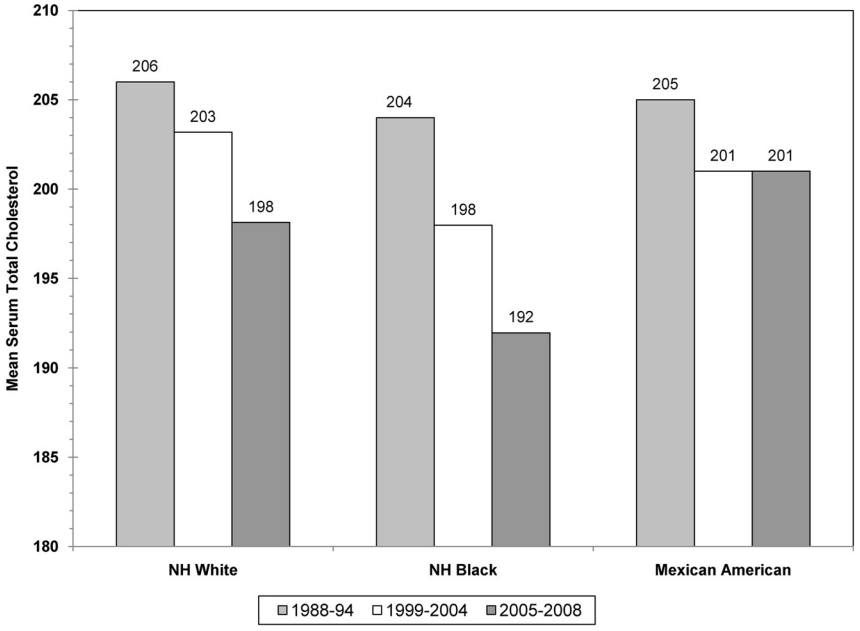

Chart 14-2. Trends in mean total serum cholesterol among adults ≥20 years of age by race and survey year (National Health and Nutrition Examination Survey: 1988–1994, 1999–2004, and 2005–2008). Values are in mg/dL. NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

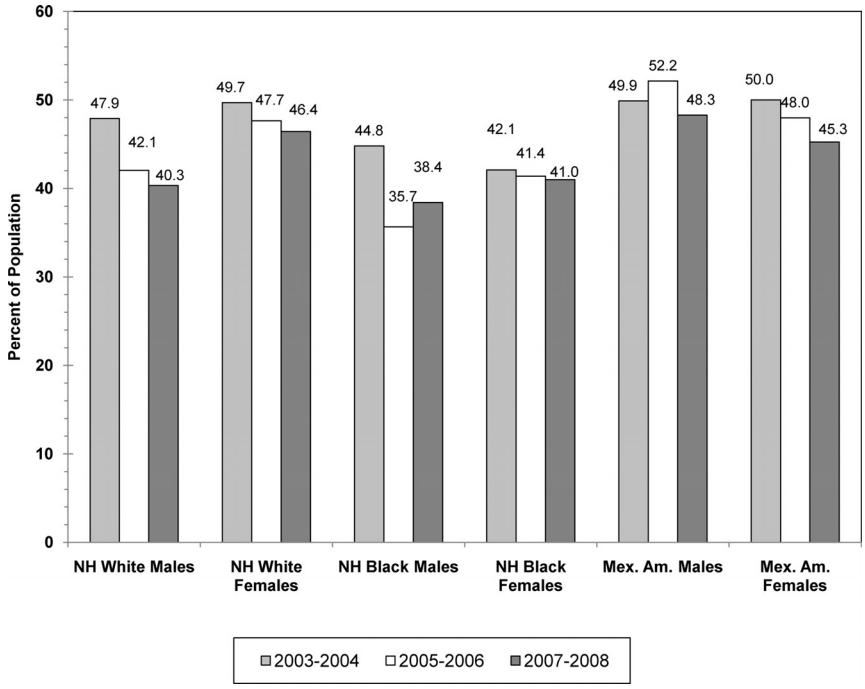

Chart 14-3. Age-adjusted trends in the prevalence of total serum cholesterol >200 mg/dL in adults ≥20 years of age by sex, race/ethnicity, and survey year (National Health and Nutrition Examination Survey 2003–2004, 2005–2006, and 2007–2008). NH indicates non-Hispanic; Mex. Am., Mexican American.

15. Risk Factor: Physical Inactivity

See Table 15-1 and Charts 15-1 through 15-4.

Prevalence

Youth

Inactivity

- The proportion of adolescents (12–19 years old) who report engaging in no regular PA is high and varies by sex and race.[1]
- Nationwide, 23.1% of adolescents were inactive during the previous 7 days, indicated by their response that they did not participate in ≥60 minutes of any kind of PA that increased their heart rate and made them breathe hard on any 1 of the previous 7 days.[1]
- Girls were more likely than boys to report inactivity (29.9% versus 17.0%).[1]
- The prevalence of inactivity was highest in black (43.6%) and Hispanic (30.5%) girls, followed by white girls (25.4%), black boys (20.6%), Hispanic boys (17.4%), and white boys (15.9%; CDC).[1]
- Nationwide, 24.9% of adolescents used a computer for activities other than school work (eg, videogames or other computer games) for ≥3 hours per day on an average school day.[1]
- A greater proportion of black and Hispanic students used computers or watched television >3 hours per day than white students.[1]

Abbreviations Used in Chapter 15

CARDIA	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
EF	ejection fraction
FMD	flow-mediated dilation
HbA_{1c}	glycosylated hemoglobin
HBP	high blood pressure
HDL	high-density lipoprotein
HF	heart failure
HR	hazard ratio
LDL	low-density lipoprotein
MEPS	Medical Expenditure Panel Survey
MET	metabolic equivalent tasks
MI	myocardial infarction
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
PA	physical activity
PAD	peripheral arterial disease
RR	relative risk

Activity Recommendations

- The proportion of students who met activity recommendations of ≥60 minutes of PA on ≥5 days of the week was 37.0% nationwide and declined from 9th (39.7%) to 12th (31.6%) grades, and at each grade level, the proportion was higher in boys than in girls.[1]
- More high school boys (45.6%) than girls (27.7%) self-reported having been physically active at least 60 minutes per day on ≥5 days; self-reported rates of activity were higher in white (39.9%) than in black (32.6%) or Hispanic (33.1%) adolescents.[1]
- A total of 15.3% of high school students met the recommendations for aerobic activity, 51.0% met the recommendations for muscle-strengthening activity, and 12.2% met the recommendations for both aerobic and muscle-strengthening activities.[2] There was a marked discrepancy between the proportion of youth (ages 6–11 years) who reported engaging in ≥60 minutes of moderate-to-vigorous PA on most days of the week and those who actually engaged in moderate-to-vigorous PA for ≥60 minutes when activity was measured objectively with accelerometers (ie, portable motion sensors that record and quantify the duration and intensity of movements) in the NHANES 2003–2004 survey.[3]
- On the basis of accelerometer counts per minute >2020, 42% of 6- to 11-year-olds accumulated ≥60 minutes of moderate-to-vigorous PA on 5 of 7 days per week, whereas only 8% of 12- to 15-year-olds and 7.6% of 16- to 19-year-olds achieved similar counts.[3]
- More boys than girls met PA recommendations (≥60 minutes of moderate to vigorous activity on most days of the week) as measured by accelerometry.[3]

Structured Activity Participation

- Despite recommendations from the National Association for Sport and Physical Education that schools should require daily physical education for students in kindergarten through 12th grade, only 33.3% of students attended physical education classes in school daily (34.6% of boys and 31.9% of girls).[1,4]
- Physical education class participation declined from the 9th through the 12th grades among boys and girls.[1]
- Among children 9 to 13 years old, 61.5% do not participate in any organized PA during nonschool hours and 22.6% do not engage in any free-time PA, according to 2002 data from the Youth Media Campaign Longitudinal Study of the CDC.[5]
- Little more than half (58.3%) of all students played on at least 1 school or community sports team in the previous year; however, the prevalence declined with increasing grade level, from 61.6% in the 9th grade to 51.1% in the 12th grade.[1]

Adults

Inactivity

- Thirty-three percent of adults (≥18 years of age) do not engage in leisure-time PA according to 2010 data from the NHIS ("no leisure-time physical activity/inactivity" refers to no sessions of light/moderate or vigorous PA of at least 10 minutes' duration).[6]
- Inactivity in 2010 was higher among women than men (35.2% versus 29.7%, age adjusted) and increased with age

from 27.1% to 32.7%, 42.2%, and 57.2% among adults 18 to 44, 45 to 64, 65 to 74, and ≥75 years of age, respectively.[6]
- Non-Hispanic black and Hispanic adults were more likely to be inactive (43.2% and 44.7%, respectively) than were non-Hispanic white adults (31.0%) on the basis of age-adjusted estimates from the 2010 NHIS.[6]
- Forty-nine percent of adults who responded to the 2010 NHIS survey did not meet either aerobic or strengthening guidelines of the 2008 Federal guidelines for PA.[6]
- Women (54.1%) were more likely than men (43.9%) to not meet the 2008 Federal PA guidelines on the basis of age-adjusted estimates from the 2010 NHIS.[6]
- The proportion of respondents who did not meet the Federal PA guidelines increased with age from 43.1% in 18- to 44-year-olds to 70.3% in adults ≥75 years of age in the 2010 NHIS.[6]
- Blacks (58.5%), American Indians/Alaska Natives (53.9%), and Asians (51.7%) were more likely to not meet the Federal PA guidelines than whites (47.7%), and Hispanic/Latino adults were more likely not to meet the Federal PA guidelines (60.1%) than non-Hispanic/non-Latino adults (47.3%) according to age-adjusted estimates from the 2010 NHIS.[6]
- The probability of not meeting the Federal PA guidelines was inversely associated with education; participants with no high school diploma (69.9%), a high school diploma (59.1%), some college (48.8%), or a bachelor's degree or higher (36.1%), respectively, did not meet the Federal PA guidelines on the basis of the 2010 NHIS.[6]

Activity Recommendations

- The proportion of adults reporting levels of PA consistent with the 2008 Physical Activity Guidelines for Americans remains low and decreases with age.[6,7] Thirty-three percent of respondents in a study examining awareness of current US PA guidelines had direct knowledge of the recommended dosage of PA (ie, frequency/duration).[8]
- The age-adjusted proportion of adults ≥18 years of age who reported engaging in regular moderate or vigorous PA as defined by the 2008 Physical Activity Guidelines for Americans was 47.2% on the basis of the 2010 NHIS; 52.1% of men and 42.6% of women met the recommendations. Prevalence for non-Hispanic whites was 51.4%, 37.3% for non-Hispanic blacks, and 36.3% for Hispanics.[6]
- The percentage of adults reporting at least 150 minutes of moderate PA or 75 minutes of vigorous PA decreased with age from 53.8% for adults 18 to 24 years of age to 23.9% for those ≥75 years of age on the basis of the 2010 NHIS.[6]
- In 2010, 24.4% of adults met the 2008 Federal PA guidelines for strengthening activity, an important component of overall physical fitness.[6] This estimate includes adults who met the strengthening guideline only or met it in combination with the aerobic guideline.
  - The percentage of men who engaged in any leisure-time strengthening activities decreased with age, from 47% at age 18 to 24 years to 16% at age ≥75 years. The percentage of women who engaged in leisure-time strengthening activities also decreased with age, from 28% at age 18 to 24 years to 11% at age ≥75 years, on the basis of the 2008 NHIS.[9]
- Adherence to PA recommendations was much lower when based on PA measured by accelerometer in NHANES 2003–2004[3]:
  - Among adults 20 to 59 years of age, 3.8% of men and 3.2% of women met recommendations to engage in moderate-to-vigorous PA (accelerometer counts >2020/min) for 30 minutes (in sessions of ≥10 minutes) on ≥5 of 7 days.
  - Among people ≥60 years of age, adherence was 2.5% in men and 2.3% in women.
- In a review examining self-reported versus actual measured PA (eg, accelerometers, pedometers, indirect calorimetry, double-labeled water, heart rate monitor), 60% of respondents self-reported higher values of activity than what was measured by use of direct methods.
  - Among men, self-reported PA was 44% greater than actual measured values; among women, self-reported activity was 138% greater than actual measured PA.[10]

Trends

Youth

- A study of 3068 youths between the ages of 14 and 24 years from 1999 to 2006 found that the prevalence of inactivity went up with age in both boys and girls.
  - Across ages, girls had a higher prevalence of physical inactivity than boys.[11]
  - In a study of 12 812 youth ages 9 to 18 years, the PA level in boys and girls declined starting at the age of 13, with a significantly greater decline in activity among girls.[12]

Adults

- Between NHANES III (1988–1994) and NHANES 2001–2006, the proportion of adults who engaged in >12 bouts of PA per month declined from 57% to 43% in men and from 49% to 43% in women.[12]
- In non-Hispanic whites, the activity level has decreased from 55.3% to 45.2%; for non-Hispanic blacks, it has decreased from 41.2% to 34.6%; and for Hispanics, the decline has been from 40.9% to 36.2%.[12]
- Accelerometry data from NHANES 2003–2006 shows that men engaged in 35 minutes of moderate activity per day, whereas for women, it was 21 minutes. More than 75% of moderate activity was accumulated in 1-minute bouts. No sex or race group had >1 bout of vigorous activity per day that lasted at least 10 minutes. Levels of activity declined sharply after the age of 50 years in all groups.[13]
- The proportion of adults meeting the 2008 Federal PA guidelines for aerobic activity (at least 150 minutes of moderate PA or 75 minutes of vigorous PA or an equivalent combination) in the 2010 NHIS was positively associated with education level: 60.4% of people with a college degree or higher met the PA guidelines compared with 27.0% of adults with less than a high school diploma.[6]
- Annual estimates of the percentage of US adults who met the muscle-strengthening criteria ranged from 17.7% (1998) to 24.4% (2010), and estimates of the percentage who met both the muscle-strengthening and aerobic criteria ranged from 14.4% (1998) to 20.7% (2010).[6,7]

CHD Risk Factors

Youth

- More girls (67.9%) than boys (55.7%) reported having exercised to lose weight or to keep from gaining weight.[1]
- White girls (72.2%) were more likely than black (54.2%) and Hispanic (66.3%) girls to report exercising to lose weight or to keep from gaining weight.[1]
- Total and vigorous PA are inversely correlated with body fat and the prevalence of obesity.[14]
- Physical inactivity was positively correlated with CHD risk factors (eg, mean arterial pressure, triglycerides, LDL, HDL, and fasting plasma glucose) in youths. Findings were similar for boys and girls.[15]

Adults

- Participants in the Diabetes Prevention Project randomized trial who met the PA goal of 150 minutes of PA per week were 44% less likely to develop DM, even if they did not meet the weight-loss target.[16]
- As a weight-loss intervention, exercise alone was associated with significant reductions in DBP (−2 mm Hg; 95% CI −4 to −1 mm Hg), triglycerides (−0.2 mmol/L; 95% CI −0.3 to −0.1 mmol/L), and fasting glucose (−0.2 mmol/L; 95% CI −0.3 to −0.1 mmol/L).[17]
- A total of 120 to 150 minutes per week of moderate-intensity activity can reduce the risk of developing metabolic syndrome and its individual components (ie, abdominal adiposity, HBP, low HDL cholesterol, high triglycerides, or high glucose).[18]
- In CARDIA, women who maintained high activity through young adulthood gained 6.1 fewer kilograms of weight and 3.8 fewer centimeters in waist circumference in middle age than those with lower activity. Highly active men gained 2.6 fewer kilograms and 3.1 fewer centimeters than their lower-activity counterparts.[19]

CHD Events and Mortality

- The PA guidelines for adults cite evidence that ≈150 minutes per week of moderate-intensity aerobic activity can reduce the risk of CVD.[20]
  - — Adherence to PA guidelines for both aerobic and muscle-strengthening activities reduces all-cause mortality risks by 27% among adults without existing chronic conditions such as DM, cancer, MI, angina, CVD, stroke, or respiratory diseases and by 45.9% among people with chronic comorbidities.[21]
  - — The RR of CHD associated with physical inactivity ranges from 1.5 to 2.4.[22]
  - — Physical inactivity is responsible for 12.2% of the global burden of MI after accounting for other CVD risk factors such as cigarette smoking, DM, hypertension, abdominal obesity, lipid profile, no alcohol intake, and psychosocial factors.[23]
  - — A 2.3% decline in physical inactivity between 1980 and 2000 prevented or postponed ≈17 445 deaths (≈5%) attributable to CHD in the United States.[24]
  - — The Nurse's Health Study of >72 000 female nurses indicated that moderate-intensity PA, such as walking, is associated with a substantial reduction in risk of total and ischemic stroke.[25]
- Longitudinal studies commonly report a graded, inverse association of PA amount and duration (ie, dose) with incident CHD and stroke.[26]
  - — In the Health Professionals Follow-Up Study, PA "dose" was inversely associated with the incidence of CHD over time, with rates declining from 46.3, 39.3, 35.9, 32.2, and 25.8 cases per 10 000 person-years according to quintiles of activity. The adjusted HR comparing the uppermost quintile of activity with the lowest was 0.72 (95% CI 0.61–0.85).[27]
  - — Metabolic equivalent tasks (MET) levels >6 were associated with a statistically significantly lower RR (RR 0.83, 95% CI 0.74–0.97 versus MET intensity of 1–3.9) of developing incident CHD in the Health Professionals Follow-Up Study of men.[27]
  - — In a meta-analysis of longitudinal studies among women, RRs of incident CHD were 0.83 (95% CI 0.69–0.99), 0.77 (95% CI 0.64–0.92), 0.72 (95% CI 0.59–0.87), and 0.57 (95% CI 0.41–0.79) across increasing quintiles of PA compared with the lowest quintile.[28]
  - — A 2003 meta-analysis of 23 studies on the association of PA with stroke indicated that compared with low levels of activity, high (RR 0.79, 95% CI 0.69–0.91) and moderate (RR 0.91, 95% CI 0.80–1.05) levels of activity were inversely associated with the likelihood of developing total stroke (ischemic and hemorrhagic).[29]
  - — In the Health Professionals Follow-Up Study, for every 3-hour per week increase in vigorous-intensity activity, the multivariate RR of MI was 0.78 (95% CI 0.61–0.98) in men. This 22% reduction of risk can be explained in part by beneficial effects of PA on HDL cholesterol, vitamin D, apolipoprotein B, and hemoglobin A1c.[30]
  - — In a 20-year study of older male veterans, an inverse, graded, and independent association between impaired exercise capacity and all-cause mortality risk was found. For each 1-MET increase in exercise capacity, mortality risk was 12% lower (HR 0.88, 95% CI 0.86–0.90). Unfit individuals who improved their fitness status had a 35% lower mortality risk (HR 0.65, 95% CI 0.46–0.93) than those who remained unfit.[31]

Secondary Prevention

- PA improves inflammatory markers in people with existing stable CHD. After a 6-week training session, C-reactive protein levels declined by 23.7% ($P<0.001$), and plasma vascular cell adhesion molecule-1 levels declined by 10.23% ($P<0.05$); there was no difference in leukocyte count or levels of intercellular adhesion molecule-1.[32]
- In a randomized trial of patients with PAD, supervised treadmill exercise training and lower-extremity resistance training were each associated with significant improvements in functional performance and quality of life compared with a usual-care control group. Exercise training

was additionally associated with improved brachial artery FMD, whereas resistance training was associated with better stair-climbing ability versus control.[33]

- The benefit of intense exercise training for cardiac rehabilitation in people with HF was tested in a trial of 27 patients with stable, medically treated HF. Intense activity (an aerobic interval-training program 3 times per week for 12 weeks) was associated with a significant 35% improvement in left ventricular EF and decreases in pro-brain natriuretic peptide (40%), left ventricular end-diastolic volume (18%), and left ventricular end-systolic volume (25%) compared with control and endurance-training groups.[34]

Costs

- The economic consequences of physical inactivity are substantial. In a summary of World Health Organization data sources, the economic costs of physical inactivity were estimated to account for 1.5% to 3.0% of total direct healthcare expenditures in developed countries such as the United States.[35]
- The 1996 MEPS was linked to self-reported activity in the 1995 NHIS. On the basis of a self-reported prevalence of inactivity of 47.5% and a prevalence of CVD of 21.5%, the direct expenditures for CVD associated with inactivity were estimated to be $23.7 billion in 2001.[36]

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Table 15-1. Met 2008 Federal Physical Activity Guidelines for Adults

Population Group	Prevalence, 2010 (Age ≥18 y), %
Both sexes	20.7
Males	25.1
Females	16.4
NH white only	21.3
Males	26.7
Females	19.1
NH black only	17.2
Males	24.6
Females	11.2
Hispanic or Latino	14.4
Mexican American	13.2
American Indian/Alaska Native only	12.5
Asian only	17.8

NH indicates non-Hispanic.

"Met 2008 federal physical activity guidelines for adults" is defined as engaging in at least 150 minutes of moderate or 75 minutes of vigorous aerobic leisure-time physical activity per week (or an equivalent combination) and engaging in leisure-time strengthening physical activities at least twice a week.

Data are age adjusted for adults ≥18 years of age.

Source: National Health Interview Survey 2010 (National Center for Health Statistics).[6]

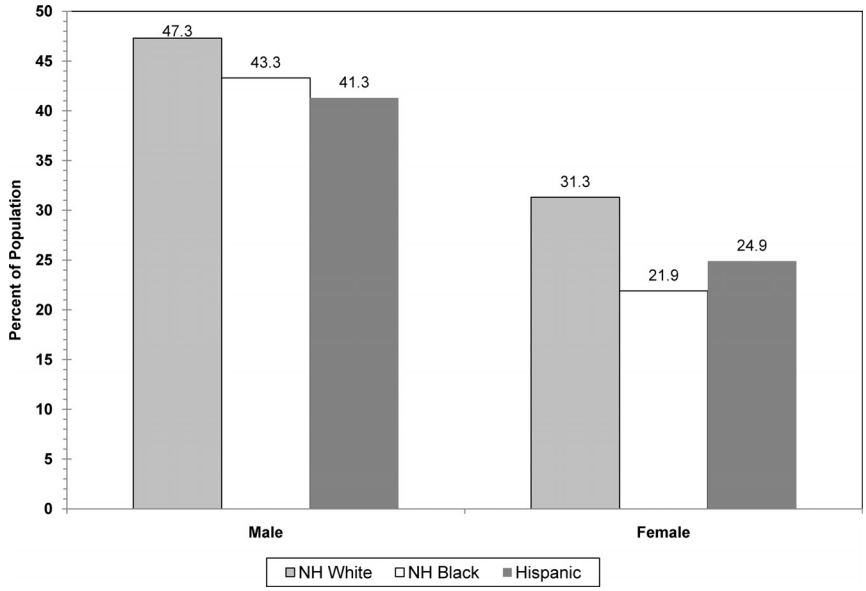

Chart 15-1. Prevalence of students in grades 9 through 12 who met currently recommended levels of physical activity during the past 7 days by race/ethnicity and sex (Youth Risk Behavior Surveillance: 2009). "Currently recommended levels" was defined as activity that increased their heart rate and made them breathe hard some of the time for a total of at least 60 minutes per day on 5 of the 7 days preceding the survey. NH indicates non-Hispanic. Data derived from *MMWR Surveillance Summaries*.[1]

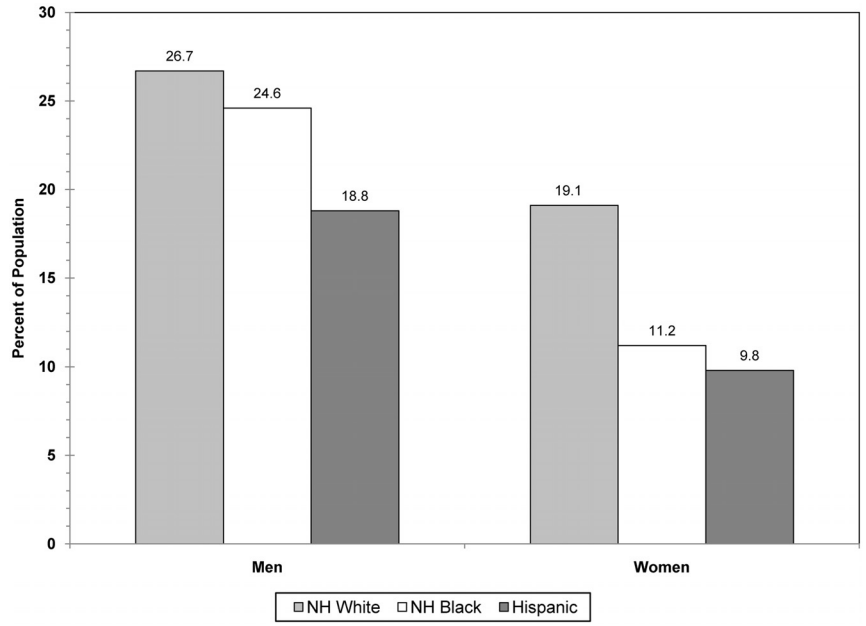

Chart 15-2. Prevalence of meeting the 2008 Federal physical activity guidelines among adults ≥18 years of age by race/ethnicity and sex (National Health Interview Survey: 2010). NH indicates non-Hispanic. Percents are age adjusted. Meeting the 2008 Federal physical activity guidelines is defined as engaging in moderate leisure-time physical activity for at least 150 minutes per week or vigorous activity at least 75 minutes per week or an equivalent combination. Source: Schiller et al.[6]

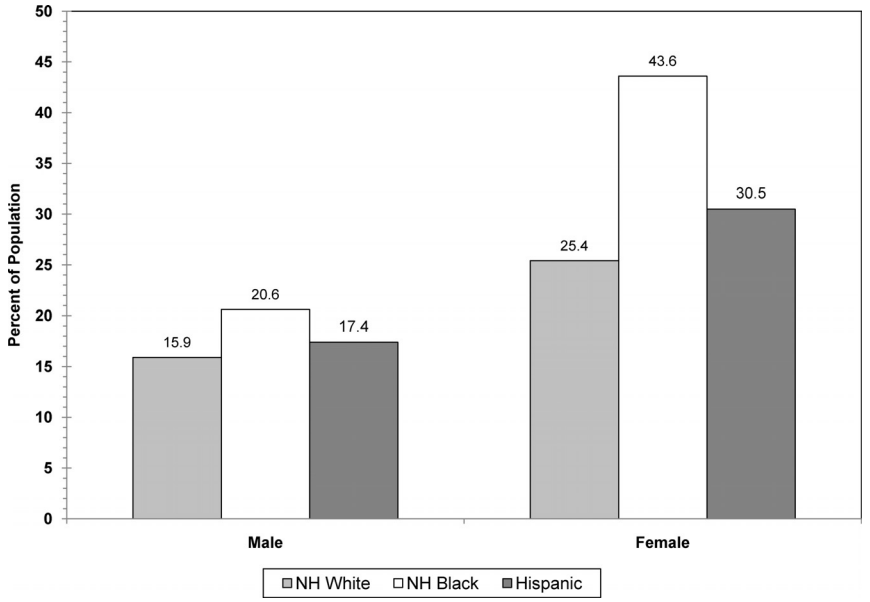

Chart 15-3. Prevalence of students in grades 9 to 12 who did not participate in at least 60 minutes of physical activity on any day by race/ethnicity and sex (Youth Risk Behavior Surveillance: 2009). NH indicates non-Hispanic. Data derived from *MMWR Surveillance Summaries*.[1]

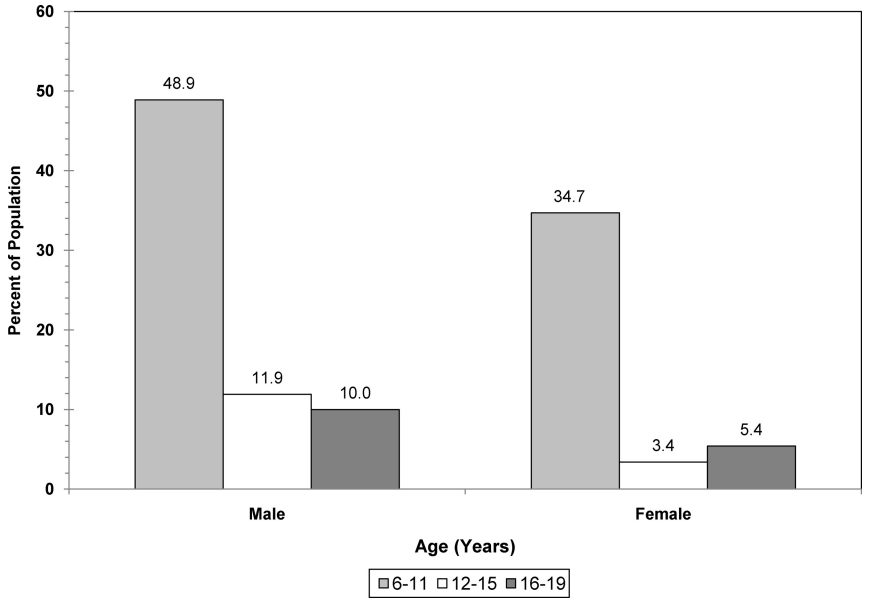

Chart 15-4. Prevalence of children 6 to 19 years of age who attained sufficient moderate-to-vigorous physical activity to meet public health recommendations (≥60 minutes per day on 5 or more of the 7 days preceding the survey), by sex and age (National Health and Nutrition Examination Survey: 2003–2004). Source: Troiano et al.[3]

16. Risk Factor: Overweight and Obesity

See Table 16-1 and Charts 16-1 through 16-3.

Prevalence

Youth

- According to nutritional surveys from the World Health Organization's Global Database on Child Growth and Malnutrition, in 2010, 43 million preschool children were either overweight or obese worldwide, and an additional 92 million were at risk of becoming overweight. Worldwide, the prevalence of childhood obesity increased from 4.2% in 1990 to 6.7% in 2010. By region, the estimated prevalence of overweight and obesity was as follows: Africa, 8.5%; Asia, 4.9%; Latin America and the Caribbean, 6.9%; Oceania 3.5%; developed countries (Europe, North America, Australia, New Zealand, and Japan), 11.7%; and developing countries, 6.1%.[1]
- The prevalence of overweight and obesity in children 2 to 5 years of age, on the basis of a BMI-for-age value ≥85th percentile of the 2000 CDC growth charts, was 16% for non-Hispanic white boys and 20% for non-Hispanic white girls, 28% for non-Hispanic black boys and 24% for non-Hispanic black girls, and 32% for Mexican American boys and 23% for Mexican American girls according to 2007 to 2008 data from NHANES (NCHS). In children 6 to 11 years of age, the prevalence was 35% for non-Hispanic white boys and 34% for non-Hispanic white girls, 36% for non-Hispanic black boys and 39% for non-Hispanic black girls, and 44% for Mexican American boys and 39% for Mexican American girls. In children 12 to 19 years of age, the prevalence was 33% for non-Hispanic white boys and 30% for non-Hispanic white girls, 33% for non-Hispanic black boys and 46% for non-Hispanic black girls, and 46% for Mexican American boys and 42% for Mexican American girls.[2]
- The prevalence of obesity in children 2 to 5 years of age, on the basis of BMI-for-age values ≥95th percentile of the 2000 CDC growth charts, was 7% for non-Hispanic white boys and 12% for non-Hispanic white girls, 11% for non-Hispanic black boys and 12% for non-Hispanic black girls, and 19% for Mexican American boys and 8% for Mexican American girls according to 2007 to 2008 data from NHANES (NCHS). In children 6 to 11 years of age, the prevalence was 21% for non-Hispanic white boys and 17% for non-Hispanic white girls, 18% for non-Hispanic black boys and 21% for non-Hispanic black girls, and 27% for Mexican American boys and 22% for Mexican American girls. In children 12 to 19 years of age, the prevalence was 17% for non-Hispanic white boys and 15% for non-Hispanic white girls, 20% for non-Hispanic black boys and 29% for non-Hispanic black girls, and 27% for Mexican American boys and 17% for Mexican American girls.[2]
- Overall, 19% of US children and adolescents 6 to 19 years of age have BMI-for-age values ≥95th percentile of the 2000 CDC growth charts for the United States (NHANES [2007–2008], NCHS).[2]
- NHANES 2003–2006 found that 11.3% of children and adolescents 2 to 19 years of age were at or above the 97th percentile of the 2000 BMI-for-age growth chart, 16.3% were ≥95th percentile, and 31.9% were ≥85th percentile.[3]
- Data from NHANES in the 2008 National Healthcare Quality Report[4] found the following:
  - — During 2003 to 2006, 39.4% of overweight (≥95th percentile of the 2000 BMI-for-age growth chart) children and teens 2 to 19 years of age were told by a doctor or health professional that they were overweight.
  - — During 2003 to 2006, overweight children 2 to 5 years of age (22.3%) and 6 to 11 years of age (35.70%) were less likely than overweight children 12 to 19 years of age (47.5%) to be told by a provider that they were overweight.
- A study of >8500 4-year-olds in the Early Childhood Longitudinal Study, Birth Cohort (National Center for Education Statistics) found that 1 in 5 were obese. Almost 13% of Asian children, 16% of white children, nearly 21% of black children, 22% of Hispanic children, and 31% of American Indian children were obese. Children were considered obese if their BMI was ≥95th percentile on the basis of CDC BMI growth charts. For 4-year-olds, that would be a BMI of ≈18 kg/m^2. Researchers did not examine reasons for the disparities.[5]
- Overweight adolescents have a 70% chance of becoming overweight adults. This increases to 80% if 1 or both parents are overweight or obese.[6]

Abbreviations for Chapter 16

BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CAD	coronary artery disease
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
FHS	Framingham Heart Study
HDL	high-density lipoprotein
HR	hazard ratio
MESA	Multi-Ethnic Study of Atherosclerosis
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHANES	National Health and Nutrition Examination Survey
NHDS	National Hospital Discharge Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NINDS	National Institute of Neurological Disorders and Stroke
NOMAS	Northern Manhattan Study
OR	odds ratio
PA	physical activity
RR	relative risk

- Childhood sociodemographic factors may contribute to sex disparities in obesity prevalence. A study of data from the National Longitudinal Study of Adolescent Health found that parental education consistently modified sex disparity in blacks. The sex gap was largest in those with low parental education (16.7% of men compared with 45.4% of women were obese) and smallest in those with high parental education (28.5% of men compared with 31.4% of women were obese). In whites, there was little overall sex difference in obesity prevalence.[7]
- The obesity epidemic is disproportionally more rampant among children living in low-income, low-education, and higher-unemployment households, according to data from the National Survey of Children's Health.[8]
- In boys and girls, on the basis of NHANES data, the prevalence of childhood obesity increases across all ranges of household education levels, although it is substantially higher among those with less education. Among boys, households headed by college graduates increased from 4.5% to 11.8% from 1998–1994 to 2005–2008, whereas those headed by individuals with less than a high school education increased from 15.3% to 21.1% over the same time period. For girls, those with a college graduate as the head of the household increased from 5.4% to 8.3% over the same time period, whereas those whose household head had less than a high school education increased from 11.4% to 20.4%.[9]
- According to the US National Longitudinal Study of Adolescent Health, 1.0% of adolescents were severely obese in 1996; the majority (70.5%) maintained this weight status into adulthood. Obese adolescents had a 16-fold increased risk of becoming severely obese adults compared with those with normal weight or those who were overweight.[10]

Adults

- Overall, 68% of US adults were overweight or obese (72% of men and 64% of women).[11]
- Among men, Mexican-Americans (80%) and non-Hispanic whites (73%) were more likely to be overweight or obese than non-Hispanic blacks (69%) according to NHANES 2007–2008.[11]
- Among women, non-Hispanic blacks (78%) and Mexican-Americans (77%) were more likely to be overweight or obese than non-Hispanic whites (61%).[11]
- Of US adults, 34% were obese (32% of men and 36% of women) according to NHANES 2007–2008.[11]
- Among men, non-Hispanic blacks (37%) and Mexican-Americans (36%) were more likely to be obese than non-Hispanic whites (32%).[11]
- Among women, non-Hispanic blacks (50%) and Mexican-Americans (45%) were more likely to be obese than non-Hispanic whites (33%).[11]
- When estimates were based on self-reported height and weight in the BRFSS/CDC survey in 2010, the prevalence of obesity ranged from 21.4% in Colorado to 34.5% in Mississippi. The median percentage by state was 27.6%.[12] Additionally, no state met the Healthy People 2010 goal of reducing obesity to 15% of adults.[13]
- The county-level prevalence of obesity in the United States ranged from 12.4% to 43.7%, with a median of 28.4% according to BRFSS/CDC 2007.[14]
- In 1998 and 1999, surveys of people in 8 states and the District of Columbia by the BRFSS study of the CDC indicated that obesity rates were significantly higher among people with disabilities, especially blacks and those 45 to 64 years of age.[15]
- Blacks ≥18 years of age (28.3%), American Indians or Alaska Natives (29.6%), and whites (36.5%) were less likely than Asians (55.0%) to be at a healthy weight on the basis of self-reported height and weight data from the 2010 NHIS.[16]
- On the basis of self-reported weights and heights, data showed that blacks ≥18 years of age (36.9%) and American Indians or Alaska Natives (39.6%) were more likely to be obese than were whites (26.8%) and Asians (11.6%), according to 2010 data from the NHIS.[16]
- Most adults in Asian subgroups were in the healthy weight range, with rates ranging from 51% for Filipino adults to 68% for Chinese adults. Although the prevalence of obesity is low within the Asian adult population, Filipino adults (14%) were more than twice as likely to be obese (BMI ≥30 kg/m^2) as Asian Indian (6%), Vietnamese (5%), or Chinese (4%) adults.[17]
- From 1999 to 2004, obese adults 45 to 64 years of age (73%) and ≥65 years of age (73.6%) were more likely than those 20 to 44 years of age (59.5%) to be told by a doctor or health professional that they were overweight. Obese adults 45 to 64 years of age and ≥65 years of age were more likely to receive advice about exercise than those 18 to 44 years of age.[4]
- Approximately 64.8% of obese adults were told by a doctor or health professional that they were overweight, according to the 2008 National Healthcare Disparities Report (on the basis of NHANES 2003–2006).[18]
- The proportion of obese adults told that they were overweight was significantly lower for non-Hispanic blacks (60.5%) and Mexican Americans (57.1%) than for non-Hispanic whites (66.4%), for middle-income people than for high-income people (62.4% versus 70.6%), and for adults with less than a high school education than for those with any college education (59.2% versus 70.3%).[18]
- A large proportion of white, black, and Hispanic participants were overweight (60% to 85%) or obese (30% to 50%), whereas fewer Chinese American participants were overweight (33%) or obese (5%), as judged by an analysis of data from MESA. These findings may be indicators of potential future increases in vascular disease burden and healthcare costs associated with the obesity epidemic.[19]

Trends

Youth

- The prevalence of BMI-for-age values ≥95th percentile of the 2000 CDC growth charts in children 6 to 11 years of age was 20% in 2007 to 2008 compared with 4.0% in 1971 to 1974. The prevalence of BMI-for-age values ≥95th percentile in adolescents 12 to 19 years of age was 18% in

2007 to 2008 compared with 6% in 1971 to 1974 in NHANES. No statistically significant linear trends in high weight for recumbent length or high BMI were found over the time periods 1999 to 2000, 2001 to 2002, 2003 to 2004, 2005 to 2006, and 2007 to 2008 among girls and boys except among the very heaviest 6- through 19-year-old boys.[2]

- Among infants and children between 6 and 23 months years of age, the prevalence of high weight for age was 7% in 1976 to 1980 and 12% in 2003 to 2006 (NHANES, NCHS).[20]
- The obesity epidemic in children continues to grow on the basis of recent data from the Bogalusa Heart Study. Compared with 1973 to 1974, the proportion of children 5 to 17 years of age who were obese was 5 times higher in 2008 to 2009.[21]

Adults

- Using 2009 self-reported BRFSS data, overall obesity prevalence was 26.7% in the United States, with rates of 27.4% in men and 26.0% in women. By race/ethnicity, the prevalence of obesity among non-Hispanic whites was 25.2%, whereas it was 36.8% among non-Hispanic blacks and 30.7% among Hispanics. There was an inverse association by education level: College graduates had a 20.8% rate of obesity, whereas those who attained less than a high school education had an obesity prevalence of 32.9%.[22]
- Analysis of the FHS, 1971 to 2001 (NHLBI), showed that among normal-weight white adults between 30 and 59 years of age, the 4-year rates of developing overweight varied from 14% to 19% in women and from 26% to 30% in men. The 30-year risk was similar for both sexes, with some variation by age. Overall, the 30-year risk for "overweight or more" exceeded 1 in 2 people, 1 in 4 for obesity, and 1 in 10 for stage II obesity (BMI $\geq$35 kg/m^2) across different age groups. The 30-year estimates correspond to the lifetime risk for "overweight or more" or obesity for participants 50 years of age.[23]
- The age-adjusted prevalence of obesity among adults increased between 1976 to 1980 and 1988 to 1994 and again between 1988 to 1994 and 1999 to 2000, on the basis of NHANES data. Over the 10-year period of 1999 to 2008, obesity showed no significant trend among women. For men, there was a significant linear trend. Obesity prevalence for men was 28% in NHANES 1999–2000 (NCHS) and 32% in NHANES 2007–2008; for women, obesity prevalence was 33% in 1999–2000 and 36% in 2007–2008.[11]
- Thirty-five percent of noninstitutionalized women 65 to 74 years of age and 27% of women $\geq$75 years of age were obese on the basis of NHANES/NCHS data in 2007 to 2008. This is an increase from 1988 to 1994, when 27% of women 65 to 74 years of age and 19% of women $\geq$75 years of age were obese. For men, in 1988 to 1994, 24% of those 65 to 74 years of age and 13% of those $\geq$75 years of age were obese compared with 40% of those 65 to 74 years of age and 26% of those $\geq$75 years of age in 2007 to 2008.[24]
- The prevalence of obesity increased by 5.6% or $\approx$2.7 million people from 1997 to 2002 among Medicare beneficiaries. By 2002, 21.4% of beneficiaries and 39.3% of disabled beneficiaries were obese compared with 16.4% and 32.5%, respectively, in 1997. The rise in obesity, along with expansions in treatment coverage, could greatly increase obesity-related Medicare spending.[25]
- The World Health Organization estimates that by 2015, the number of overweight people globally will increase to 2.3 billion, and 700 million will be obese. Globally, at least 20 million children <5 years of age were overweight in 2005. Once considered a problem only in high-income countries, overweight and obesity are now dramatically on the rise in low- and middle-income countries, particularly in urban settings.[26]

Morbidity

- Overweight children and adolescents are at increased risk for future adverse health effects, including the following[27]:
  - — Increased prevalence of traditional cardiovascular risk factors such as hypertension, hyperlipidemia, and DM.
  - — Poor school performance, tobacco use, alcohol use, premature sexual behavior, and poor diet.
  - — Other associated health conditions, such as asthma, hepatic steatosis, sleep apnea, stroke, some cancers (breast, colon, and kidney), musculoskeletal disorders, and gallbladder disease.
- According to data from the Bogalusa Heart Study and the Young Finns study, adolescents with high BMI in the overweight or obese range are at a 2.5-fold increased risk of developing metabolic syndrome, a 2.2-fold increased risk of high carotid IMT, and a 3.4-fold increased risk of DM in adulthood.[28]
- According to data from the Staff Periodic Examination Center of the Israeli Army Medical Corps, elevated BMI during adolescence was associated with DM (HR 2.76) and CHD diagnosed via angiography (HR 5.43); only the association with CHD persisted after BMI adjustment.[29]
- The increasing prevalence of obesity is driving an increased incidence of type 2 DM. Data from the FHS indicate a doubling in the incidence of DM over the past 30 years, most dramatically during the 1990s and primarily among individuals with a BMI >30 kg/m^2.[30]
- Obesity was the most powerful predictor of DM in the Nurses' Health Study. Women with a BMI of $\geq$35 kg/m^2 had an RR for DM of 38.8 compared with women with a BMI of <23 kg/m^2.[31]
- Overweight and obesity were associated with increased risk for CVD in the FHS. The age-adjusted relative risk for CVD was increased by 21% in men and 20% in women among those who were overweight and by 46% in men and 64% in women among those who were obese.[32]
- Abdominal obesity is an independent risk factor for ischemic stroke in all race/ethnic groups. This effect is larger for those <65 years of age (OR 4.4) than for those >65 years of age (OR 2.2; NOMAS, NINDS).[33]
- A recent comparison of risk factors in both the Honolulu Heart Program and the FHS (NHLBI) showed that a BMI increase of $\approx$3 kg/m^2 raised the risk of hospitalized thromboembolic stroke by 10% to 30%.[34]

- Obesity is also a strong predictor of sleep-disordered breathing, itself strongly associated with the development of CVD, as well as with myriad other health conditions, including numerous cancers, nonalcoholic fatty liver disease, gallbladder disease, musculoskeletal disorders, and reproductive abnormalities.[35]
- A recent meta-analysis of 15 prospective studies demonstrated the increased risk for Alzheimer disease or vascular dementia and any dementia was 1.35 and 1.26 for overweight, respectively, and 2.04 and 1.64 for obesity, respectively.[36]
- A randomized clinical trial of 130 severely obese adult individuals randomized to either 12 months of diet and PA or only 6 months of PA resulted in 12.1 and 9.9 kg, respectively, of weight loss at 1 year, with improvements in waist circumference, visceral fat, BP, and insulin resistance.[37]
- A meta-analysis of 58 prospective studies demonstrated associations with BMI, waist circumference, and waist-to-hip ratio with CHD (HR 1.29–1.30), stroke (HR 1.20–1.25), and CVD (HR 1.23–1.25) per 1-standard deviation higher values, although risk prediction was not improved with the inclusion of adiposity variables.[38]

Mortality

- Elevated childhood BMIs in the highest quartile were associated with premature death as an adult in a cohort of 4857 American Indian children during a median follow-up of 23.9 years.[39]
- Among adults, obesity was associated with nearly 112 000 excess deaths (95% CI 53 754–170 064) relative to normal weight in 2000. Grade I obesity (BMI 30 to <35 kg/m^2) was associated with almost 30 000 of these excess deaths (95% CI 8534–68 220) and grade II to III obesity (BMI ≥ 35 kg/m^2) with $>82\,000$ (95% CI 44 843–119 289). Underweight was associated with nearly 34 000 excess deaths (95% CI 15 726 to 51 766). As other studies have found,[40] overweight (BMI 25 to <30 kg/m^2) was not associated with excess deaths.[41]
- Overweight was associated with significantly increased mortality resulting from DM or kidney disease and was not associated with increased mortality resulting from cancer or CVD in an analysis of 2004 data from NHANES. Obesity was associated with significantly increased mortality caused by CVD, some cancers, and DM or kidney disease. Obesity was associated with 13% of CVD deaths in 2004.[42]
- Data from NHANES 1988–1994 were studied to determine estimates of excess deaths associated with BMI and other anthropometric variables. Estimates for all-cause mortality, obesity-related causes of death, and other causes of death showed no statistically significant or systematic differences between BMI and other variables.[43]
- In a collaborative analysis of data from almost 900 000 adults in 57 prospective studies, mostly in western Europe and North America, overall mortality was lowest at $\approx$22.5 to 25 kg/m^2 in both sexes and at all ages, after exclusion of early follow-up and adjustment for smoking status. Above this range, each 5-kg/m^2-higher BMI was associated with $\approx$30% higher all-cause mortality, and no specific cause of death was inversely associated with BMI. Below 22.5 to 25 kg/m^2, the overall inverse association with BMI was predominantly related to strong inverse associations for smoking-related respiratory disease, and the only clearly positive association was for ischemic heart disease.[44]
- In a meta-analysis of 1.46 million white adults, over a mean follow-up period of 10 years, all-cause mortality was lowest at BMI levels of 20.0 to 24.9 kg/m^2. Among women, compared with a BMI of 22.5 to 24.9 kg/m^2, the HR for death was as follows: BMI 15.0 to 18.4 kg/m^2, 1.47; 18.5 to 19.9 kg/m^2, 1.14; 20.0 to 22.4 kg/m^2, 1.0; 25.0 to 29.9 kg/m^2, 1.13; 30.0 to 34.9 kg/m^2, 1.44; 35.0 to 39.9 kg/m^2, 1.88; and 40.0 to 49.9 kg/m^2, 2.51. Similar estimates were observed in men.[45]
- Overweight and obesity were associated with large decreases in life expectancy in an analysis of data from the FHS (NHLBI). Forty-year-old female nonsmokers lost 3.3 years and 40-year-old male nonsmokers lost 3.1 years of life expectancy because of overweight. Among 40-year-old nonsmokers, women lost 7.1 years and men lost 5.8 years because of obesity. Obese female smokers lost 7.2 years and obese male smokers lost 6.7 years compared with normal-weight nonsmokers.[46]
- Recent calculations based on NHANES data from 1978 to 2006 suggest that the gains in life expectancy from smoking cessation are beginning to be outweighed by the loss of life expectancy from obesity.[47]
- As a result of the increasing prevalence of obesity, the number of quality-adjusted life years lost as a result of obesity is similar to or greater than that lost as a result of smoking, according to data from the BRFSS.[48]
- Recent estimates suggest that reductions in smoking, cholesterol, BP, and PA levels resulted in a gain of 2 770 500 life-years; however, these gains were reduced by a loss of 715 000 life-years caused by the increased prevalence of obesity and DM.[49]

Cost

- Among children and adolescents, annual hospital costs related to obesity were $127 million between 1997 and 1999.[50]
- According to 1 study, overall estimates show that the annual medical burden of obesity has increased to almost 10% of all medical spending and could amount to $147 billion per year in 2008 (in 2008 dollars).[51]
- If current trends in the growth of obesity continue, total healthcare costs attributable to obesity could reach $861 to $957 billion by 2030, which would account for 16% to 18% of US health expenditures.[52]
- According to NHANES I data linked to Medicare and mortality records, obese 45-year-olds had lifetime Medicare costs of $163 000 compared with $117 000 among those with normal weight by the time they reached 65 years of age.[53]
- The total excess cost related to the current prevalence of adolescent overweight and obesity is estimated to be $254 billion ($208 billion in lost productivity secondary to

premature morbidity and mortality and $46 billion in direct medical costs).[54]

Bariatric Surgery

- Patients with BMI >40 kg/m^2 or >35 kg/m^2 with an obesity-related comorbidity are eligible for gastric bypass surgery, which is typically performed as either a Roux-en-Y gastric bypass or a biliopancreatic diversion.
- According to the 2006 NHDS, the incidence of bariatric surgery was estimated at 113 000 cases per year, with costs of ≈1.5 billion dollars annually.[55]
- Among obese Swedish patients undergoing bariatric surgery and followed up for up to 15 years, maximum weight loss was 32%. The risk of death was 0.76 among those who underwent bariatric surgery compared with matched control subjects.[56] Among 641 patients followed up for 10 years compared with 627 matched control subjects, after 2 years of follow-up, 72% of the surgically treated patients versus 21% of the control patients had remission of their DM; at 10 years of follow-up, results were 36% and 13%, respectively. Similar results have been observed for hypertension, elevated triglycerides, and low HDL cholesterol.[57]
- According to retrospective data from the United States, among 9949 patients who underwent gastric bypass surgery, after a mean of 7 years, long-term mortality was 40% lower among the surgically treated patients than among obese control subjects. Specifically, cancer mortality was reduced by 60%, DM mortality by 92%, and CAD mortality by 56%. Death rates attributable to accidents and suicide were higher (58%) in the surgery group.[58]
- A recent retrospective cohort from the Veterans Affairs medical system showed that in a propensity-matched analysis, bariatric surgery was not associated with reduced mortality compared with obese control subjects (time-adjusted HR 0.94, 95% CI 0.64–1.39).[59]

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Table 16-1. Overweight and Obesity

Population Group	Prevalence of Overweight and Obesity in Adults, 2005–2008: Age ≥20 y	Prevalence of Obesity in Adults, 2005–2008: Age ≥20 y	Prevalence of Overweight and Obesity in Children, 2007–2008: Ages 2–19 y	Prevalence of Obesity in Children, 2007–2008: Ages 2–19 y	Cost, 2008*
Both sexes, n (%)	149 300 000 (67.3)	75 000 000 (33.7)	23 600 000 (31.7)	12 600 000 (16.9)	$147 Billion
Males	78 000 000 (72.4)	34 900 000 (32.4)	12 200 000 (32.1)	6 800 000 (17.8)	. . .
Females	71 300 000 (62.3)	40 100 000 (35.2)	11 400 000 (31.3)	5 800 000 (15.9)	. . .
NH white males, %	72.3	32.1	29.5	15.7	. . .
NH white females, %	59.3	32.8	29.2	14.9	. . .
NH black males, %	70.8	37.0	33.0	17.3	. . .
NH black females, %	77.7	51.0	39.0	22.7	. . .
Mexican American males, %	77.5	31.4	41.7	24.9	. . .
Mexican American females, %	75.1	43.4	36.1	16.5	. . .

NH indicates non-Hispanic; ellipses (. . .), data not available.

Data for white and black males and females are for non-Hispanics. Overweight and obesity in adults is defined as body mass index (BMI) ≥25 kg/m^2. Obesity in adults is defined as BMI ≥30 kg/m^2. In children, overweight and obesity are based on BMI-for-age values at or above the 85th percentile of the 2000 Centers for Disease Control and Prevention (CDC) growth charts. In children, obesity is based on BMI-for-age values at or above the 95th percentile of the CDC growth charts. In January 2007, the American Medical Association's Expert Task Force on Childhood Obesity recommended new definitions for overweight and obesity in children and adolescents[62]; however, statistics based on this new definition are not yet available.

*Data from *Health Affairs*.[51]

Sources: Age-adjusted National Health and Nutrition Examination Survey (NHANES) 2005–2008 (National Center for Health Statistics), National Heart, Lung, and Blood Institute, and unpublished data. Estimates from NHANES 2005–2008 (National Center for Health Statistics) were applied to 2008 population estimates. In children, age-adjusted NHANES 2007–2008 data were applied to 2006 population estimates.[2,11]

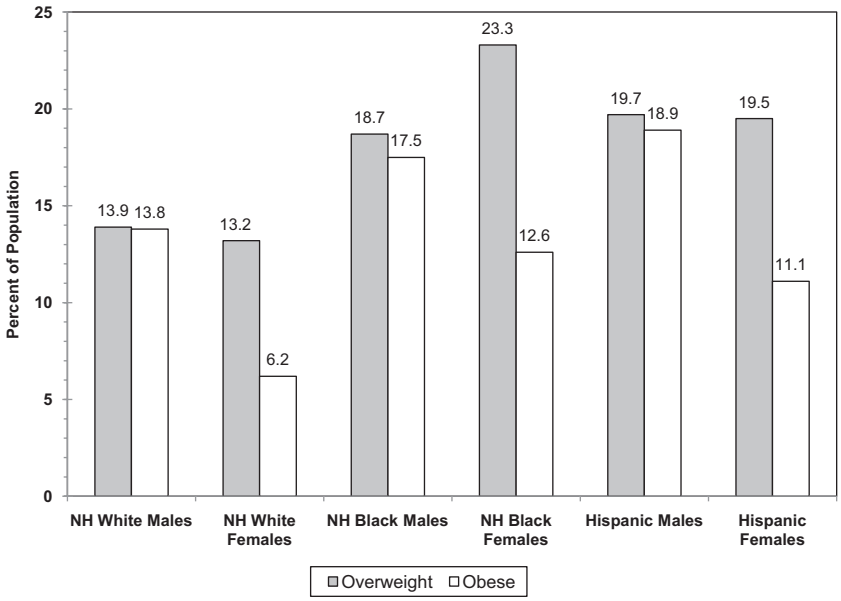

Chart 16-1. Prevalence of overweight and obesity among students in grades 9 through 12 by sex and race/ethnicity. NH indicates non-Hispanic. Data derived from Youth Risk Behavior Surveillance–United States, 2009, Table 90.[60]

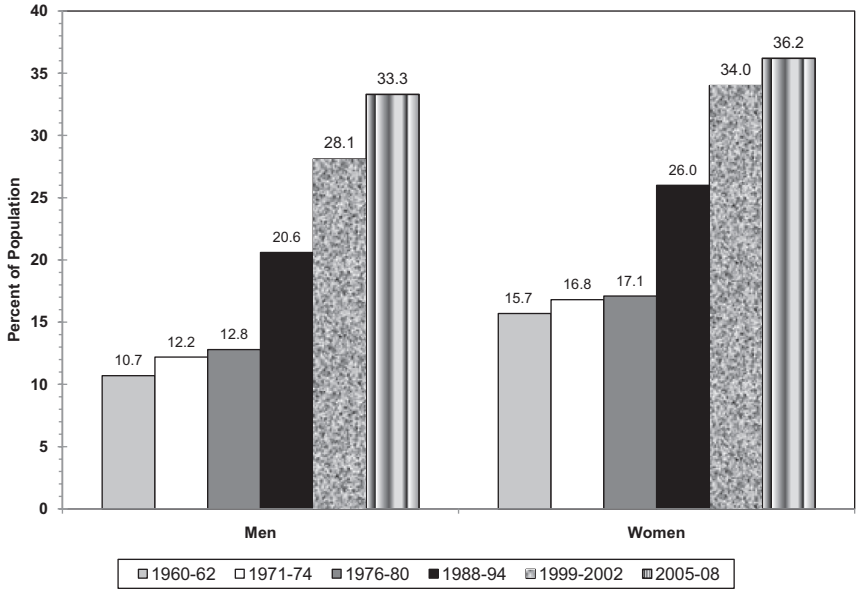

Chart 16-2. Age-adjusted prevalence of obesity in adults 20 to 74 years of age by sex and survey year (National Health Examination Survey: 1960–1962; National Health and Nutrition Examination Survey: 1971–1974, 1976–1980, 1988–1994, 1999–2002, and 2005–2008). Obesity is defined as a body mass index of 30.0 kg/m^2. Data derived from Health, United States, 2010 (National Center for Health Statistics).[61]

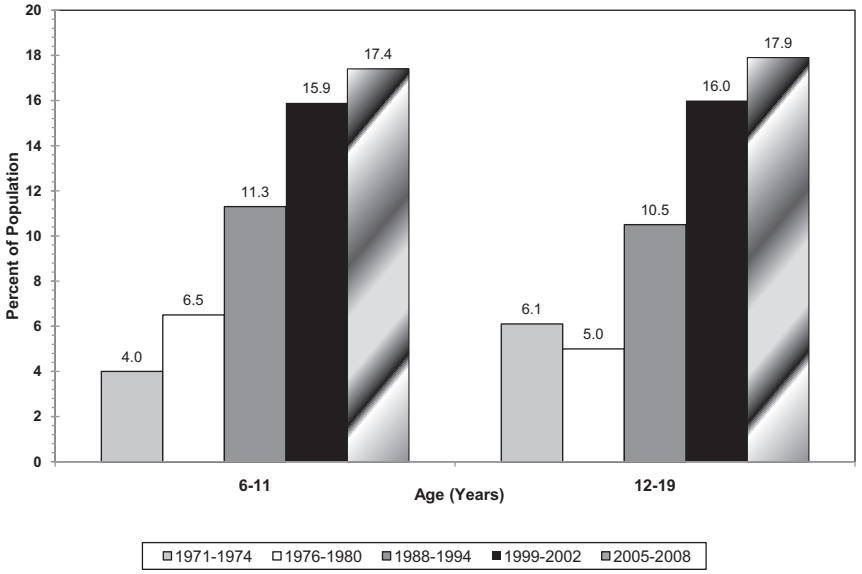

Chart 16-3. Trends in the prevalence of obesity among US children and adolescents by age and survey year (National Health and Nutrition Examination Survey: 1971–1974, 1976–1980, 1988–1994, 1999–2002 and 2005–2008). Data derived from Health, United States, 2010 (National Center for Health Statistics).[61]

17. Risk Factor: Diabetes Mellitus

ICD-9 250; ICD-10 E10 to E14. See Table 17-1 and Charts 17-1 through 17-4.

Prevalence

Youth

- In SEARCH, the prevalence of DM in youths <20 years of age in 2001 in the United States was 1.82 cases per 1000 youths (0.79 per 1000 among youths 0–9 years of age and 2.80 per 1000 among youths 10–19 years of age). Non-Hispanic white youths had the highest prevalence (1.06 per 1000) in the younger group. Among youths 10 to 19 years of age, black youths (3.22 per 1000) and non-Hispanic white youths (3.18 per 1000) had the highest rates, followed by American Indian youths (2.28 per 1000), Hispanic youths (2.18 per 1000), and Asian/Pacific Islander youths (1.34 per 1000). Among younger children, type 1 DM accounted for ≥80% of DM; among older youths, the proportion of type 2 DM ranged from 6% (0.19 per 1000 for non-Hispanic white youths) to 76% (1.74 per 1000 for American Indian youths). This translates to 154 369 youths with physician-diagnosed DM in 2001 in the United States, for an overall prevalence estimate for DM in children and adolescents of ≈0.18%.[1]
- Approximately 186 000 people <20 years of age have DM. Each year, ≈15 000 people <20 years of age are diagnosed with type 1 DM. Healthcare providers are finding more and more children with type 2 DM, a disease usually diagnosed in adults ≥40 years of age. Children who develop type 2 DM are typically overweight or obese and have a family history of the disease. Most are American Indian, black, Asian, or Hispanic/Latino.[2]
- Among adolescents 10 to 19 years of age diagnosed with DM, 57.8% of blacks were diagnosed with type 2 versus type 1 DM compared with 46.1% of Hispanic and 14.9% of white youths.[3]
- According to the Bogalusa Heart Study, a long-term follow-up study of youths aging into adulthood, youths who were prediabetic or who had DM are more likely to have a constellation of metabolic disorders in young adulthood (19–44 years of age), including obesity, hypertension, dyslipidemia, and metabolic syndrome, all of which predispose to CHD.[4]

Adults

- On the basis of data from NHANES 2005–2008 (NCHS; unpublished NHLBI tabulation; Table 17-1), an estimated 18.3 million Americans ≥20 years of age have physician-diagnosed DM. An additional 7.1 million adults have undiagnosed DM, and ≈81.5 million adults have prediabetes (eg, fasting blood glucose of 100 to <126 mg/dL). The prevalence of prediabetes in the US adult population is nearly 37%.
- Data from NHANES 2005–2006 (NCHS) showed the prevalence of diagnosed DM in adults ≥65 years of age to be 17.0%. The prevalence of undiagnosed DM was 14.6% (based on fasting glucose or oral glucose tolerance testing).[5]
- Among Americans ≥20 years of age, 11.3% have diagnosed DM. Men ≥20 years of age have a slightly higher prevalence (11.8%) than women (10.8%).[6]
- After adjustment for population age differences, 2007 to 2009 national survey data for people ≥20 years of age indicate that 7.1% of non-Hispanic whites, 8.4% of Asian Americans, 11.8% of Hispanics, and 12.6% of non-Hispanic blacks had diagnosed DM.[6]
- Compared with non-Hispanic white adults, the risk of diagnosed DM was 18% higher among Asian Americans,

Abbreviations Used in Chapter 17

ACS	acute coronary syndrome
AMI	acute myocardial infarction
ARIC	Atherosclerosis Risk in Communities study
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention
CHD	coronary heart disease
CHS	Cardiovascular Health Study
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
ECG	electrocardiographic
ESRD	end-stage renal disease
FHS	Framingham Heart Study
HbA1c	glycosylated hemoglobin
HD	heart disease
HDL	high-density lipoprotein
HR	hazard ratio
ICD-9	*International Classification of Diseases, 9th Revision*
ICD-10	*International Classification of Diseases, 10th Revision*
LDL	low-density lipoprotein
MEPS	Medical Expenditure Panel Survey
MESA	Multi-Ethnic Study of Atherosclerosis
MI	myocardial infarction
NAMCS	National Ambulatory Medical Care Survey
NCHS	National Center for Health Statistics
NH	non-Hispanic
NHAMCS	National Hospital Ambulatory Medical Care Survey
NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NSTEMI	non–ST-segment–elevation myocardial infarction
OR	odds ratio
PA	physical activity
PAR	population-attributable risk
RR	relative risk
SBP	systolic blood pressure
SEARCH	Search for Diabetes in Youth Study
STEMI	ST-segment–elevation myocardial infarction
UA	unstable angina

66% higher among Hispanics/Latinos, and 77% higher among non-Hispanic blacks.[6]
- In 2004 to 2006, the prevalence of diagnosed DM was more than twice as high for Asian Indian adults (14%) as for Chinese (6%) or Japanese (5%) adults.[7]
- Type 2 DM accounts for 90% to 95% of all diagnosed cases of DM in adults.[6]
- The prevalence of DM increased by 8.2% from 2000 to 2001. From 1990 to 2001, the prevalence of those diagnosed with DM increased 61%.[8]
- On the basis of 2010 BRFSS (CDC) data, the prevalence of adults who reported ever having been told by a physician that they had DM ranged from 5.3% in Alaska to 13.2% in Alabama. The median percentage among states was 8.7%.[9]
- The CDC analyzed data from 1994 to 2004 collected by the Indian Health Service that indicated that the age-adjusted prevalence per 1000 population of DM increased 101.2% among American Indian/Alaska Native adults <35 years of age (from 8.5% to 17.1%). During this time period, the prevalence of diagnosed DM was greater among females than males in all age groups.[10]
- On the basis of projections from NHANES/NCHS studies between 1984 and 2004, the total prevalence of DM in the United States is expected to more than double from 2005 to 2050 (from 5.6% to 12.0%) in all age, sex, and race/ethnicity groups. Increases are projected to be largest for the oldest age groups (for instance, increasing by 220% among those 65–74 years of age and by 449% among those ≥75 years of age). DM prevalence is projected to increase by 99% among non-Hispanic whites, by 107% among non-Hispanic blacks, and by 127% among Hispanics. The age/race/ethnicity group with the largest increase is expected to be blacks ≥75 years of age (increase of 606%).[11]
- According to NHIS data from 1997 to 2008, the prevalence of DM was higher among Asian Americans (4.3% to 8.2%) than whites (3.8% to 6.0%), despite lower BMI levels (23.6 versus 26.1 kg/m^2 in the earliest time period) among Asians.[12]
- The prevalence of DM for all age groups worldwide was estimated to be 2.8% in 2000 and is projected to be 4.4% in 2030. The total number of people with DM is projected to rise from 171 million in 2000 to 366 million in 2030.[13]
- According to international survey and epidemiological data from 2.7 million participants, the prevalence of DM in adults increased from 8.3% (in men) and 7.5% (in women) in 1980 to 9.8% (men) and 9.2% (women) in 2008. The number of individuals affected with DM increased from 153 million in 1980 to 347 million in 2008.[14]

Incidence

Youths

- In the SEARCH study, the incidence of DM in youths overall was 24.3 per 100 000 person-years. Among children <10 years of age, most had type 1 DM, regardless of race/ethnicity. The highest rates of incident type 1 DM were observed in non-Hispanic white youths (18.6, 28.1, and 32.9 per 100 000 person-years for age groups of 0–4, 5–9, and 10–14 years, respectively). Overall, type 2 DM was relatively infrequent, with the highest rates (17.0–49.4 per 100 000 person-years) seen among 15- to 19-year-old minority groups.[3]

Adults

- A total of 1.9 million new cases of DM were diagnosed in people ≥20 years of age in 2006.[6]
- Data from Framingham, MA, indicate a doubling in the incidence of DM over the past 30 years, most dramatically during the 1990s. Among adults 40 to 55 years of age in each decade of the 1970s, 1980s, and 1990s, the age-adjusted 8-year incidence rates of DM were 2.0%, 3.0%, and 3.7% among women and 2.7%, 3.6%, and 5.8% among men, respectively. Compared with the 1970s, the age- and sex-adjusted OR for DM was 1.40 in the 1980s and 2.05 in the 1990s (P for trend=0.0006). Most of the increase in absolute incidence of DM occurred in individuals with a BMI ≥30 kg/m^2 (P for trend=0.03).[15]
- DM incidence in adults also varies markedly by race. Over 5 years of follow-up in 45- to-84-year-olds in the MESA, 8.2% of the cohort developed DM. The cumulative incidence was highest in Hispanics (11.3%), followed by black (9.5%), Chinese (7.7%), and white (6.3%) participants.[16]

Mortality

DM mortality in 2008 was 70 553. Any-mention mortality in 2008 was 231 402 (NHLBI tabulation of NCHS mortality data).

- The 2007 overall underlying-cause death rate attributable to DM was 22.5. Death rates per 100 000 people were 24.6 for white males, 45.9 for black males, 17.2 for white females, and 40.2 for black females (NCHS, Health Data Interactive[17]).
- According to data from the National Diabetes Information Clearinghouse, the National Institute of Diabetes and Digestive and Kidney Diseases, and the National Institutes of Health:
  - At least 68% of people >65 years of age with DM die of some form of HD; 16% die of stroke.
  - HD death rates among adults with DM are 2 to 4 times higher than the rates for adults without DM.[6]
- In a collaborative meta-analysis of 820 900 individuals from 97 prospective studies, DM was associated with the following risks: all-cause mortality, HR 1.80 (95% CI 1.71–1.90); cancer death, HR 1.25 (95% CI 1.19–1.31); and vascular death, HR 2.32 (95% CI 2.11–2.56). In particular, DM was associated with death attributable to the following cancers: liver, pancreas, ovary, colorectal, lung, bladder, and breast. A 50-year-old with DM died on average 6 years earlier than an individual without DM.[18]
- FHS/NHLBI data show that having DM significantly increased the risk of developing CVD (HR 2.5 for women and 2.4 for men) and of dying when CVD was present (HR 2.2 for women and 1.7 for men). Diabetic men and women ≥50 years of age lived an average of 7.5 and 8.2 years less than their nondiabetic equivalents. The differences in life expectancy free of CVD were 7.8 and 8.4 years, respectively.[19]
- Analysis of data from NHANES 1971–2000 found that men with DM experienced a 43% relative reduction in the age-adjusted mortality rate, which was similar to that of nondiabetic men. Among women with DM, however,

mortality rates did not decrease, and the difference in mortality rates between diabetic and nondiabetic women doubled.[20]

- During 1979 to 2004, DM death rates for black youths 1 to 19 years of age were approximately twice those for white youths. During 2003 to 2004, the annual average DM death rate per 1 million youths was 2.46 for black youths and 0.91 for white youths.[21]
- Analysis of data from the FHS from 1950 to 2005 found reductions in all-cause and CVD mortality among men and women with and without DM; however, all-cause and CVD mortality rates among individuals with DM remain ≈2-fold higher than for individuals without DM.[22]

Awareness

- The National Institute of Diabetes and Digestive and Kidney Diseases estimates that 25.8 million Americans (8.3% of the population) have DM.[6]
- Analysis of NHANES/NCHS data from 1988–1994 to 2005–2006 in adults ≥20 years of age showed that 40% of those with DM did not know they had it.[5] Although the prevalence of diagnosed DM has increased significantly over the past decade, the prevalences of undiagnosed DM and impaired fasting glucose have remained relatively stable. Minority groups remain disproportionately affected.[23]
- Analysis of NHANES/NCHS data collected during 2005 to 2008 indicated that the prevalence of DM was 8.2% among people ≥20 years of age. Prevalence of DM was defined as people who were told by a physician or other health professional that they have DM. Of the estimated 18.3 million adults with DM, 73.3% were told they had DM or were undergoing treatment and 26.7% (5.7 million) were unaware of the diagnosis. Of 7 895 000 people being treated (37.3% of the diabetic population), one third (2 604 000) had their DM under control (ie, they were undergoing treatment and had fasting plasma glucose <126 mg/dL), and 25.0% (5.3 million) were being treated but did not have their DM under control (fasting plasma glucose ≥126 mg/dL). An estimated 13.3 million individuals with DM are not treated. The untreated and unaware population (5.6 million) was 26.7% of the diabetic population (NHLBI tabulation of NHANES 2003–2006; Chart 17-4).

Aftermath

- Although the exact date of DM onset can be difficult to determine, duration of DM appears to affect CVD risk. Longitudinal data from Framingham, MA, suggest that the risk factor–adjusted RR of CHD is 1.38 (95% CI 0.99–1.92) times higher and the risk for CHD death is 1.86 (95% CI 1.17–2.93) times higher for each 10-year increase in duration of DM.[24]
- DM increases the risk of stroke, with the RR ranging from 1.8 to almost 10.0.[25,26] DM increases ischemic stroke incidence at all ages, but this risk is most prominent before 55 years of age in blacks and before 65 years of age in whites.[26]
- Ischemic stroke patients with DM are younger, more likely to be black, and more likely to have hypertension, MI, and high cholesterol than nondiabetic patients.[26] On the basis of data from the NCHS/NHIS from 1997 to 2005[27]:
  - During 1997 to 2005, the estimated number of people ≥35 years of age with DM with a self-reported cardiovascular condition increased 36%, from 4.2 million in 1997 to 5.7 million in 2005; however, the age-adjusted prevalence of self-reported CVD conditions among people with diagnosed DM ≥35 years of age decreased 11.2%, from 36.6% in 1997 to 32.5% in 2005.
  - During 1997 to 2005, age-adjusted CVD prevalence was higher among men than women, among whites than blacks, and among non-Hispanics than Hispanics. Among women, the age-adjusted prevalence decreased by 11.2%; among men, it did not decrease significantly. Among blacks, the age-adjusted prevalence of self-reported CVD decreased by 25.3%; among whites, no significant decrease occurred; among non-Hispanics, the rate decreased by 12%. No clear trends were detected among Hispanics. If the total number of people with DM and self-reported CVD increased over this period but proportions with self-reported CVD declined, the data suggest that the mean age at which people have been diagnosed is decreasing, or the higher CVD mortality rate among older diabetic individuals is removing them from ability to self-report CVD. These and other data show a consistent increase over time in the United States of the number of people with DM and CVD.
- Statistical modeling of the use and effectiveness of specific cardiac treatments and of changes in risk factors between 1980 and 2000 among US adults 25 to 84 years of age showed that the age-adjusted death rate for CHD decreased from 543 to 267 deaths per 100 000 population among men and from 263 to 134 deaths per 100 000 population among women. Approximately 47% of this decrease was attributed to treatments, and ≈44% was attributed to changes in risk factors, although reductions were offset in part by increases in BMI and the prevalence of DM, which accounted for an increased number of deaths (8% and 10%, respectively).[28] An analysis from the Cooper Clinic in Dallas, TX, of exercise ECG responses and CVD mortality in 2854 men with DM reported 441 deaths (210 CVD and 133 CHD) over a follow-up of 16 years. That analysis showed that equivocal and abnormal exercise ECG responses were associated with higher risk of all-cause, CVD, and CHD mortality. Across normal, equivocal, and abnormal exercise ECG groups, age- and examination year–adjusted CHD mortality rates per 10 000 person-years were 23.0, 48.6, and 69.0, respectively (P for trend <0.001), and risk factor–adjusted HRs were 1.00, 1.68 (95% CI 1.01–2.77), and 2.21 (95% CI 1.41–3.46; P for trend <0.001), respectively.[29]
- A subgroup analysis was conducted of patients with DM enrolled in randomized clinical trials that evaluated ACS therapies. The data included 62 036 patients from Throm-

bolysis in Myocardial Infarction (TIMI) studies (46 577 with STEMI and 15 459 with UA/NSTEMI). Of these, 17.1% had DM. Modeling showed that mortality at 30 days was significantly higher among patients with DM than among those without DM who presented with UA/NSTEMI (2.1% versus 1.1%; $P \leq 0.001$) and STEMI (8.5% versus 5.4%; $P=0.001$), with adjusted risks for 30-day mortality in DM versus no DM of 1.78 for UA/NSTEMI (95% CI 1.24–2.56) and 1.40 (95% CI 1.24–1.57) for STEMI. DM was also associated with significantly higher mortality 1 year after UA/NSTEMI or STEMI. By 1 year after ACS, patients with DM presenting with UA/NSTEMI had a risk of death that approached that of patients without DM presenting with STEMI (7.2% versus 8.1%).[30]

- Data from the ARIC study of the NHLBI found that DM was a weaker predictor of CHD in blacks than in whites.[31]
- Data from Framingham, MA, show that despite improvements in CVD morbidity and mortality, DM continues to elevate CVD risk. Participants 45 to 64 years of age from the FHS original and offspring cohorts who attended examinations in 1950 to 1966 ("earlier" time period) and 1977 to 1995 ("later" time period) were followed up for incident MI, CHD death, and stroke. Among participants with DM, the age- and sex-adjusted CVD incidence rate was 286.4 per 10 000 person-years in the earlier period and 146.9 per 10 000 person-years in the later period, a 35.4% decline. HRs for DM as a predictor of incident CVD were not significantly different in the earlier (risk factor–adjusted HR 2.68, 95% CI 1.88–3.82) versus later (HR 1.96, 95% CI 1.44–2.66) periods.[32] Thus, although there was a 50% reduction in the rate of incident CVD events among adults with DM, the absolute risk of CVD remained 2-fold greater than among people without DM.[32]
  - — Data from these earlier and later time periods in Framingham also suggest that the increasing prevalence of DM is leading to an increasing rate of CVD, resulting in part from CVD risk factors that commonly accompany DM. The age- and sex-adjusted HR for DM as a CVD risk factor was 3.0 in the earlier time period and 2.5 in the later time period. Because the prevalence of DM has increased over time, the PAR for DM as a CVD risk factor increased from 5.4% in the earlier time period to 8.7% in the later time period (attributable risk ratio 1.62; $P=0.04$). Adjustment for CVD risk factors (age, sex, hypertension, current smoking, high cholesterol, and obesity) weakened this attributable risk ratio to 1.5 ($P=0.12$).[33]
  - — Other data from Framingham show that over 30 years, CVD among women with DM was 54.8% among normal-weight women but 78.8% among obese women. Among normal-weight men with DM, the lifetime risk of CVD was 78.6%, whereas it was 86.9% among obese men.[34]
- Other studies show that the increased prevalence of DM is being followed by an increasing prevalence of CVD morbidity and mortality. New York City death certificate data for 1989 to 1991 and 1999 to 2001 and hospital discharge data for 1988 to 2002 show increases in all-cause and cause-specific mortality between 1990 and 2000, as well as in annual hospitalization rates for DM and its complications among patients hospitalized with AMI and/or DM. During this decade, all-cause and cause-specific mortality rates declined, although not for patients with DM; rates increased 61% and 52% for diabetic men and women, respectively, as did hospitalization rates for DM and its complications. The percentage of all AMIs occurring in patients with DM increased from 21% to 36%, and the absolute number more than doubled, from 2951 to 6048. Although hospital days for AMI fell overall, for those with DM, they increased 51% (from 34 188 to 51 566). These data suggest that increases in DM rates threaten the long-established nationwide trend toward reduced coronary artery events.[35]
- In an analysis of provincial health claims data for adults living in Ontario, Canada, between 1992 and 2000, the rate of patients admitted for AMI and stroke decreased to a greater extent in the diabetic than the nondiabetic population (AMI, −15.1% versus −9.1%, $P=0.0001$; stroke, −24.2% versus −19.4%, $P=0.0001$). Diabetic patients experienced reductions in case fatality rates related to AMI and stroke similar to those without DM (−44.1% versus −33.2%, $P=0.1$; −17.1% versus −16.6%, $P=0.9$, respectively) and similarly comparable decreases in all-cause mortality. Over the same period, the number of DM cases increased by 165%, which translates to a marked increase in the proportion of CVD events occurring among patients with DM: AMI, 44.6%; stroke, 26.1%; AMI deaths, 17.2%; and stroke deaths, 13.2%.[36]
- In the same data set, the transition to a high-risk category (an event rate equivalent to a 10-year risk of 20% or an event rate equivalent to that associated with previous MI) occurred at a younger age for men and women with DM than for those without DM (mean difference 14.6 years). For the outcome of AMI, stroke, or death resulting from any cause, diabetic men and women entered the high-risk category at 47.9 and 54.3 years of age, respectively. The data suggest that DM confers a risk equivalent to aging 15 years. In North America, diverse data show lower rates of CVD among diabetic people, but as the prevalence of DM has increased, so has the absolute burden of CVD, especially among middle-aged and older individuals.[37]
- DM accounted for 44% of the new cases of end-stage renal disease (ESRD) in 2007. According to data from the US Renal Data System and BRFSS from 1996 to 2007, the incidence rate of ESRD attributed to DM decreased from 304.5 per 100 000 to 199.1 per 100 000).[38]
- According to NHANES data, the prevalence of diabetic kidney disease has increased from 2.2% in NHANES III to 3.3% in NHANES 2005–2008. These increases were observed in direct proportion to increases in DM.[39]
- HbA_{1c} levels ≥6.5% can be used to diagnose DM.[40] In the population-based ARIC study, HbA_{1c} levels ≥6.5% had a 14-year follow-up, multivariable-adjusted HR of 16.5 (95% CI 14.2–19.1) for diagnosed DM and 1.95 (95% CI 1.53–2.48) for CHD relative to those with HbA_{1c} <5.0%.[41]

- According to data from the ARIC study and NHANES III, the sensitivity and specificity for diagnosing DM (compared with a single fasting glucose measurement of at least 126 mg/dL) were 47% and 98%, respectively.

Risk Factors

- Data from the 2004 National Healthcare Disparities Report (Agency for Healthcare Research and Quality, US Department of Health and Human Services) found that only approximately one third of adults with DM received all 5 interventions to reduce risk factors recommended for comprehensive DM care in 2001. The proportion receiving all 5 interventions was lower among blacks than whites and among Hispanics than non-Hispanic whites.[42]
  - In multivariable models that controlled for age, sex, income, education, insurance, and residence location, blacks were 38% less likely and Hispanics were 33% less likely than their respective comparison groups to receive all recommended risk factor interventions in 2001.[42]
- Between NHANES III 1988–1994 (NCHS) and NHANES 1999–2002 (NCHS), considerable differences were found among ethnic groups in glycemic control rates among adults with type 2 DM. Among non-Hispanic whites, the control rates were 43.8% in 1988 to 1994 and 48.4% in 1999 to 2002. For non-Hispanic blacks, the rates were 41.2% and 36.5%, respectively. For Mexican Americans, the respective rates were 34.5% and 34.2%.[43]
- In 1 large academic medical center, outpatients with type 2 DM were observed during an 18-month period for proportions of patients who had HbA_{1c} levels, BP, or total cholesterol levels measured; who had been prescribed any drug therapy if HbA_{1c} levels, SBP, or LDL cholesterol levels exceeded recommended treatment goals; and who had been prescribed greater-than-starting-dose therapy if these values were above treatment goals. Patients were less likely to have cholesterol levels measured (76%) than HbA_{1c} levels (92%) or BP (99%; $P<0.0001$ for either comparison). The proportion of patients who received any drug therapy was greater for above-goal HbA_{1c} (92%) than for above-goal SBP (78%) or LDL cholesterol (38%; $P<0.0001$ for each comparison). Similarly, patients whose HbA_{1c} levels were above the treatment goal (80%) were more likely to receive greater-than-starting-dose therapy than were those who had above-goal SBP (62%) and LDL cholesterol levels (13%; $P<0.0001$).[44]
  - Data from the same academic medical center also showed that CVD risk factors among women with DM were managed less aggressively than among men with DM. Women were less likely than men to have HbA_{1c} <7% (without CHD: adjusted OR for women versus men 0.84, $P=0.005$; with CHD: 0.63, $P<0.0001$). Women without CHD were less likely than men to be treated with lipid-lowering medication (0.82; $P=0.01$) or, when treated, to have LDL cholesterol levels <100 mg/dL (0.75; $P=0.004$) and were less likely than men to be prescribed aspirin (0.63; $P<0.0001$). Women with DM and CHD were less likely than men to be prescribed aspirin (0.70, $P<0.0001$) and, when treated for hypertension or hyperlipidemia, were less likely to have BP levels <130/80 mm Hg (0.75, $P<0.0001$) or LDL cholesterol levels <100 mg/dL (0.80, $P=0.006$).[45]
- In 2001 to 2002, among adults ≥18 years of age with DM, 50.2% were not at goal for HbA_{1c} (<7%), 64.6% were not at goal for LDL cholesterol (<100 mg/dL), and 53% were not at goal for BP (<130/80 mm Hg). Moreover, 48.6% were not at recommended levels of triglycerides (<150 mg/dL in women). Only 5.3% of men and 12.7% of women were simultaneously at goal for HbA_{1c}, LDL cholesterol, and BP.[46]
- Analysis of data from the CHS of the NHLBI found that lifestyle risk factors, including PA level, dietary habits, smoking habits, alcohol use, and adiposity measures, assessed late in life, were each independently associated with risk of new-onset DM. Participants whose PA level and dietary, smoking, and alcohol habits were all in the low-risk group had an 82% lower incidence of DM than all other participants. When absence of adiposity was added to the other 4 low-risk lifestyle factors, incidence of DM was 89% lower.[47]
- Aggressive treatment of hypertension is recommended for adults with DM to prevent cardiovascular complications. Between NHANES III (1984–1992) and NHANES 1999–2004, the proportion of patients with DM whose BP was treated increased from 76.5% to 87.8%, and the proportion whose BP was controlled nearly doubled (from 15.9% to 29.6%).[48]
- According to 2007 data from the BRFSS, only 25% of adults with DM achieved recommended levels of total PA based on the 2007 American Diabetes Association guidelines.[49]

Hospitalizations

Youth

- Nationwide Inpatient Sample data from 1993 to 2004 were analyzed for individuals 0 to 29 years of age with a diagnosis of DM. Rates of hospitalizations increased by 38%. Hospitalization rates were higher for females (42%) than for males (29%). Inflation-adjusted total charges for DM hospitalizations increased 130%, from $1.05 billion in 1993 to $2.42 billion in 2004.[50]

Hypoglycemia

- Hypoglycemia is a common side effect of DM treatment, typically defined as a blood glucose level <50 mg/dL; severe hypoglycemia is additionally defined as patients needing assistance to treat themselves.
- In the ADVANCE (Action in Diabetes and Vascular Disease: Preterax and Diamicron Modified Release Controlled Evaluation) trial, 2.1% of patients had an episode of severe hypoglycemia. Severe hypoglycemia was associated with an increased risk of major macrovascular events (HR

2.88, 95% CI 2.01–4.12), cardiovascular death (HR 2.68, 95% CI 1.72–4.19), and all-cause death (HR 2.69, 95% CI 1.97–3.67), including nonvascular outcomes. The lack of specificity of hypoglycemia with vascular outcomes suggests that it might be a marker for susceptibility. Risk factors for hypoglycemia included older age, DM duration, worse renal function, lower BMI, lower cognitive function, multiple glucose-lowering medications, and randomization to the intensive glucose control arm.[51]

Cost

- In 2007, the direct ($116 billion) and indirect ($58 billion) cost attributable to DM was $174 billion.[6] These estimates include not just DM as a primary diagnosis but also DM-related long-term complications that are attributed to DM.[52]
- A study of data from NHANES 2003–2006, Ingenix Research DataMart, 2003–2005 NAMCS, the 2003–2005 NHAMCS, the 2004–2005 Nationwide Inpatient Sample, and the 2003–2005 MEPS found that the estimated economic cost of undiagnosed DM in 2007 was $18 billion, including medical costs of $11 billion and indirect costs of $7 billion.[53]
- According to 2003–2005 MEPS data (household component data), reductions in DM and hypertension of 5% could save ≈9 billion dollars annually in the short-term. Longer term, savings could total nearly 25 billion dollars.[54]

Type 1 DM

- Type 1 DM constitutes 5% to 10% of DM in the United States.[55]
- A long-term study of patients with type 1 DM from 1966 showed that risk of mortality was 7 times greater than that of the general population.[56]
- According to 30-year mortality data from Allegheny County, PA, those with type 1 DM have a mortality rate 5.6 times higher than the general population.[57]
- The leading cause of death among patients with type 1 DM is CVD, which accounted for 22% of deaths among the Allegheny County, PA, type 1 DM registry, followed by renal (20%) and infectious (18%) causes.[58]
- Long-term follow-up data from the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Study Research Group showed that intensive versus conventional treatment in the Diabetes Control and Complications Trial was associated with a 42% reduced risk of CVD ($P=0.02$) and a 57% reduced risk of the composite end point ($P=0.02$; included nonfatal MI, stroke, and CVD death).[59]
- Observational data from the Swedish National Diabetes Register showed that most CVD risk factors were more adverse among patients with HbA_{1c} between 8.0% and 11.9% than among those with HbA_{1c} between 5.0% and 7.9%. Per 1% unit increase in HbA_{1c}, the HR of fatal and nonfatal CHD was 1.30 in multivariable adjusted models and 1.27 for fatal and nonfatal CVD. Among patients with HbA_{1c} 8.0% to 11.9% compared with those with HbA_{1c} 5.0% to 7.9%, the HR of fatal/nonfatal CHD was 1.71 and the risk of fatal/nonfatal CVD was 1.59.[60]
- Among 2787 patients from the EURODIAB Prospective Complications Study, age, waist-hip ratio, pulse pressure, non-HDL cholesterol, microalbuminuria, and peripheral and autonomic neuropathy were risk factors for all-cause, CVD, and non-CVD mortality.[61]

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Table 17-1. Diabetes

Population Group	Prevalence of Physician-Diagnosed DM, 2008: Age ≥20 y	Prevalence of Undiagnosed DM, 2008: Age ≥20 y	Prevalence of Prediabetes, 2008: Age ≥20 y	Incidence of Diagnosed DM: Age ≥20 y	Mortality (DM), 2008†: All Ages	Hospital Discharges, 2009 All Ages	Cost, 2007‡
Both sexes	18 300 000 (8.0%)	7 100 000 (3.1%)	81 500 000 (36.8%)	1 600 000§	70 553	688 000	$174 Billion
Males	8 300 000 (7.9%)	4 400 000 (4.1%)	48 100 000 (44.9%)		35 346 (50.1%)*	313 000	
Females	10 000 000 (8.2%)	2 700 000 (2.3%)	33 400 000 (28.8%)		35 207 (49.9%)*	375 000	
NH white males	6.8%	3.9%	45.4%		28 598		
NH white females	6.5%	1.9%	27.9%		27 295		
NH black males	14.3%	4.8%	31.6%		5457		
NH black females	14.7%	4.0%	27.1%		6607		
Mexican American males	11.0%	6.3%	44.9%				
Mexican American females	12.7%	3.8%	34.3%				

DM indicates diabetes mellitus; and NH, non-Hispanic.

Undiagnosed DM is defined as those whose fasting glucose is ≥126 mg/dL but who did not report being told by a healthcare provider that they had DM. Prediabetes is a fasting blood glucose of 100 to <126 mg/dL (impaired fasting glucose); prediabetes includes impaired glucose tolerance.

*These percentages represent the portion of total DM mortality that is for males vs females.

†Mortality data are for whites and blacks and include Hispanics.

‡Centers for Disease Control and Prevention, National Diabetes Fact Sheet, 2011.[6]

Sources: Prevalence: Prevalence of diagnosed and undiagnosed diabetes: National Health and Nutrition Examination Survey 2005–2008, National Center for Health Statistics (NCHS), and National Heart, Lung, and Blood Institute. Percentages for racial/ethnic groups are age-adjusted for Americans ≥20 years of age. Age-specific percentages are extrapolations to the 2008 US population estimates. Incidence: National Institute of Diabetes and Digestive and Kidney Diseases estimates. Mortality: NCHS. These data represent underlying cause of death only. Hospital discharges: National Hospital Discharge Survey, NCHS; data include those inpatients discharged alive, dead, or status unknown.

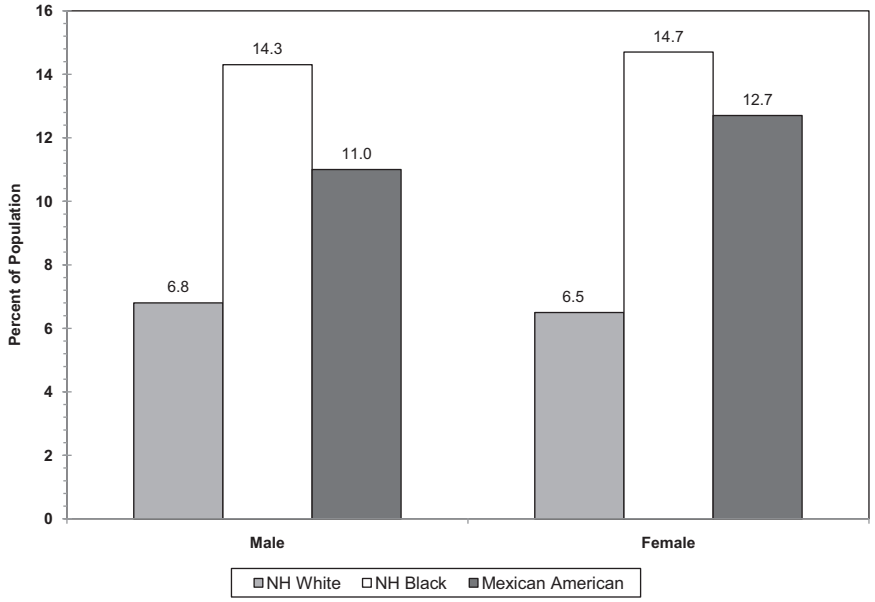

Chart 17-1. Age-adjusted prevalence of physician-diagnosed diabetes mellitus in adults ≥20 years of age by race/ethnicity and sex (National Health and Nutrition Examination Survey: 2005–2008). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

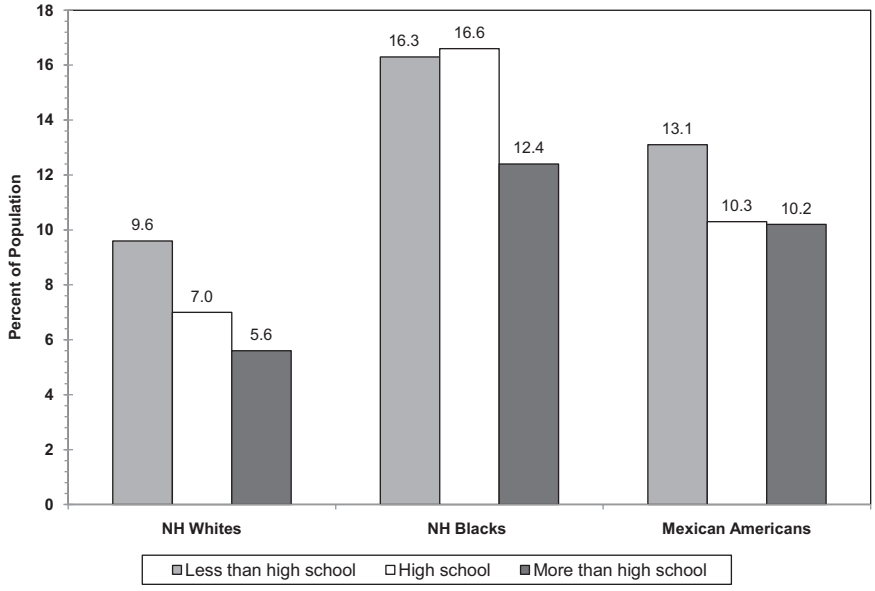

Chart 17-2. Age-adjusted prevalence of physician-diagnosed type 2 diabetes mellitus in adults ≥20 years of age by race/ethnicity and years of education (National Health and Nutrition Examination Survey: 2005–2008). NH indicates non-Hispanic. Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

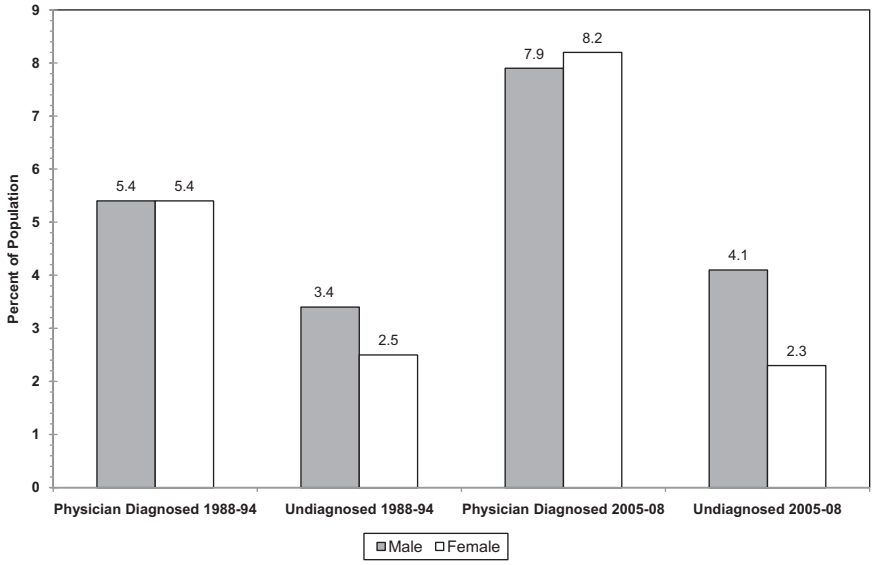

Chart 17-3. Trends in diabetes mellitus prevalence in adults ≥20 years of age by sex (National Health and Nutrition Examination Survey: 1988–1994 and 2005–2008). Source: National Center for Health Statistics, National Heart, Lung, and Blood Institute.

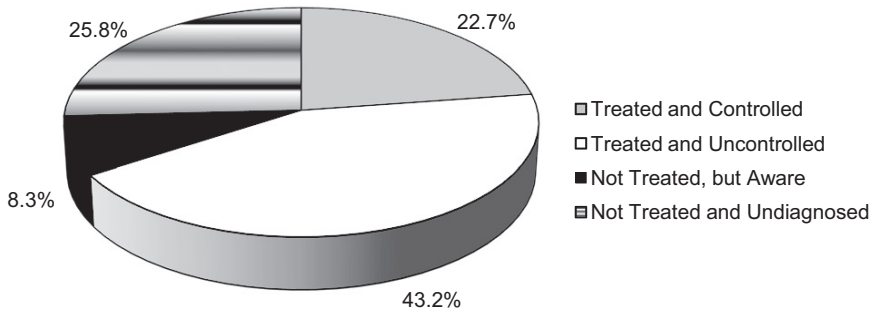

Chart 17-4. Diabetes mellitus awareness, treatment, and control (National Health and Nutrition Examination Survey: 2005–2008). Source: National Heart, Lung, and Blood Institute.

18. End-Stage Renal Disease and Chronic Kidney Disease

ICD-10 N18.0. See Tables 18-1 through 18-3.

ESRD is a condition that is most commonly associated with DM and/or HBP, occurs when the kidneys are functioning at a very low level, and is currently defined as the receipt of chronic renal replacement treatment such as hemodialysis, peritoneal dialysis, or kidney transplantation. The ESRD population is increasing in size and cost as those with CKD transition to ESRD and as a result of changing practice patterns in the United States.

- Data from the 2010 annual report of the US Renal Data System showed that in 2008, the prevalence of ESRD was 547 982, with 70% of these prevalent cases being treated with hemodialysis.[1]
- In 2008, 112 476 new cases of ESRD were reported.[1]
- In 2008, 17 413 kidney transplants were performed.[1]
- Data from a large cohort of insured patients found that in addition to established risk factors for ESRD, lower hemoglobin levels, higher serum uric acid levels, self-reported history of nocturia, and family history of kidney disease are independent risk factors for ESRD.[2]
- Data from a large insured population revealed that among adults with a glomerular filtration rate (GFR) >60 mL · min^{-1} · 1.73 m^{-2} and no evidence of proteinuria or hematuria at baseline, risks for ESRD increased dramatically with higher baseline BP level, and in this same patient population, BP-associated risks were greater in men than in women and in blacks than in whites[3] (Table 18-1).
- Compared with white patients with similar levels of kidney function, black patients are much more likely to progress to ESRD and are on average 10 years younger when they reach ESRD.[4,5]
- Results from a large community-based population showed that higher BMI also independently increased the risk of ESRD. The higher risk of ESRD with overweight and obesity was consistent across age, sex, and race and in the presence or absence of DM, hypertension, or known baseline kidney disease[6] (Table 18-2).

Abbreviations Used in Chapter 18

BMI	body mass index
BP	blood pressure
CHD	coronary heart disease
CHF	congestive heart failure
CI	confidence interval
CKD	chronic kidney disease
CVD	cardiovascular disease
DM	diabetes mellitus
eGFR	estimated glomerular filtration rate
ESRD	end-stage renal disease
GFR	glomerular filtration rate
HBP	high blood pressure
HF	heart failure
ICD-10	*International Classification of Diseases, 10th Revision*
JNC V	fifth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
MI	myocardial infarction
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
PAD	peripheral arterial disease
RR	relative risk

Age, Sex, Race, and Ethnicity

- The median age of the population with ESRD in 2008 varied across different racial/ethnic groups: 57.4 years for blacks, 58.0 years for Native American, 59.3 years for Asians, and 60.6 years for whites.[1]
- Treatment of ESRD is more common in men than in women.[1]
- Blacks, Hispanics, Asian Americans, and Native Americans have significantly higher rates of ESRD than do whites/Europeans. Blacks represent nearly 32% of treated patients with ESRD.[1]

Chronic Kidney Disease

Prevalence

- CKD, defined as reduced GFR, excess urinary protein excretion, or both, is a serious health condition and a worldwide public health problem. The incidence and prevalence of CKD are increasing in the United States and are associated with poor outcomes and a high cost to the US healthcare system. Controversy exists about whether CKD itself independently causes incident CVD, but it is clear that people with CKD, as well as those with ESRD, represent a population at very high risk for CVD events. In fact, individuals with CKD are more likely to die of CVD than to transition to ESRD. The US Renal Data System estimates that by 2020, >700 000 Americans will have ESRD, with >500 000 requiring dialysis and >250 000 receiving a transplant.
- The National Kidney Foundation Kidney Disease Outcome Quality Initiative developed guidelines in 2002 that provided a standardized definition for CKD. Prevalence estimates may differ depending on assumptions used in obtaining estimates, including which equation is used to estimate GFR and methods for measuring proteinuria.[7] The most recent US prevalence estimates of CKD, with the use of Kidney Disease Outcome Quality Initiative guidelines, come from NHANES 1999–2004 (NCHS) in adults ≥20 years of age[8]:
  - The prevalence of CKD (stages I to V)[9] is 16.8%.[8] This represents an increase from the 14.5% prevalence estimate from NHANES 1988–1994 (NCHS; recalculated).[8]
  - The prevalence of GFR ≥90 mL · min^{-1} · 1.73 m^{-2} with kidney damage (ie, presence of albuminuria) is 5.7%.
  - The prevalence of stage II CKD (estimated glomerular filtration rate [eGFR] 60–89 mL · min^{-1} · 1.73 m^{-2} with kidney damage) is 5.4%.
  - The prevalence of stage III CKD (eGFR 30–59 mL · min^{-1} · 1.73 m^{-2}) is 5.4%.

— The prevalence of stages IV and V CKD (eGFR <29 $mL \cdot min^{-1} \cdot 1.73\ m^{-2}$) is 0.4%.

- Nearly 26 million people (13%) in the United States have CKD, and most are undiagnosed.[10] Another 20 million are at increased risk for CKD.[11]

Demographics

- Using current definitions, the prevalence of CKD is higher with older age[1]:
  - — 6.0% for those 20 to 39 years of age
  - — 11.6% for those 40 to 59 years of age
  - — 38.8% for those ≥60 years of age
- CKD prevalence was greater among those with DM (43.8%) and hypertension (29.4%) than among those without these chronic conditions.[1]
- The prevalence of CKD was slightly higher among Mexican Americans (18.7%) and non-Hispanic blacks (19.9%) than among non-Hispanic whites (16.1%). This disparity was most evident for those with stage I CKD; non-Hispanic whites had a CKD prevalence of 4.2% compared with prevalences among Mexican Americans and non-Hispanic blacks of 10.2% and 9.4%, respectively.[8]

Risk Factors

- Many traditional CVD risk factors are also risk factors for CKD, including older age, male sex, hypertension, DM, smoking, and family history of CVD.
- Recent evidence suggests that BMI is associated with worsening CKD.
  - — In a cohort of 652 African American individuals with hypertensive nephrosclerosis, BMI was independently associated with urine total protein and albumin excretion.[12]
- In addition, both the degree of CKD (ie, eGFR) and urine albumin are strongly associated with the progression from CKD to ESRD. In addition, urine albumin level is associated with progression to CKD across all levels of reduced eGFR.[13]
- Other risk factors include systemic conditions such as autoimmune diseases, systemic infections, and drug exposure, as well as anatomically local conditions such as urinary tract infections, urinary stones, lower urinary tract obstruction, and neoplasia. Even after adjustment for these risk factors, excess CVD risk remains.[14]

ESRD/CKD and CVD

- CVD is the leading cause of death among those with ESRD, although the specific cardiovascular cause of death may be more likely to be arrhythmic than an AMI, end-stage heart failure, or stroke.
  - — CVD mortality is 5 to 30 times higher in dialysis patients than in subjects from the general population of the same age, sex, and race.[15,16]
  - — Individuals with less severe forms of kidney disease are also at significantly increased CVD risk independent of typical CVD risk factors.[17]
  - — CKD is a risk factor for recurrent CVD events.[18]
- Studies from a broad range of cohorts demonstrate an association between reduced eGFR and elevated risk of CVD, CVD outcomes, and all-cause death[17,19–24] that appears to be largely independent of other known major CVD risk factors.
- Although clinical practice guidelines recommend management of mineral and bone disorders secondary to CKD, a recent meta-analysis suggests that there is no consistent association between calcium and parathyroid hormone and the risk of death or cardiovascular events.[25]
- Any degree of albuminuria, starting below the microalbuminuria cutpoint, has been shown to be an independent risk factor for cardiovascular events, CHF hospitalization, PAD, and all-cause death in a wide variety of cohorts.[26–31]
  - — A recent meta-analysis of 21 published studies of albuminuria involving 105 872 participants (730 577 person-years) from 14 studies with urine albumin/creatinine ratio measurements and 1 128 310 participants (4 732 110 person-years) from 7 studies with urine dipstick measurements showed that excess albuminuria or proteinuria is independently associated with a higher risk of CVD and all-cause mortality.[32]
  - — People with both albuminuria/proteinuria and reduced eGFR are at particularly high risk for CVD, CVD outcomes, and death.[33]
  - — The exact reasons why CKD and ESRD increase the risk of CVD have not been completely delineated but are clearly multifactorial and likely involve pathological alterations in multiple organ systems and pathways.

Cost: ESRD

- The total annual cost of treating ESRD in the United States was $26.8 billion in 2008, representing nearly 6% of the total Medicare budget.[1]
- The total annual cost associated with CKD has not been determined accurately to date.

Cystatin C: Kidney Function and CVD

Serum cystatin C, another marker of kidney function, has been proposed to be a more sensitive indicator of kidney function than serum creatinine and creatinine-based estimating formulas at higher levels of GFR. It is a low-molecular-weight protein produced at a constant rate by all nucleated cells and appears not to be affected significantly across age, sex, and levels of muscle mass. Cystatin C is excreted by the kidneys, filtered through the glomerulus, and nearly completely reabsorbed by proximal tubular cells.[34] Several equations have been proposed using cystatin C alone and in combination with serum creatinine to estimate kidney function.[35,36]

All-Cause Mortality

Elevated levels of cystatin C have been shown to be associated with increased risk for all-cause mortality in studies from a broad range of cohorts.[37–39]

- In addition to GFR and urine albumin-to-creatinine ratio, cystatin C provides incremental information for the prediction of ESRD and mortality.
  - In a recent analysis of 26 643 US adults, the addition of cystatin C to the combination of creatinine and albumin-to-creatinine ratio resulted in a significant improvement in the prediction of both all-cause mortality and the development of ESRD.[40]

Cardiovascular Disease

- Data from a large national cohort found higher values of cystatin C to be associated with prevalent stroke, angina, and MI,[41] as well as higher BMI.[42]
- Elevated cystatin C was an independent risk factor for HF,[43,44] PAD events,[45] clinical atherosclerosis, and subclinical measures of CVD in older adults,[46] as well as for cardiovascular events among those with CHD.[37,47]
- In several diverse cohorts, elevated cystatin C has been found to be associated with CVD-related mortality,[39,48,49] including sudden cardiac death.[50]

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Table 18-1. BP and the Adjusted Risk of ESRD Among 316 675 Adults Without Evidence of Baseline Kidney Disease

JNC V BP Category	Adjusted RR (95% CI)
Optimal	1.00 (Reference)
Normal, not optimal	1.62 (1.27–2.07)
High normal	1.98 (1.55–2.52)
Hypertension	
Stage 1	2.59 (2.07–3.25)
Stage 2	3.86 (3.00–4.96)
Stage 3	3.88 (2.82–5.34)
Stage 4	4.25 (2.63–6.86)

BP indicates blood pressure; ESRD, end-stage renal disease; JNC V, fifth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; RR, relative risk; and CI, confidence interval.

Table 18-2. Multivariable Association Between BMI and Risk of ESRD Among 320 252 Adults

BMI, kg/m^2	Adjusted RR (95% CI)
18.5–24.9 (Normal weight)	1.00 (Reference)
25.0–29.9 (Overweight)	1.87 (1.64–2.14)
30.0–34.9 (Class I obesity)	3.57 (3.05–4.18)
35.0–39.9 (Class II obesity)	6.12 (4.97–7.54)
≥40.0 (Extreme obesity)	7.07 (5.37–9.31)

BMI indicates body mass index; ESRD, end-stage renal disease; RR, relative risk; and CI, confidence interval.

Table 18-3. Adjusted Hazard Ratio (95% CI) for Death of Any Cause, Cardiovascular Events, and Hospitialization Among 1 120 295 Ambulatory Adults, According to the Estimated GFR*

Estimated GFR, $mL \cdot min^{-1} \cdot 1.73\ m^{-2}$	Death of Any Cause	Any Cardiovascular Event	Any Hospitalization
≥60†	1.00	1.00	1.00
45–59	1.2 (1.1–1.2)	1.4 (1.4–1.5)	1.1 (1.1–1.1)
30–44	1.8 (1.7–1.9)	2.0 (1.9–2.1)	1.5 (1.5–1.5)
15–29	3.2 (3.1–3.4)	2.8 (2.6–2.9)	2.1 (2.0–2.2)
<15	5.9 (5.4–6.5)	3.4 (3.1–3.8)	3.1 (3.0–3.3)

CI indicates confidence interval; GFR, glomerular filtration rate.

*The analyses were adjusted for age, sex, income, education, use or nonuse of dialysis, and presence or absence of prior coronary heart disease, prior chronic heart failure, prior ischemic stroke or transient ischemic attack, prior peripheral arterial disease, diabetes mellitus, hypertension, dyslipidemia, a serum albumin level of ≤3.5 g/dL, dementia, cirrhosis or chronic liver disease, chronic lung disease, documented proteinuria, and prior hospitalizations.

†This group served as the reference group.

19. Metabolic Syndrome

- Metabolic syndrome refers to a cluster of risk factors for CVD and type 2 DM. Although several different definitions for metabolic syndrome have been proposed, the International Diabetes Federation, NHLBI, AHA, and others recently proposed a harmonized definition for metabolic syndrome.[1] By this definition, metabolic syndrome is diagnosed when ≥3 of the following 5 risk factors are present (most but not all people with DM will be classified as having metabolic syndrome by this definition because they will have at least 2 other factors besides the glucose criterion; many will prefer to separate those with DM into a separate group for risk stratification or treatment purposes):
  - Fasting plasma glucose ≥100 mg/dL or undergoing drug treatment for elevated glucose.
  - HDL cholesterol <40 mg/dL in men or <50 mg/dL in women or undergoing drug treatment for reduced HDL cholesterol.
  - Triglycerides ≥150 mg/dL or undergoing drug treatment for elevated triglycerides.
  - Waist circumference ≥102 cm in men or ≥88 cm in women in the United States.
  - BP ≥130 mm Hg systolic or ≥85 mm Hg diastolic or undergoing drug treatment for hypertension or antihypertensive drug treatment in a patient with a history of hypertension.
- Identification of metabolic syndrome represents a call to action for the healthcare provider and patient to address the underlying lifestyle-related risk factors, including abdominal obesity, physical inactivity, and atherogenic diet, as well as clinical management to address the characteristic atherogenic dyslipidemia, elevated BP, elevated glucose, and/or prothrombotic state that are common to people with metabolic syndrome. A multidisciplinary team of healthcare professionals is desirable to adequately address these multiple issues in patients with the metabolic syndrome.[2]

Abbreviations Used in Chapter 19

AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities
BMI	body mass index
BP	blood pressure
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
DM	diabetes mellitus
FRS	Framingham Risk Score
HDL	high-density lipoprotein
HR	hazard ratio
HF	heart failure
LDL	low-density lipoprotein
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart, Lung, and Blood Institute
OR	odds ratio
PA	physical activity
RR	relative risk

Adults

The following estimates include many of those who have DM, in addition to those with metabolic syndrome without DM.

- Prevalence of metabolic syndrome varies by the definition used, with definitions such as that from the International Diabetes Federation that suggest lower thresholds for defining central obesity in European whites, Asians, and Hispanics resulting in higher prevalence estimates.[3]
- On the basis of NHANES 2003–2006 data and National Cholesterol Education Program/Adult Treatment Panel III guidelines, ≈34% of adults ≥20 years of age met the criteria for metabolic syndrome.[4]
- Also based on NHANES 2003–2006 data[4]:
  - The age-adjusted prevalence was 35.1% for men and 32.6% for women.
  - Among men, the age-specific prevalence ranged from 20.3% among people 20 to 39 years of age to 40.8% for people 40 to 59 years of age and 51.5% for people ≥60 years of age. Among women, the age-specific prevalence ranged from 15.6% among people 20 to 39 years of age to 37.2% for people 40 to 59 years of age and 54.4% for those ≥60 years of age.
  - The age-adjusted prevalences of people with metabolic syndrome were 37.2%, 25.3%, and 33.2% for non-Hispanic white, non-Hispanic black, and Mexican American men, respectively. Among women, the percentages were 31.5%, 38.8%, and 40.6%, respectively.
  - The age-adjusted prevalence was ≈53% higher among non-Hispanic black women than among non-Hispanic black men and ≈22% higher among Mexican American women than among Mexican American men.
- The prevalence of metabolic syndrome is also high among immigrant Asian Indians, ranging between 26.8% and 38.2% depending on the definition used.[5]
- The prevalence of metabolic syndrome among pregnant women increased to 26.5% during 1999–2004 from 17.8% during 1988 to 1994.[6]
- Despite its prevalence, the public's recognition of metabolic syndrome is limited.[7]

Children/Adolescents

- An AHA scientific statement about metabolic syndrome in children and adolescents was released in 2009.[8]
- Metabolic syndrome should be diagnosed with caution in children and adolescents, because metabolic syndrome categorization in adolescents is not stable. Approximately half of the 1098 adolescent participants in the Princeton School District Study diagnosed with pediatric Adult Treatment Panel III metabolic syndrome lost the diagnosis over 3 years of follow-up.[9]

- Additional evidence of the instability of the diagnosis of metabolic syndrome in children exists. In children 6 to 17 years of age participating in research studies in a single clinical research hospital, the diagnosis of metabolic syndrome was unstable in 46% of cases after a mean of 5.6 years of follow-up.[10]
- On the basis of NHANES 1999–2002 data, the prevalence of metabolic syndrome in adolescents 12 to 19 years of age was 9.4%, which represents ≈2.9 million people. It was 13.2% in boys, 5.3% in girls, 10.7% in whites, 5.2% in blacks, and 11.1% in Mexican Americans.[11]
- In 1999 to 2004, ≈4.5% of US adolescents 12 to 17 years of age had metabolic syndrome according to the definition developed by the International Diabetes Federation.[12] In 2006, this prevalence would have represented ≈1.1 million adolescents 12 to 17 years of age with metabolic syndrome. It increased from 1.2% among those 12 to 13 years of age to 7.1% among those 14 to 15 years of age and was higher among boys (6.7%) than girls (2.1%). Furthermore, 4.5% of white adolescents, 3.0% of black adolescents, and 7.1% of Mexican American adolescents had metabolic syndrome. The prevalence of metabolic syndrome remained relatively stable during successive 2-year periods: 4.5% for 1999 to 2000, 4.4% to 4.5% for 2001 to 2002, and 3.7% to 3.9% for 2003 to 2004.
- In 1999 to 2002, among overweight or obese adolescents, 44% had metabolic syndrome.[11] In 1988 to 1994, two thirds of all adolescents had at least 1 metabolic abnormality.[13]
- Of 31 participants in the NHLBI Lipid Research Clinics Princeton Prevalence Study and the Princeton Follow-Up Study who had metabolic syndrome at baseline, 21 (68%) had metabolic syndrome 25 years later.[14] After adjustment for age, sex, and race, the baseline status of metabolic syndrome was significantly associated with an increased risk of having metabolic syndrome during adulthood (OR 6.2, 95% CI 2.8–13.8).
- In the Bogalusa Heart Study, 4 variables (BMI, homeostasis model assessment of insulin resistance, ratio of triglycerides to HDL cholesterol, and mean arterial pressure) considered to be part of the metabolic syndrome clustered together in blacks and whites and in children and adults.[15] The degree of clustering was stronger among adults than children. The clustering of rates of change in the components of the metabolic syndrome in blacks exceeded that in whites.
- Cardiovascular abnormalities are associated with metabolic syndrome in children and adolescents.[16,17]

Risk

Adults

- Consistent with 2 earlier meta-analyses, a recent meta-analysis of prospective studies concluded that metabolic syndrome increased the risk of developing CVD (summary RR 1.78, 95% CI 1.58–2.00).[18] The risk of CVD tended to be higher in women (summary RR 2.63) than in men (summary RR 1.98; $P=0.09$). On the basis of results from 3 studies, metabolic syndrome remained a predictor of cardiovascular events after adjustment for the individual components of the syndrome (summary RR 1.54, 95% CI 1.32–1.79). A more recent meta-analysis among 87 studies comprising 951 083 subjects showed an even higher risk of CVD associated with metabolic syndrome (summary RR 2.35, 95% CI 2.02–2.73), with significant increased risks (RRs ranging from 1.6 to 2.9) for all-cause mortality, CVD mortality, MI, and stroke, as well as for those with metabolic syndrome without DM.[19]
- In one of the earlier studies among US adults, mortality follow-up of the second NHANES showed a stepwise increase in risk of CHD, CVD, and total mortality across no disease, metabolic syndrome (without DM), DM, prior CVD, and those with CVD and DM, with an HR for CHD mortality of 2.02 (95% CI 1.42–2.89) associated with metabolic syndrome. Increases in risk were also seen across the number of metabolic syndrome risk factors.[20]
- Several studies suggest that the FRS is a better predictor of incident CVD than metabolic syndrome.[21–23] In the San Antonio Heart Study, the area under the receiver-operating characteristic curve was 0.816 for the FRS and 0.811 for the FRS plus the metabolic syndrome.[21] Furthermore, the sensitivity for CVD at a fixed specificity was significantly higher for the FRS than for the metabolic syndrome. In ARIC, metabolic syndrome did not improve the risk prediction achieved by the FRS.[22] In the British Regional Heart Study, the area under the receiver-operating characteristic curve for the FRS was 0.73 for incident CHD during 10 years of follow-up, and the area under the receiver-operating characteristic curve for the number of metabolic syndrome components was 0.63.[23] For CHD events during 20 years of follow-up, the areas under the receiver-operating characteristic curves were 0.68 for the FRS and 0.59 for the number of metabolic syndrome components.
- Estimates of relative risk for CVD generally increase as the number of components of metabolic syndrome increases.[23] Compared with men without an abnormal component in the Framingham Offspring Study, the HRs for CVD were 1.48 (95% CI 0.69–3.16) for men with 1 or 2 components and 3.99 (95% CI 1.89–8.41) for men with ≥3 components.[24] Among women, the HRs were 3.39 (95% CI 1.31–8.81) for 1 or 2 components and 5.95 (95% CI 2.20–16.11) for ≥3 components. Compared with men without a metabolic abnormality in the British Regional Heart Study, the HRs were 1.74 (95% CI 1.22–2.39) for 1 component, 2.34 (95% CI 1.65–3.32) for 2 components, 2.88 (95% CI 2.02–4.11) for 3 components, and 3.44 (95% CI 2.35–5.03) for 4 or 5 components.[23]
- The cardiovascular risk associated with the metabolic syndrome varies on the basis of the combination of metabolic syndrome components present. Of all possible ways to have 3 metabolic syndrome components, the combination of central obesity, elevated BP, and hyperglycemia conferred the greatest risk for CVD (HR 2.36, 95% CI 1.54–3.61) and mortality (HR 3.09, 95% CI 1.93–4.94) in the Framingham Offspring Study.[25]
- Data from the Aerobics Center Longitudinal Study indicate that risk for CVD mortality is increased in men without

DM who have metabolic syndrome (HR 1.8, 95% CI 1.5–2.0); however, among those with metabolic syndrome, the presence of DM is associated with even greater risk for CVD mortality (HR 2.1, 95% CI 1.7–2.6).[26] Analysis of data from NCHS was used to determine the number of disease-specific deaths attributable to all nonoptimal levels of each risk factor exposure by age and sex. The results of the analysis of dietary, lifestyle, and metabolic risk factors show that targeting a handful of risk factors has large potential to reduce mortality in the United States.[27]

- In addition to CVD, the metabolic syndrome has also been associated with incident AF[28] and HF.[29]
- The metabolic syndrome is associated with increased healthcare use and healthcare-related costs among individuals with and without DM. Overall, healthcare costs increase by ≈24% for each additional metabolic syndrome component present.[30]

Children

- Few prospective pediatric studies have examined the future risk for CVD or DM according to baseline metabolic syndrome status. Data from 771 participants 6 to 19 years of age from the NHLBI's Lipid Research Clinics Princeton Prevalence Study and the Princeton Follow-Up Study showed that the risk of developing CVD was substantially higher among those with metabolic syndrome than among those without this syndrome (OR 14.6, 95% CI 4.8–45.3) who were followed up for 25 years.[14]
- Another analysis of 814 participants of this cohort showed that those 5 to 19 years of age who had metabolic syndrome at baseline had an increased risk of having DM 25 to 30 years later compared with those who did not have the syndrome at baseline (OR 11.5, 95% CI 2.1–63.7).[31]
- Additional data from the Princeton Follow-Up Study, the Fels Longitudinal Study, and the Muscatine Study suggest that the absence of components of the metabolic syndrome in childhood had a high negative predictive value for the development of metabolic syndrome or DM in adulthood.[32]

Risk Factors

- In prospective or retrospective cohort studies, the following factors have been reported as being directly associated with incident metabolic syndrome, defined by 1 of the major definitions: age,[31,33–35] low educational attainment,[33,36] low socioeconomic status,[37] smoking,[36–39] low levels of PA,[36–42] low levels of physical fitness,[40,43–45] intake of soft drinks,[46] intake of diet soda,[47] magnesium intake,[48] energy intake,[42] carbohydrate intake,[33,38,49] total fat intake,[33,49] Western dietary pattern,[47] meat intake,[47] intake of fried foods,[47] heavy alcohol consumption,[50] abstention from alcohol use,[33] parental history of DM,[31] long-term stress at work,[51] pediatric metabolic syndrome,[31] obesity or BMI,[33,34,38,42,52] childhood obesity,[53] waist circumference,[35,49,54–57] intra-abdominal fat,[58] gain in weight or BMI,[33,59] change in weight or BMI,[35,38,60] weight fluctuation,[61] BP,[35,49,56,62] heart rate,[63] homeostasis model assessment,[54,64] fasting insulin,[54] 2-hour insulin,[54] proinsulin,[54] fasting glucose or hyperglycemia,[35,54,56] 2-hour glucose,[54] impaired glucose tolerance,[54] triglycerides,[35,49,52,54–56] low HDL cholesterol,[35,49,53,54,56] oxidized LDL,[64] uric acid,[60,65] γ-glutamyltransferase,[60,66,67] alanine transaminase,[60,66,68,69] plasminogen activator inhibitor-1,[70] aldosterone,[70] leptin,[71] C-reactive protein,[72,73] adipocyte–fatty acid binding protein,[74] and free testosterone index.[75]
- The following factors have been reported as being inversely associated with incident metabolic syndrome, defined by 1 of the major definitions, in prospective or retrospective cohort studies: muscular strength,[76] change in PA or physical fitness,[38,43] alcohol intake,[36,42] Mediterranean diet,[77] dairy consumption,[47] insulin sensitivity,[54] ratio of aspartate aminotransferase to alanine transaminase,[68] total testosterone,[75,78,79] sex hormone–binding globulin,[75,78,79] and Δ5-desaturase activity.[80]
- Furthermore, men were more likely than women to develop metabolic syndrome,[33,35] and blacks were shown to be less likely to develop metabolic syndrome than whites.[33]

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20. Nutrition

See Tables 20-1 and 20-2 and Charts 20-1 through 20-3.

This chapter of the update highlights national nutritional intake data, focusing on foods, nutrients, dietary patterns, and other dietary factors that are related to cardiometabolic health. It is intended to examine current intakes, trends and changes in intakes, and estimated effects on disease to support and further stimulate efforts to monitor and improve dietary habits in relation to cardiovascular health.

Prevalence

Foods and Nutrients: Adults

See Table 20-1; NHANES 2005–2008; personal communication with D. Mozaffarian (July 2011).

The dietary consumption by US adults of selected foods and nutrients related to cardiometabolic health is detailed in Table 20-1 according to sex and race or ethnic subgroups:

- Average consumption of whole grains by white and black men and women was between 0.5 and 0.8 servings per day, with only between 3% and 5% of white and black adults meeting guidelines of ≥3 servings per day. Average whole grain consumption by Mexican Americans was ≈2 servings per day, with 21% to 27% consuming ≥3 servings per day.
- Average fruit consumption ranged from 1.1 to 1.8 servings per day in these sex and race or ethnic subgroups; 9% to 11% of whites, 6% to 7% of blacks, and 8% to 10% of Mexican Americans met guidelines of ≥2 cups per day. When 100% fruit juices were included, the number of servings consumed and the proportions of adults consuming ≥2 cups per day approximately doubled.
- Average vegetable consumption ranged from 1.3 to 2.2 servings per day; 6% to 7% of whites, 3% of blacks, and 3% of Mexican Americans consumed ≥2 ½ cups per day. The inclusion of vegetable juices and sauces generally produced little change in these consumption patterns.
- Average consumption of fish and shellfish was lowest among white women (1.2 servings per week) and highest among black men and women (1.7 servings per week); ≈75% to 80% of all adults in each sex and race or ethnic subgroup consumed <2 servings per week. Approximately 10% to 13% of whites, 14% to 15% of blacks, and 12% of Mexican Americans consumed ≥250 mg of eicosapentaenoic acid and docosahexaenoic acid per day.
- Average consumption of nuts, legumes, and seeds was ≈2 to 3 servings per week among white and black men and women and 6 servings per week among Mexican American men and women. Approximately 20% of whites, 15% of blacks, and 40% of Mexican Americans met guidelines of ≥4 servings per week.
- Average consumption of processed meats was lowest among Mexican American women (1.8 servings per week) and highest among black men (3.6 servings per week). Between 36% (Mexican American women) and 66% (black men) of adults consumed ≥1 serving per week.
- Average consumption of sugar-sweetened beverages ranged from ≈7 servings per week among white women to 16 servings per week among Mexican American men. Approximately 50% and 33% of white men and women, 73% and 65% of black men and women, and 76% and 62% of Mexican American men and women, respectively, consumed >36 oz (4.5 8-oz servings) per week.
- Average consumption of sweets and bakery desserts ranged from ≈4 servings per day (Mexican American men) to 7 servings per day (white men). Approximately two thirds of white and black men and women and half of all Mexican American men and women consumed >2.5 servings per week.
- Between 33% and 50% of adults in each sex and race or ethnic subgroup consumed <10% of total calories from saturated fat, and between 58% and 70% consumed <300 mg of dietary cholesterol per day.
- Only 4% to 7% of whites, 2% to 4% of blacks, and 9% to 11% of Mexican Americans consumed ≥28 g of dietary fiber per day.
- Only 8% to 11% of whites, 9% to 11% of blacks, and 13% to 19% of Mexican Americans consumed <2.3 g of sodium per day. In 2005, the US Department of Health and Human Services and US Department of Agriculture recommended

Abbreviations Used in Chapter 20

ALA	α-linoleic acid
BMI	body mass index
BP	blood pressure
BRFSS	Behavioral Risk Factor Surveillance System
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	diastolic blood pressure
DHA	docosahexaenoic acid
DM	diabetes mellitus
EPA	eicosapentaenoic acid
GISSI	Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico
GFR	glomerular filtration rate
HD	heart disease
HDL	high-density lipoprotein
HEI	Healthy Eating Index
LDL	low-density lipoprotein
NA	not available
NH	non-Hispanic
NHANES	NAtional Health and Nutrition Examination Survey
PA	physical activity
PREMIER	Prospective Registry Evaluating Myocardial Infarction: Events and Recovery
PUFA	polyunsaturated fatty acid
RR	relative risk
SBP	systolic blood pressure
SD	standard deviation
WHI	Women's Health Initiative

that adults in specific groups, including people with hypertension, all middle-aged and older adults, and all blacks, should consume ≤1.5 g of sodium per day. In 2005 to 2006, the majority (69.2%) of US adults belonged to ≥1 of these specific groups in whom sodium consumption should be <1.5 g/d.[1]

Foods and Nutrients: Children and Teenagers

See Table 20-2; NHANES 2005–2008; personal communication with D. Mozaffarian (July 2011).

The dietary consumption by US children and teenagers of selected foods and nutrients related to cardiometabolic health is detailed in Table 20-2:

- Average whole grain consumption was low, ranging from 0.4 to 0.6 servings per day, with <4% of all children in different age and sex subgroups meeting guidelines of ≥3 servings per day.
- Average fruit consumption was low and decreased with age: ≈1.5 servings per day in younger boys and girls (5–9 years of age), 1.3 servings per day in adolescent boys and girls (10–14 years of age), and 0.9 servings per day in teenage boys and girls (15–19 years of age). The proportion meeting guidelines of ≥2 cups per day was also low and decreased with age: ≈8% in those 5 to 9 years of age, 7% to 8% in those 10 to 14 years of age, and 4% in those 15 to 19 years of age. When 100% fruit juices were included, the number of servings consumed approximately doubled or tripled, and proportions consuming ≥2 cups per day were 29% to 36% of those 5 to 9 years of age, 22% to 26% of those 10 to 14 years of age, and 21% to 22% of those 15 to 19 years of age.
- Average vegetable consumption was low, ranging from 0.9 to 1.1 servings per day, with <2% of children in different age and sex subgroups meeting guidelines of ≥2 ½ cups per day.
- Average consumption of fish and shellfish was low, ranging between 0.5 and 0.7 servings per week in all age and sex groups. Among all ages, only 10% to 13% of children and teenagers consumed ≥2 servings per week.
- Average consumption of nuts, legumes, and seeds ranged from 1.3 to 1.4 servings per week among 5- to 9-year-olds, 1.4 to 2.1 servings per week among 10- to 14-year-olds, and 0.8 to 1.1 servings per week among 15- to 19-year-olds. Only between 7% and 14% of children in different age and sex subgroups consumed ≥4 servings per week.
- Average consumption of processed meats ranged from 2.1 to 3.2 servings per week; was uniformly higher than the average consumption of nuts, legumes, and seeds; and was up to 6 times higher than the average consumption of fish and shellfish. Between 40% and 54% of children consumed ≥2 servings per week.
- Average consumption of sugar-sweetened beverages was higher in boys than in girls and was ≈8 servings per week in 5- to 9-year-olds, 11 to 13 servings per week in 10- to 14-year-olds, and 14 to 18 servings per week in 15- to 19-year-olds. This was generally considerably higher than the average consumption of whole grains, fruits, vegetables, fish and shellfish, or nuts, legumes, and seeds. Only between 17% (boys 15–19 years of age) and 42% (boys and girls 5–9 years of age) of children consumed <4.5 servings per week.
- Average consumption of sweets and bakery desserts was ≈8 to 10 servings per week in 5- to 9-year-olds and 10- to 14-year-olds and 6 to 8 servings per week in 15- to 19-year-olds. From 82% (girls 5–9 years of age) to 58% (boys 15–19 years of age) of youths consumed >2.5 servings per week.
- Average consumption of eicosapentaenoic acid and docosahexaenoic acid was low, ranging from ≈45 to 75 mg/d in boys and girls at all ages. Only between 3% and 7% of children and teenagers at all ages consumed ≥250 mg/d.
- Average consumption of saturated fat was between 11% and 12% of calories, and average consumption of dietary cholesterol was ≈230 mg/d. Approximately one fifth to one third of children consumed <10% energy from saturated fat, and ≈80% consumed <300 mg of dietary cholesterol per day.
- Average consumption of dietary fiber ranged from 12 to 14 g/d. Less than 2% of children in all different age and sex subgroups consumed ≥28 g/d.
- Average consumption of sodium ranged from 3.1 to 3.4 g/d. Between 7% and 12% of children in different age and sex subgroups consumed <2.3 g/d.

Energy Balance

Energy balance, or consumption of total calories appropriate for needs, is determined by the balance of average calories consumed versus expended, with this balance depending on multiple factors, including calories consumed, PA, body size, age, sex, and underlying basal metabolic rate. Thus, one individual may consume relatively high calories but have negative energy balance (as a result of even greater calories expended), whereas another individual may consume relatively few calories but have positive energy balance (because of low calories expended). Given such variation, the most practical and reasonable method to assess energy balance in populations is to assess changes in weight over time (Trends section).

- Average daily caloric intake in the United States is ≈2500 calories in adult men and 1800 calories in adult women (Table 20-1). In children and teenagers, average caloric intake is higher in boys than in girls and increases with age in boys (Table 20-2). Trends in energy balance are described below. The average US adult gains approximately 1 lb per year. In an analysis of >120 000 US men and women followed up for up to 20 years, changes in intakes of different foods and beverages were linked to long-term weight in very different ways.[2] Foods and beverages most strongly linked to weight gain included potatoes, sugar-sweetened beverages, processed meats, red meats, refined grains (eg, white bread, low-fiber breakfast cereals), and sweets/desserts. In contrast, increasing the intake of several foods was linked to relative weight loss over time, including nuts, whole grains, fruits, vegetables, and yogurt.
- Other nutritional determinants of positive energy balance (more calories consumed than expended), as determined by adiposity or weight gain, include larger portion sizes[3,4] and

greater consumption of fast food and commercially prepared meals.[5–9]

- Preferences for portion size are associated with BMI, socioeconomic status, eating in fast food restaurants, and television watching.[10,11] Portion sizes are larger at fast food restaurants than at home or at other restaurants.[12]
- In 1999 to 2000, 41% of US adults consumed ≥3 commercially prepared meals per week.[6] Between 1999 and 2004, 53% of Americans consumed an average of 1 to 3 restaurant meals per week, and 23% consumed ≥4 restaurant meals per week.[13] Spending on food away from home, including restaurant meals, catered foods, and food eaten during out-of-town trips, increased from 26% of average annual food expenditures in 1970% to 42% in 2004.[13] Macronutrient composition of the overall diet or of specific foods, such as percent calories from total fat, does not appear to be strongly associated with energy balance as ascertained by weight gain or loss.[2,14–16] In contrast, dietary quality, as characterized by higher or lower intakes of specific foods and beverages, is strongly linked to weight gain (above).[2]
- Preliminary evidence suggests that consumption of *trans* fat may be associated with energy imbalance as assessed by changes in adiposity or weight, as well as more specific adverse effects on visceral adiposity, but such data are still emerging.[17–19]
- Other individual factors associated with positive energy balance (weight gain) include greater television watching (particularly as related to greater food consumption)[2,20–24] and lower average sleep duration.[2,25]
- Randomized controlled trials of weight loss in obese individuals generally show modestly greater weight loss with low-carbohydrate versus low-fat diets at 6 months, but at 1 year, such differences diminish, and a diet that focuses on dietary quality and whole foods may be most successful.[26–28]
- A comparison of BRFSS data in 1996 and 2003 suggested a shift in self-reported dietary strategies to lose weight, with the proportion focusing on calorie restriction increasing from 11.3% to 24.9% and the proportion focusing on restricting fat consumption decreasing from 41.6% to 29.1%.[29]
- A 2007 to 2008 national survey of 1082 retail stores in 19 US cities found that energy-dense snack foods/beverages were present in 96% of pharmacies, 94% of gas stations, 22% of furniture stores, 16% of apparel stores, and 29% to 65% of other types of stores.[30]
- Societal and environmental factors independently associated with energy imbalance (weight gain), via either increased caloric consumption or decreased expenditure, include education, income, race/ethnicity, and local conditions such as availability of grocery stores, types of restaurants, safety, parks and open spaces, and walking or biking paths.[31–33] PA is covered in Chapter 15 of this update.

Dietary Patterns

In addition to individual foods and nutrients, overall dietary patterns can be used to assess more global dietary quality. Different dietary patterns have been defined, including the Healthy Eating Index (HEI), Alternative HEI, Western versus prudent dietary patterns, Mediterranean dietary pattern, and DASH-type diet.

- In 1999 to 2004, only 19.4% of hypertensive US adults were following a DASH-type diet (based on intake of fiber, magnesium, calcium, sodium, potassium, protein, total fat, saturated fat, and cholesterol). This represented a decrease from 26.7% of hypertensive US adults in 1988 to 1994.[34]
- Among older US adults (≥60 years of age) in 1999 to 2002, 72% met guidelines for dietary cholesterol intake, but only between 18% and 32% met guidelines for the HEI food groups (meats, dairy, fruits, vegetables, and grains). On the basis of the HEI score, only 17% of older US adults consumed a good-quality diet. Higher HEI scores were seen in white adults and individuals with greater education; lower HEI scores were seen in black adults and smokers.[35]
- Nearly 75 000 women 38 to 63 years of age in the Nurses' Health Study without a history of CVD or DM were followed up from 1984 to 2004. It was found that a greater adherence to the Mediterranean diet, as reflected by a higher Alternate Mediterranean Diet Score, was associated with a lower risk of incident CHD and stroke in women.[36]

Dietary Supplements

Use of dietary supplements is common in the United States among both adults and children:

- More than half (53%) of US adults in 2003 to 2006 used dietary supplements, with the most common supplement being multivitamins and multiminerals (40% of men and women reporting use).[37] It has been shown that most supplements are taken daily and for at least 2 years. Supplement use was associated with older age, higher education, greater PA, wine intake, lower BMI, and white race.[38]
- One third (32%) of US children (birth to 18 years of age) used dietary supplements in 1999 to 2002, with the highest use (48.5%) occurring among 4- to 8-year-olds. The most common supplements were multivitamins and multiminerals (58% of supplement users). The primary nutrients supplemented (either by multivitamins and/or individual vitamins) included vitamin C (29% of US children), vitamin A (26%), vitamin D (26%), calcium (21%), and iron (19%). Supplement use was associated with higher family income, a smoke-free home environment, lower child BMI, and less screen time (television, video games, or computers).[39]
- In a 2005 to 2006 telephone survey of US adults, 41.3% were making or had made in the past a serious weight-loss attempt. Of these, one third (33.9%) had used a dietary supplement for weight loss, with such use being more common in women (44.9%) than in men (19.8%) and in blacks (48.7%) or Hispanics (41.6%) than in whites (31.2%); in those with high school education or less (38.4%) than in those with some college or more (31.1%); and in those with household income <$40 000 per year (41.8%) than in those with higher incomes (30.3%).[40]

- Multiple trials of most dietary supplements, including folate, vitamin C, and vitamin E, have generally shown no significant effect on CVD risk. The major exceptions are long-chain omega-3 fatty acids (fish oil), for which 3 large randomized controlled trials that included populations with and without established HD have shown significant reductions in risk of CVD events at doses of 1 to 2 g/d (Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico [GISSI]-Prevenzione, Japan Eicosapentaenoic Acid Lipid Intervention Study, and GISSI-HF).[41–43] A few other smaller trials of fish oil have not shown significant effects on CVD risk, perhaps related to insufficient statistical power.[44] Another multicenter randomized trial conducted in a population with diabetic nephropathy found that B vitamin supplementation containing folic acid (2.5 mg/d), vitamin B_6 (25 mg), and vitamin B_{12} (1 mg/d) resulted in a greater decrease in GFR and an increase in MI and stroke compared with placebo.[45]

Trends

Energy Balance

Energy balance, or consumption of total calories appropriate for needs, has been steadily worsening in the United States over the past several decades, as evidenced by the dramatic increases in overweight and obesity among both children and adults across broad cross sections of sex, race/ethnicity, geographic residence, and socioeconomic status.[46,47]

- Although trends in total calories consumed are difficult to quantify exactly because of differing methods of serial national dietary surveys over time, multiple lines of evidence indicate that average total energy consumption has increased by at least 200 kcal/d per person in the past 3 decades.
- Data from NHANES indicate that between 1971 and 2004, average total energy consumption among US adults increased by 22% in women (from 1542–1886 kcal/d) and by 10% in men (from 2450–2693 kcal/d; Chart 20-1). These increases are supported by data from the Nationwide Food Consumption Survey (1977–1978) and the Continuing Surveys of Food Intake (1989–1998).[12]
- The increases in calories consumed during this time period are attributable primarily to greater average carbohydrate intake, particularly of starches, refined grains, and sugars (Foods and Nutrients section). Other specific changes related to increased caloric intake in the United States include larger portion sizes, greater food quantity and calories per meal, and increased consumption of sugar-sweetened beverages, snacks, commercially prepared (especially fast food) meals, and higher-energy-density foods.[6,12,48–52]
- Between 1977 to 1978 and 1994 to 1996, the average portion sizes for nearly all foods increased at fast food outlets, other restaurants, and home. These included a 33% increase in the average portion of Mexican food (from 408 to 541 calories), a 34% increase in the average portion of cheeseburgers (from 397 to 533 calories), a 36% increase in the average portion of French fries (from 188 to 256 calories), and a 70% increase in the average portion of salty snacks such as crackers, potato chips, pretzels, puffed rice cakes, and popcorn (from 132 to 225 calories).[12]
- Among US children 2 to 7 years of age, an estimated energy imbalance of only 110 to 165 kcal/d (the equivalent of one 12- to 16-oz bottle of soda/cola) was sufficient to account for the excess weight gain between 1988 to 1994 and 1999 to 2002.[53]

Foods and Nutrients

Several changes in foods and nutrients have occurred over time. Selected changes are highlighted:

Macronutrients

- Starting in 1977 and continuing until the most recent dietary guidelines revision in 2005, a major focus of US dietary guidelines was reduction of total dietary fat.[52] During this time, average total fat consumption declined as a percent of calories from 36.9% to 33.4% in men and from 36.1% to 33.8% in women (Chart 20-1).
- Dietary guidelines during this time also emphasized carbohydrate consumption (eg, as the base of the Food Guide Pyramid),[54] which increased from 42.4% to 48.2% of calories in men and from 45.4% to 50.6% of calories in women (Chart 20-1). Evaluated as absolute intakes, the increase in total calories consumed during this period was attributable primarily to the greater consumption of carbohydrates, both as foods (starches and grains) and as beverages.[55,56]

Sugar-Sweetened Beverages

- Between 1965 and 2002, the average percentage of total calories consumed from beverages in the United States increased from 11.8% to 21.0% of energy, which represents an overall absolute increase of 222 cal/d per person.[51] This increase was largely caused by increased consumption of sugar-sweetened beverages and alcohol: Average consumption of fruit juices went from 20 to 39 kcal/d; of milk, from 125 to 94 kcal/d; of alcohol, from 26 to 99 kcal/d; of sweetened fruit drinks, from 13 to 38 kcal/d; and of soda/cola, from 35 to 143 kcal/d (Chart 20-2).
- In addition to increased overall consumption, the average portion size of a single sugar-sweetened beverage increased by >50% between 1977 and 1996, from 13.1 to 19.9 fl oz.[12]
- Among children and teenagers (2–19 years of age), the largest increases in consumption of sugar-sweetened beverages between 1988 to 1994 and 1999 to 2004 were seen among black and Mexican American youths compared with white youths.[52]

Fruits and Vegetables

- Between 1994 and 2005, the average consumption of fruits and vegetables declined slightly, from a total of 3.4 to 3.2 servings per day. The proportions of men and women consuming combined fruits and vegetables ≥5 times per day were low (≈20% and 29%, respectively) and did not change during this period.[57]

Morbidity and Mortality

Effects on Cardiovascular Risk Factors

In randomized controlled trials, dietary habits affect multiple cardiovascular risk factors, including both established risk

factors (SBP, DBP, LDL cholesterol levels, HDL cholesterol levels, glucose levels, and obesity/weight gain) and novel risk factors [eg, inflammation, cardiac arrhythmias, endothelial cell function, triglyceride levels, lipoprotein(a) levels, and heart rate]:

- A DASH dietary pattern with low sodium reduced SBP by 7.1 mm Hg in adults without hypertension and by 11.5 mm Hg in adults with hypertension.[58]
- Compared with the low-fat DASH diet, DASH-type diets that increased consumption of either protein or unsaturated fat had similar or greater beneficial effects on CVD risk factors. Compared with a baseline usual diet, each of the DASH-type diets, which included various percentages (27%–37%) of total fat and focused on whole foods such as fruits, vegetables, whole grains, and fish, as well as potassium and other minerals and low sodium, reduced SBP by 8 to 10 mm Hg, DBP by 4 to 5 mm Hg, and LDL cholesterol by 12 to 14 mg/dL. The diets that had higher levels of protein and unsaturated fat also lowered triglyceride levels by 16 and 9 mg/dL, respectively.[59]
- In a meta-analysis of randomized controlled trials, consumption of 1% of calories from *trans* fat in place of saturated fat, monounsaturated fat, or polyunsaturated fat increased the ratio of total to HDL cholesterol by 0.031, 0.054, and 0.67; increased apolipoprotein B levels by 3, 10, and 11 mg/L; decreased apolipoprotein A-1 levels by 7, 5, and 3 mg/L; and increased lipoprotein(a) levels by 3.8, 1.4, and 1.1 mg/L, respectively.[60]
- In meta-analyses of randomized controlled trials, consumption of eicosapentaenoic acid and docosahexaenoic acid for ≥12 weeks lowered SBP by 2.1 mm Hg[61] and lowered resting heart rate by 2.5 beats per minute.[62]
- In a pooled analysis of 25 randomized trials totaling 583 men and women both with and without hypercholesterolemia, nut consumption significantly improved blood lipid levels.[63] For a mean consumption of 67 g/d of nuts, total cholesterol was reduced by 10.9 mg/dL (5.1%), LDL cholesterol by 10.2 mg/dL (7.4%), and the ratio of total cholesterol to HDL-cholesterol by 0.24 (5.6% change; $P<0.001$ for each). Triglyceride levels were also reduced by 20.6 mg/dL (10.2%) in subjects with high triglycerides (≥150 mg/dL). Different types of nuts had similar effects.[63] A review of cross-sectional and prospective cohort studies suggests that higher intake of sugar-sweetened beverages is associated with greater visceral fat and higher risk of type 2 DM.[64] In the PREMIER study, a prospective analysis of the 810 participants indicated that a reduction in sugar-sweetened beverages of 1 serving per day was associated with a reduction in SBP of 1.8 mm Hg (95% CI 1.2–2.4) and a reduction in DBP of 1.1 mm Hg (95% CI 0.7–1.4).[65]
- In a randomized controlled trial, compared with a low-fat diet, 2 Mediterranean dietary patterns that included either virgin olive oil or mixed nuts lowered SBP by 5.9 and 7.1 mm Hg, plasma glucose by 7.0 and 5.4 mg/dL, fasting insulin by 16.7 and 20.4 pmol/L, the homeostasis model assessment index by 0.9 and 1.1, and the ratio of total to HDL cholesterol by 0.38 and 0.26 and raised HDL cholesterol by 2.9 and 1.6 mg/dL, respectively. The Mediterranean dietary patterns also lowered levels of C-reactive protein, interleukin-6, intercellular adhesion molecule-1, and vascular cell adhesion molecule-1.[66]

Effects on Cardiovascular Outcomes

Because dietary habits affect a broad range of established and novel risk factors, estimation of the impact of nutritional factors on cardiovascular health by considering only a limited number of pathways (eg, only effects on lipids, BP, and obesity) will systematically underestimate or even misconstrue the actual total impact on health. Randomized controlled trials and prospective observational studies can better quantify the total effects of dietary habits on clinical outcomes:

- In the WHI randomized clinical trial (n=48 835), reduction of total fat consumption from 37.8% energy (baseline) to 24.3% energy (at 1 year) and 28.8% energy (at 6 years) had no effect on incidence of CHD (RR 0.98, 95% CI 0.88–1.09), stroke (RR 1.02, 95% CI 0.90–1.15), or total CVD (RR 0.98, 95% CI 0.92–1.05) over a mean of 8.1 years.[67] This was consistent with null results of 4 prior randomized clinical trials (below) and multiple large prospective cohort studies (below) that indicated little effect of total fat consumption on risk of CVD.[68]
- In 3 separate meta-analyses of prospective cohort studies, the largest of which included 21 studies with up to 2 decades of follow-up, saturated fat consumption overall had no significant association with incidence of CHD, stroke, or total CVD.[69–71] However, in a pooled individual-level analysis of 11 prospective cohort studies, the specific exchange of polyunsaturated fat consumption in place of saturated fat was associated with lower CHD risk, with 13% lower risk for a 5% energy exchange (RR 0.87, 95% CI 0.70–0.97).[72] These findings are consistent with a meta-analysis of randomized controlled trials in which increased polyunsaturated fat consumption in place of saturated fat reduced CHD risk, with 10% lower risk for a 5% energy exchange (RR 0.90, 95% CI 0.83–0.97).[73]
- In a pooled analysis of individual-level data from 11 prospective cohort studies in the United States, Europe, and Israel that included 344 696 participants, each 5% higher energy consumption of carbohydrate in place of saturated fat was associated with a 7% higher risk of CHD (RR 1.07, 95% CI 1.01–1.14).[72] Each 5% higher energy consumption of monounsaturated fat in place of saturated fat was not significantly associated with CHD risk.[72]
- In a meta-analysis of prospective cohort studies, each 2% of calories from *trans* fat was associated with a 23% higher risk of CHD (RR 1.23, 95% CI 1.11–1.37).[74]
- In meta-analyses of prospective cohort studies, each daily serving of fruits or vegetables was associated with a 4% lower risk of CHD (RR 0.96, 95% CI 0.93–0.99) and a 5% lower risk of stroke (RR 0.95, 95% CI 0.92–0.97).[75,76]
- In a meta-analysis of prospective cohort studies, greater whole grain intake (2.5 compared with 0.2 servings per day) was associated with a 21% lower risk of CVD events (RR 0.79, 95% CI 0.73–0.85), with similar estimates for specific CVD outcomes (HD, stroke, fatal CVD) and in sex-specific analyses. In contrast, refined grain intake was

not associated with lower risk of CVD (RR 1.07, 95% CI 0.94–1.22).[77]

- In a meta-analysis of 16 prospective cohort studies that included 326 572 generally healthy individuals in Europe, the United States, China, and Japan, fish consumption was associated with significantly lower risk of CHD mortality.[78] Compared with no consumption, an estimated 250 mg of long-chain omega-3 fatty acids per day was associated with 35% lower risk of CHD death ($P<0.001$). In a meta-analysis of 17 prospective cohort studies and 3 case-control studies that included >1.2 million participants from multiple countries, consumption of unprocessed red meat was not significantly associated with incidence of CHD or DM. In contrast, each 50-g serving per day of processed meats (eg, sausage, bacon, hot dogs, deli meats) was associated with higher incidence of both CHD (RR 1.42, 95% CI 1.07–1.89) and DM (RR 1.19, 95% CI 1.11–1.27).[79]
- In a meta-analysis of 6 prospective observational studies, higher consumption of nuts was associated with significantly lower incidence of CHD (comparing higher to low intake: RR 0.70, 95% CI 0.57–0.82).[70]
- Higher consumption of dairy or milk products is associated with lower incidence of DM and trends toward lower risk of stroke.[70,80,81] Some limited evidence suggests that these associations are stronger for low-fat dairy or milk than for other dairy products. Dairy consumption is unassociated with risk of CHD.[70,81]
- Higher estimated consumption of dietary sodium was not associated with lower CVD mortality in NHANES,[82] although such findings may be limited by changes in behaviors that result from underlying risk (reverse causation). In a post hoc analysis of the Trials of Hypertension Prevention, participants randomized to low-sodium interventions had a 25% lower risk of CVD (RR 0.75, 95% CI 0.57–0.99) after 10 to 15 years of follow-up after the original trials.[83]
- Among 88 520 generally healthy women in the Nurses' Health Study who were 34 to 59 years of age in 1980 and were followed up from 1980 to 2004, regular consumption of sugar-sweetened beverages was independently associated with higher incidence of CHD, with 23% and 35% higher risk with 1 and ≥2 servings per day, respectively, compared with <1 per month.[84] Among the 15 745 participants in the ARIC study, the OR for developing CKD was 2.59 for participants who had a serum uric acid level >9.0 mg/dL and who drank >1 sugar-sweetened soda per day.[85]
- In a cohort of 380 296 US men and women, greater versus lower adherence to a Mediterranean dietary pattern, characterized by higher intakes of vegetables, legumes, nuts, fruits, whole grains, fish, and unsaturated fat and lower intakes of red and processed meat, was associated with a 22% lower cardiovascular mortality (RR 0.78, 95% CI 0.69–0.87).[86] In a cohort of 72 113 US female nurses, a dietary pattern characterized by higher intakes of vegetables, fruits, legumes, fish, poultry, and whole grains was associated with a 28% lower cardiovascular mortality (RR 0.72, 95% CI 0.60–0.87), whereas a dietary pattern characterized by higher intakes of processed meat, red meat, refined grains, French fries, and sweets/desserts was associated with a 22% higher cardiovascular mortality (RR 1.22, 95% CI 1.01–1.48).[87] Similar findings have been seen in other cohorts and for other outcomes, including development of DM and metabolic syndrome.[88–92]
- In one report that used consistent and comparable risk assessment methods and nationally representative data, the mortality effects in the United States of 12 modifiable dietary, lifestyle, and metabolic risk factors were assessed. High dietary salt consumption was estimated to be responsible for 102 000 annual deaths, low dietary omega-3 fatty acids for 84 000 annual deaths, high dietary *trans* fatty acids for 82 000 annual deaths, and low consumption of fruits and vegetables for 55 000 annual deaths.[93]

Cost

The US Department of Agriculture forecast that the Consumer Price Index for all food would increase 4.5% to 5.5% in 2008 as retailers continued to pass on higher commodity and energy costs to consumers in the form of higher retail prices. The Consumer Price Index for food increased 4.0% in 2007, the highest annual increase since 1990. Prices for foods eaten at home increased 4.2% in 2007, whereas prices for foods eaten away from home increased by 3.6%.[52]

- The proportion of total US food expenditures for meals outside the home, as a share of total food dollars, increased from 25% in 1957 to 38% in 1977 to 49% in 2007[54] (Chart 20-3).
- The proportion of sales of meals and snacks from fast food restaurants compared with total meals and snacks away from home increased from 5% in 1958 to 28% in 1977 to 37% in 2007.[94]
- As a proportion of income, food has become less expensive over time in the United States. As a share of personal disposable income, average (mean) total food expenditures by families and individuals have decreased from 23.5% (1947) to 18.4% (1957) to 13.4% (1977) to 9.8% (2007). For any given year, the share of disposable income spent on food is inversely proportional to absolute income: The share increases as absolute income levels decline.[94]
- Among 154 forms of fruits and vegetables priced with ACNielsen Homescan data, more than half were estimated to cost 25 cents per serving. Consumers could meet a recommendation of 3 servings of fruits and 4 servings of vegetables daily for a total cost of 64 cents per day.[94]
- An overview of the costs of various strategies for primary prevention of CVD determined that the estimated costs per year of life gained were between $9800 and $18 000 for statin therapy, ≥$1500 for nurse screening and lifestyle advice, $500 to $1250 for smoking cessation, and $20 to $900 for population-based healthy eating.[95]
- Each year, >$33 billion in medical costs and $9 billion in lost productivity resulting from HD, cancer, stroke, and DM are attributed to poor nutrition.[96–100]

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Table 20-1. Dietary Consumption (Mean±SD) in 2005–2008 Among US Adults ≥20 Years of Age of Selected Foods and Nutrients Related to Cardiometabolic Health[96–99]

	NH White Men		NH White Women		NH Black Men		NH Black Women		Mexican American Men		Mexican American Women	
	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*
Foods												
Whole grains, servings/d	0.7±0.7	4.6	0.8±0.7	5.3	0.5±0.5	3.1	0.6±0.6	4.1	2.1±1.6	27.4	1.7±1.4	21.0
Fruits, servings/d	1.3±1.3	8.9	1.6±1.4	10.7	1.1±1.4	6.2	1.2±1.3	7.0	1.4±1.3	8.0	1.8±1.5	10.4
Fruits including 100% juices, servings/d	2.4±2.3	23.3	2.5±2.1	24.9	2.8±0.9	26.6	2.9±1.2	26.7	2.8±2.3	23.9	3.2±2.6	28.7
Vegetables including starch, servings/d	1.9±1.1	6.3	2.2±1.1	7.2	1.6±0.9	3.2	1.8±1.0	3.6	1.3±0.7	2.6	1.6±0.7	2.5
Vegetables including starch and juices/ sauces, servings/d	2.2±1.2	8.8	2.5±1.3	9.6	1.7±0.9	3.9	1.9±1.1	4.9	1.7±0.8	3.9	1.9±0.7	4.8
Fish and shellfish, servings/wk	1.5±1.4	22.0	1.2±0.6	19.1	1.7±1.3	23.0	1.7±0.9	25.2	1.6±1.3	20.0	1.4±1.3	19.7
Nuts, legumes, and seeds, servings/wk	2.7±2.0	20.3	2.4±1.9	19.9	2.3±1.5	15.9	1.8±0.4	14.5	6.3±6.8	41.2	5.8±3.6	39.9
Processed meats, servings/wk	3.2±1.9	46.1	2.0±1.0	60.1	3.6±2.2	43.8	2.7±2.2	52.1	2.1±2.2	60.8	1.8±2.2	64.2
Sugar-sweetened beverages, servings/wk	9.9±11.6	50.0	6.6±10.9	66.7	13.8±9.0	27.2	11.8±8.9	34.8	15.6±10.3	24.2	10.0±8.8	37.9
Sweets and bakery desserts, servings/wk	6.5±4.8	35.4	7.4±4.5	32.2	6.0±4.1	42.0	6.8±3.5	38.6	3.7±2.9	55.1	5.5±0.9	48.4
Nutrients												
Total calories, kcal/d	2520±659	NA	1757±455	NA	2371±722	NA	1749±568	NA	2400±703	NA	1798±528	NA
EPA/DHA, g/d	0.129±0.138	13.0	0.109±0.138	10.2	0.146±0.131	15.2	0.146±0.102	13.8	0.146±0.102	12.1	0.119±0.102	12.0
ALA, g/d	1.35±0.33	25.5	1.52±0.50	72.2	1.32±0.38	23.4	1.43±0.33	68.0	1.21±0.23	16.5	1.34±0.27	64.1
n-6 PUFA, % energy	7.1±1.2	NA	7.5±1.6	NA	7.3±1.6	NA	7.6±1.5	NA	6.7±0.9	NA	6.9±1.4	NA
Saturated fat, % energy	11.4±2.2	33.3	11.4±2.1	36.0	10.8±1.7	39.8	10.6±2.0	43.3	10.1±2.0	50.4	10.4±1.7	48.5
Dietary cholesterol, mg/d	277±90	66.9	274±83	69.5	303±123	61.8	317±106	57.7	323±142	58.8	310±120	61.2
Total fat, % energy	34.1±5.1	54.2	33.9±4.7	53.6	33.8±4.7	51.1	33.5±4.6	54.2	31.6±5.1	65.7	31.8±4.9	65.4
Carbohydrate, % energy	47.3±7.2	NA	49.7±6.6	NA	48.6±6.2	NA	50.7±6.3	NA	50.3±6.7	NA	52.3±6.5	NA
Dietary fiber, g/d	15.0±5.0	4.2	17.2±5.8	7.0	13.1±4.6	2.4	14.2±5.0	3.8	17.7±6.1	9.3	18.9±4.7	11.2
Sodium, g/d	3.3±0.6	10.5	3.5±0.6	8.3	3.2±0.5	11.3	3.4±0.5	8.9	3.0±0.7	19.4	3.2±0.6	12.7

SD indicates standard deviation; NH, non-Hispanic; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; ALA, α-linoleic acid; n-6-PUFA, ω-6-polyunsaturated fatty acid; and NA, not available.

Based on data from NHANES 2005–2006 and 2007–2008, derived from two 24-hour dietary recalls per person, with population SDs adjusted for within-person vs between-person variation. All values are energy-adjusted using individual regressions or percent energy, and for comparability, means and proportions are reported for a 2000-kcal/d diet. To obtain actual mean consumption levels, the group means for each food or nutrient can be multiplied by the group-specific total calories (kcal/d) divided by 2000 kcal/d.

*Guidelines adjusted to a 2000-kcal/d diet. Whole grains (characterized as minimum 1.1 g of fiber per 10 g of carbohydrate), 3 or more 1-oz equivalent (1 oz of bread; 1 cup of dry cereal; 1/2 cup of cooked rice, pasta, or cereal) servings per day (Dietary Guidelines for Americans[98]); fish or shellfish, 2 or more 100-g (3.5-oz) servings per week[98]; fruits, 2 cups per day[99]; vegetables, 2 1/2 cups per day, including up to 3 cups per week of starchy vegetables[99]; nuts, legumes, and seeds, 4 or more 50-g servings per week[98]; processed meats (bacon, hot dogs, sausage, processed deli meats), 2 or fewer 100-g (3.5-oz) servings per week (1/4 of discretionary calories)[99]; sugar-sweetened beverages (defined as ≥50 cal/8 oz, excluding whole juices), ≤36 oz per week (≈1/4 of discretionary calories) [98,99]; sweets and bakery desserts, 2.5 or fewer 50-g servings per week (≈1/4 of discretionary calories)[98,99]; EPA/DHA, ≥0.250 g/d[101]; ALA, ≥1.6/1.1 g/d (men/women)[100]; saturated fat, <10% energy[99]; dietary cholesterol, <300 mg/d[99]; total fat, 20% to 35% energy[99]; dietary fiber, ≥28/d[99]; and sodium, <2.3 g/d.[99]

Table 20-2. Dietary Consumption (Mean±SD) in 2005–2008 Among US Children and Teenagers of Selected Foods and Nutrients Related to Cardiometabolic Health

	Boys (5–9 y)		Girls (5–9 y)		Boys (10–14 y)		Girls (10–14 y)		Boys (15–19 y)		Girls (15–19 y)	
	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*	Average Consumption	% Meeting Guidelines*
Foods												
Whole grains, servings/d	0.5±0.5	2.7	0.5±0.3	1.1	0.6±0.6	4.0	0.5±0.4	1.8	0.4±0.4	1.6	0.5±0.5	2.6
Fruits, servings/d	1.5±1.1	8.3	1.5±0.8	8.5	1.2±1.0	6.9	1.4±1.1	8.4	0.9±0.8	3.9	0.9±0.8	4.6
Fruits including 100% juices, servings/d	3.3±1.7	35.7	3.1±1.4	28.5	2.4±1.7	21.7	2.8±1.9	26.0	2.2±1.7	21.1	2.4±1.7	21.7
Vegetables including starch, servings/d	0.9±0.4	0.5	1.0±0.5	0.9	1.0±0.6	1.1	1.1±0.6	1.6	1.0±0.1	1.1	1.1±0.2	1.1
Vegetables including starch and juices/ sauces, servings/d	1.1±0.4	0.8	1.1±0.6	1.3	1.1±0.6	1.2	1.3±0.6	1.9	1.3±0.9	1.5	1.3±0.4	2.4
Fish and shellfish, servings/wk	0.5±0.7	9.9	0.7±0.7	11.7	0.9±0.7	13.5	0.6±0.7	10.3	0.7±0.9	11.0	0.7±0.9	12.2
Nuts, legumes, and seeds, servings/wk	1.4±0.4	12.5	1.3±2.5	9.6	2.1±2.8	14.1	1.4±1.0	10.1	1.1±1.0	9.4	0.8±1.0	6.8
Processed meats, servings/wk	2.3±1.1	55.5	2.1±1.0	60.0	2.6±1.0	54.9	2.3±1.0	52.1	3.2±1.5	45.6	2.4±1.0	55.8
Sugar-sweetened beverages, servings/wk	8.5±5.9	38.6	8.3±5.1	37.9	13.3±7.0	23.5	10.9±7.3	31.6	18.2±11.1	16.7	13.9±10.1	32.4
Sweets and bakery desserts, servings/wk	10.1±2.1	19.5	9.3±2.1	18.3	9.0±2.1	23.5	8.1±2.0	30.2	6.0±5.2	41.9	8.2±5.2	31.5
Nutrients												
Total calories, kcal/d	1946±328	NA	1743±330	NA	2139±403	NA	1849±432	NA	2670±903	NA	1845±453	NA
EPA/DHA, g/d	0.045±0.025	3.1	0.056±0.025	5.9	0.074±0.030	7.3	0.052±0.030	4.7	0.071±0.022	5.2	0.065±0.021	5.7
ALA, g/d	1.12±0.15	9.5	1.15±0.20	46.3	1.11±0.20	9.7	1.19±0.28	49.1	1.14±0.18	13.2	1.34±0.18	59.2
n-6 PUFA, % energy	6.4±1.1	NA	6.5±1.0	NA	6.5±1.0	NA	6.7±0.9	NA	6.4±0.6	NA	7.1±1.3	NA
Saturated fat, % energy	11.7±1.4	24.9	11.8±0.8	21.3	11.6±0.7	27.8	11.5±1.8	28.6	11.8±1.4	24.8	11.3±1.7	34.1
Dietary cholesterol, mg/d	225±69	81.6	239±57	78.4	245±57	76.6	226±114	81.6	244±114	76.9	240±114	77.7
Total fat, % energy	33.0±3.3	67.6	33.3±3.0	66.6	33.1±2.6	65.6	33.1±4.2	61.6	33.6±3.1	58.9	33.3±4.9	57.0
Carbohydrate, % energy	54.5±4.1	NA	53.8±3.7	NA	53.1±3.6	NA	53.8±4.9	NA	51.4±4.1	NA	52.9±6.3	NA
Dietary fiber, g/d	13.6±2.3	0.2	13.9±2.2	0.7	13.3±3.4	1.1	13.9±3.3	1.8	11.9±2.4	0.6	13.3±2.9	1.9
Sodium, g/d	3.1±0.3	8.2	3.2±0.4	8.7	3.2±0.2	9.8	3.3±0.2	7.4	3.2±0.4	11.9	3.4±0.5	9.1

SD indicates standard deviation; NA, not available; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; ALA, α-linoleic acid; and n-6-PUFA, ω-6-polyunsaturated fatty acid.

Based on data from NHANES 2005–2006 and 2007–2008, derived from two 24-hour dietary recalls per person, with population SDs adjusted for within-person vs between-person variation. All values are energy-adjusted using individual regressions or percent energy, and for comparability, means and proportions are reported for a 2000-kcal/d diet. To obtain actual mean consumption levels, the group means for each food or nutrient can be multiplied by the group-specific total calories (kcal/d) divided by 2000 kcal/d.

*See Table 20-1 for food group, serving size, and guideline definitions. For different age and sex subgroups here, the guideline cutpoints are standardized to a 2000-kcal/d diet to account for differences in caloric intake in these groups.

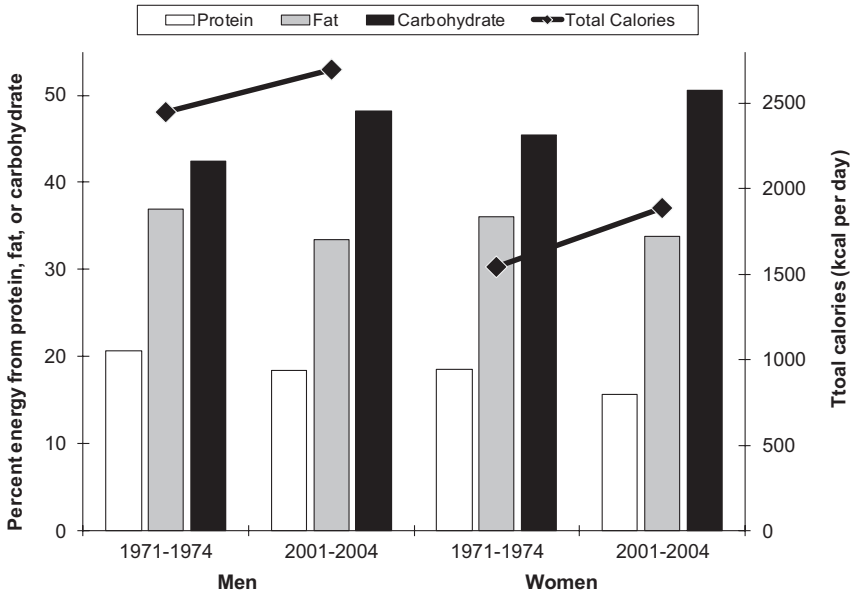

Chart 20-1. Age-adjusted trends in macronutrients and total calories consumed by US adults (20–74 years of age), 1971–2004. Data derived from National Center for Health Statistics.[13]

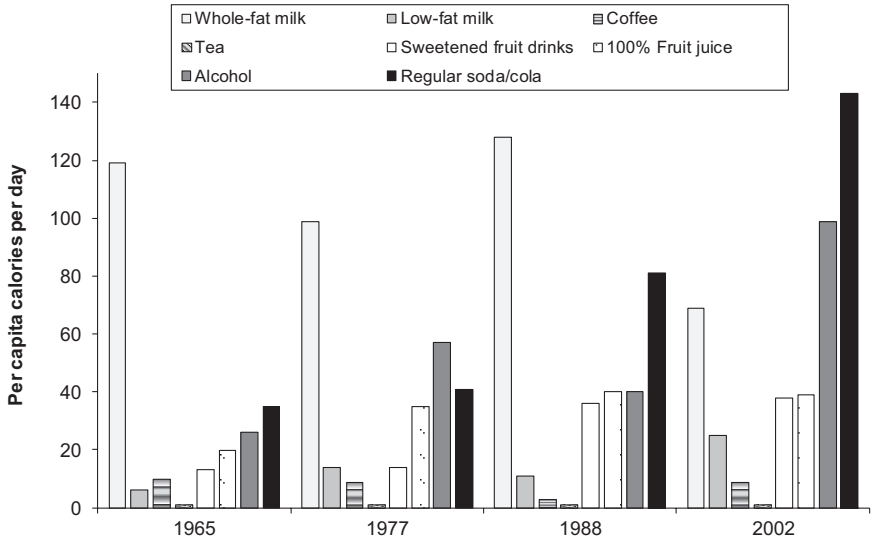

Chart 20-2. Per capita calories consumed from different beverages by US adults (19 years of age), 1965–2002. Data derived from Nationwide Food Consumption Surveys (1965, 1977–1978), National Health and Nutrition Examination Survey (1988–1994, 1999–2002), and Duffey and Popkin.[51]

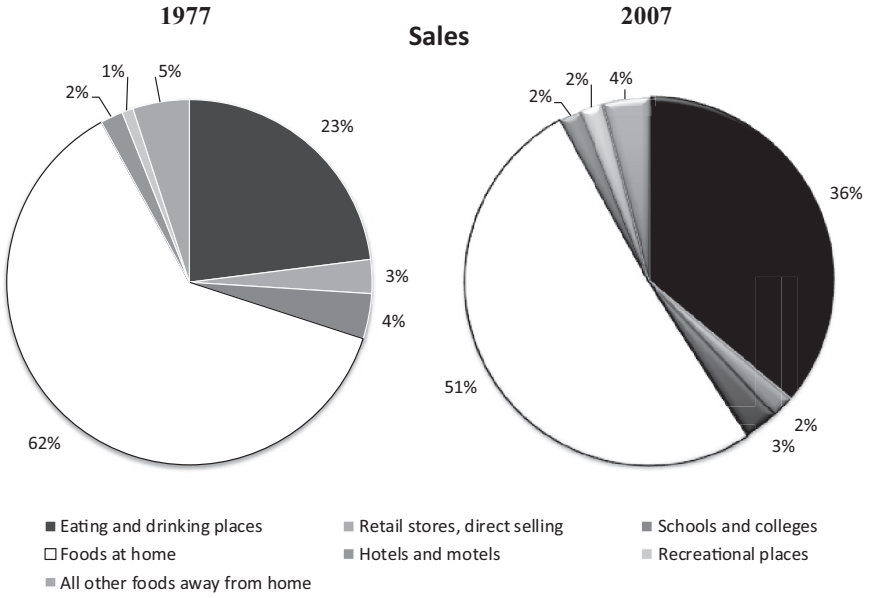

Chart 20-3. Total US food expenditures away from home and at home, 1977 and 2007. Data derived from US Department of Agriculture Economic Research Service.[54]

21. Quality of Care

See Tables 21-1 through 21-13.

The Institute of Medicine defines quality of care as "the degree to which health services for individuals and populations increase the likelihood of desired health outcomes and are consistent with current professional knowledge."[1] The Institute of Medicine has defined 6 specific domains for improving health care, including care that is safe, effective, patient-centered, timely, efficient, and equitable.

In the following sections, data on quality of care will be presented based on the 6 domains of quality as defined by the Institute of Medicine. This is intended to highlight current care and to stimulate efforts to improve the quality of cardiovascular care nationally. Where possible, data are reported from recently published literature or standardized quality indicators from quality-improvement registries (ie, those consistent with the methods for quality performance measures endorsed by the ACC and the AHA).[2] Additional data on aspects of quality of care, such as adherence to ACC/AHA clinical practice guidelines, are also included to provide a spectrum of quality-of-care data. The data selected are meant to provide examples of the current quality of care as reflected by the Institute of Medicine domain and are not meant to be comprehensive given the sheer number of publications yearly.

- The *safety domain* has been defined as avoiding injuries to patients from the care that is intended to help them. The following are several publications that have focused on safety issues:
  - — In a small, single-center study conducted over a 2-month period in the cardiac care unit of a tertiary center, Rahim et al[3] demonstrated that iatrogenic adverse events were common (99 of 194 patients), of which bleeding (27%) was the most common preventable iatrogenic adverse event.
  - — Using the National Cardiovascular Data Registry Cath-PCI registry, Tsai et al[4] found that almost one fourth of dialysis patients undergoing PCI (n=22 778) received a contraindicated antithrombotic agent, specifically enoxaparin, eptifibatide, or both. Patients who received a contraindicated antithrombotic agent had an increased risk of in-hospital bleeding (OR 1.63, 95% CI 1.35–1.98) and a trend toward increased mortality (OR 1.15, 95% CI 0.97–1.36).[4]
  - — Using data from the Acute Coronary Treatment and Intervention Outcomes Registry-GWTG (ACTION Registry-GWTG), Mathews and colleagues developed a contemporary model to stratify in-hospital bleeding risk for patients after STEMI and NSTEMI.[5] The 12 factors associated with major bleeding in the model were heart rate, baseline hemoglobin, female sex, baseline serum creatinine, age, electrocardiographic changes, HF or shock, DM, PAD, body weight, SBP, and home warfarin use. The risk model discriminated well in the derivation (C statistic=0.73) and the validation (C statistic=0.71) cohorts, and the risk score for major bleeding corresponded well with observed bleeding.[5]

Abbreviations Used in Chapter 21

ACC	American College of Cardiology
ACEI	angiotensin-converting enzyme inhibitor
ACS	acute coronary syndrome
ACTION	Acute Coronary Treatment and Intervention Outcomes Registry
ADP	adenosine diphosphate
AHA	American Heart Association
AMI	acute myocardial infarction
ARB	angiotensin receptor blocker
BMI	body mass index
BP	blood pressure
CABG	coronary artery bypass grafting
CAD	coronary artery disease
CI	confidence interval
COURAGE	Clinical Outcomes Utilizing Revascularization and AGgressive drug Evaluation trial
CPR	cardiopulmonary resuscitation
CRUSADE	Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the ACC/AHA Guidelines
D2B	door-to-balloon
DM	diabetes mellitus
DVT	deep venous thrombosis
ECG	electrocardiogram
ED	emergency department
EF	ejection fraction
EMS	emergency medical services
GP	glycoprotein
GWTG	Get With The Guidelines
HbA_{1c}	hemoglobin A_{1c}
HF	heart failure
HIQR	Hospital Inpatient Quality Reporting Program
IV	intravenous
LDL	low-density lipoprotein
LV	left ventricular
LVEF	left ventricular ejection fraction
LVSD	left ventricular systolic dysfunction
N/A	not applicable
NHANES	National Health and Nutrition Examination Survey
NM	not measured
NSTEMI	non–ST-elevation myocardial infarction
OR	odds ratio
PAD	peripheral arterial disease
PCI	percutaneous coronary intervention
ROC	Resuscitation Outcomes Consortium
RR	relative risk
SBP	systolic blood pressure
SCD-HeFT	Sudden Cardiac Death in Heart Failure Trial
SD	standard deviation
STEMI	ST-elevation myocardial infarction
tPA	tissue-type plasminogen activator
UFH	unfractionated heparin
VF	ventricular fibrillation
VHA	Veterans Health Administration

— In a random sample of medical and surgical long-term care adult patients in Massachusetts hospitals, López et al[6] assessed the association between disclosure of an adverse event and patients' perception of quality of care. Overall, only 40% of adverse events were disclosed. Higher quality ratings were associated with disclosure of an adverse event. Conversely, lower patient perception of quality of care was associated with events that were preventable and with events that caused discomfort.[6]

— The AHA published a scientific statement[7] about medication errors in acute cardiovascular and stroke patients and classified medication errors into the following categories:

  ○ Improper dosing or timing, or delivery of an incorrect or unnecessary medication.
  ○ Administration to the wrong patient (errors of omission).
  ○ Failure to prescribe appropriate medication therapy or needed monitoring of medication therapy (errors of omission).

— Recommendations were also made that could improve medication safety in cardiovascular care.

• *Effective care* has been defined as providing services based on scientific knowledge to all who could benefit and refraining from providing services to those not likely to benefit. It also encompasses monitoring results of the care provided and using them to improve care for all patients.[1] There are many quality-improvement registries that have been developed for inpatient cardiovascular/stroke care, and the data on these are provided in subsequent tables. Similar efforts are under way for quality-of-care registries in the outpatient setting. In 2011, the AHA published a policy statement for expanding the applications of existing and future clinical registries.[8] This statement discusses recommendations on ensuring high-quality data, linking clinical registries with supplemental data, integrating registries with electronic health records, safeguarding privacy, securing adequate funding, and developing a business model to initiate and sustain these registries.

— In the CRUSADE registry, 1 in 10 patients (10.3%) had a documented contraindication to reperfusion. Primary reasons for contraindications were identified as absence of an ischemic indication (53.8%), bleeding risk (16.7%), patient-related reasons (25.3%), and other (4.2%). Conversely, 7.2% of patients with STEMI without a reperfusion contraindication did not have reperfusion therapy administered, and this was associated with greater in-hospital mortality.[9]

— According to data from NHANES 1988–1994 and 1999–2008, rates of hypertension have increased from 23.9% in 1988 to 1994 to 29.0% in 2007 to 2008, and hypertension control has increased from 27.3% in 1988 to 1994 to 50.1% in 2007 to 2008. In addition, among patients with hypertension, BP has decreased from 143.0/80.4 to 135.2/74.1 mm Hg.[10]

— The AHA and the ACC Foundation Task Force on Performance Measures published a scientific statement that provides new insights into the methodology of performance measures. It covers topics such as the use of exceptions in performance measures, modification and retirement of performance measures, new insights into the implementation of performance measures, use of composite measures, and the challenges associated with the concept of shared accountability.[11]

— The National Quality Forum is a nonprofit organization that aims to improve the quality of health care for all. Recognizing that adherence can impact the effectiveness of therapies, the National Quality Forum has adopted several performance measures related to medication adherence/persistence, including angiotensin-converting enzyme inhibitor/angiotensin receptor blocker use and persistence among patients with CAD who are at high risk for coronary events, persistence of β-blocker treatment after a heart attack for patients with AMI, and adherence to lipid-lowering medication.[12]

— Outcome measures of 30-day mortality and 30-day readmission after hospitalization for AMI or HF have been developed that adjust for patient mix (eg, comorbidities) so that comparisons can be made across hospitals.[13–16] Using national Medicare data from July 2005 through June 2008, the median (10th, 90th percentile) hospital risk-standardized mortality rate was 16.6% (14.7%, 18.4%) for AMI and 11.1% (9.4%, 13.1%) for HF. The median risk-standardized readmission rate was 19.9% (18.8%, 21.1%) for AMI and 24.4% (22.3%, 27.0%) for HF. For various hospital characteristics (number of beds, ownership, teaching status, bypass surgery facility), there were high- and low-performing hospitals in all categories.[13]

— A study of 30 947 patients admitted with ischemic strokes showed that admission to a designated stroke center compared with admission to a nondesignated hospital was associated with more frequent use of thrombolytic therapy (4.8% versus 1.7%, $P<0.001$) and lower 30-day all-cause mortality (10.1% versus 12.5%, $P<0.001$).[17]

— A study of 458 hospitals participating in the Society of Thoracic Surgeons National Cardiac Database showed that an intervention of receiving quality-improvement educational material designed to influence the prescription rates of 4 medication classes (aspirin, β-blockers, lipid-lowering therapy, and angiotensin-converting enzyme inhibitors) after CABG discharge in addition to site-specific feedback reports led to a significant improvement in adherence for all 4 secondary prevention medications at the intervention sites compared with the control sites.[18]

— Inpatient ACS, HF, and stroke quality-of-care measures data, including trends in care data, where available from national registries, are given in Tables 21-1 through 21-6.

— In 2011, ACC Foundation/AHA/American Medical Association–Physician Consortium for Performance Improvement performance measures for CAD and hy-

pertension were published.[19] The 9 performance measures for CAD care included BP control, lipid control, symptom and activity assessment, symptom management, tobacco use (screening, cessation, and intervention), antiplatelet therapy, β-blocker therapy, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker therapy, and cardiac rehabilitation patient referral from an outpatient setting. For hypertension care, the performance measures included BP control. This set was an update to the 2005 ACC Foundation/AHA performance measures for CAD and hypertension and included modifications to 7 of the 2005 performance measures. Screening for DM was retired from the CAD set published in 2005, whereas symptom management and cardiac rehabilitation referral were added to the 2011 CAD set.

— Selected outpatient quality-of-care measures from the National Committee for Quality Assurance for 2009 appear in Table 21-7.

— Quality-of-care measures for patients who had out-of-hospital cardiac arrest and were enrolled in the Resuscitation Outcomes Consortium (ROC) cardiac epistry in 2010 (ROC Investigators, unpublished data, June 20, 2011) are given in Table 21-8 for individuals of any age and in Table 21-9 for children.

- *Patient-centered care* has been defined as the provision of care that is respectful of and responsive to individual patient preferences, needs, and values and that ensures that patient values guide all clinical decisions. Dimensions of patient-centered care include the following: (1) Respect for patients' values, preferences, and expressed needs; (2) coordination and integration of care; (3) information, communication, and education; (4) physical comfort; (5) emotional support; and (6) involvement of family and friends. Studies focusing on some of these aspects of patient-centered care are highlighted below.

  — The Clinical Outcomes Utilizing Revascularization and AGgressive drug Evaluation (COURAGE) trial,[20] which investigated a strategy of PCI plus optimal medical therapy versus optimal medical therapy alone, demonstrated that both groups had significant improvement in health status during follow-up. By 3 months, health status scores had increased in the PCI group compared with the medical therapy group to 76±24 versus 72±23 for physical limitation ($P=0.004$), 77±28 versus 73±27 for angina stability ($P=0.002$), 85±22 versus 80±23 for angina frequency ($P<0.001$), 92±12 versus 90±14 for treatment satisfaction ($P<0.001$), and 73±22 versus 68±23 for quality of life ($P<0.001$). The PCI plus optimal medical therapy group had a small but significant incremental benefit compared with the optimal medical therapy group early on, but this benefit disappeared by 36 months.

  — In the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT)[21] of single-lead implantable cardioverter-defibrillator versus amiodarone for moderately symptomatic HF, patients with implantable cardioverter-defibrillators had improvement in quality of life compared with medical therapy patients at 3 and 12 months but not at 30 months. Implantable cardioverter-defibrillator shocks in the month preceding a scheduled assessment were associated with a decrease in quality of life in multiple domains. The authors concluded that the presence of a single-lead implantable cardioverter-defibrillator was not associated with any detectably adverse quality of life during 30 months of follow-up.

  — Peikes et al[22] reported on 15 care-coordination programs as part of a Medicare demonstration project for patients with congestive HF, CAD, DM, and other conditions. Thirteen of the 15 programs did not show a difference in hospitalization rates, and none of the programs demonstrated a net savings. The interventions tested varied significantly, but the majority of the interventions included patient education to improve adherence to medication, diet, exercise, and self-care regimens and improving care coordination through various approaches. These programs overall had favorable effects on none of the adherence measures and only a few of the many quality-of-care indicators examined. The authors concluded that programs with substantial in-person contact that target moderately to severely ill patients can be cost-neutral and improve some aspects of care.

  — Hernandez et al[23] showed that patients with outpatient follow-up within 7 days of discharge for an HF hospitalization were less likely to be readmitted within 30 days in the GWTG-HF registry of patients who were ≥65 years of age. The median length of stay was 4 days (interquartile range 2–6 days), and 21.3% of patients were readmitted within 30 days. At the hospital level, the median percentage of patients who had early follow-up after discharge from the index hospitalization was 38.3% (interquartile range 32.4%–44.5%).

  — Smolderen et al[24] assessed whether health insurance status affects decisions to seek care for AMI. Uninsured and insured patients with financial concerns were more likely to delay seeking care during AMI and had prehospital delays of >6 hours (48.6% of uninsured patients and 44.6% of insured patients with financial concerns compared with 39.3% of insured patients without financial concerns). Lack of health insurance and financial concerns about accessing care among those with health insurance were each associated with delays in seeking emergency care for AMI.

  — Using a cohort (n=192) nested within a randomized trial at a university-affiliated ambulatory practice, Murray et al[25] demonstrated that refill adherence of <40% was associated with a 3-fold higher incidence of hospitalization for HF than a refill adherence of ≥80% ($P=0.002$). In multivariable analysis, prescription label–reading skills were associated with a lower incidence of HF-specific emergency care (incidence rate ratio 0.76, 95% CI 0.19–0.69), and participants with adequate health literacy had a lower risk of hospitalization for HF (incidence rate ratio 0.34, 95% CI 0.15–0.76).

- The *timely care* domain relates to reducing waits and sometimes harmful delays for both those who receive and those who give care. Timeliness is an important characteristic of any service and is a legitimate and valued focus of improvement in health care and other industries.
  - — Data from the CRUSADE national quality-improvement initiative showed that median delay from onset of symptoms to hospital presentation for patients presenting with NSTEMI was 2.6 hours and was significantly associated with in-hospital mortality but did not change over time from 2001 to 2006.[26]
  - — Bradley et al[27] demonstrated that participation in the Door-to-Balloon (D2B) Alliance led to a reduction in door-to-balloon time to within 90 minutes for patients with STEMI. By March 2008, >75% of patients had door-to-balloon times of ≤90 minutes compared with only approximately one fourth of patients in April 2005.
  - — Using data between 2005 and 2007 from the National Cardiovascular Data Catheterization PCI registry, Wang et al demonstrated that among STEMI patients, only 10% of the transfer patients received PCI within ≤90 minutes (versus 63% for direct-arrival patients; $P<0.0001$).[28]
  - — Data on time to reperfusion for STEMI or ischemic stroke are provided from national registries in Table 21-10.
  - — Among patients who experienced in-hospital cardiac arrest and were enrolled in the AHA National Cardiopulmonary Registry (now GWTG-Resuscitation):
    - ○ Chan et al[29] demonstrated significant variation in timely defibrillation (<2 minutes) for patients with in-hospital cardiac arrest among 200 hospitals participating in the National Registry of Cardiopulmonary Resuscitation. Adjusted rates of delayed defibrillation varied from 2.4% to 50.9% of in-hospital cardiac arrests. The variations in defibrillation rates were largely unexplained by traditional hospital factors.
    - ○ Survival did not improve with use of an automated external defibrillator compared with a manual defibrillator.[30]
    - ○ Among those who experienced pulseless in-hospital cardiac arrest with an initial shockable rhythm in 2010 (GWTG Investigators, unpublished data, June 20, 2011), 90.5% of adults and 87.5% of children received a defibrillation attempt within 3 minutes.
- *Efficiency* has been defined as avoiding waste, in particular waste of equipment, supplies, ideas, and energy. In an efficient healthcare system, resources are used to get the best value for the money spent.
  - — The AHA and ACC have jointly developed a scientific statement that outlines standards for measures to be used for public reporting of efficiency in health care. The group identified 4 standards important to the development of any efficiency performance measure, including (1) integration of quality and cost, (2) valid cost measurement and analysis, (3) no or minimal incentive to provide poor-quality care, and (4) no or proper attribution of the measure. In the statement, 4 examples were provided of hospital-based efficiency measures, as well as information on how each of the measures fared within the 4 domains recommended. The examples were length of stay, 30-day readmission, hospitalization costs, and nonrecommended imaging tests.[30a]
  - — At an urban, tertiary care, academic medical center ED, elements of departmental work flow were redesigned to streamline patient throughput before implementation of a fully integrated ED information system with patient tracking, computerized charting and order entry, and direct access to patient historical data from the hospital data repository. Increasing the clinical information available at the bedside and improving departmental work flow through ED information system implementation and process redesign led to decreased patient throughput times and improved ED efficiency (eg, the length of stay for all patients [from arrival to time patient left the ED] decreased by 1.94 hours, from 6.69 [n=508] before the intervention to 4.75 [n=691] after the intervention; $P<0.001$).[31]
  - — Himmelstein et al[32] analyzed whether more-computerized hospitals had lower costs of care or administration or better quality to address a common belief that computerization improves healthcare quality, reduces costs, and increases administrative efficiency. They found that hospitals that increased computerization faster had more rapid administrative cost increases ($P=0.0001$); however, higher overall computerization scores correlated weakly with better quality scores for AMI ($r=0.07$, $P=0.003$) but not for HF, pneumonia, or the 3 conditions combined. In multivariate analyses, more-computerized hospitals had slightly better quality. The authors concluded that hospital computing might modestly improve process measures of quality but does not reduce administrative or overall costs.
- *Equitable care* means the provision of care that does not vary in quality because of personal characteristics such as sex, ethnicity, geographic location, and socioeconomic status. The aim of equity is to secure the benefits of quality health care for all the people of the United States. With regard to equity in caregiving, all individuals rightly expect to be treated fairly by local institutions, including healthcare organizations.
  - — Chan et al[33] demonstrated that rates of survival to discharge were lower for black patients (25.2%) than for white patients (37.4%). Lower rates of survival to discharge for blacks reflected lower rates of both successful resuscitation (55.8% versus 67.4%) and postresuscitation survival (45.2% versus 55.5%). Adjustment for the hospital site at which patients received care explained a substantial portion of the racial differences in successful resuscitation (adjusted RR 0.92, 95% CI 0.88–0.96; $P<0.001$) and eliminated the racial differences in postresuscitation survival (adjusted RR 0.99, 95% CI 0.92–1.06; $P=0.68$). The authors concluded that much of the racial

difference was associated with the hospital center in which black patients received care.

— Cohen et al[34] demonstrated that among hospitals engaged in a national quality monitoring and improvement program, evidence-based care for AMI appeared to improve over time for patients irrespective of race/ethnicity, and differences in care by race/ethnicity care were reduced or eliminated. They analyzed 142 593 patients with AMI (121 528 whites, 10 882 blacks, and 10 183 Hispanics) at 443 hospitals participating in the GWTG-CAD program. Overall, defect-free care was 80.9% for whites, 79.5% for Hispanics (adjusted OR versus whites 1.00, 95% CI 0.94–1.06; $P=0.94$), and 77.7% for blacks (adjusted OR versus whites 0.93, 95% CI 0.87–0.98; $P=0.01$). A significant gap in defect-free care was observed for blacks during the first half of the study but was no longer present during the remainder of the study. Overall, progressive improvements in defect-free care were observed regardless of race/ethnic groups.

— According to NHANES 1999–2006, 45% of adults had at least 1 of 3 chronic conditions (hypertension, hypercholesterolemia, or DM), 13% had 2 of these conditions, and 3% of adults had all 3 conditions. Non-Hispanic black people were more likely than non-Hispanic white and Mexican-American people to have at least 1 of the 3 conditions. In 15% of US adults, ≥1 of the 3 conditions is undiagnosed.[35]

— Thomas et al[36] analyzed data among hospitals that voluntarily participated in the AHA's GWTG-HF program from January 2005 through December 2008. They demonstrated that relative to white patients, Hispanic and black patients hospitalized with HF were significantly younger (median age 78, 63, and 64 years, respectively) but had lower EFs (mean EF 41.1%, 38.8%, and 35.7%, respectively) with a higher prevalence of DM (40.2%, 55.7%, and 43.8%, respectively) and hypertension (70.6%, 78.4%, and 82.8%, respectively). The provision of guideline-based care was comparable for white, black, and Hispanic patients. Black (1.7%) and Hispanic (2.4%) patients had lower in-hospital mortality than white patients (3.5%). Improvement in adherence to all-or-none HF measures increased annually from year 1 to year 3 for all 3 racial/ethnic groups.[36]

— GWTG data by race, sex, and ethnicity are provided in Tables 21-11 through 21-13.

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Table 21-1. Acute Coronary Syndrome Quality-of-Care Measures, 2010

Quality-of-Care Measure	VHA*	National Data From HIQR Program†	ACTION-GWTG STEMI‡	ACTION–GWTG NSTEMI‡
Aspirin within 24 h of admission	99	98.6	98	96
Aspirin at discharge	99	98.6	99	97
β-blockers within 24 h of admission, among AMI and angina patients	97	R	NM	NM
β-blockers at discharge	99	98.3	97	95
Lipid-lowering medication at discharge	NM	NM	95§	90§
Lipid therapy at discharge if LDL cholesterol >100 mg/dL	98‖	NM	NM	NM
ARB/ACEI at discharge for patients with LVEF <40%	98	94.7	89	84
ACEI at discharge for AMI patients	NM	NM	78	70
Adult smoking cessation advice/counseling	99	98.5	99	98
Cardiac rehabilitation referral for AMI patients	NM	NM	82	70

VHA indicates Veterans Health Administration; HIQR, Hospital Inpatient Quality Reporting; ACTION-GWTG, Acute Coronary Treatment and Intervention Outcomes Registry–Get With The Guidelines; STEMI, ST-elevation myocardial infarction; NSTEMI, non–ST-elevation myocardial infarction; R, retired in 2009; AMI, acute myocardial infarction; NM, not measured; LDL, low-density lipoprotein; ARB, angiotensin receptor blocker; ACEI, angiotensin-converting enzyme inhibitor; and LVEF, left ventricular ejection fraction.

Values are percentages.

*VHA: AMI patients.

†HIQR Program includes data from all payers, including Medicare and Medicaid.

‡ACTION Registry: STEMI and NSTEMI patients are reported separately. Patients must be admitted with acute ischemic symptoms within the previous 24 hours, typically reflected by a primary diagnosis of STEMI or NSTEMI. Patients who are admitted for any other clinical condition are not eligible.

§Denotes statin use at discharge. Use of any other lipid-lowering agent was 11% for STEMI patients and 14% for NSTEMI patients.

‖Lipid-lowering therapy among patients with LDL cholesterol >130 mg/dL.

Table 21-2. HF Quality-of-Care Measures, 2010

Quality-of-Care Measure	National Data From HIQR Program*	AHA GWTG-HF	VHA
LVEF assessment	97.8	98†	100
ARB/ACEI at discharge for patients with LVSD	94.7	94.2†	96
Complete discharge instructions	88.9	93.3†	97
Adult smoking cessation advice/counseling	98.5	99.3†	99
β-blockers at discharge for patients with LVSD, no contraindications	NM	94.8†	NM
Anticoagulation for AF or atrial flutter, no contraindications	NM	70.2	95

HF indicates heart failure; HIQR, Hospital Inpatient Quality Reporting; AHA GWTG-HF, American Heart Association's Get With The Guidelines–Heart Failure; VHA, Veterans Health Administration; LVEF, left ventricular ejection fraction; ARB/ACEI, angiotensin receptor blocker/angiotensin-converting enzyme inhibitor; LVSD, left ventricular systolic dysfunction; NM, not measured; and AF, atrial fibrillation.

Values are percentages.

In the GWTG registry, mechanical ventilation was required in 3.0% of patients. In-hospital mortality rate was 3.0%, and mean length of hospital stay was 5.5 days (median 4.0 days).

*HIQR Program includes data from all payers, including Medicare and Medicaid.

†Indicates the 5 key performance measures targeted in GWTG-HF. The composite quality-of-care measure for 2010 was 95.7%. The composite quality-of-care measure indicates performance on the provision of several elements of care. It is computed by summing the numerators for each key performance measure across the population of interest to create a composite numerator (all the care that was given), summing the denominators for each measure to form a composite denominator (all the care that should have been given), and reporting the ratio (the percentage of all the needed care that was given).

Table 21-3. Time Trends in GWTG-ACS Quality-of-Care Measures, 2006–2010

Quality-of-Care Measure	2006	2007	2008	2009	2010*
Aspirin within 24 h of admission	94.7	92.8	91.2	90.9	97
Aspirin at discharge	94.4	95.8	94.9	95.5	98
β-blockers at discharge	92.8	94.6	94.5	94.9	96
Lipid-lowering medication at discharge	84.5	85.6	81.6	86.8	92
Lipid therapy at discharge if LDL cholesterol $>$100 mg/dL	89.1	90.7	91.9	92.5	NM
ARB/ACEI at discharge for patients with LVEF $<$40%	87.3	91.1	91.9	91.9	86
ACEI at discharge for AMI patients	72.6	71.0	66.6	65.9	73
Adult smoking cessation advice/counseling	94.3	97.4	98.4	98.4	98
Cardiac rehabilitation referral for AMI patients	71.1	63.6	52.0	49.1	75

GWTG-ACS indicates Get With The Guidelines–Acute Coronary Syndrome; LDL, low-density lipoprotein; NM, not measured; ARB/ACEI, angiotensin receptor blocker/angiotensin-converting enzyme inhibitor; LVEF, left ventricular ejection fraction; and AMI, acute myocardial infarction.

Values are percentages.

*Measures from 2006–2009 are from the American Heart Association (AHA) GWTG-Coronary Artery Disease (CAD) registry. 2010 Measures are from the AHA ACTION registry (Acute Coronary Treatment and Intervention Outcomes Registry; the AHA's GWTG-CAD has now merged into the ACTION registry).

In the ACTION registry, the unadjusted in-hospital mortality rate for 2010 was 4.8% (95% confidence interval 4.6% to 4.9%; excludes transfer-out patients).

Table 21-4. Time Trends in GWTG-HF Quality-of-Care Measures, 2006–2010

Quality-of-Care Measure	2006	2007	2008	2009	2010
LVEF assessment*	93.8	96.2	96.8	98.2	98
ARB/ACEI at discharge for patients with LVSD*	85.5	89.1	91.6	93.0	94.2
Complete discharge instructions*	78.8	84.8	88.5	90.9	93.3
Adult smoking cessation advice/counseling*	90.8	94.7	97.1	97.6	99.3
β-blockers at discharge for patients with LVSD, no contraindications*	89.9	90.2	92.5	92.7	94.8
Anticoagulation for atrial fibrillation or atrial flutter, no contraindications	62.9	61.6	60.7	68.9	70.2

GWTG-HF indicates Get With The Guidelines–Heart Failure; LVEF, left ventricular ejection fraction; ARB/ACEI, angiotensin receptor blocker/angiotensin-converting enzyme inhibitor; and LVSD, left ventricular systolic dysfunction.

Values are percentages.

In the GWTG registry, mechanical ventilation was required in 3.5% of patients. In-hospital mortality was 3.0%, and mean length of hospital stay was 5.5 days (median 4.0 days).

*Indicates the 5 key achievement measures targeted in GWTG-HF. The composite quality-of-care measure for 2010 was 95.7%. The composite quality-of-care measure indicates performance on the provision of several elements of care. It is computed by summing the numerators for each key achievement measure across the population of interest to create a composite numerator (all the care that was given), summing the denominators for each measure to form a composite denominator (all the care that should have been given), and reporting the ratio (the percentage of all the needed care that was given).

Table 21-5. Time Trends in GWTG-Stroke Quality-of-Care Measures, 2006–2010

Quality-of-Care Measure	2006	2007	2008	2009	2010
IV tPA in patients who arrived $\leq$2 h after symptom onset, treated in $\leq$3 h*	55.8	60.2	63.9	73.1	76.2
IV tPA in patients who arrived $<$3.5 h after symptom onset, treated in $\leq$4.5 h†	N/A	N/A	N/A	N/A	42.5
IV tPA door-to-needle time $\leq$60 min	22.5	24.9	25.9	28.0	29.5
Thrombolytic complications: IV tPA and life-threatening, serious systemic hemorrhage	20.8	17.3	16.1	15.1	13.1
Antithrombotics $<$48 h after admission*	94.8	95.8	96.0	96.2	96.3
DVT prophylaxis by second hospital day*	85.3	88.9	92.2	92.7	92.2
Antithrombotics at discharge*	94.1	95.1	97.0	97.8	97.7
Anticoagulation for atrial fibrillation at discharge*	88.2	89.5	93.1	93.5	93.5
Therapy at discharge if LDL cholesterol $>$100 mg/dL or LDL cholesterol not measured or on therapy at admission*	70.3	76.3	82.1	86.2	88.1
Counseling for smoking cessation*	86.1	92.2	94.3	96.2	96.7
Lifestyle changes recommended for BMI $>$25 kg/m^2	42.5	45.7	51.7	57.3	57.8
Composite quality-of-care measure	85.9	88.9	91.7	93.3	93.7

GWTG-Stroke indicates Get With The Guidelines–Stroke; IV, intravenous; tPA, tissue-type plasminogen activator; N/A, not applicable; DVT, deep venous thrombosis; LDL, low-density lipoprotein; and BMI, body mass index.

Values are percentages.

In-hospital mortality for the 2010 patient population was 6.6% percent, and mean length of hospital stay was 4.9 days (median 3.0 days).

*Indicates the 7 key achievement measures targeted in GWTG-Stroke.

†New quality measure subsequent to the European Cooperative Acute Stroke Study III.

Table 21-6. Additional ACTION-GWTG Quality-of-Care Metrics for ACS Care (2010)

Quality Metrics	Overall	STEMI	NSTEMI
ECG within 10 min of arrival	61	73	55
Aspirin within 24 h of arrival	97	98	96
Any anticoagulant use*	93	95	91
Dosing error			
UFH dose	54	55	54
Enoxaparin dose	12	12	12
GP IIb/IIIa inhibitor dose	8	8	8
ADP receptor inhibitor† on discharge	82	93	74
Prescribed statins on discharge	92	95	90
Adult smoking cessation advice/counseling	98	99	98
Cardiac rehabilitation referral	75	82	70
In-hospital mortality‡ (95% CI)	4.8 (4.6–4.9)	5.9 (5.7–6.2)	4 (3.8–4.1)

ACTION-GWTG indicates Acute Coronary Treatment and Intervention Outcomes Registry–Get With The Guidelines; ACS, acute coronary syndrome; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non–ST-segment elevation myocardial infarction; UFH, unfractionated heparin; GP, glycoprotein; ADP, adenosine diphosphate; and CI, confidence interval.

Values are percentages.

*Includes UFH, low-molecular-weight heparin, bivalirudin, or fondaparinux use.

†Includes clopidogrel or prasugrel.

‡Excludes transfer-out patients.

Table 21-7. National Committee for Quality Assurance Health Plan Employer Data and Information Set Measures of Care

	Commercial	Medicare	Medicaid
AMI			
β-blocker persistence*	74.4	82.6	76.6
Cholesterol management for patients with cardiovascular disease			
Cholesterol screening	88.4	88.4	80.7
LDL cholesterol control (<100 mg/dL)	59.2	55.7	41.2
Hypertension			
BP <140/90 mm Hg	64.1	59.8	55.3
DM			
HbA_{1c} testing	89.2	89.6	80.6
HbA_{1c} >9.0%	28.2	28	44.9
Eye examination performed	56.5	63.5	52.7
LDL cholesterol screening	85	87.3	74.2
LDL cholesterol <100 mg/dL	47	50	33.5
Monitoring nephropathy	82.9	88.6	76.9
BP <130/80 mm Hg	33.9	33.3	32.2
BP <140/90 mm Hg	65.1	60.5	59.8
Advising smokers to quit	79.5	77.9	74.3
BMI percentile assessment in children and adolescents	35.4	N/A	30.3
Nutrition counseling (children and adolescents)	41	N/A	41.9
Counseling for physical activity (children and adolescents)	36.5	N/A	32.5
BMI assessment for adults	41.3	38.8	34.6
Physical activity discussion in older adults (≥65 y)	N/A	51.3	N/A

AMI indicates acute myocardial infarction; LDL, low-density lipoprotein; BP, blood pressure; DM, diabetes mellitus; HbA_{1c}, hemoglobin A_{1c}; BMI, body mass index; and N/A, not available or not applicable.

Values are percentages.

*β-Blocker persistence: Received persistent β-blocker treatment for 6 months after AMI hospital discharge.

Table 21-8. Quality of Care for Out-of-Hospital Resuscitation

	Bystander CPR, % (95% CI)	Time to First EMS Defibrillator Turned On, Mean (SD), min
EMS-treated, nontraumatic cardiac arrest	41.0 (39.7–42.3)	8.7 (4.6)
Bystander-witnessed VF	60.2 (56.6–63.9)	7.6 (3.2)

CPR indicates cardiopulmonary resuscitation; CI, confidence interval; EMS, emergency medical services; SD, standard deviation; and VF, ventricular fibrillation.

Table 21-9. Quality of Care for Out-of-Hospital Resuscitation of Children

	Bystander CPR, % (95% CI)	Time to First EMS Defibrillator Turned On, Mean (SD), min
EMS-treated, nontraumatic cardiac arrest	56.3 (49.8–62.8)	8.8 (3.6)
Bystander-witnessed VF	75.0 (45.0–100)	7.2 (1.2)

CPR indicates cardiopulmonary resuscitation; CI, confidence interval; EMS, emergency medical services; SD, standard deviation; and VF, ventricular fibrillation.

Table 21-10. Timely Reperfusion for ACS and Stroke 2010

Quality-of-Care Measure	VHA*	National Data From HIQR Program†	ACTION-GWTG STEMI‡	GWTG-Stroke
STEMI				
tPA within 30 min	58§	57.1	62	N/A
Percutaneous coronary intervention within 90 min	67	90.4	91	N/A
Stroke				
IV tPA in patients who arrived ≤2 h after symptom onset, treated in ≤3 h	N/A	N/A	N/A	76.2
IV tPA in patients who arrived <3.5 h after symptom onset, treated in ≤4.5 h	N/A	N/A	N/A	42.5
IV tPA door-to-needle time ≤60 min	N/A	N/A	N/A	29.5

ACS indicates acute coronary syndrome; VHA, Veterans Health Administration; HIQR, Hospital Inpatient Quality Reporting; ACTION-GWTG, Acute Coronary Treatment and Intervention Outcomes Registry–Get With The Guidelines; STEMI, ST-elevation myocardial infarction; GWTG-Stroke, Get With The Guidelines–Stroke; tPA, tissue-type plasminogen activator; N/A, not applicable; and IV, intravenous.

Values are percentages.

*VHA: acute myocardial infarction patients.

†HIQR Program includes data from all payers, including Medicare and Medicaid.

‡ACTION Registry: STEMI and NSTEMI patients are reported separately. Patients must be admitted with acute ischemic symptoms within the previous 24 hours, typically reflected by a primary diagnosis of STEMI or NSTEMI. Patients who are admitted for any other clinical condition are not eligible.

§Indicates low number.

Table 21-11. Quality of Care by Race/Ethnicity and Sex in the ACTION Registry, 2010

Quality-of-Care Measure	White	Black	Other	Men	Women
Aspirin at admission	98	97	97	98	97
Aspirin at discharge	98	97	97	98	97
β-blockers at discharge	96	95	95	96	95
Time to PCI ≤90 min for STEMI patients	93	88	91	93	91
ARB/ACEI at discharge for patients with LVEF <40%	86	86	86	86	86
Statins at discharge	98	97	98	98	97

ACTION indicates Acute Coronary Treatment and Intervention Outcomes Network; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction; ARB/ACEI angiotensin receptor blocker/angiotensin-converting enzyme inhibitor; and LVEF, left ventricular ejection fraction.

Values are percentages.

Table 21-12. Quality of Care by Race/Ethnicity and Sex in the GWTG-HF Program

Quality-of-Care Measure	White	Black	Hispanic	Men	Women
Complete set of discharge instructions*	92.8	93.8	93.3	93.6	92.9
Measure of LV function*	98.9	97.0	98.3	98.0	97.9
ACEI or ARB at discharge for patients with LVSD, no contraindications*	93.2	95.5	94.9	93.8	94.4
Smoking cessation counseling, current smokers*	99.2	99.6	99.3	99.3	99.3
β-blockers at discharge for patients with LVSD, no contraindications*	94.7	95.3	94.7	95.1	94.5
Hydralazine/nitrates at discharge for patients with LVSD, no contraindications		12.5		13.5†	10.7†
Anticoagulation for atrial fibrillation or atrial flutter, no contraindications	74.7	68.2	69.2	72.0	68.1
Composite quality-of-care measure	95.9	95.9	95.8	95.7	95.6

GWTG-HF indicates Get With The Guidelines–Heart Failure; LV, left ventricular; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; and LVSD, LV systolic dysfunction.

Values are percentages.

*Indicates the 5 key achievement measures targeted in GWTG-HF.

†For black patients only.

Table 21-13. Quality of Care by Race/Ethnicity and Sex in the GWTG-Stroke Program

Quality-of-Care Measure	White	Black	Hispanic	Male	Female
IV tPA in patients who arrived $\leq$2 h after symptom onset, treated in $\leq$3 h*	76.0	75.4	77.5	76.0	76.4
IV tPA in patients who arrived $<$3.5 h after symptom onset, treated in $\leq$4.5 h	41.6	43.1	47.8	43.3	41.7
IV tPA door-to-needle time $\leq$60 min	29.3	27.3	32.8	31.8	27.3
Thrombolytic complications: IV tPA and life-threatening, serious systemic hemorrhage	12.3	16.0	15.2	13.5	12.8
Antithrombotics $<$48 h after admission*	96.6	95.9	95.5	96.7	96.1
DVT prophylaxis by second hospital day*	92.2	92.4	91.1	92.5	91.9
Antithrombotics at discharge*	97.9	97.2	96.9	98.0	97.4
Anticoagulation for atrial fibrillation at discharge*	93.6	92.9	93.4	94.0	93.1
Therapy at discharge if LDL $>$100 mg/dL or LDL not measured or on therapy at admission*	88.0	88.6	87.9	90.2	86.3
Counseling for smoking cessation*	96.8	96.7	95.6	96.7	96.6
Lifestyle changes recommended for BMI $>$25 kg/m^2	57.5	57.9	61.3	58.2	57.4
Composite quality-of-care measure	93.8	93.8	93.0	94.4	93.1

GWTG-Stroke indicates Get With The Guidelines–Stroke; IV, intravenous; tPA, tissue-type plasminogen activator; DVT, deep venous thrombosis; LDL, low-density lipoprotein; and BMI, body mass index.

Values are percentages.

*Indicates the 7 key performance measures targeted in GWTG-Stroke.

22. Medical Procedures

See Tables 22-1 and 22-2 and Charts 22-1 through 22-3.

- The total number of inpatient cardiovascular operations and procedures increased 22%, from 6 133 000 in 1999 to 7 453 000 in 2009 (NHLBI computation based on NCHS annual data). Data from the NHDS were examined for trends from 1990 to 2004 for use of PCI and CABG and in-hospital mortality rate attributable to PCI and CABG by sex.[1]
  - — Discharge rates (per 10 000 population) for PCI increased 58%, from 37.2 in 1990 to 1992 to 59.2 in 2002 to 2004.
  - — Discharge rates for CABG increased from 34.1 in 1990 to 1992 to 38.6 in 1996 to 1998, then declined to 25.2 in 2002 to 2004.
  - — In 1990 to 1992, discharge rates for CABG were 53.5 for males and 18.1 for females; these rates increased through 1996–1998, then declined to 38.8 and 13.6, respectively, in 2002 to 2004. The magnitude of these declines decreased by age decile and were essentially flat for both men and women 75 years of age.
  - — PCI discharge rates increased from 54.5 for males and 23.0 for females to 83.0 and 38.7 over the 15-year time interval. In 2002 to 2004, discharge rates for men and women 65 to 74 years of age were 135.1 and 64.0, respectively. For those 75 years of age, the rates were 128.7 and 69.0, respectively.
  - — In-hospital mortality rate (deaths per 100 CABG discharges) declined from 4.3 to 3.5 in 2002 to 2004 despite an increase in Charlson comorbidity index. The mortality rate declined in all age and sex subsets, but especially in women.
- Data from the Acute Care Tracker database were used to estimate the population-based rates per 100 000 population for PCI and CABG procedures from 2002 to 2005, standardized to the 2005 US population[2]:
  - — Adjusted for age and sex, the overall rate for coronary revascularization declined from 382 to 358 per 100 000. PCI rates during hospitalization increased from 264 to 267 per 100 000, whereas CABG rates declined from 121 to 94.
- Data from men and women enrolled in Medicare from 1992 to 2001 suggest that efforts to eliminate racial disparities in the use of high-cost cardiovascular procedures (PCI, CABG, and carotid endarterectomy) were unsuccessful.[3]
  - — In 1992, among women, the age-standardized rates of carotid endarterectomy were 1.59 per 1000 enrollees for whites and 0.64 per 1000 enrollees for blacks. By 2002, the rates were 2.42 per 1000 enrollees among white women and 1.15 per 1000 enrollees among black women. For men, the difference in rates between whites and blacks remained the same. In 1992, the rates were 3.13 per 1000 enrollees among white men and 0.82 per 1000 enrollees among black men; in 2001, the rates were 4.42 and 1.44, respectively.

Abbreviations Used in Chapter 22

AHA	American Heart Association
CABG	coronary artery bypass graft
CHF	congestive heart failure
D2B	door-to-balloon
GWTG-CAD	Get With The Guidelines–Coronary Artery Disease
HD	heart disease
ICD-9-CM	*International Classification of Diseases, 9th Revision, Clinical Modification*
NCHS	National Center for Health Statistics
NHDS	National Hospital Discharge Survey
NHLBI	National Heart, Lung, and Blood Institute
PCI	percutaneous coronary intervention
STEMI	ST-elevation myocardial infarction
TOF	tetralogy of Fallot

Cardiac Catheterization and PCI

- From 1999 to 2009, the number of cardiac catheterizations decreased slightly, from 1 271 000 to 1 072 000 annually (NHLBI tabulation, NHDS, NCHS).
- In 2009, an estimated 596 000 patients underwent PCI (previously referred to as percutaneous transluminal coronary angioplasty, or PTCA) procedures in the United States (NHLBI tabulation, NHDS, NCHS).
- In 2009, ≈66% of PCI procedures were performed on men, and ≈53% were performed on people ≥65 years of age (NHDS, NCHS).
- In-hospital death rates for PCI have remained stable although comorbidities increased for patients who received the procedure.[1]
- In 2006, ≈76% of stents implanted during PCI were drug-eluting stents compared with 24% that were bare-metal stents.[4]
- In a study of nontransferred patients with STEMI treated with primary PCI from July 2006 to March 2008, there was significant improvement over time in the percentage of patients receiving PCI within 90 minutes, from 54.1% from July to September 2006 to 74.1% from January to March 2008, among hospitals participating in the GWTG-CAD program. This improvement was seen whether or not hospitals joined the D2B Alliance during that period.[5]

Cardiac Open Heart Surgery

The NHDS (NCHS) estimates that in 2009, in the United States, 242 000 patients underwent a total of 416 000 coronary artery bypass procedures (defined by procedure codes). CABG volumes have declined nationally since 1998. Risk-adjusted mortality for CABG has declined significantly over the past decade.

- Data from the Society of Thoracic Surgeons' National Adult Cardiac Database, which voluntarily collects data from ≈80% of all hospitals that perform CABG in the

United States, indicate that a total of 158 008 procedures involved CABG in 2010.[6]

- Data from the Society of Thoracic Surgeons' National Adult Cardiac Database document a 50% decline in the risk-adjusted mortality rate despite a significant increase in preoperative surgical risk.[7]

Congenital Heart Surgery, 2006 to 2010 (From the Society of Thoracic Surgeons)

There were 103 664 procedures performed from July 2006 to June 2010. The in-hospital mortality rate was 3.2% in 2010. The 5 most common diagnoses were the following: patent ductus arteriosus (7.4%); hypoplastic left heart syndrome (6.9%); ventricular septal defect, type 2 (6.3%); cardiac, other (5.3%); and TOF (4.9%).[8]

Congenital Heart Surgery, 1998 to 2002 (From Society of Thoracic Surgeons)

There were 16 920 procedures performed from 1998 to 2002 at 18 centers. In 2002, there were 4208 procedures performed. The in-hospital mortality rate ranged from 5.7% in 1998 to 4.3% in 2002. Of these procedures, ≈46% were performed in children >1 year old, ≈32% in infants between 29 days and 1 year of age, and ≈22% in neonates (<29 days old). The conditions for which these procedures were most commonly performed were the following: patent ductus arteriosus (6.5%), ventricular septal defect (6.4%), and TOF (6.0%).

Heart Transplantations

In 2010, 2333 heart transplantations were performed in the United States. There were 272 transplant hospitals in the United States, 132 of which performed heart transplantations (based on Organ Procurement and Transplantation Network data as of June 8, 2011).

- Of the recipients in 2010, 73.0% were male, and 67.0% were white; 19.9% were black, whereas 8.5% were Hispanic; 25.0% were <35 years of age, 18.4% were 35 to 49 years of age, and 56.6% were ≥50 years of age.
- As of June 3, 2011, for transplants that occurred between 1997 and 2004, the 1-year survival rate for males was 88.0%, and for females, it was 86.2%; the 3-year rates were 79.3% for males and 77.2% for females; and the 5-year rates were 73.2% for males and 69.0% for females. The 1-, 3-, and 5-year survival rates for white cardiac transplant patients were 87.6%, 79.7%, and 73.3%, respectively. For black patients, they were 86.2%, 73.1%, and 64.0%, respectively. For Hispanic patients, they were 88.9%, 78.7%, and 73.1%, respectively.
- As of June 8, 2011, 3183 patients were on the transplant waiting list for a heart transplant, and 66 patients were on the list for a heart/lung transplant.

Cardiovascular Healthcare Expenditures

An analysis of claims and enrollment data from the Continuous Medicare History Sample and from physician claims from 1995 to 2004 was used to evaluate the conditions that contributed to the most expensive 5% of Medicare beneficiaries.[9]

- Ischemic HD, CHF, and cerebrovascular disease, respectively, constituted 13.8%, 5.9%, and 5.7% of the conditions of all beneficiaries in 2004. In patients in the top 5% overall for all expenditures, the respective figures were 39.1%, 32.7%, and 22.3% for these cardiovascular conditions.

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Table 22–1. 2009 National Healthcare Cost and Utilization Project Statistics: Mean Hospital Charges and In-Hospital Death Rates and Mean Length of Stay for Various Cardiovascular Procedures

Procedure	Mean Hospital Charges, $	In-Hospital Death Rate, %	Mean Length of Stay, d
Total vascular and cardiac surgery and procedures	66 703	2.89	6.0
Cardiac revascularization (bypass)	124 404	1.75	9.1
PCI	60 309	0.95	2.9
Diagnostic cardiac catheterization	36 905	0.90	3.7
Pacemakers	61 015	1.21	4.9
Implantable defibrillators	134 904	0.62	5.1
Endarterectomy	32 689	0.38	2.5
Valves	171 270	3.90	11.0
Heart transplantations	540 125	4.81	44.0

PCI indicates percutaneous coronary intervention.

Data derived from the Agency for Healthcare Research and Quality, Healthcare Cost and Utilization Project.[10]

Table 22–2. Estimated* Inpatient Cardiovascular Operations, Procedures, and Patient Data by Sex and Age: United States, 2009 (in Thousands)

		Sex			Age, y			
Operation/Procedure/Patients	ICD-9-CM Procedure Codes	All	Male	Female	<15 y	15–44 y	45–64 y	≥65 y
Valves	35.1, 35.2, 35.99	139	81	58	3†	12	34	89
Angioplasty	36.0, 00.66	1133	745	388	. . .	55	471	608
PCI (patients)	36.06, 36.07, 00.66	596	394	202	. . .	29	249	319
PCI	00.66	605	400	205	. . .	29	255	321
PCI with stents	36.06, 36.07	528	345	183	. . .	26	216	287
Cardiac revascularization‡	36.1–36.3	416	305	111	. . .	14	162	240
Cardiac revascularization (patients)	36.1–36.3	242	175	67	. . .	8†	91	144
Cardiac catheterization	37.21–37.23	1072	622	449	5†	80	450	537
Pacemakers	37.7, 37.8, 00.50, 00.53	397	193	204	4†	18	47	328
Pacemaker devices	(37.8, 00.53)	174	83	91	2†	4†	19	149
Pacemaker leads	(37.7, 00.50)	223	110	113	2†	14	28	179
Implantable defibrillators	37.94–37.99, 00.51, 00.54	116	86	30	2†	8†	43	65
Endarterectomy	38.12	93	52	41	. . .	. . .	21	72
Total vascular and cardiac surgery and procedures§‖	35–39, 00.50–00.51, 00.53–00.55, 00.61–00.66	7453	4154	3299	252	715	2600	3886

ICD-9-CM indicates *International Classification of Diseases, 9th Revision, Clinical Modification*; PCI, percutaneous coronary intervention; and ellipses (. . .), data not available.

These data do not reflect any procedures performed on an outpatient basis. Many more procedures are being performed on an outpatient basis. Some of the lower numbers in this table compared with 2006 probably reflect this trend. Data include procedures performed on newborn infants.

*Breakdowns are not available for some procedures, so entries for some categories do not add to totals. These data include codes for which the estimated number of procedures is <5000. Categories with such small numbers are considered unreliable by the National Center for Health Statistics and in some cases may have been omitted.

†Estimate should be used with caution because it may be unreliable or does not meet standards of reliability or precision.

‡Because ≥1 procedure codes are required to describe the specific bypass procedure performed, it is impossible from these (mixed) data to determine the average number of grafts per patient.

§Totals include procedures not shown here.

‖This estimate includes angioplasty and stent insertions for noncoronary arteries.

Data derived from the National Hospital Discharge Survey/National Center for Health Statistics, 2009. Estimates are based on a sample of inpatient records from short-stay hospitals in the United States.

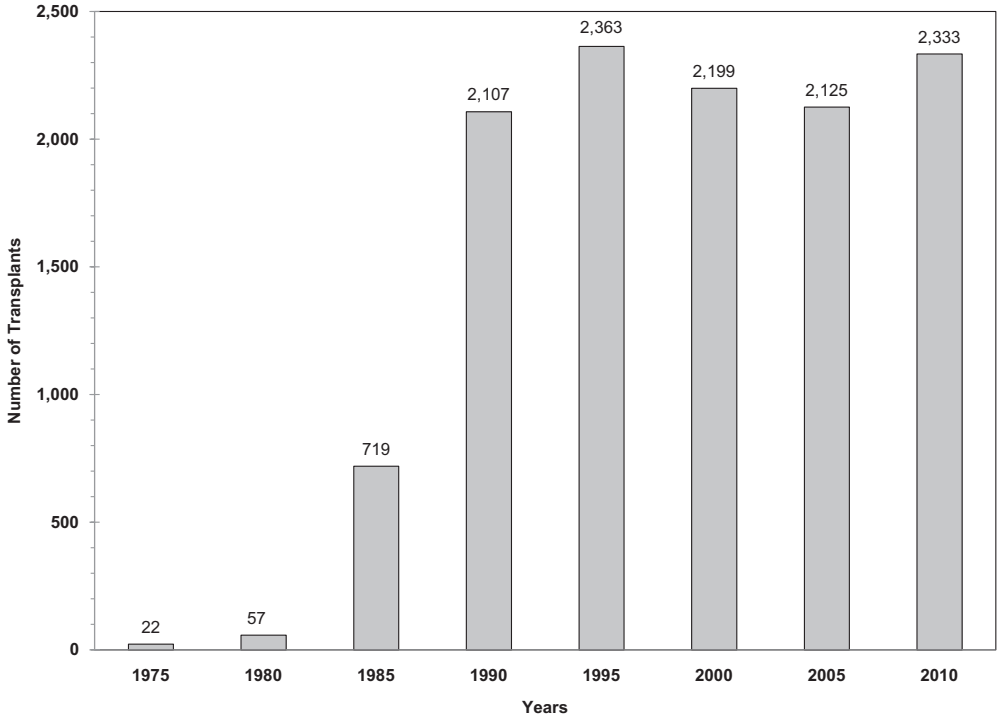

Chart 22-1. Trends in heart transplantations (United Network for Organ Sharing: 1975–2010). Source: United Network for Organ Sharing, scientific registry data.

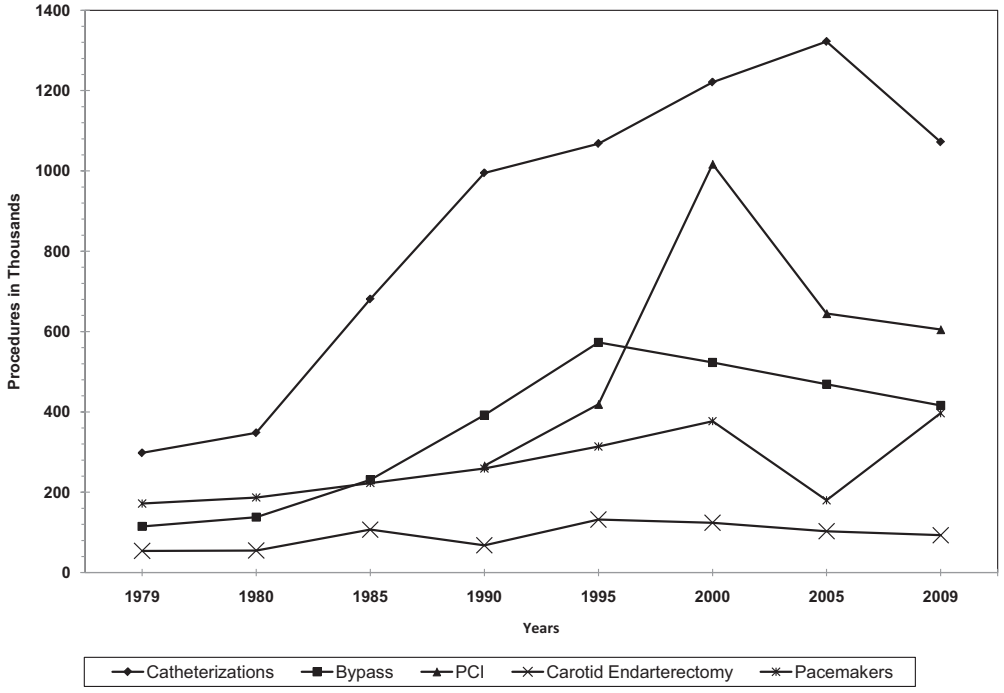

Chart 22-2. Trends in cardiovascular procedures, United States: 1979–2009. PCI indicates percutaneous coronary intervention. Note: Inpatient procedures only. Source: National Hospital Discharge Survey, National Center for Health Statistics, and National Heart, Lung, and Blood Institute.

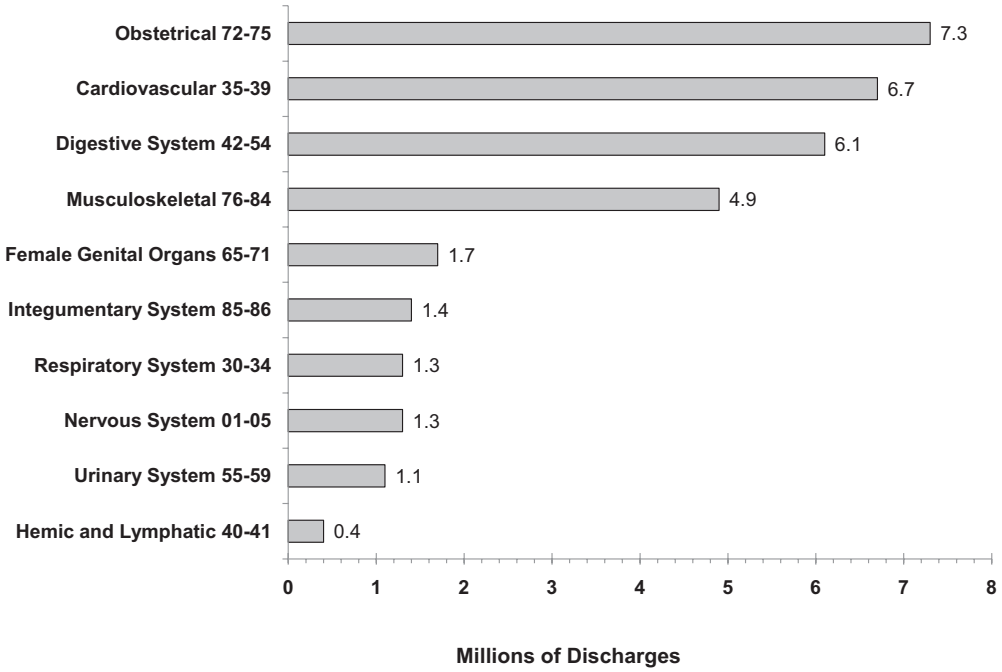

Chart 22-3. Number of surgical procedures in the 10 leading diagnostic groups, United States: 2009. Source: National Hospital Discharge Survey/National Center for Health Statistics and National Heart, Lung, and Blood Institute.

23. Economic Cost of Cardiovascular Disease

See Tables 23-1 and 23-2 and Charts 23-1 through 23-4.

The annual direct and indirect cost of CVD and stroke in the United States is an estimated $297.7 billion (Table 23-1; Chart 23-1). This figure includes $179 billion in expenditures (direct costs, which include the cost of physicians and other professionals, hospital services, prescribed medication, and home health care, but not the cost of nursing home care) and $118.5 billion in lost future productivity attributed to premature CVD and stroke mortality in 2008 (indirect costs).

The direct costs for CVD and stroke are the healthcare expenditures for 2008 available on the Web site of the nationally representative MEPS of the Agency for Healthcare Research and Quality.[1] Details on the advantages or disadvantages of using MEPS data are provided in the Heart Disease and Stroke Statistics–2011 Update.[2] Indirect mortality costs are estimated for 2008 by multiplying the number of deaths that year attributable to CVD and strokes, in age and sex groups, by estimates of the present value of lifetime earnings for those age and sex groups as of 2008. Mortality data are from the National Vital Statistics System of the NCHS.[3] The present values of lifetime earnings are unpublished estimates furnished by the Institute on Health and Aging, University of California at San Francisco, by Wendy Max, PhD, on April 18, 2011. Those estimates have a 3% discount rate, the recommended percentage.[4] The discount rate removes the effect of inflation in income over the lifetime of earnings. The estimates are for 2007, inflated to 2008 by 3% to account for the 2007 to 2008 change in hourly worker compensation in the business sector reported by the Bureau of Labor Statistics.[5]

The indirect costs exclude lost productivity costs attributable to CVD and stroke illness during 2008 among workers, people keeping house, people in institutions, and people unable to work. Those morbidity costs were substantial in very old studies, but an adequate update could not be made.

Most Costly Diseases

CVD and stroke accounted for 16% of total health expenditures in 2008, more than any major diagnostic group.[1,6] That is also the case for indirect mortality costs. By way of comparison, CVD total direct and indirect costs shown in Table 23-1 are higher than the official National Cancer Institute estimates for cancer and benign neoplasms in 2008, which were cited as $228 billion total ($93 billion in direct costs, $19 billion in indirect morbidity costs, and $116 billion in indirect mortality costs).[7]

Table 23-2 shows direct and indirect costs for CVD by sex and by 2 broad age groups. Chart 23-2 shows total direct costs for the 14 leading chronic diseases in the MEPS list. HD is the most costly condition.[1]

Projections

The AHA developed methodology to project future costs of care for HBP, CHD, HF, stroke, and all other CVD.[8] By 2030, 40.5% of the US population is projected to have some form of CVD.[8] Between 2012 and 2030, total direct medical costs of CVD are projected to triple, from $309 billion to $834 billion. Indirect costs (attributable to lost productivity) for all CVDs are estimated to increase from $185 billion in 2012 to $284 billion in 2030, an increase of 53%. Charts 23-3 and 23-4 show further detail of projected total costs of CVD. These data indicate that CVD prevalence and costs are projected to increase substantially. It is important to underscore that differences exist between these estimates and those stated above. These apparent discrepancies largely reflect methodological differences and emphasize that the importance of cost projections resides in the documentation of profoundly adverse trends, which constitute an urgent call to action and must be reversed, rather than in the calculation of precise numbers.

Abbreviations Used in Chapter 23

AHA	American Heart Association
CHD	coronary heart disease
COPD	chronic obstructive pulmonary disease
CVD	cardiovascular disease
HBP	high blood pressure
HD	heart disease
HF	heart failure
MEPS	Medical Expenditure Panel Survey
NCHS	National Center for Health Statistics

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Table 23-1. Estimated Direct and Indirect Costs (in Billions of Dollars) of CVD and Stroke: United States, 2008

	Heart Disease*	Stroke	Hypertensive Disease†	Other Circulatory Conditions	Total Cardiovascular Disease
Direct costs‡					
Hospital inpatient stays	54.0	9.1	6.2	10.4	79.7
Hospital emergency department visits	7.3	0.9	1.7	0.9	10.8
Hospital outpatient or office-based provider visits	16.9	1.8	13.0	4.7	36.4
Home health care	7.6	5.8	5.1	0.9	19.4
Prescribed medicines	9.7	1.2	21.3	0.7	32.9
Total expenditures	95.5	18.8	47.3	17.6	179.2
Indirect costs§					
Lost productivity/mortality‖	94.8	15.5	3.3	4.9	118.5
Grand totals	190.3	34.3	50.6	22.5	297.7

CVD indicates cardiovascular disease.

Numbers do not add to total because of rounding.

*This category includes coronary heart disease, heart failure, part of hypertensive disease, cardiac dysrhythmias, rheumatic heart disease, cardiomyopathy, pulmonary heart disease, and other or ill-defined heart diseases.

†Costs attributable to hypertensive disease are limited to hypertension without heart disease.

‡Medical Expenditure Panel Survey healthcare expenditures are estimates of direct payments for care of a patient with the given disease provided during the year, including out-of-pocket payments and payments by private insurance, Medicaid, Medicare, and other sources. Payments for over-the-counter drugs are not included. These estimates of direct costs do not include payments attributed to comorbidities. Total cardiovascular disease costs are the sum of costs for the 4 diseases but with some duplication.

§The Statistics Committee agreed to suspend presenting estimates of lost productivity attributable to morbidity until a better estimating method can be developed.

‖Lost future earnings of persons who died in 2008, discounted at 3%.

Sources: Estimates from the Household Component of the Medical Expenditure Panel Survey of the Agency for Healthcare Research and Quality for direct costs (2008).[1] Indirect mortality costs are based on 2008 counts of deaths by the National Center for Health Statistics and an estimated present value of lifetime earnings furnished for 2007 by Wendy Max (Institute for Health and Aging, University of California, San Francisco, 2011) and inflated to 2008 from change in worker compensation reported by the Bureau of Labor Statistics.[5]

All estimates prepared by Thomas Thom and Michael Mussolino, National Heart, Lung, and Blood Institute.

Table 23-2. Costs of Total CVD in Billions of Dollars by Age and Sex: United States, 2008

Cost	Total	Male	Female	Age <65 y	Age ≥65 y
Direct	179.2	88.6	90.7	86.1	93.2
Indirect mortality	118.5	88.5	30.0	102.5	16.0
Total	297.7	177.1	120.7	188.6	109.2

CVD indicates cardiovascular diseases and stroke.

Numbers may not add to total because of rounding.

Source: Medical Expenditure Panel Survey, 2008 (direct costs), and mortality data from the National Center for Health Statistics, present value of lifetime earnings from the Institute for Health and Aging, University of California, San Francisco, and hourly compensation data from the Bureau of Labor Statistics (indirect costs).

All estimates prepared by Thomas Thom and Michael Mussolino, National Heart, Lung, and Blood Institute.

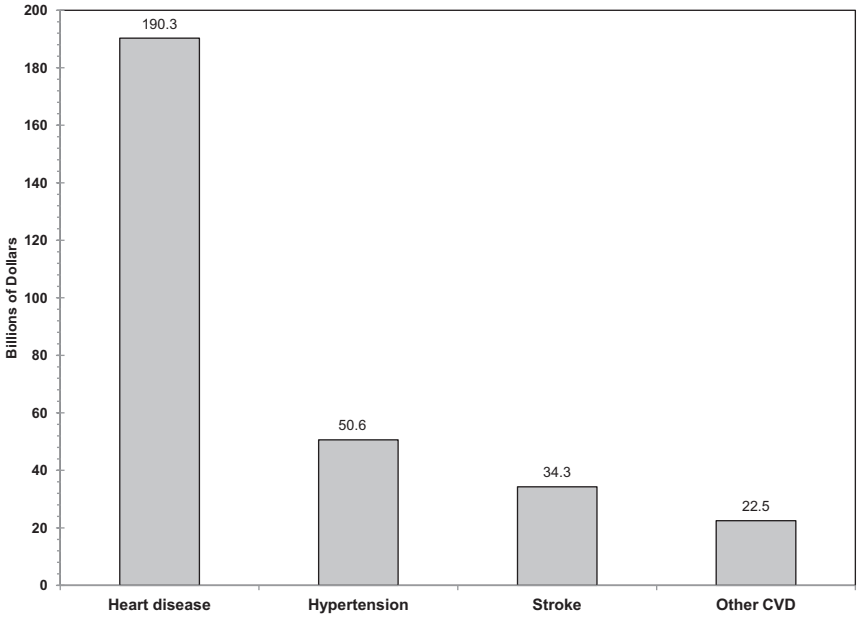

Chart 23-1. Direct and indirect costs (in billions of dollars) of major cardiovascular diseases (CVD) and stroke (United States: 2008). Source: National Heart, Lung, and Blood Institute.

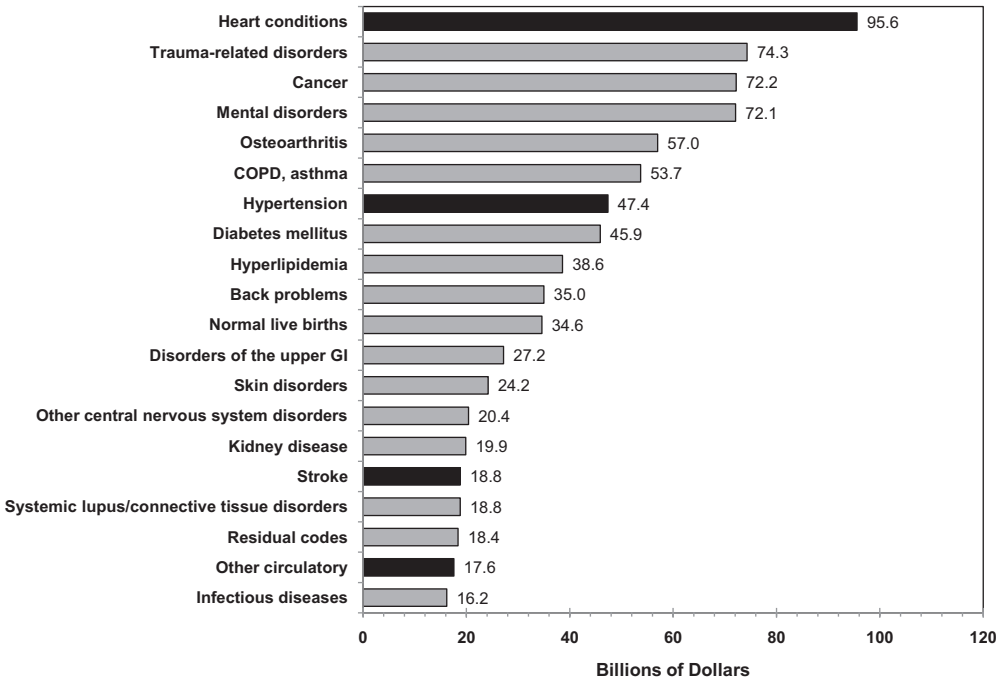

Chart 23-2. The 20 leading diagnoses for direct health expenditures, United States, 2008 (in billions of dollars). COPD indicates chronic obstructive pulmonary disease; GI, gastrointestinal tract. Source: National Heart, Lung, and Blood Institute; estimates are from the Medical Expenditure Panel Survey, Agency for Healthcare Research and Quality, and exclude nursing home costs.

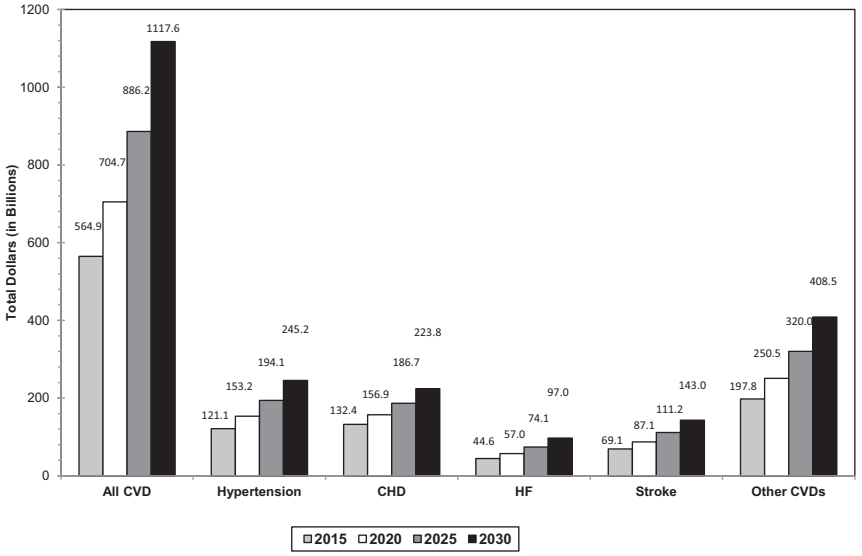

Chart 23-3. Projected total costs of cardiovascular disease (CVD), 2015–2030 (in billions 2008$) in the United States. CHD indicates coronary heart disease; HF, heart failure. Data derived from Heidenreich et al[8] with permission of the publisher. Copyright © 2011, American Heart Association.

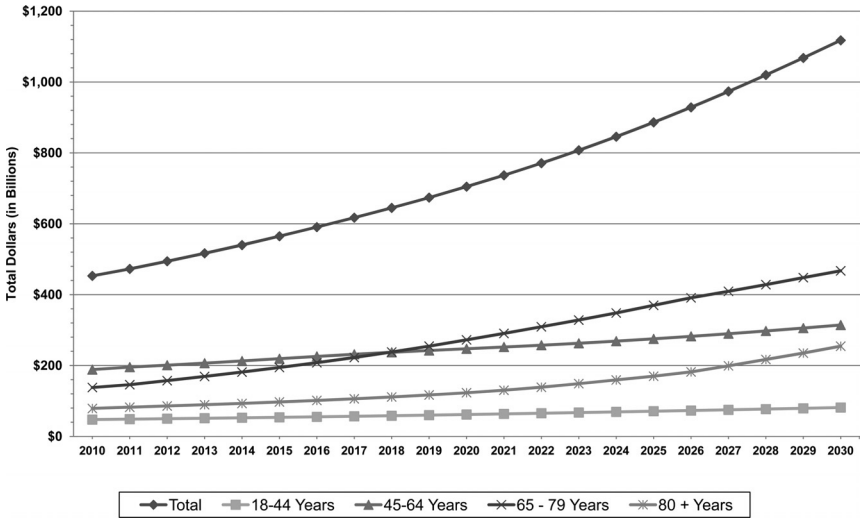

Chart 23-4. Projected total (direct and indirect) costs of total cardiovascular disease by age (2010 $ in billions). Unpublished data tabulated by American Heart Association using methods described in Heidenreich et al.[8]

24. At-a-Glance Summary Tables

See Tables 24-1 through 24-4.

Sources: See the following summary tables and charts for complete details:

- Total cardiovascular disease–Table 3-1.
- Coronary heart disease–Table 5-1.
- Stroke–Table 6-1.
- High blood pressure–Table 7-1.
- Congenital heart defects–Table 8-1.
- Heart failure–Table 9-1.
- Smoking–Table 13-1.
- Blood cholesterol–Table 14-1.
- Physical activity–Table 15-1.
- Overweight/obesity–Table 16-1; Chart 16-1.
- Diabetes mellitus–Table 17-1.

Table 24-1. Males and CVD: At-a-Glance Table

Diseases and Risk Factors	Both Sexes	Total Males	White Males	Black Males	Mexican American Males
Total CVD					
Prevalence, 2008*	82.6 M (36.2%)	39.9 M (37.4%)	37.4%	44.8%	30.7%
Mortality, 2008†	811.9 K	392.2 K	335.2 K	46.8 K	N/A
CHD					
Prevalence, CHD, 2008*	16.3 M (7.0%)	8.8 M (8.3%)	8.5%	7.9%	6.3%
Prevalence, MI, 2008*	7.9 M (3.1%)	4.8 M (4.3%)	4.3%	4.3%	3.0%
Prevalence, AP, 2008*	9.0 M (3.9%)	4.0 M (3.8%)	3.8%	3.3%	3.6%
New and recurrent CHD‡§	1.26 M	740.0 K	675.0 K	70.0 K	N/A
New and recurrent MI§	935.0 K	565.0 K	N/A	N/A	N/A
Incidence, AP (stable angina)‖	500.0 K	320.0 K	N/A	N/A	N/A
Mortality, 2008, CHD†	405.3 K	216.2 K	189.4 K	21.4 K	N/A
Mortality, 2008, MI†	133.0 K	71.7 K	63.0 K	6.9 K	N/A
Stroke					
Prevalence, 2008*	7.0 M (3.0%)	2.8 M (2.7%)	2.4%	4.5%	2.0%
New and recurrent strokes†	795.0 K	370.0 K	325.0 K	45.0 K	N/A
Mortality, 2008†	134.1 K	53.5 K	44.5 K	7.2 K	N/A
HBP					
Prevalence, 2008*	76.4 M (33.5%)	36.5 M (34.1%)	33.9%	43.0%	27.8%
Mortality, 2008†	61.0 K	26.8 K	19.6 K	6.4 K	N/A
HF					
Prevalence, 2008*	5.7 M (2.4%)	3.1 M (3.0%)	2.7%	4.5%	2.3%
Mortality, 2008†	56.8 K	23.0 K	20.3 K	2.4 K	N/A
Smoking					
Prevalence, 2010¶	44.1 M (19.3%)	23.7 M (21.2%)	23.0%	23.4%	15.2%#
Blood cholesterol					
Prevalence, 2008					
Total cholesterol ≥200 mg/dL*	98.8 M (44.4%)	45.0 M (41.8%)	41.2%	37.0%	50.1%
Total cholesterol ≥240 mg/dL*	33.6 M (15.0%)	14.6 M (13.5%)	13.7%	9.7%	16.9%
LDL cholesterol ≥130 mg/dL*	71.3 M (31.9%)	35.3 M (32.5%)	30.5%	34.4%	41.9%
HDL cholesterol <40 mg/dL*	41.8 M (18.9%)	30.8 M (28.6%)	29.5%	16.6%	31.7%
PA**					
Prevalence, 2010¶	20.7%	25.1%	26.7%	24.6%	N/A
Overweight and obesity					
Prevalence, 2008					
Overweight and obesity, BMI ≥25.0 Kg/m^2*	149.3 M (67.3%)	78.0 M (72.4%)	72.3%	70.8%	77.5%
Obesity, BMI ≥30.0 Kg/m^2*	75.0 M (33.7%)	34.9 M (32.4%)	32.1%	37.0%	31.4%
DM					
Prevalence, 2008					
Physician-diagnosed DM*	18.3 M (8.0%)	8.3 M (7.9%)	6.8%	14.3%	11.0%
Undiagnosed DM*	7.1 M (3.1%)	4.4 M (4.1%)	3.9%	4.8%	6.3%
Prediabetes*	81.5 M (36.8%)	48.1 M (44.9%)	45.4%	31.6%	44.9%
Incidence, diagnosed DM*	1.6 M	N/A	N/A	N/A	N/A
Mortality, 2008†	70.6 K	35.3 K	28.6 K	5.5 K	N/A

CVD indicates cardiovascular disease; M, millions; K, thousands; N/A, data not available; CHD, coronary heart disease (includes heart attack, angina pectoris chest pain, or both); MI, myocardial infarction (heart attack); AP, angina pectoris (chest pain); HBP, high blood pressure; HF, heart failure; LDL, low-density lipoprotein; HDL, high-density lipoprotein; PA, physical activity; BMI, body mass index; and DM, diabetes mellitus.

*Age ≥20 y.

†All ages.

‡New and recurrent MI and fatal CHD.

§Age ≥35 y.

‖Age ≥45 y.

¶Age ≥18 y.

#Hispanic.

**Met 2008 Federal PA guidelines for adults.

Table 24-2. Females and CVD: At-a-Glance Table

Diseases and Risk Factors	Both Sexes	Total Females	White Females	Black Females	Mexican American Females
Total CVD					
Prevalence, 2008*	82.6 M (36.2%)	42.7 M (35.0%)	33.8%	47.3%	30.9%
Mortality, 2008†	811.9 K	419.7 K	360.4 K	49.8 K	N/A
CHD					
Prevalence, CHD, 2008*	16.3 M (7.0%)	7.5 M (6.1%)	5.8%	7.6%	5.6%
Prevalence, MI, 2008*	7.9 M (3.1%)	3.1 M (2.2%)	2.1%	2.2%	1.1%
Prevalence, AP, 2008*	9.0 M (3.9%)	5.0 M (4.0%)	3.7%	5.6%	3.7%
New and recurrent CHD‡§	1.26 M	515.0 K	445.0 K	65.0 K	N/A
New and recurrent MI§	935.0 K	370.0 K	N/A	N/A	N/A
Incidence, AP (stable angina)‖	500.0 K	180.0 K	N/A	N/A	N/A
Mortality, 2008, CHD†	405.3 K	189.1 K	164.5 K	20.5 K	N/A
Mortality, 2008, MI†	133.0 K	61.3 K	52.9 K	7.1 K	N/A
Stroke					
Prevalence, 2008*	7.0 M (3.0%)	4.2 M (3.3%)	3.3%	4.4%	2.7%
New and recurrent strokes†	795.0 K	425.0 K	365.0 K	60.0 K	N/A
Mortality, 2008†	134.1 K	80.6 K	68.8 K	9.5 K	N/A
HBP					
Prevalence, 2008*	76.4 M (33.5%)	39.9 M (32.7%)	31.3%	45.7%	28.9%
Mortality, 2008†	61.0 K	34.2 K	26.3 K	7.0 K	N/A
HF					
Prevalence, 2008*	5.7 M (2.4%)	2.6 M (2.0%)	1.8%	3.8%	1.3%
Mortality, 2008†	56.8 K	33.8 K	30.2 K	3.1 K	N/A
Smoking					
Prevalence, 2010¶	44.1 M (19.3%)	20.4 M (17.5%)	20.5%	16.7%	9.0%#
Blood cholesterol					
Prevalence, 2008					
Total cholesterol ≥200 mg/dL*	98.8 M (44.4%)	53.8 M (46.3%)	47.0%	41.2%	46.5%
Total cholesterol ≥240 mg/dL*	33.6 M (15.0%)	19.0 M (16.2%)	16.9%	13.3%	14.0%
LDL cholesterol ≥130 mg/dL*	71.3 M (31.9%)	36.0 M (31.0%)	32.0%	27.7%	31.6%
HDL cholesterol <40 mg/dL*	41.8 M (18.9%)	11.0 M (9.7%)	10.1%	6.6%	12.2%
PA**					
Prevalence, 2010¶	20.7%	16.4%	19.1%	11.2%	N/A
Overweight and obesity					
Prevalence, 2008					
Overweight and obesity, BMI ≥25.0 Kg/m^{2}*	149.3 M (67.3%)	71.3 M (62.3%)	59.3%	77.7%	75.1%
Obesity, BMI ≥30.0 Kg/m^{2}*	75.0 M (33.7%)	40.1 M (35.2%)	35.2%	51.0%	43.4%
DM					
Prevalence, 2008					
Physician-diagnosed DM*	18.3 M (8.0%)	10.0 M (8.2%)	6.5%	14.7%	12.7%
Undiagnosed DM*	7.1 M (3.1%)	2.7 M (2.3%)	1.9%	4.0%	3.8%
Prediabetes*	81.5 M (36.8%)	33.4 M (28.8%)	27.9%	27.1%	34.3%
Incidence, diagnosed DM*	1.6 M	N/A	N/A	N/A	N/A
Mortality, 2008†	70.6 K	35.2 K	27.3 K	6.6 K	N/A

CVD indicates cardiovascular disease; M, millions; K, thousands; N/A, data not available; CHD, coronary heart disease (includes heart attack, angina pectoris chest pain, or both); MI, myocardial infarction (heart attack); AP, angina pectoris (chest pain); HBP, high blood pressure; HF, heart failure; LDL, low-density lipoprotein; HDL, high-density lipoprotein; PA, physical activity; BMI, body mass index; and DM, diabetes mellitus.

*Age ≥20 y.

†All ages.

‡New and recurrent MI and fatal CHD.

§Age ≥35 y.

‖Age ≥45 y.

¶Age ≥18 y.

#Hispanic.

**Met 2008 Federal PA guidelines for adults.

Table 24-3. Race/Ethnicity and CVD: At-a-Glance Table

Diseases and Risk Factors	Both Sexes	Whites		Blacks		Mexican Americans		Hispanics/ Latinos		Asians: Both Sexes	American Indian/ Alaska Native: Both Sexes
		Males	Females	Males	Females	Males	Females	Males	Females		
Total CVD											
Prevalence, 2008*	82.6 M (36.2%)	37.4%	33.8%	44.8%	47.3%	30.7%	30.9%	N/A	N/A	N/A	N/A
Mortality, 2008†	811.9 K	335.2 K	360.4 K	46.8 K	49.8 K	N/A	N/A	N/A	N/A	N/A	N/A
CHD											
Prevalence, CHD, 2008*	16.3 M (7.0%)	8.5%	5.8%	7.9%	7.6%	6.3%	5.6%	5.8%‖		3.9%‖	4.1%‖#
Prevalence, MI, 2008*	7.9 M (3.1%)	4.3%	2.1%	4.3%	2.2%	3.0%	1.1%	N/A	N/A	N/A	N/A
Prevalence, AP, 2008*	9.0 M (3.9%)	3.8%	3.7%	3.3%	5.6%	3.6%	3.7%	N/A	N/A	N/A	N/A
New and recurrent CHD‡§	1.26 M	675.0 K	445.0 K	70.0 K	65.0 K	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, CHD, 2008†	405.3 K	189.3 K	164.5 K	21.4 K	20.5 K	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, MI, 2008†	133.0 K	63.0 K	52.9 K	7.0 K	7.1 K	N/A	N/A	N/A	N/A	N/A	N/A
Stroke											
Prevalence, 2008*	7.0 M (3.0%)	2.4%	3.3%	4.5%	4.4%	2.0%	2.7%	2.0%‖		1.3%‖	N/A
New and recurrent strokes†	795.0 K	325.0 K	365.0 K	45.0 K	60.0 K	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, 2008†	134.1 K	44.4 K	68.8 K	7.2 K	9.5 K	N/A	N/A	N/A	N/A	N/A	N/A
HBP											
Prevalence, 2008*	76.4 M (33.5%)	33.9%	31.3%	43.0%	45.7%	27.8%	28.9%	21.5%‖		19.4%‖	21.8%‖
Mortality, 2008†	61.0 K	19.6 K	26.3 K	6.4 K	7.0 K	N/A	N/A	N/A	N/A	N/A	N/A
HF											
Prevalence, 2008*	5.7 M (2.4%)	2.7%	1.8%	4.5%	3.8%	2.3%	1.3%	N/A	N/A	N/A	N/A
Mortality, 2008†	56.8 K	20.3 K	30.2 K	2.4 K	3.1 K	N/A	N/A	N/A	N/A	N/A	N/A
Smoking											
Prevalence, 2010‖	44.1 M (19.3%)	23.0%	20.5%	23.4%	16.7%	12.0%		15.2%	9.0%	9.3%	26.6%
Blood cholesterol											
Prevalence, 2008											
Total cholesterol ≥200 mg/dL*	98.8 M (44.4%)	41.2%	47.0%	37.0%	41.2%	50.1%	46.5%	N/A	N/A	N/A	N/A
Total cholesterol ≥240 mg/dL*	33.6 M (15.0%)	13.7%	16.9%	9.7%	13.3%	16.9%	14.0%	N/A	N/A	N/A	N/A
LDL cholesterol ≥130 mg/dL*	71.3 M (31.9%)	30.5%	32.0%	34.4%	27.7%	41.9%	31.6%	N/A	N/A	N/A	N/A
HDL cholesterol <40 mg/dL*	41.8 M (18.9%)	29.5%	10.1%	16.6%	6.6%	31.7%	12.2%	N/A	N/A	N/A	N/A
PA¶											
Prevalence, 2010‖	20.7%	21.3%		17.2%		13.2%		14.4%		17.8%	12.5%
Overweight and obesity											
Prevalence, 2008											
Overweight and obesity, BMI ≥25.0kg/m^2*	149.3 M (67.3%)	72.3%	59.3%	70.8%	77.7%	77.5%	75.1%	N/A	N/A	N/A	N/A
Overweight and obesity, BMI ≥30.0kg/m^2*	75.0 M (33.7%)	32.1%	32.8%	37.0%	51.0%	31.4%	43.4%	N/A	N/A	N/A	N/A
DM											
Prevalence, 2008											
Physician-diagnosed DM*	18.3 M (8.0%)	6.8%	6.5%	14.3%	14.7%	11.0%	12.7%	N/A	N/A	N/A	N/A
Undiagnosed DM*	7.1 M (3.1%)	3.9%	1.9%	4.8%	4.0%	6.3%	3.8%	N/A	N/A	N/A	N/A
Prediabetes*	81.5 M (36.8%)	45.4%	27.9%	31.6%	27.1%	44.9%	34.3%	N/A	N/A	N/A	N/A
Incidence, diagnosed DM*	1.6 M	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Mortality, 2008†	70.6 K	28.6 K	27.3 K	5.5 K	6.6 K	N/A	N/A	N/A	N/A	N/A	N/A

CVD indicates cardiovascular disease; M, millions; N/A, data not available; K, thousands; CHD, coronary heart disease (includes heart attack, angina pectoris chest pain, or both); MI, myocardial infarction (heart attack); AP, angina pectoris (chest pain); HBP, high blood pressure; HF, heart failure; LDL, low-density lipoprotein; HDL, high-density lipoprotein; PA, physical activity; BMI, body mass index; and DM, diabetes mellitus.

*Age >20 y.

†All ages.

‡New and recurrent MI and fatal CHD.

§Age ≥35 y.

‖Age ≥18 y.

¶Met 2008 Federal PA guidelines for adults.

#Figure not considered reliable.

Table 24-4. Children, Youth, and CVD: At-a-Glance Table

				NH Whites		NH Blacks		Mexican Americans	
Diseases and Risk Factors	Both Sexes	Total Males	Total Females	Males	Females	Males	Females	Males	Females
Congenital cardiovascular defects									
Mortality, 2008*	3.4 K	1.8 K	1.6 K	1.4 K	1.2 K	0.3 K	0.3 K	N/A	N/A
Smoking, %									
High school students, grades 9–12									
Current cigarette smoking, 2009	19.5	19.8	19.1	22.3	22.8	10.7	8.4	19.4†	16.7†
Current cigar smoking, 2009	14.0	18.6	8.8	21.0	8.0	13.9	11.5	15.8†	9.5†
Blood cholesterol, mg/dL									
Mean total cholesterol									
Ages 4–11 y	164.5	163.8	165.2	163.9	165.6	165.7	162.3	160.7	161.5
Ages 12–19 y	159.2	156.3	162.3	155.9	162.3	157.7	163.6	156.9	161.3
Mean HDL cholesterol									
Ages 4–11 y	54.7	55.6	53.6	54.7	52.8	61.4	58.1	53.6	51.1
Ages 12–19 y	51.6	49.3	54.0	48.1	53.3	54.6	56.9	48.3	53.5
Mean LDL cholesterol									
Ages 12–19 y	88.5	87.1	89.9	87.6	89.8	88.8	92.6	88.4	88.8
PA‡									
Prevalence, grades 9–12, 2009§									
Met currently recommended levels of PA, %	37.0	45.6	27.7	47.3	31.3	43.3	21.9	41.3†	24.9†
Overweight and obesity									
Prevalence, 2008									
Children and adolescents, ages 2–19 y, overweight or obese	23.6 M (31.7%)	12.2 M (32.1%)	11.4 M (31.3%)	29.5%	29.2%	33.0%	39.0%	41.7%	36.1%
Children and adolescents, age 2–19 y, obese§	12.6 M (16.9%)	6.8 M (17.8%)	5.8 M (15.9%)	15.7%	14.9%	17.3%	22.7%	24.9%	16.5%

CVD indicates cardiovascular disease; NH, non-Hispanic; K, thousands; N/A, data not available; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PA, physical activity; and M, millions.

Overweight indicates a body mass index in the 95th percentile of the Centers for Disease Control and Prevention 2000 growth chart.

*All ages.

†Hispanic.

‡Regular leisure-time PA.

§Data derived from Eaton DK, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, Harris WA, Lowry R, McManus T, Chyen D, Lim C, Whittle L, Brener ND, Wechsler H; Centers for Disease Control and Prevention (CDC). Youth risk behavior surveillance—United States, 2009. *MMWR Surveill Summ*. 2010;59:1–142.

25. Glossary

- *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 (2008), 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, reduce the cost of health care, improve patient safety, decrease the number of medical errors, and broaden access to essential services. AHRQ sponsors and conducts research that provides evidence-based information on healthcare outcomes, quality, cost, use, and access. The information helps healthcare decision makers (patients, clinicians, health system leaders, and policy makers) make more informed decisions and improve the quality of healthcare services. AHRQ conducts the Medical Expenditure Panel Survey (MEPS; ongoing).
- *Bacterial endocarditis*—An infection of the heart's inner lining (endocardium) or of the heart valves. The bacteria that most often cause endocarditis are streptococci, staphylococci, and enterococci.
- *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 the height in meters (kg/m^2).
- *Centers for Disease Control and Prevention/National Center for Health Statistics (CDC/NCHS)*—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 to …) (ongoing)
  - National Health Interview Survey (NHIS) (ongoing)
  - National Hospital Discharge Survey (NHDS) (ongoing)
  - National Ambulatory Medical Care Survey (NAMCS) (ongoing)
  - National Hospital Ambulatory Medical Care Survey (NHAMCS) (ongoing)
  - National Nursing Home Survey (periodic)
  - National Home and Hospice Care Survey (periodic)
- *Centers for Medicare & Medicaid Services (CMS), formerly Health Care Financing Administration (HCFA)*—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 (I21–I22), other acute ischemic (coronary) heart disease (I24), angina pectoris (I20), atherosclerotic cardiovascular disease (I25.0), and all other forms of chronic ischemic CHD (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 codes I00–I99)*—Included as part of what the AHA calls "cardiovascular disease." ("Total cardiovascular disease" in this Glossary.)
- *Diseases of the heart*—Classification 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 (I29–I49, I50.1–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.
- *Health Care Financing Administration (HCFA)*—See Centers for Medicare & Medicaid Services (CMS).
- *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 Mexican Americans or Mexicans, as reported by government agencies or specific studies. In many cases, data for all Hispanics are more difficult to obtain.
- *Hospital discharges*—The number of inpatients (including newborn infants) discharged from short-stay hospitals for whom some type of disease was the first-listed diagnosis. Discharges include those discharged alive, dead, or "status unknown."
- *International Classification of Diseases (ICD) codes*—A classification system in standard use in the United States.

The *International Classification of Diseases* 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.

- *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.
- *Major cardiovascular diseases*—Disease classification commonly reported by the NCHS; represents ICD codes I00 to I78. The AHA does not use "major cardiovascular diseases" for any calculations. See "Total cardiovascular disease" in this Glossary.
- *Metabolic syndrome*—The metabolic syndrome is defined* as the presence of any 3 of the following 5 diagnostic measures: Elevated waist circumference (≥102 cm in men or ≥88 cm in women), elevated triglycerides (≥150 mg/dL [1.7 mmol/L] or drug treatment for elevated triglycerides), reduced high-density lipoprotein (HDL) cholesterol (<40 mg/dL [0.9 mmol/L] in men, <50 mg/dL [1.1 mmol/L] in women, or drug treatment for reduced HDL 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*—Incidence and prevalence rates are both measures of morbidity (ie, measures of various effects of disease on a population).
- *Mortality*—Mortality data for states can be obtained from the NCHS Web site (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 a year, are 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 to …) (ongoing)
  - — Honolulu Heart Program (HHP) (1965–1997)
  - — Cardiovascular Health Study (CHS; 1988 to …) (ongoing)
  - — Atherosclerosis Risk in Communities (ARIC) study (1985 to …) (ongoing)
  - — Strong Heart Study (SHS) (1989–1992, 1991–1998)
  - — The NHLBI also published reports of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure and the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III, or ATP III).
- *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 activity generally refers to the subset of physical activity that enhances health.
- *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 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 categories, 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 whites, blacks, American Indians or Alaska Natives, and Asian or Pacific Islanders 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

*According to criteria established by the American Heart Association/National Heart, Lung, and Blood Institute and published in *Circulation* (*Circulation*. 2005;112:2735–2752).

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, Q20–Q28)*—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). When data are available, we include congenital cardiovascular defects (Q20–Q28).
- *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 train of events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury." Contributing cause of death would be any other disease or condition that the decedent may also have had.

Correction

In the article by Roger et al, "Heart Disease and Stroke Statistics—2012 Update: A Report From the American Heart Association," which published ahead of print on December 15, 2011, in *Circulation* (10.1161/CIR.0b013e31823ac046), a correction is needed.

On page e15, in the Writing Group Disclosures table, for William B. Borden, the disclosure entries under the "Research Grant," "Other Research Support," "Speakers' Bureau/Honoraria," and "Ownership Interest" columns read "NIH†." The disclosure entries in these columns should read, "None."

The online version of the article is available at:
http://circ.ahajournals.org/content/early/2011/12/15/CIR.0b013e31823ac046